



Disaster and Trauma eBook



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Synonyms, Key Words, and Related Terms

air medical transport, air transport services, flight physician, flight nurse, aeromedical crew

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History of Aeromedical Transport

Transport of injured patients by air can be traced to World War I, when a French fighter plane was used to evacuate a wounded Serb. Fixed-wing transport had limited use until World War II, when the Allies evacuated large numbers of casualties by air, primarily in C-47 transports.

The modern era of aeromedical transport began during World War II, when helicopters transported wounded patients in Burma. During the Korean and Vietnamese conflicts, helicopter aeromedical transport significantly reduced battlefield mortality and became an important and highly visible part of the military trauma system. In Korea, more than 20,000 wounded soldiers were transported in litters strapped to the skids of Bell-47 and Sikorsky S-51 helicopters. In Vietnam's Operation Dustoff, 800,000 patients were aeromedically transported.

The successful aeromedical experience in Vietnam proved the ability of helicopters to transport injured patients directly from trauma scenes to specialized trauma care centers (see [Picture 1](#)). Awareness of the role of military aeromedical transport made the extension of helicopter use to the civilian arena inevitable.

The first aeromedical transport program was established in 1972 at St Anthony's Hospital in Denver, Colo. Since then, the number of aeromedical programs has grown steadily, reaching more than 160 today. Some services that began with single rotor-wing aircraft have added additional helicopters, fixed-wing aircraft, or ground critical care transport capability. Aeromedical transport services now provide much more than trauma scene response assistance. Air transport services also transfer patients from EDs or inpatient units of referring hospitals to receiving tertiary care centers. Fixed-wing transports (see [Picture 2](#)) are particularly useful for long-distance (ie, >150-200 miles) interfacility transports and can operate in weather conditions that may restrict rotor-wing aircraft.

As prehospital and transport medicine become more integrated, air medical service providers are cooperating or combining with ground interfacility and critical care transport services. In some cases, a single service trains and provides medical crew staffing for rotor-wing, fixed-wing, and ground transport vehicles. Patient acuity and logistical factors (eg, distance) determine the optimal mode of transport to the receiving center. Integration of air and ground transport modes represents the maturation of air transport as a vital component in the development and operation of regional trauma and medical care systems.

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Organizational Models of Air Transport Services

The evolution of aeromedical transport over the past 2 decades has produced multiple organizational models. Each model has inherent advantages and disadvantages, and different regions and different emergency medical services (EMS) system needs may require different models of transport.

The most common organizational model in the US is the hospital-based aeromedical program. A hospital, usually a tertiary or academic center, sponsors and owns the program. Aircraft, pilots, and maintenance services often are provided to the hospital by an aviation vendor with EMS experience. In some cases, multiple hospitals share the sponsorship and costs of an air transport service, creating a consortium program.

A second organizational model, which has been used successfully for years, is the public service agency structure. Government agencies (eg, Maryland State Police) deploy these aircraft, and they may serve in roles outside the medical setting. Military aircraft, used on an as-available basis when no civilian transport is obtainable, constitute an additional form of governmental air transport.

The private service organizational model most commonly is seen in the form of a fixed-wing service based at an airport. These services generally do not respond to accident scenes. They may use on-call medical staff, or they may simply provide pilots and aircraft for use by hospital-based medical crew performing long-range medical transports.

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Aircraft

Early air transport programs typically used single-engine rotor-wing craft. In the past decade, these relatively small, light, and economical aircraft largely have been replaced by twin-engine helicopters (see [Pictures 3-8](#)). While generally more expensive to purchase and operate, twin-engine helicopters have advantages over single-engine helicopters, including a larger carrying capacity, larger cabin spaces with improved patient access, potential for 2-patient transports, faster transport speed, and improved safety. In addition, hot-loads (loading patients with rotors turning) are accomplished more easily with the rear clamshell doors or side-loading schemes available in larger helicopters (see [Pictures 9](#), [Picture 10](#)).

Weather and related aviation considerations play an important role in aeromedical EMS systems. Most programs fly under visual flight rules (VFR), which state that pilots must have visibility sufficient to fly without relying on instruments. Upgrading aircraft and pilots to instrument flight rules (IFR) capability is expensive and may require pilot-copilot operations but increases overall safety margins and reduces weather-related flight refusals. The ability to fly in hostile weather is an advantage of fixed-wing aircraft.

Clearly, different aircraft are suited for different EMS system and regional needs. A program performing 5-minute transports from accident scenes has markedly different needs than a program responsible for transporting patients within a tertiary care center's 300-mile catchment radius. In many cases, a region's needs are optimally served by a combination of rotor-wing and fixed-wing aircraft.

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Flight Crew

Just as varying types of aircraft have served well in aeromedical transport, different flight crew configurations have been used successfully. Though a few programs use a single medical flight crew model and some larger helicopters can accommodate 3 crew members, the vast majority of air transport helicopters are staffed with a 2-person medical flight crew. Interprogram variability results from the different training and background of medical crew members.

Most models incorporate a flight nurse experienced with emergency and critical care. Many of these flight nurses are cross-qualified as emergency medical technicians (EMTs). In addition to the flight nurse, air transport helicopters may be staffed with a second flight nurse, a paramedic, a respiratory therapist, or a resident- or attending-level physician. In some programs, medical crew composition changes with the nature of the flight (eg, neonatal intensive care transport requires specialized personnel, when available).

Consideration of optimal configuration of the aeromedical crew has generated long-lasting and lively debate. Many experts believe that a critical care nursing or emergency medicine background combined with the roadside expertise of the paramedic generates the ideal flight team. Other commentators, supporting the nurse-nurse configuration, remark that a team of paramedic-qualified flight nurses offers the best of both worlds. Transport services using respiratory therapists as medical crew contend that, since airway management is a prime concern of prehospital and transport medicine, specialists in intubation and ventilator management are valuable flight crew members. Finally, aeromedical crews in European (and some American) programs who regularly fly with physicians believe that the presence of a physician improves decision making and technical care.

Not surprisingly, individuals in aeromedical services tend to strongly support their program's particular favored crew configuration. Much literature, little of it substantive, has addressed the issue of optimal crew configuration. In essence, the questions of what constitutes the best aeromedical flight configuration and whether onboard physicians are desirable have no clear answers.

With nonphysician personnel in various aeromedical programs able to perform complex interventions (eg, tube thoracostomy, subclavian venous cannulation, neuromuscular blockade-enhanced orotracheal intubation), the distinction between a physician and the nonphysician crew is diminished. Unfortunately, no studies have been performed to evaluate possible outcome differences between patients transported by a physician crew and those transported by a nonphysician crew. Definitive conclusions regarding the superiority of a particular crew configuration cannot be drawn until such studies are completed. In the meantime, air transport service personnel may be well advised to continue the methods that have best served their patients. However, they should continue reviewing crew configuration research with an open mind.

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Air Transport of Trauma Patients

Aeromedical transport types can be subdivided into trauma and nontrauma transfers. Since aeromedical transport began in military operations, much of the early research regarding air transport use has focused on trauma flights and evacuation of injured patients.

The first study to address outcomes in civilian helicopter transport used a mathematical algorithm (ie, Trauma and Injury Severity Score [TRISS]) to generate predicted outcomes for a study population of injured patients. Actual outcomes were compared to outcomes predicted by TRISS calculations. Actual mortality was determined identical to predicted mortality for patients transported by ground. For patients undergoing aeromedical scene transport, however, actual mortality was significantly lower than that predicted by TRISS. The study concluded that aeromedical scene transport resulted in improved outcome for injured trauma patients. The study postulated that the higher level of care (eg, endotracheal intubation) provided by flight crews was responsible for improved outcome in aeromedical patients.

Subsequent TRISS-based studies provided more data supporting use of helicopters for both scene and interfacility transport of injured patients. Unsurprisingly, these studies suggested that the patients most likely to benefit from helicopter transport were those with the highest TRISS-predicted mortality. While the TRISS studies and subsequent investigations using other forms of retrospective acuity matching provide evidence of the benefits of aeromedical transport, a definitive demonstration of outcome improvement has not been shown. Considerable logistical and political hurdles complicate randomization of injured patients to air or ground transport.

Amidst the uncontrolled and often chaotic setting of the trauma scene, one of the most important decisions is whether the patient's condition warrants helicopter transport. While this decision always incorporates a considerable amount of judgment, national organizations have attempted to provide regional EMS systems with a framework to facilitate generation of protocols for scene helicopter dispatch. For example, [Air Medical Physician Association \(AMPA\)](#) suggests consideration of geographic factors, traffic, patient acuity, and weather as well as additional factors that may affect transport times. As an added consideration, ground EMS transport of trauma patients from some rural regions to urban trauma centers results in deprivation of regional EMS coverage; this should be avoided whenever possible.

AMPA guidelines also call for consideration of aeromedical scene response in cases of multiple casualty or prolonged extrication. Regional EMS systems establish their own specific criteria for triage of the trauma scene patient to helicopter transport. These criteria vary but usually incorporate mechanism-of-injury factors, acuity scoring (eg, Trauma Score [TS], Glasgow Coma Scale [GCS]), and logistic variables. The [National Association of EMS Physicians \(NAEMSP\)](#) recommends helicopter scene transport for patients with a TS less than 12, GCS score less than 10, systolic BP less than 90 mm Hg, respiration less than 10 per minute or more than 35, heart rate less than 60 beats per minute or more than 120, or unresponsiveness to verbal stimuli.

Helicopter scene transport of patients with penetrating trauma has proved controversial. Since most penetrating trauma occurs within city limits, air transport often saves little time over ground EMS transport. Helicopter use in this setting is best reserved for cases in which patients are likely to benefit from time savings or from the higher level of care provided by the flight crew.

Utilization review (UR) for scene and interfacility aeromedical transport of injured patients is hampered

by a lack of concrete data demonstrating which patients benefit from helicopter transfer. AMPA recommends considering factors such as predicted mortality and the need for complex intratransport or posttransport medical and surgical care. At minimum, all trauma aeromedical transports are routinely reviewed by most programs to ensure that transported patients meet criteria for trauma center care.

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Nontrauma Aeromedical Transport

Nontrauma flights comprise a significant proportion of the activity of most air transport services. The nontrauma category includes a wide variety of diagnoses. Distribution depends on referral patterns and types of tertiary care provided at the receiving institutions of the air transport service.

In many regions, cardiac patients constitute the majority of nontrauma air transports. Research has demonstrated safety of transporting cardiac patients by air, even in cases where thrombolytic therapy has been administered. Positive experience with transport of these patients has resulted in important roles for aeromedical and ground transport, which often are provided by the same service, in the development of regional cardiac care networks.

One reason aeromedical services can provide safe transport for cardiac patients is the high level of intratransport care provided by the in-flight medical crew. Air transport services' pharmacologic, monitoring, and mechanical ventilation capabilities are such that ICU-to-ICU transports can be achieved without a significant decrease in quality of care during transport. In fact, many programs can accommodate advanced technology (eg, intra-aortic balloon pump).

Aeromedical transport also has proven to be a safe and effective means to achieve tertiary care transfer of patients of all ages with serious medical, surgical, or obstetric conditions. Unfortunately, the inherently varied nature of the nontrauma population limits objective demonstration of outcome improvement resulting from air transport of these patients. Accordingly, nontrauma transfers should be triaged to air transport on a case-by-case basis using established regional protocols designed to maximize patient benefit and maintain cost-effectiveness for the EMS systems and hospitals involved.

Pictures



Picture 1: Aeromedical helicopter on rooftop helipad at an urban trauma center

Picture type: Photo



Picture 2: Interior of a fixed-wing aeromedical aircraft

Picture type: Photo



Picture 3: AS365N2 Dauphin aeromedical helicopter landing at a trauma scene

Picture type: Photo





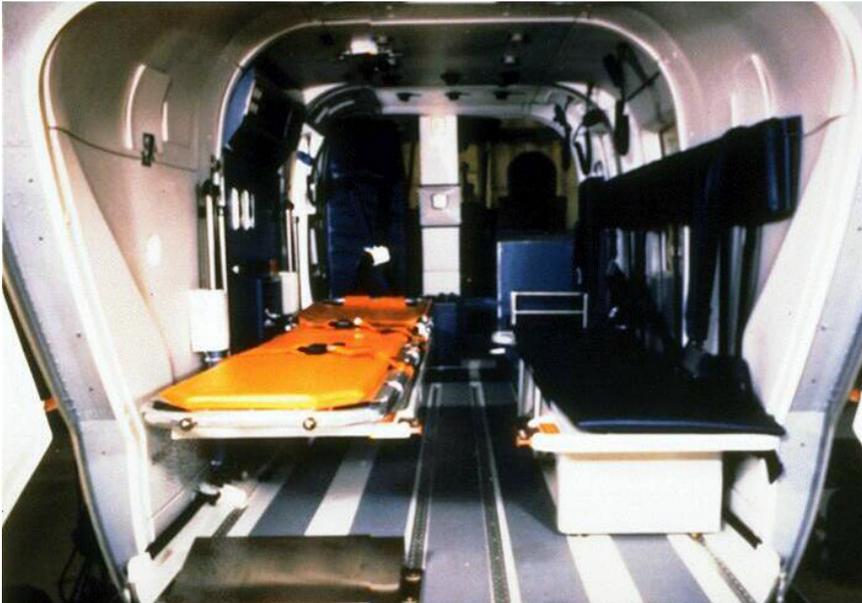
Picture 4: AS365N2 Dauphin: Interior view looking towards left side

Picture type: Photo



Picture 5: Skid-type landing gear on this BK-117 enhances the aircraft's ability to land in rough terrain. (Courtesy UMass LifeFlight)

Picture type: Photo



Picture 6: Interior configurations of some aircraft, such as the pictured BK-117, allow 2-patient transports.

Picture type: Photo



Picture 7: Agusta 109-Max aeromedical helicopter (Courtesy Dartmouth-Hitchcock Air Response Team)

Picture type: Photo



Picture 8: Sikorsky S-76 aeromedical helicopter (Courtesy University of Kentucky Aeromedical Service)

Picture type: Photo



Picture 9: Hot-loading a trauma scene patient in a BK-117 with clamshell rear doors

Picture type: Photo



Picture 10: Side-loading mechanism in the AS365N2 Dauphin

Picture type: Photo

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Disaster Planning

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Introduction to Disaster Planning: the Scope and Nature of the Problem

Disasters are not uncommon; 69% of persons living in the southeastern US have reported exposure to some traumatic event. A major disaster occurs somewhere in the world almost daily; however, to most people, disasters of the type discussed in this chapter are unusual events.

There is evidence that disasters may be increasing, as are their costs. The American Red Cross recently reported that it responded to more US disasters in the year 2000 than at any time in American historyâ€ 55 shelters were opened in 27 states in response to disasters in December 2000 alone.

Still, the low probability of a major catastrophe leads to a certain degree of complacency and underestimation of the impact of such an event. The results of this attitude are frequently felt after the cataclysmic episode subsides. Indeed, several authors note that the best time to propose major changes for disaster preparedness, including its funding, is immediately following a major disaster, even if it has occurred in a remote jurisdiction.

In the US, large multiple-casualty events are exceptionally rare by world standards. Only 6 disasters in US history have resulted in more than 1000 fatalities ([see Table 1](#)). The vast majority of major events

have resulted in fewer than 40 injuries. In the first two thirds of this century, less than 17,000 deaths resulted from natural disasters. More than 3 times as many individuals died in motor vehicle accidents (MVAs) in 1967 alone.

When a disaster strikes, the general population expects public service agencies and other branches of the local, state, or federal government to rapidly mobilize to help the community. Preservation of life and health are of paramount importance to those individuals affected by these disasters. For this reason, medical professionals must be included in all phases of disaster planning as well as in the immediate response to these events.

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Classifying Disasters

Natural versus technological disasters

Disasters are classified in a variety of ways. A common system divides incidents into natural and technological (human-made) disasters. For planning purposes, this system provides little conceptual help, and there are frequent crossovers. For example, artificial structures may collapse as the result of hurricanes or earthquakes. Fire and rescue personnel had to contend with flood water while fighting structural fires in Grand Forks, North Dakota.

Certain generalizations may be made about natural disasters. Tornadoes may be quite lethal but are generally short-lived. Hurricanes cut a wider swath than tornadoes, tend to last longer, and have more long-term recovery effects. Tornadoes, hurricanes, and floods tend to occur in certain geographic locations. Volcanoes also may be quite lethal but have become more predictable in recent years. The most devastating natural phenomena are earthquakes. The recent (January 2001) earthquake in India has claimed at least 30,000 lives, and the death toll may be as high as 100,000. Additionally, the widespread destruction and long-term effects of earthquakes tend to impact a much larger population than most natural disasters. This situation could worsen with time with continued global urbanization, especially in underdeveloped countries prone to these phenomena.

Technological disasters tend to be more contained but can be quite lethal. Fires have caused some of the largest numbers of casualties in this country. Toxic spills (eg, in Bhopal, India) and nuclear mishaps (Chernobyl) have caused short- and long-term havoc, death, and destruction.

War and terrorism

Other incidents with potential for mass casualties and disaster include war and terrorism. Although the world has yet to experience a terrorist-related nuclear disaster, the raw materials and technology exist to

develop nuclear devices as small portable units. No location is immune from the devastating effects of terrorism. These activities in recent years have become more lethal and with no forewarning. Domestic and foreign examples include the bombings of the Murrah Federal Building in Oklahoma City and the World Trade Center in New York City and the Sarin nerve agent attack on the Tokyo, Japan, subway system. A great fear during the Persian Gulf Crisis was the possibility of a biological weapons release by the Iraqi army.

Classifying disasters

Disasters are often classified by the resultant anticipated necessary response.

- A Level I disaster is one in which local emergency response personnel and organizations are able to contain and deal effectively with the disaster and its aftermath.
- A Level II disaster requires regional efforts and mutual aid from surrounding communities.
- A Level III disaster is of such a magnitude that local and regional assets are overwhelmed, requiring statewide or federal assistance.

Disaster preparation

Various methods have been developed to assist planners in disaster preparation. One, a modification of the Injury Severity Score, is based on cause, effect, area involved, number of casualties, and other parameters. The potential injury creating event (PICE) system is designed to identify common aspects of a disaster and of response capabilities. Such systems are especially valuable tools in planning for disaster mitigation.

The PICE system uses 4 modifiers to describe a particular disaster (see [Table 2](#)). The first modifier describes the potential for additional casualties. The second identifies the degree to which local resources are disrupted. The third modifier identifies the geographic boundaries of involvement. The final modifier, crisis staging, indicates the likelihood of needing outside assistance to augment or replace local resources. Note that these descriptors would change for identical disasters depending on the location of the event and the availability of resources.

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Definitions and Terminology

Disaster medicine is difficult to conceptualize. It is broadly defined in several ways.

The World Health Organization defines a disaster as a "sudden ecological phenomenon of sufficient magnitude to require external assistance." The American College of Emergency Physicians states that a

disaster has occurred "when the destructive effects of natural or man-made forces overwhelm the ability of a given area or community to meet the demand for health care." Other definitions exist, but the common denominator calls for a disruption of such magnitude that the organization, infrastructure, and resources of a community are unable to return to normal operations following the event without outside assistance.

In contrast to disasters, multiple casualty incidents (MCIs) have as their primary effects morbidity and mortality to individuals, while the community infrastructure remains relatively intact. A passenger train accident with 500 injured or dead occupants is considered an MCI. If, however, this morbidity and mortality were the result of the release of chlorine gas from a hazardous material accident, a much higher potential for additional casualties would exist. Normal operations and activities of daily living would be disrupted for a longer period. This is considered a disaster by most authors.

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Phases of Disaster Response

A disaster cycle has 4 phases, and all responses must pass through each. These are (1) mitigation, (2) planning, (3) response, and (4) recovery. Pitfalls during transitions occur throughout the phases. Generalized awareness, proper planning, and contingencies may reduce their overall effect.

Mitigation

In certain cases, some of the devastating effects of disasters can be reduced before the actual event. For example, evacuations may be orchestrated before hurricanes or floods. Early warning allows residents to seek shelter from tornadoes. Sprinkler systems in businesses and homes can reduce overall risk of total fire destruction.

Planning

Disaster planning is discussed more thoroughly in [External and Internal Planning](#). It cannot be stressed enough, however, that a disaster plan is not synonymous with disaster planning. Many communities have detailed "paper" plans, which, when tested, are found to be either based on faulty assumptions or to be totally unworkable in the heat of the initial response.

Response

A number of events occur during initial response to a disaster. If there is forewarning, certain of these events may take place even before the event. Unfortunately, significant forewarning is rare.

Activation

Notification and initial response

During this phase, organizations involved in disaster response and the potentially affected populations are notified. In the event that the disaster is anticipated, this phase takes place even before the disaster. Many locations in hurricane areas require more than 24 hours for full evacuation.

Organization of command and scene assessment

Once the activation phase has begun, the prearranged command and staff structure (for details, see [Incident command system](#) below) for responding to the disaster should be arranged and initial communications nets established. This is one of the most crucial steps to take once the disaster occurs.

Historically, much valuable time is lost during a disaster response while the central system coordinating the response effort is being prepared. During this phase, initial reports leading to overall scene assessment begin to arrive. For static disasters, required response assets may need to be determined. Often, the only initially known fact is that the disaster is an ongoing process. However, even this fact is important in determining whether outside assistance is needed, leading to timely activation of those resources.

Implementation

Search and rescue

Depending on the structure and function of the incident command system (ICS), search and rescue may fall under the direction of fire, emergency medical service (EMS), or police (security) forces. In contained, geographically localized incidents, the search and rescue effort is fairly straightforward. In larger disasters, especially ones that are ongoing or may involve terrorist activities, a cooperative approach is necessary and the very act of search and rescue must be highly organized to ensure adequate and complete coverage of all areas.

Extrication, triage, stabilization, and transport

Extrication has evolved into a fire services function in most of the country. In addition to specialized technical and trench rescue teams, fire services have more experience with building collapse and secondary hazards (eg, floods, fires) than other organizations.

The concept of triage involves providing the most help for as many as possible. A complete description of triage is beyond the scope of this review. Medical personnel are accustomed to providing extensive,

definitive care to every patient. When confronted by a number of patients simultaneously in a disaster situation, it is easy to become overwhelmed, even for an experienced disaster worker. Triage must occur at multiple levels, and patients must be reassessed during every step of the process.

Transport must be both organized and orchestrated. During recent civilian disasters and even in Operation Desert Storm, the majority of critically injured individuals were taken to only 1 or 2 receiving facilities, which were almost overwhelmed. This occurred at a time when other facilities sat dormant awaiting patients.

Definitive scene management

While scene control and containment may be relatively simple in a local, static disaster, dynamic and paralytic disasters may take several days to contain and stabilize. As the length of time of the disaster increases, additional resources must be made available, as rescue crews reach exhaustion, supplies become spent, and additional hazards develop.

Recovery

The recovery phase is frequently underemphasized in disaster plans, but it is crucial for the affected community. During this phase, some semblance of order is restored, public utilities are reestablished, and infrastructure begins to operate effectively. Scene withdrawal and a return to normal operations usually occur simultaneously. Treatment of the responders is also vitally important during this phase. Critical incident stress debriefing has evolved for this purpose (see [Critical Incident Stress Management](#)).

Debriefing

Valuable lessons may be learned during debriefing. It is of utmost importance to obtain as much information as possible from all parties involved in the disaster response effort. Without full disclosure, similar weak responses will impede future efforts.

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External and Internal Planning

External planning

A disaster plan encompassing both local and regional areas must focus on 3 possible scenarios:

1. The disaster occurs within the region and is confined and controlled with existing resources.

2. The disaster occurs in a neighboring region, and regional assets are requested through mutual aid agreements.
3. The disaster area is the region and requires state or federal assistance for an effective response.

Incident command system

After a series of fires in California in the 1970s, the Fire Suppression Services developed the ICS concept to organize an effective response to major disasters. The ICS structure includes 5 functional units of command, operations, logistics, planning, and finance (see [Picture 1](#)). Most disaster plans include similar organizational structures that are modified depending on normal operations of the various agencies.

In developing a disaster plan, leaders should remember that it is impossible to plan for all contingencies; therefore, plans must be relatively general and expandable. Most disasters that can be contained using local or regional resources have fewer than 100 fatalities and fewer than 500 major casualties. Obviously, these numbers vary according to the community's supportive resources. If plans are developed for large-scale disasters, they should focus on the first 48 hours of the disaster. Although state and federal assistance is available for these larger incidents, it is rare for these assistance teams to arrive during the first day. Research suggests that virtually all fatalities occur during the first 24 hours.

Rehearsal

All phases of the disaster response must be addressed in a disaster plan. Functional job descriptions and responsibilities of all agencies and organizations involved should be delineated clearly. More importantly, these plans should be exercised and rehearsed. The ideal exercise includes participation by all parties involved. Since these exercises, by their very nature, disrupt normal operations and are costly in personnel and material utilization, disaster agencies frequently resort to tabletop exercises. This is a simulation of an emergency situation for training and testing plans and procedures that does not involve movement of response resources. Tabletop exercises are excellent training tools, because they allow people in leadership positions to work through major problems without the cost of running vehicles, using staff and volunteer time, or using supplies. They can quickly highlight areas of weakness where additional support may be needed.

Organization

As part of the Federal Response Plan, the National Disaster Medical System was developed in the 1980s by the Department of Defense, the Veteran's Administration, the Federal Emergency Management Agency, and the Department of Health and Human Services. The Federal Response Plan calls for the development and response of up to 12 functional units to assist, but not direct, the disaster response initiative on declaration of a state of emergency by a territory or state government (see [Table 3](#)). Approximately 1000 stateside beds were identified in preparation for Desert Storm, although no simulation exercise was performed, leading to criticism from the Government Accounting Agency.

Disaster medical assistance teams (DMATs) are groups composed of approximately 30 volunteers including physicians, nurses, EMS personnel, and others who are transported to disaster sites to participate in the triage, stabilization, transport, and treatment of patients. The utility of teams that are geared toward emergency response (such as the Marine Corps Chemical and Biological Immediate Response Team [CBIRT]) has recently been questioned because of the delay in their arrival for situations with immediate need. DMATs responded to the Oklahoma City Federal Building bombing and have prestaged at certain critical events, such as the Atlanta Olympic Games and the 1995 Democratic National Convention.

Internal planning

Hospital disaster planners must take into account the scenarios previously described, including the possibility that the disaster may involve the hospital. Such rare events as mass decontamination, multiple triage and staging areas within the confines of the hospital, recall of personnel, and adequate supplies and resupply must be anticipated. The Joint Commission on Accreditation of Hospitals (JCAHO) requires hospitals to exercise disaster plans periodically and to form disaster committees. These committees should comprise key departments within the hospital, including administration, nursing services, security, communications, laboratory, radiology, medical records, and maintenance/engineering.

The hospital disaster plan should include protocols and policies that meet the following needs:

- Recognition and notification
- Assessment of hospital capabilities
- Personnel recall
- Establishment of a facility control center
- Maintenance of accurate records
- Public relations
- Equipment resupply

New, more stringent requirements for health care organizations were approved by JCAHO in 2000 and went into effect as of 1 January 2001. Probably most significant are the requirements to integrate hospital disaster planning into community plans, to ensure that disaster programs address all phases of the disaster cycle, and to have the capability to evacuate the entire hospital staff and patients and relocate and operate from an independent facility. Discussions are continuing with JCAHO to further strengthen requirements concerning decontamination, polices, and training in response to terrorist activities involving chemical,

biological, radiological, nuclear, and explosive agents.

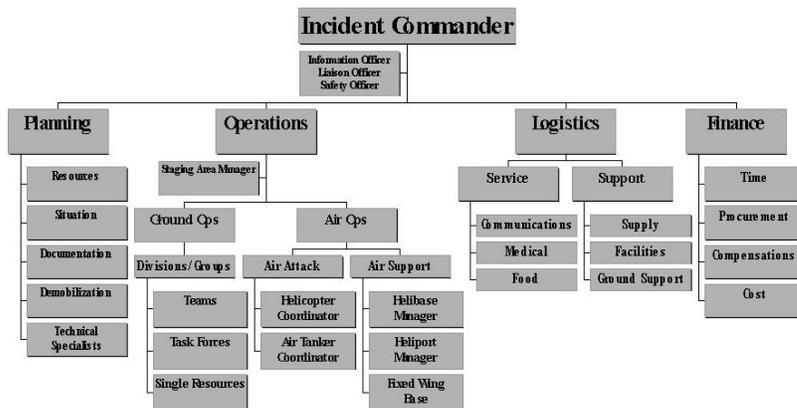
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Summary

Disaster planning is a regional effort. Every jurisdiction should plan for MCIs and disasters. All plans must be simple and based on normal daily operations of the various components involved in the disaster plan. Personnel potentially involved must be familiar with the disaster plan. It should be exercised frequently, even if only by paper or tabletop drills. Contingency plans for mutual assistance and state or federal response also must be considered and reviewed.

Pictures

Incident Command System



Picture 1: Incident command system organizational chart

Picture type: Graph

US Disasters with Greater than 1,000 Casualties

| Year | Event | Deaths |
|------|------------------------|--------|
| 1865 | Steamship Explosion | 1547 |
| 1871 | Forest Fire, Wisconsin | 1182 |
| 1889 | Flood, Pennsylvania | >2000 |
| 1900 | Hurricane, Texas | 8,000 |
| 1904 | Steamship Fire | 1021 |
| 1928 | Hurricane, Florida | 2000 |

Picture 2: Major US disasters (deaths >1000)

Picture type: Photo

Potential Injury Creating Events (PICE) Algorithm

| A | B | C | Stage |
|---------|------------|---------------|-------|
| Stable | Static | Local | 0 |
| Dynamic | Disruptive | Regional | I |
| | Paralytic | National | II |
| | | International | III |

Picture 3: Potential injury-creating event algorithm

Picture type: Graph

Federal Response Plan

| Function | Responsible Agency |
|------------------------|---------------------------------|
| • Transportation | Department of Transportation |
| • Communications | National Communications System |
| • Public Works | USA Corps of Engineers |
| • Fire Suppression | Dept of Agriculture |
| • Information/Planning | FEMA |
| • Mass Care | American Red Cross |
| • Resource support | Government Services Agency |
| • Health/Medical | Dept of Health/Human Services |
| • Urban Search/Rescue | FEMA |
| • HAZMAT | Environmental Protection Agency |
| • Food Services | Dept of Agriculture |
| • Energy | Dept of Energy |

Picture 4: Federal response plan functional components

Picture type: Photo

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Continuous Quality Improvement in Ems

Continuous quality improvement (CQI) is the sum of all activities undertaken to assess and improve the products and services provided throughout the entire Emergency Medical Services (EMS) system. The goal is to deliver a service that is timely, consistent, appropriate, compassionate, cost-effective and, most importantly, beneficial to the patient's outcome and/or comfort.

Since the inception of modern EMS in the 1970s, the dilemma of providing effective quality assurance for prehospital care by nonphysicians has plagued the EMS community. The EMS environment, with its many limitations, presents multiple challenges for the process of quality management. Over the last 30 years, as a result of poor planning and coordination, the EMS system has evolved into a patchwork of various-sized providers, often belonging to combinations of agencies such as fire services, police, fire and EMS, municipal EMS, private entities, and volunteer organizations. A single system may include a police dispatcher; a fire dispatcher; a volunteer first responder; firefighters for extrication and transport; a private agency paramedic to render patient care; and a physician from hospital A providing on-line medical control while the patient is transported to hospital B. Such complex structures require close coordination between the medical and operational entities to maintain efficiency and resolve conflicts.

Quality management in such systems is hampered severely unless a central body exists to coordinate

various CQI activities. This body should have appropriate authority and representation from the various system components and from the local medical community and public safety agencies.

Major progress in quality assurance has been achieved recently in various EMS systems by following the footsteps of other service agencies and by applying the principles of CQI developed for industrial models by W. Edward Deming and Joseph M. Juran. With its proactive systems-evaluation approach, CQI has revolutionized the way quality of service is assessed and improved. CQI recognizes that the majority of problems result from a failure in the process of providing the service rather than from the providers themselves. Instead of focusing on crisis management, CQI focuses on providing quality service that meets the customer's needs.

Focus of CQI

CQI dictates that quality be built into the system with clearly defined, positively reinforced expectations and good communications. Quality improvement can exist only through constant monitoring of performance in a system that is well designed. Faulty processes must be improved proactively to minimize the chances of problems. Quality management is performed from within the system, by an external, independent, and unbiased body comprising representatives of consumers, public safety agencies, and the medical community.

The primary focus of CQI is to assure that field staff members provide the highest quality of care. The entire system must be supportive of this goal. Measurable standards that reflect the expectations of the system are invaluable in providing the proper tools and environment for success.

Whenever the service provided falls below the expected standards, the following process is implemented:

- The problem is identified.
- A corrective course of action is established and implemented.
- The outcome is measured.

Instead of targeting the "bad apples," CQI targets the care system. Change may be indicated for protocols, policies, procedures, staffing patterns, communications, equipment, and educational programs. By learning from what was done right and wrong, CQI seeks to improve care provided for all patients, not just for those receiving deficient care.

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Protocols and Standards

Establishing clear goals, standards, policies, and protocols right from the beginning that comply with

national, state, regional, and local regulations is important. Measurable standards are developed to respond to the specific needs and limitations of the local environment, including geographical constraints, resource availability, and political issues. These standards serve as guidelines to provide accountability of the medical community. Support of all the agencies involved with providing quality medical care can be achieved with clearly communicated standards and protocols. Consensus among the various agencies is essential in development and enforcement of standards. The medical director has the responsibility of addressing standards dealing with patient care.

Patient care protocol standards

Treatment protocols describe the authority and responsibility of EMTs and provide guidance for medical education and care. They define an acceptable, standardized approach to each commonly encountered patient problem. This allows field staff to provide care with consistent quality. Optimal care and medical accountability require standardized protocols, algorithms, and standing orders to outline specific actions that can be undertaken by a provider without contacting a physician for orders. Any deviation from these standing orders must be considered a breach of duty and may result in an inquiry.

Time standards

Goals are set to optimize response time, on-scene time, and transport time for both emergency and nonemergency services without reducing the quality of services provided. Other time standards may include, but are not limited to, (1) the amount of time required to access the system, (2) dispatch time, (3) medical intervention time, and (4) the amount of time it takes for crews to return back into service.

Procedural standards

These standards describe the circumstances indicated to perform a particular procedure. Specifics of the procedure are communicated, such as how, where, and when to do a procedure and the amount of time required to perform it. Expected results, adverse effects, alternative approaches, and possible complications are described within the procedural standards.

Equipment standards

Patient care could be hindered if the provider is not equipped with the proper working tools. Any new equipment is evaluated in light of its intended use, appropriateness, and field performance prior to purchasing. The system also should be in compliance with the manufacturer's recommendations for scheduled maintenance.

Field performance standards

These are minimum standards that all employees must meet while performing their duties. They include areas of clinical competency, adherence to protocols and policies, personal behavior, manners behind the

wheel, and general professionalism.

Educational standards

Providers need to be trained to meet the expectations and requirements of the system in programs that comply with regional and national standards. Minimum requirements for initial training, testing, and continuing education also must be established.

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Education

Once standards have been established, they must be communicated and taught to all personnel involved. Similar education should take place each time a standard is changed. Education is used to reinforce proper patient care, update the provider regarding newer treatment modalities and equipment, and remedy perceived deficiencies. Mechanisms should be established to measure the effectiveness of the information given and of the educators themselves. Physician involvement is essential to assure appropriate utilization of skills and equipment.

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Data Collection and Analysis

EMS system administrators should constantly evaluate system performance and the level of care delivered. Information is gathered to assess various components of the system. This is particularly difficult because of a lack of consistent, reliable, accurate, and uniform data among the different agencies that constitute the system. Data sources may include standardized EMS run reports, dispatch logs, hospital patient records, incident reports, public safety records, testing, and surveys to assess perception and satisfaction. Patient confidentiality should be assured. Data are to be tracked, recorded, and used to identify areas needing improvement. Providers should be congratulated when standards are met or exceeded. When standards are not met, the reasons are to be determined, and a correction plan is to be established, implemented, and evaluated.

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Quality Evaluation and Improvement

The primary focus of the CQI is to assure that field staff members provide the highest quality patient care. Staff members should be assured that the system in which they work is supportive of this goal. Evaluation of EMS system performance must take place regionally and locally within individual agencies. This evaluation should reflect appropriate priorities and should be accomplished in a positive fashion to maintain morale and to avoid the perception that "Big Brother" is watching.

Prospective evaluation

Prospective evaluation is the most effective process to ensure quality of care in EMS because it can prevent mistakes. The evaluation process involves issuing of credentials to providers and reviewing of the various components of the system, including policies and protocols. The system should exhibit a dynamic process of review and modification of the different system components in anticipation of future needs.

Achieving provider credentials

Issuing credentials to qualified providers is important to ensure the quality of care provided. A comprehensive background check is invaluable to verify the applicant's education, employment history, certification, experience, clinical performance, driving skills, personal interactions with patients and co-workers, and possible criminal record. In addition, the applicant must undergo an evaluation process that includes interviews with several key evaluators and satisfactory performance on an entrance examination that has both written and practical components.

After successful completion of the process to attain proper credentials, the new provider should complete an in-house orientation program, as well as a field preceptor program, to familiarize the new employee with the system. The provider will be evaluated throughout these programs. A provider who fails any of the exams or field performance reviews may be considered for retesting at the discretion of the medical director. Subsequent failures will lead to reconsideration of employment.

After successful completion of the above requirements, the new provider should be partnered with an experienced EMT in a mentoring program. The probationary period should be no less than 6 months. To maintain credentials, the provider needs to complete ongoing educational requirements, have routine performance reviews, and pass periodic skill evaluations.

Training of other care providers

Dispatchers should be trained in the same fashion as field providers. They should comply with local and regional requirements and be able to provide appropriate prearrival instructions.

Sufficient education and experience in EMS are essential for on-line and off-line medical direction providers. To be effective, they must possess an appreciation of the system and awareness of provider skill limitations.

Concurrent evaluation

On-scene review of care reinforces positive behavior and helps prevent and correct errors before bad habits develop. Concurrent performance review should take place during orientation and on a continuing basis through review by the medical director and other supervisors.

On-line medical control provides an important means of concurrent assessment of the EMTs' evaluation and treatment. Feedback from receiving facilities is necessary to measure the appropriateness of providers' assessment and care.

Retrospective evaluation

Retrospective evaluation is the most time-consuming, most often practiced, and least valuable type of review. Information gathered retrospectively often is incomplete and inconsistent.

Critique sessions

Here, the medical director or designee periodically reviews selected runs of interest with the providers. Discussion often surrounds the appropriateness of medical care and adherence to standards. These sessions should be used as an educational vehicle to review standards, protocols, policies, and procedures. They also provide a great forum to solicit feedback on the practicality of certain standards in the field.

Patient encounter review/audits

Although some systems advocate a 100% chart review, such a review is time-consuming and inefficient. Reviewing a representative, randomized sample of encounters (eg, 1 of every 10) is sufficient to assess (1) dispatch management of the emergency call, (2) field documentation, (3) documentation completeness, (4) appropriateness of patient assessment, (5) ability to formulate differential diagnoses and arrive at a working clinical impression, (6) appropriateness of treatment, and (7) timeliness of service. Some services can elect to pay more attention to certain high-risk encounters, such as the following:

- Respiratory and cardiopulmonary arrests
- Multisystem trauma
- Use of specialized advanced life support (ALS) procedures
- Use of on-scene aeromedical evacuation
- Situations when ALS personnel transfer patient care to basic life support (BLS) personnel
- Patient refusals of treatment
- Transport treatment of minors and use of the automatic external defibrillator (AED) and semi-automatic external defibrillator (SAED)

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Exploration Process

An inquiry is a fact-finding process conducted when a weakness in an employee's performance is suggested. Review of the circumstances involved must confirm that the weakness was not system related. The inquiry may reveal that the weakness is related to suboptimal equipment, staffing, protocols, or policies.

Timeliness and confidentiality are essential to the inquiry process. Severity of the allegation and the usefulness of information sought should dictate the extent of the inquiry. Ascertaining relevant information may be as simple as a phone call to the involved employee(s) for clarification. If satisfactory answers are provided to all questions, the process may end there. However, an in-depth review of all related documents and tapes, as well as formal interviews of all parties involved, may be necessary. Inquiries made in person should be conducted with at least 2 senior representatives of the CQI team.

The provider involved should be counseled in private, and a mutually acceptable corrective course of action should begin. Commonly, remediation is limited to a training or educational process and rarely involves disciplinary action. Once the remediation program is completed, the case is considered closed, and all records of the incident are kept confidential. The involved parties should be made aware of all elements of the resolution plan, except those that pertain to another employee.

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Summary

Continuous quality improvement is a vital component of an EMS system. Patients must receive high-quality emergency medical care in the field. The leadership of the medical director and the medical community is crucial in realizing this goal. High-quality patient care requires a comprehensive, up-to-date knowledge base; access to the tools necessary to do the job; and strong motivation.

The CQI team should constantly scrutinize system performance and patient care provided to determine areas needing improvement.

When a goal is not met, the following process should be implemented:

- Identify the problem.
- Establish a corrective plan of action.

- Execute the plan.
- Measure the outcome.

More often than not, the problem is the result of a system deficiency. The system is improved by refining standards and providing appropriate equipment. Providing ongoing education to personnel on use of equipment and specifics of the standards and protocols is essential to providing quality patient care.

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Introduction

The history of EMS extends back to the biblical story of the Good Samaritan. Accounts of ancient wars reveal many examples of organized methods of transportation and care of the sick and injured. Historical archives suggest that Caesar designated battlefield medics among his troops. Napoleon's chief surgeons developed "les ambulances volantes," consisting of horse-drawn wagons staffed with battlefield caregivers. Similar systems, commonly operated by hospitals and funeral homes, were used in various American cities soon after the end of the Civil War.

It was not until the late 1960s to early 1970s that the modern era of EMS was created, with coordinated transport and prehospital interventions, to provide earlier, more intensive care to the community. In the late 1960s, Pantridge established mobile units staffed by physicians and nurses to extend the coronary care unit to the prehospital setting.

The 1966 National Highway Safety Act authorized the US Department of Transportation to fund communication and education for EMS services as well as purchases of ambulances and equipment. Congress enacted the Emergency Medical Services Systems Act of 1973 (public law 93-154), which funded and authorized the Department of Health, Education, and Welfare to develop EMS systems throughout the country. More than 300 regional EMS management entities were designated to develop a systematic approach to EMS care. The US EMS system trained and empowered physician surrogates (ie,

paramedics) to deliver prehospital patient care.

Public law 93-154 identified the following 15 components as essential to an EMS system:

- Communications
- Training
- Manpower
- Mutual aid
- Transportation
- Accessibility
- Facilities
- Critical care units
- Transfer of care
- Consumer participation
- Public education
- Public safety agencies
- Standard medical records
- Independent review and evaluation
- Disaster linkage

The initial EMS design subsequently was proven deficient in many respects, including medical direction and accountability, prevention, rehabilitation, financing, and operational and patient care protocols. EMS systems continued to be refined in the 1980s and 1990s, even after most federal funding had ended.

Successful EMS systems are designed to meet the needs of the communities they serve. The state provides laws that broadly outline what is prudent, safe, and acceptable. To be effective, EMS systems must be planned and operated at the local level.

Communities need to identify their individual needs and resources, develop funding mechanisms, and become involved on all levels in structuring the system. A governing body or council should be established to organize, direct, and coordinate all system components. The council consists of representatives from the local medical, EMS, consumer, and public safety agencies to ensure consensus in developing policies and settling disputes. The EMS system must provide equal access to all and remain protected from forces that serve the interests of only one group.

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Medical Direction

Physician input, leadership, and oversight are essential in ensuring that the medical care provided is safe,

effective, and in accordance with accepted standards. Physicians must be empowered and involved in planning, implementing, overseeing, and evaluating all components of the system. Medical direction is characterized as either immediate (on-line) or organizational (off-line).

On-line medical direction provides emergency medical technicians (EMTs) with clinical consultation in the field, either in person or, more commonly, via radio or telephone communication. This responsibility is delegated primarily by the medical director to physicians who staff local EDs. The base station facility providing on-line medical control is required to monitor all advanced life support (ALS) communications, provide field consultations, and notify receiving facilities of incoming patients. Physicians providing on-line direction should be trained appropriately and be familiar with the operations and limitations of the system.

The medical director assumes authority and responsibility for off-line medical direction. In cooperation with the local medical community, the medical director is responsible for developing standards, protocols, policies, and procedures; developing training programs; issuing credentials and providing evaluations; and implementing a process for continuous quality improvement.

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Communications

A comprehensive communications plan is essential to provide the community access to system dispatch and to provide the EMT access to medical direction and additional resources. Establishing the universal 911 emergency telephone number has greatly improved the system's accessibility. Additional advancements have been made with the introduction of enhanced 911 systems, which automatically provide the dispatcher with the caller's address and telephone number. Using enhanced 911, callers can obtain services even if they are unable to communicate with dispatch.

Emergency medicine dispatch includes assessment of patient location and status, provision of prearrival instructions, coordination with public service agencies, setting priorities for use of resources (ALS, basic life support [BLS]), and response mode (lights, sirens). Two-way radios and cellular phone links are used to establish communication between the base, field, and one or more hospitals. The communication plan should define EMS equipment, training, frequencies, protocols, policies, and etiquette.

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Transportation and Facilities

Ground vehicles provide most EMS transportation. Ambulances should be constructed according to federal standards and be equipped appropriately to provide basic or advanced care.

Air transport consists of a helicopter or airplane equipped for BLS or ALS. Aircraft are used to transport patients over great distances, decrease total prehospital time, and reach patients in locations where access is difficult. Operational standards are established to delineate the equipment needed, the number of personnel and level of certification required, the response-time criteria, and the destination for each transport.

On-line medical direction should be obtained for all calls that result in no transport. This includes cases in which providers decide not to transport, the patient refuses care, or the patient is triaged to a lower level of care. Otherwise, the provider may be perceived as practicing medicine without a license and could be charged with such an offense.

Interfacility transportation occurs once the patient has been examined and stabilized. Patients are transported in compliance with regional protocols and federal laws (ie, Consolidated Omnibus Budget Reconciliation Act [COBRA], Emergency Medical Treatment and Active Labor Act [EMTALA]). Legislation dictates that medically unstable patients be transferred only when the transfer is expected to have a positive effect on outcome.

Patients should be transported to the closest, most appropriate facility. Receiving facilities are required to have the capabilities to treat patients, stabilize their conditions, and improve their outcomes. Patients in stable condition may be transported to the hospital of their choice, as long as the transport meets regional point-of-entry protocols, has the approval of on-line medical control, and does not overburden the system unnecessarily.

Specialized facilities required to care for the severely injured are not available in every hospital. Local communities need to establish regional protocols to provide clear guidance for the transport of patients in unstable condition to categorized facilities. Patients who are in unstable condition because of special problems, such as trauma and burns, can be transported to regionally designated hospitals, bypassing closer facilities.

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Training and Protocols

Providers must be trained to meet expectations and requirements in programs that comply with regional and national standards. Training includes didactic, clinical, and field components. Most states require that candidates pass written and practical examinations prior to certification. Additionally, EMTs are required to receive continuing didactic and clinical education to maintain certification.

Education also is used to reinforce proper patient care, update standards and protocols, and remedy perceived deficiencies in patient care. Physician involvement is essential to assure appropriate utilization of skills and equipment. The EMS system also provides community education, such as public courses in cardiopulmonary resuscitation (CPR), first aid, child safety, and EMS access.

Protocols are developed to deal with operational, administrative, and patient care issues. They define a standardized, acceptable approach to commonly encountered problems. Protocols should reflect regional and national standards, as well as the uniqueness and limitations of the local environment. The medical director has the responsibility to address protocols dealing with patient care, such as triage and treatment.

Triage assesses the condition of each patient, sorts patients into treatment categories, and optimizes use of field resources for treatment and transportation. In addition, triage addresses the level of provider capability and the level of response needed to care for the patient. Specific triage criteria are essential during multiple casualty incidents to facilitate the screening, prioritization, treatment, and transport of patients.

Treatment protocols describe the authority and responsibility of providers and offer guidance for medical evaluation and care. Optimal care and medical accountability require standardized protocols, algorithms, and standing orders that outline specific actions providers can take without contacting a physician for orders. Any deviation from these standing orders must be considered a breach of duty and must result in an audit. On-line medical direction is crucial in systems requiring decision making to provide guidance and assume some of the patient-care responsibilities.

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Continuous Quality Improvement

Continuous quality improvement (CQI) is the sum of all activities undertaken to assess and improve the products and services EMS provides. The goal is to influence patient outcomes positively by delivering timely, consistent, appropriate, compassionate, and cost-effective services. CQI ensures that the field staff provides the highest quality of care and that the system supports this goal. Quality should be monitored from within the EMS system and by an external, independent, and unbiased body that involves the consumer, government, and medical communities. Standardized protocols, policies, performance, and documentation are invaluable in constructing a successful CQI process.

Quality evaluation is prospective, concurrent, and retrospective. Prospective evaluation is the most effective process to ensure quality in EMS, because it has the potential to prevent mistakes. The system must be scrutinized constantly to determine areas requiring refinement and improvement. When goals and standards are not met, CQI staff members must identify the problem, establish and implement a corrective course of action, and measure the outcome. Concurrent evaluation occurs on-scene or on-line.

Staff members observe performance, encourage positive behavior, and correct problems before bad habits develop. Retrospective evaluation is the least valuable and most time-consuming review. It includes critique sessions and reviews of patient encounter tapes and charts.

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Disaster Planning and Community Relations

The EMS system is an integral element of disaster preparedness and planning. It plays an important role in initial response and transportation and is essential in establishing a regional disaster preparedness plan in coordination with public safety agencies, government, and the medical community. The plan should address disaster management, communication, treatment, and destination of casualties. Periodic disaster drills serve to assess performance, refine management, and educate personnel and the community.

Public support is invaluable in constructing a successful EMS system; involvement is required to plan a system that works for everyone. Consumers need to be well informed of the benefits of having an EMS system and how to gain access to it.

Public education programs are essential to inform the community on ways to access the EMS system properly. They also are important in preparing laypersons to render first aid while waiting for EMS. These programs should be coordinated with local public safety and volunteer agencies to project a unified message and achieve maximum impact.

The EMS system must have strong ties with many agencies inside and outside the community. Cooperation is essential with public safety agencies, which are most frequently the first to respond to an emergency and may provide all or part of EMS care.

Mutual aid agreements should be developed with neighboring communities to provide assistance when one system is disabled or overburdened. These arrangements ensure uninterrupted patient care in the event of natural disasters or other emergency situations.

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Summary

An EMS system is a comprehensive, coordinated program that delivers prompt response, appropriate care, and safe transport in medical emergencies. EMS should be designed to fulfill the needs of the local community and to provide equal access for all patients. Consumers, in conjunction with the local medical

community, EMS, and public safety agencies, should help organize and govern the system. This council and the medical director must ensure the presence and function of all the systems and their necessary components.

The EMS system is only as strong as its weakest component. It should be evaluated continuously and modified to maximize quality, optimize efficiency, and minimize cost.

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Ems and Cardiac Arrest

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Traumatic Cardiac Arrest

Victims of traumatic cardiac arrest rarely survive outside the hospital. If the patient has suffered blunt trauma, cardiac arrest prior to reaching the hospital carries a 99% mortality rate in spite of ongoing efforts at resuscitation after arrival at the hospital. In victims of penetrating trauma, cardiac arrest prior to reaching the hospital produces virtually the same negative outcome. However, some studies suggest that patients who arrive at the hospital just after or while losing vital signs may have a better outcome if an open thoracotomy and aortic cross-clamping are performed while efforts at resuscitation continue. Some studies report a successful outcome approximately 4% of the time.

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Medical Cardiac Arrest

In patients suffering cardiac arrest outside the hospital, the outcomes and survival rates are unclear. Numerous articles present conflicting data. Indeed, data collection has been fraught with difficulties, creating problems for efforts to compare data and survival rates between studies. Comparison of data from various studies has been complicated by differences in EMS system capabilities, response times, documentation of down times, bystander intervention with cardiopulmonary resuscitation (CPR), and documentation of rhythm upon presentation. The outcome variables studied have included such factors as patient survival, hospital admission, and discharge from the hospital neurologically intact. Recent efforts

to standardize definitions and data collection have resulted in the uniform adoption of the Utstein criteria for use by EMS researchers studying outcomes for patients suffering cardiac arrest outside the hospital.

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Factors Affecting Survival

Comparison of data between studies has shown a survival range from 4.9-22%. This disparity is due, in part, to different measures of outcome employed to define survival. In spite of the limitations in interpretation of these studies, several factors appear to be associated with an increased chance of survival in cases of out-of-hospital cardiac arrest.

Patients presenting with an initial rhythm indicating ventricular fibrillation have the highest rate of survival. One rural system in northwest Washington State documented a 43% survival rate when the presenting rhythm was ventricular fibrillation or ventricular tachycardia. Other studies have shown that, in 56% of patients, immediate defibrillation by EMS of patients in ventricular fibrillation has resulted in a pulse-generating rhythm that was maintained through hospital discharge. This drops to 6% with the third defibrillation attempt. Other studies report that 28% of patients converted to normal rhythm if the defibrillation attempt occurred within 12 minutes after cardiac arrest. Cardiac arrest cases in which rapid bystander CPR is administered have a higher survival rate than cases without this treatment. Many studies report that survival is extremely unlikely if the presenting cardiac rhythm is asystole.

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Out-of-Hospital Termination of Efforts

Recent research supports acceptance of termination of resuscitative efforts in the field by authority of prehospital personnel in the field or of family members or physicians by radio command order. This generally occurs when out-of-hospital efforts at resuscitation include prolonged advanced life support measures and the patient is in asystole. This is consistent for medical and traumatic cardiac arrests, particularly in rural areas where transport times to a hospital are prolonged. Some EMS services also provide family grief counseling in these circumstances.

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Ems and Mass Gatherings

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Introduction

Public gatherings represent special challenges to EMS. Emergency medical care delivered in this setting is inconsistent and can fall short of the need in the absence of adequate planning, training, and personnel.

Surprisingly little data are available from which to plan the emergency medical needs for public events. No recognized standards or guidelines exist for providing emergency medical services at public mass gatherings.

Most authorities define a mass gathering as a group exceeding 1000 persons. Many times that number often gathers for large events. This chapter discusses planning for such an event. Both prehospital and physician issues will be reviewed.

Nine major elements should be considered when planning such an event, as follows:

- Crowd size
- Personnel
- Medical triage and facilities
- Medical care
- Public information and education

- Medical records
- Mutual aid
- Data collection
- Other factors, including public access, disaster planning, weather, and event duration

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Structures and Location

Virtually every imaginable structure has served as a venue for mass gatherings. Papers have been written about events at the Calgary and Los Angeles Olympic Games, Vancouver World Expo, Indianapolis Speedway, NFL football games, and college sporting events. Early literature discussed care at the 1969 antiwar demonstrations and the first concert at Woodstock.

The type of structure and surrounding geography play significant roles in the planning for the event. Most patients are treated at a predetermined site where they must either go by themselves or be brought. Planners must consider the following:

- Transport of patients to treatment sites
- Routes to treatment sites
- Routes EMS personnel may take to reach patients
- Optimal placement of treatment sites
- Number of sites needed
- Possible obstructions to patient flow
- Egress routes (way out) to other facilities
- Availability of other facilities
- Environmental realities of the location

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Nature of the Event

Crowds come to mass gatherings out of common interest in the event. The participants at a rock concert are likely to be very different from those attending the Indianapolis 500. While gatherings are commonly collections of healthy individuals, some are not. Fulde reported on care at a "Concert For Life" in Australia. The event was held to benefit a cardiac research center and an AIDS service. It attracted large numbers of cardiac patients, including transplant recipients, and people suffering from AIDS.

The type of patient encounter is determined largely by the type of crowd. Most involve minor problems such as headache, minor abrasions, lacerations, or burns. In addition, problems specific to the type of event are likely. For example, individuals affected by various drugs are likely to present at rock concerts. Typical presentations by event type include the following:

Rock concerts - Drugs, alcohol, minor trauma

Auto races and Olympic Games - Serious trauma, heat- and alcohol-related problems. Athletes and primary competitors may have their own medical teams.

Demonstrations - Injuries and tear gas

Professional/college sporting events - Minor injuries, intoxication, heat-related problems, and cardiac problems, including arrest

Citizen sporting events, runs, cross-country ski races - Heat-related illness, exhaustion, and cold-related illness

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Crowd Size and Demographics

Usage rates (ie, the number of people treated per 1000 attendees) vary widely. At the 1986 Vancouver World's Expo, the average was 3.9. At the Los Angeles Olympics, the rates varied from a low of 0.68 at the soccer venue to a high of 6.8 at the rowing/canoeing venue. When usage is divided into acuity levels, as was done at the 1988 Calgary Olympics, most are of low acuity.

As a general rule, volume is a function of the number of attendees and the duration of the event. Some authors note that usage rates are likely to be higher in settings where groups are allowed to move about more freely.

In settings attracting older spectators, the likelihood of serious medical problems is higher. One author suggests that, although the provision of primary first aid is admirable, assuring the availability of on-site defibrillators, particularly automated external defibrillators, is more advantageous.

Most authors admit that a written record will not be kept for most trivial visits. All agree that good records should be kept for all visits except the most trivial. Standard items include demographic data, brief medical history, type of illness or injury, treatment rendered and disposition. Various databases can be used, if required. Everyone agrees that good records should be kept for those patients whose illness or

injury warrants transportation to a hospital.

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Environmental Factors

Warm weather events increase the likelihood of heat-related problems among both the athletes and the spectators. Several years ago, the American College of Sports Medicine issued a position statement that strenuous events be postponed or canceled at certain wet bulb temperatures, a combination of several environmental factors. Heat exhaustion is much more common than heat stroke, but even well-trained athletes suffer heat stroke. Warm weather also increases the total number of participants.

Hypothermia may occur in settings that do not necessarily involve extremely cold temperatures. It is particularly likely in mass gatherings involving water, such as triathlons or citizen swimming events (ie, "swim meets"). The presence of rain markedly increases the likelihood of hypothermia.

Staffing and on-site personnel

The overwhelming majority of patients can be triaged and treated effectively by registered nurses and paramedics. Several authors suggest that the ideal situation would be accurate triage by nonphysicians with rapid referral of appropriate cases.

Ratios of 1-2 physicians for every 50,000 people and 2 paramedics or 1 paramedic/EMT team for every 10,000 people have been recommended. Anticipated usage rates, based on previous experience, allow modification of staffing ratios.

Several authors have suggested that part of nonphysician staff preparation include cardiopulmonary resuscitation (CPR) training and instruction in the use of the automated external defibrillator.

Personnel must know how to communicate with one another. As with disaster and mass casualty incidents, radios provide the bulk of the communication. Land-line phones are helpful if available. Cellular phones also may be considered.

The issue occasionally is raised about the need for physicians to be present at the site of mass gatherings. Most patients do not need physician care, but physicians should be available. The higher the risk of significant trauma, the more likely the event sponsor is to have a physician on the scene.

Physicians with EMS experience should be involved in the writing of protocols, policies, and standing orders. Regional medical command requirements should be considered.

Advocates of physician presence at mass gatherings note the following:

- Overall cost of care should be lower (assuming volunteer physicians) because fewer patients will be referred to hospitals.
- The likelihood of serious trauma has been the impetus for having physician care available at auto races and other events where the risk is great.
- The potential of a long distance to definitive care makes the physician's presence valuable.
- The event sponsor will be much more comfortable with a physician present than without one.
- If a physician is present, hospital impact may be decreased, disaster response enhanced, and media coverage improved.

Physician considerations

The choice of medical discipline varies. Clearly the services of an orthopedist or sports medicine physician are required sometimes, but the emergency physician is the logical choice for medical doctor coverage according to several authors.

Wackerle, writing about the related topic of disaster medicine, said, "Emergency physicians are usually the most appropriate for this role, because they are familiar with the system and personnel providing care before hospitalization; they are practiced in rapid assessment, basic treatment, and triage and they have a good working rapport with other specialists needed during the response."

Malpractice coverage must be addressed. Commonly, physicians must obtain a rider from their insurer.

Physicians usually must obtain or hold a license in the state where the event is to be held, although some states grant temporary privileges in certain situations. The physician must be sure of the scope of practice permitted in those cases.

Regardless of whether the physician medical director is present at the event, the physician has the responsibility of laying out the ground rules. Written standard operating procedures must be explained and understood by personnel. Personnel also should understand that the medical director has certain expectations involving commitment, behavior, and performance.

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Transportation

Transportation raises several issues, including the following:

- How does the patient get to the triage/treatment area?

- What route will the patient take to get there?
- What directions need to be provided to the crowd directing them? Do directions need to be provided in more than one language?
- Where will the triage/treatment areas be located?
- If litters are to be used, where will they be located and who will use them?
- Where will the ambulances be staged?
- What roads are available to the ambulances?
- What obstructions, such as physical, structural, or weather related, could the ambulances encounter?
- How many ambulances should be kept at the site?
- From where will backup vehicles be drawn?
- Where will patients be taken? How are hospitals to be notified? Potential receiving sites should be notified of mass gatherings far in advance of the event.

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Equipment

In addition to transport equipment such as litters and ambulances, a well-stocked basic life support (BLS) or advanced life support (ALS) vehicle, depending on provider capability, will carry most of the needed supplies, including the following:

- Automated external defibrillators, monitor(s)
- Airway equipment
- Endotracheal tubes, laryngoscopes, and blades
- Cricothyrotomy supplies
- Oxygen equipment
- Nebulizer with medications for nebulizer treatments
- Bag/valve/mask
- Suction
- Bandages
- Immobilization devices
- Intravenous (IV) fluids, catheters, administration sets
- Syringes, needles
- Stethoscopes
- Handheld radio
- Pneumothorax kit with Heimlich valve
- Medications including but not limited to atropine, naloxone, morphine, lidocaine, dextrose (D50), epinephrine, albuterol, adenosine, nitroglycerine, furosemide, and diphenhydramine

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Interagency Coordination

Agencies that should or may need to be involved in the planning of care for mass gatherings include the following:

- Local, regional EMS
- Fire Department
- Red Cross
- Police Department
- Local hospitals

As with mass casualty incidents, advance planning is essential to assure delivery of needed health care. Anyone who may be involved with interagency coordination should help with the planning. For those agencies that may but are unlikely to be required, written agreements for their participation should be obtained.

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Ems and Terrorism

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Introduction

Emergency Medical Services (EMS) often co-manage initial scenes in many medium- to large-scale incidents involving large numbers of casualties. Occasionally, EMS also manages the scene until state or federal agencies arrive. Even though relatively few terrorist-related incidents have occurred in the US, EMS agencies must plan for and anticipate such incidents.

Many authorities consider the US a very high-profile target. For many Americans, the bombing of the Federal Building in Oklahoma City in 1995 brought home the tragic realization that America is not immune to terrorism. Philip Stern, an expert on terrorism with the Fairfax Group, said, "As an open society and a democracy this country is particularly vulnerable. We have free passage, coast-to-coast, anyone can apply for a visa to visit and the population is enormous and diversified."

Some feel that such an inviting atmosphere makes terrorist activities inevitable. A report released in 1997 by the US Senate Permanent Subcommittee on Investigations in 1997 said, "It is not a matter of IF, but WHEN such an event will occur. Many of the terrorist groups of today appear more and more likely to utilize weapons of mass destruction."

Perhaps even more disturbing, some feel that terrorists are tending toward more technologically advanced weapons and communications. Use of the Internet and information on the World Wide Web

facilitates tremendous technological advancement in destructive potential.

All of these issues underscore the importance of proper education in EMS and advance planning. Terrorist activity may have various mechanisms, including chemical or biological weapons and explosives, as well as new methods of destruction. Full response to a terrorist incident requires in-depth interagency planning involving police; fire; EMS; regional, state, and national emergency organizations; as well as a wide array of ancillary services. Assign each agency respective roles as soon as possible, well in advance of potential terrorist acts.

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Aspects of Terrorism

Determining the actual extent of the terrorist threat in the US is difficult, as it is poorly defined and rapidly changing. James Alan Fox, Dean of Northeastern University's Criminal Justice Program in Boston, believes the 2 current motivations for terrorism are revenge and attention.

Several terrorist incidents in the US highlight the lethal nature of terrorist acts: the World Trade Center bombing, the Atlanta Olympic bombing, and the Oklahoma Federal Building bombing.

In 1993, the World Trade Center in New York City was bombed, killing 6 people and injuring more than 1000. In April 1995, the Federal Building in Oklahoma City was bombed, killing 168 people. A pipe bomb was detonated in Centennial Park during the 1996 Olympic Games in Atlanta, Georgia, killing 2 people and wounding more than 100. Such events emphasize the potential loss of life and property caused by terrorist activities.

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Preplanning

Effective response to a terrorist incident hinges on comprehensive planning and interagency cooperation. Address and resolve jurisdictional issues well in advance. After a plan has been devised, update it regularly to reflect changes in resources, population, terrorist activities, or potential targets. Local police, fire, EMS, and Disaster & Emergency Services (DES) agencies should form the first line of response. Other agencies that may be involved include the following:

- Secret Service
- Bureau of Alcohol, Tobacco, and Firearms (BATF)

- State disaster agencies
- Military or reserve units
- Specialized medical units

Depending on circumstances, state or federal response agencies, such as the Emergency Broadcast System, Federal Aviation Administration, or National Weather Service may need to become involved.

An act of terrorism in a metropolitan area may cause major health and medical consequences that could rapidly overwhelm virtually all local health facilities; thus, ongoing contact with state and national agencies is recommended highly. The Federal Bureau of Investigation (FBI) is the federally designated lead agency in a confirmed domestic terrorist event. Depending on location, however, some federal agencies may not be on-site for 24 hours or more.

On a national level, the US Government has established the National Disaster Medical System (NMDS). This organization assists in providing medical care and transportation for disaster victims. The NDMS comprises sections of the Department of Health and Human Resources, the Department of Veterans Affairs, the Department of Defense, and the Federal Emergency Management Agency (FEMA). Any state can enlist the services of NDMS, which provides assistance at the disaster site, evacuates patients, and finds beds for evacuated patients. Congress also has established a Domestic Preparedness Program that provides enhanced training for local first responders and forms metropolitan medical strike teams in major cities.

In evaluating sites of potential terrorist activity, consider the release of chemical or biological agents into crowded and contained areas, such as sports stadiums, office or public buildings, and transportation systems (eg, the Tokyo subway Sarin incident). Such places provide tempting targets despite on-site security. Rapid identification of the chemical or biological agent is critical to proper disposition of patients and to management of affected areas. Disasters of human agency, particularly involving hazardous materials (HAZMAT), radioactive materials, or chemical agents, may produce unfamiliar medical problems that cannot be identified rapidly in readily available emergency medicine (EM) literature. Utilize all possible resources early in the incident to ensure proper identification of the agent and prompt initiation of proper protocols.

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Incident Management

Most of the principles of incident management are similar to those of mass casualty incidents. Primary concern in potential terrorist incidents is to secure the area and to ascertain the severity and nature of the threat. Keep in mind that delayed explosives or materials intended to harm rescue workers may have been planted at the site. Primary and secondary perimeters must be established and secured. Determine if

a cleared, downwind perimeter is needed, and establish one if required.

Early involvement of support and ancillary services, mutual aid agencies, and local agencies in the planning process is prudent. After identifying the potential threat, determine which type of protective equipment is necessary. Emphasis must be placed on decontamination and protection of rescuers and victims.

After establishing a decontamination and triage area, rescuers should put on appropriate protective clothing before entering the affected area and beginning rescue efforts. The first focus is on supportive care with emphasis on aggressive airway control and decontamination. Issues associated with simultaneous containment, neutralization, and/or decontamination may be addressed by ancillary agencies. Following initial triage, patients are given primary or aggressive aid depending on their presentation and the resources available. Decontaminate and transport patients to a facility that has been informed about the etiology of the incident as soon as feasible. Secure and clean the area to complete the physical response. Recordkeeping, analysis of the incident, and investigations conclude the complete response.

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Selected Agents of Terrorism: Conventional Explosives

An explosive is a normally stable material that, when introduced into a chemical reaction, converts rapidly from a solid or liquid to an expanding gas. Explosives are categorized as either low grade or high grade. Low-grade explosives burn quite rapidly. Black powder, the original low-grade explosive, served as the basis for development of smokeless gunpowder and some rocket propellants. Other examples of low-grade explosives are nitrostarch, nitrocellulose, and commercial fireworks.

High-grade explosives, also termed "detonating explosives," are more stable than low-grade explosives, frequently requiring trauma or shock for detonation. Nitroglycerin is the original high-grade explosive. Ammonium nitrate is another example of the early types of detonating explosives. Composition B, C-3, C-4, and TNT were developed later. Other examples include Amatol 80/20, RDX, PETN, and dynamite. Initiating high-grade explosives are a separate class of very sensitive high-grade explosives, such as lead styphnate and lead azide.

The Unabomber, known for constructing and using pipe bombs, is an example of a terrorist who targeted specific victims. The pipe bomb is easy to construct using readily available components and published step-by-step instructions. The Unabomber was very careful in his choice of components used to assemble bombs, complicating the search for his identity.

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Selected Agents of Terrorism: Chemical Agents

Chemical agents were first used extensively in World War I with dramatic results against unprepared troops. Although far less lethal than conventional explosives, chemical weapons can affect and incapacitate large numbers of troops in a short time. Chemical warfare agents were defined by the United Nations in 1969 as "chemical substances, whether gaseous, liquid or solid, which may be employed because of their direct toxic effects on man, animals, or plants." The ready availability of precursors of modern chemical weapons and copious documentation on their preparation make the use of chemical weapons for terrorist actions far more likely than use of nuclear or biological weapons. In addition, potential terrorists could easily locate a chemical production facility, sabotage it using chemical or conventional explosives, and allow ambient winds to spread the toxins. The resultant environmental contamination would fulfill many terrorists' objectives of generating fear, trepidation, and panic among the population.

Chemical agents are separated into 2 broad categories, lethal and nonlethal. Lethal agents include cyanides, nerve agents, vesicants, and choking agents. Nonlethal agents include lacrimating, emesis-inducing, and incapacitating agents.

Lethal chemical agents

Cyanide compounds

Although they have been used since World War I, cyanide compounds are highly volatile, rendering them less useful than chlorine. The military designates hydrogen cyanide (AC) and cyanogen chloride (CK) as the substances used in warfare. Cyanide and its compounds are among the most rapidly acting chemical agents. Signs and symptoms include air hunger, hyperpnea, apnea, seizures, coma, and death. Cyanide poisoning is treated with a combination of nitrites and thiosulfates available in commercial kits. Protective masks, gowns, and gloves are necessary until the patient is decontaminated completely.

Nerve agents

Nerve agents are chemicals that inhibit acetylcholinesterase irreversibly. They combine with acetylcholine (ACH) to prevent transmission at the neuromuscular junction and affect both the sympathetic and parasympathetic nervous systems. Salivation, lacrimation, urination, defecation, and emesis (SLUDGE) are common signs. Muscarinic effects may cause the most serious complications, including bronchoconstriction, laryngospasm, and respiratory depression or arrest. These nerve agents may be delivered by droplet, vapor, or both. Symptoms of skin exposure appear much more slowly than those from inhalation. Onset of symptoms varies from 1 minute to a few hours. If cyanide compounds are

inhaled or absorbed through the mucus membranes, death can occur in 1-10 minutes.

Therapy includes atropine sulfate, with as much as 10-40 mg required in some instances. Currently, the US Army uses 2-PAM (pralidoxime chloride), although it is not completely effective. It is least effective against Soman (GD). Pretreatment with pyridostigmine competitively inhibits the nerve agent. Pyridostigmine combines reversibly with ACH, which can resume neurotransmission after disassociation. However, such pretreatment does not protect against seizures.

Current nerve agents include Tabun (GA), Sarin (GB), Soman (GD), and VX. Local decontamination of these agents includes washing with soap and water. Health care providers should wear full protective gear until the patient is cleared by an environmental health specialist. VX, considered the most toxic of the nerve agents, is also the most difficult to decontaminate because of its low volatility.

Choking agents

Causing pronounced irritation to the upper and lower respiratory tracts, these agents are potentially dangerous because of a period of latency. A victim with dyspnea and mild chest discomfort may deteriorate after several hours to apnea and subsequent death. Chlorine is a widely used chemical that falls into the choking agent class. Chlorine causes upper- and lower-respiratory irritation, lacrimation, chest pain, dyspnea, coughing, laryngeal edema, pulmonary damage, and pulmonary edema.

Treatment is symptomatic with nebulized sodium bicarbonate. Decontaminate by copious flushing of affected areas with water. Medical providers need no special protection from chlorine-exposed patients.

Vesicant agents

Vesicant agents, also termed blistering agents, may be toxic to the lungs, eyes, and mucous membranes. They are named for their tendency to cause blisters. Mustard gas is the best known vesicant, originally used in World War I. It is a primary tissue irritant and has no significant allergenic component. Lesions are primarily cutaneous, but respiratory, ocular, and GI manifestations may occur, as well as cough, bloody sputum, and dyspnea. Areas of exposure become erythematous and progress to bullae, similar to toxic epidermal necrolysis. Symptoms may not occur for several hours after exposure. No antidote is available.

Other vesicants include sulfur mustard (HD), nitrogen mustard (HN), agent T, and phosgene oxime (CX). Lewisite, unlike mustard and mustard derivatives, causes immediate pain and skin irritation.

Medical providers require protective masks and clothing for patient management. Decontaminate by blotting and cleansing with soap and water. Avoid scrubbing and hot water.

Nonlethal chemical agents

Lacrimator agents

Lacrimator agents (tear gases) are widely used by law enforcement and the military. The most common effects are nasal and ocular discharges, photophobia, and burning sensations in the mucous membranes. Prolonged exposure may produce tightness in the chest, shortness of breath, and malaise and may cause vesiculations or bullae. Physical injuries may be observed from explosive discharge or kinetic effects of projectiles. At least 1 death has been attributed to lacrimator agents.

Most patients can be decontaminated fully by undressing, showering, and washing with soap and water. Medical personnel should use protective masks, gowns, and gloves, since lacrimator agents are transmitted by physical contact.

Types of lacrimator agents include bromobenzyl cyanide (CA), ortho-chlorobenzylidenemalonitrile (CS), dibenzoxazepine (CR), 2-chloroacetophenone (CN), chloroacetophenone in chloroform (CNC), and chloroacetophenone and chloropicrin in chloroform (CNS).

Emesis-inducing agents

Emesis agents, also termed nausea gases, are not used routinely in the US. They produce respiratory and skin irritation effects similar to those of lacrimator agents, as well as profound nausea. Examples of such compounds include adamsite (DM), diphenylcyanoarsine (DC), and diphenylchloroarsinine (DA).

No decontamination is required in the field, and diluted bleach solution has proven effective for definitive cleaning. Ordinary clothing protects against these agents; chemical insert masks and standard gloves are adequate.

Incapacitating agents

The possibility of a nonlethal incapacitating agent has long intrigued military commanders. Several agents have been tested, including lysergic acid diethylamide (LSD), mescaline, psilocybin, and psilocin. The only successful agent in production is benzilate (BZ).

BZ is a delayed onset (1-4 h) agent causing tachycardia, dizziness, vomiting, blurred vision, stupor, confusion, and random activity. Affected persons may be docile, belligerent, stuporous, or confused. They may appear intoxicated.

Decontaminate by washing with soap and water or with dilute bleach solution. Protective masks with charcoal filters provide adequate protection. Gloves are not necessary, since the agent is not absorbed through the skin.

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Selected Agents of Terrorism: Biological Agents

The Biological and Toxin Weapons Convention of 1972 banned the development, production, and stockpiling of biological weapons not required for peaceful intentions. The US, UK, Soviet Union, and 67 other nations signed this document. Despite the fact that no biological agents have been used officially in warfare to date, the prospect of their use raises many concerns. Terrorism's history suggests the potential for the use of biological agents. Many authorities fear the use of biological agents more than the use of chemical agents, since antidotes and specific countermeasures are available for some chemical weapons. Use of biological agents in terrorist acts potentially could cause tens of thousands of casualties and cost the US economy billions of dollars.

Various scenarios involving use of biological weapons are possible, from a sudden epidemic to a subacute, prolonged pandemic. Pathogens might be disseminated without anyone's realizing it until after the incubation period ends, by then exposing hundreds or thousands of civilians. Anticipating and controlling the dissemination of biological weapons may be difficult, causing complications for terrorists and intended victims. The effects of a biological agent also could evolve as a slowly developing, hard-to-categorize cluster of widely scattered cases, inadvertently allowing further dissemination of the pathogen until the connection is recognized.

Certain aspects of a disease outbreak may combine to prompt suspicion of terrorist activity, including temporal patterns of illness, selected populations of victims, clinical presentation of illness, certain strains or species of pathogens, geographic location, morbidity or mortality patterns, antimicrobial resistance patterns, residual infectivity, route of exposure, weather or climate conditions, incubation period, or concurrence with other terrorist activities.

The number of potential biological agents is nearly impossible to estimate. Agents range from simple viruses to bacteria and compounds derived from vertebrate animals. Biological agents are classified into 2 broad groups, infectious and noninfectious.

Infectious agents

This group includes viruses, bacteria, protozoa, and fungi. The list of potential pathogens is extremely long, although an abbreviated list of agents can be considered that is based on previous use as agents during wartime. The synopsis presented for each disease is meant only as an overview. Consult definitive texts for complete information.

Anthrax

Caused by *Bacillus anthracis*, anthrax causes a necrotizing hemorrhagic mediastinitis. Death usually occurs in 24-36 hours. Standard precautions, including masks, gowns, gloves, and isolation, are

sufficient. Vaccine is available.

Brucellosis

Highly infectious *Brucella* species are less commonly fatal than anthrax. Fever, malaise, osteomyelitis, and genitourinary (GU) infections may occur. Endocarditis typically is the cause of death. Standard precautions are sufficient. Include precautions against direct contact if draining lesions are present.

Encephalitis viruses

Venezuelan, Eastern, and Western equine encephalitis viruses are likely to be used in weapons. Fever, headache, confusion, obtundation, dysphasia, seizures, paresis, and death may be observed. The Eastern variety has the highest mortality rate at 50-75%. A vaccine for Venezuelan equine encephalitis (VEE) is available, and effective vaccines for the others are currently in development. Standard precautions are sufficient; however, mosquito control is suggested, since mosquitoes are a vector.

Clostridium botulinum

An epidemic of descending and progressive bulbar and skeletal paralysis in afebrile patients may suggest botulinum poisoning. Respiratory failure is the most frequent cause of death. Since an antitoxin is available, standard precautions are sufficient.

Yersinia pestis (plague)

Plague usually manifests as pneumonia, culminating in respiratory failure and shock. A vaccine is available, yet precautions are required against pulmonary and droplet exposure.

Coxiella burnetii (Q fever)

A zoonotic disease with domestic livestock as vectors, Q fever varies in its manifestations. Fever, chills, and headache are the most common symptoms, although malaise, diaphoresis, and myalgia often are observed. Mortality rate is low, even when the disease is untreated. A vaccine is under investigation. Standard precautions are sufficient.

Rift valley fever

A hemorrhagic virus infection, Rift valley fever manifests with symptoms such as malaise, fever, prostration, generalized vascular permeability, and abnormal circulatory regulation. Several different varieties of hemorrhagic viruses are documented, including Ebola. Most are highly infectious and cause morbidity. Some carry high mortality rates. Depending on the strain, a vaccine may be available. Precautions against direct contact are recommended. Additional precautions may be necessary if massive hemorrhage is present.

Smallpox virus

Smallpox may be a viable biological weapon, since the last natural case occurred in 1977 and the smallpox vaccine no longer is produced. Aerosol exposure causes viremia, malaise, fever, headache, delirium, and prolonged rash. Morbidity is caused primarily by secondary bacterial pneumonia. Precautions against aerosol infection are necessary.

Francisella tularensis (tularemia)

Tularemia, a zoonotic disease, occurs in ulceroglandular or typhoidal form. Typhoidal form manifests as fever, prostration, and respiratory symptoms. Mortality rate in typhoidal tularemia is approximately 35%. Standard precautions are sufficient. A vaccine is available as an investigational drug.

Noninfectious agents

Allergic agents come from a variety of sources, including mite and insect particles, feathers, epithelium, hair, urine, feces, and powdered enzymes. Problems caused by these agents may include respiratory symptoms, conjunctivitis, and/or dermatitis.

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Conclusion

Responding to a terrorist event can represent a tremendous drain on resources for all agencies involved. The potential for death and destruction is tremendous. Agencies responsible for responding to terrorist events can reduce potential injury, illness, and death only through complete and ongoing planning.

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Hazmat

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Synonyms, Key Words, and Related Terms

hazardous materials, HAZMAT

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Introduction

One of the most challenging aspects of providing emergency medical care is attending to patients who have been contaminated with hazardous materials (HAZMATs). HAZMATs are defined as substances that have the potential to harm a person or the environment upon contact. HAZMATs can be gases, liquids, or solids and include radioactive and chemical materials. Biological organisms, such as viruses and bacteria, are not included as HAZMATs in this article.

Most hospitals in the US lack plans or facilities for attending to patients exposed to HAZMAT, even though this problem is becoming more common. Federal statutes require hospitals to participate in the planning and care of persons exposed to HAZMAT and to protect exposed employees.

The potential for exposure to HAZMATs in the US is significant. More than 60,000 chemicals are produced annually in the US, of which approximately 2,000 are considered hazardous by the US Department of Transportation (DOT). More than 4 billion tons of chemicals are transported yearly by surface, air, or water routes. These shipments are initiated from more than 100,000 different locations, with more than 1 million people directly involved in the transportation process. More than 500,000 shipments of HAZMATs are made every day, totaling approximately 1.5 billion tons per year.

Incidence of HAZMAT exposures cannot be ascertained accurately because a national reporting system does not exist. However, available information suggests that HAZMAT exposures are not infrequent injuries. In 1994, the Chemical Manufacturers Association emergency response phone system (Chemical Transportation Emergency Center [CHEMTREC]) logged more than 200,000 calls, of which 8,000 involved HAZMAT emergencies.

CHEMTREC is a public service of the Chemical Manufacturers Association. However, CHEMTREC is neither intended nor equipped to function as a general information source.

In an attempt to better define the magnitude of this problem, the Agency for Toxic Substances Disease Registry developed the Hazardous Substances Emergency Events Surveillance System in 1990. State health departments contracted with the Agency for Toxic Substances Disease Registry to investigate and report all hazardous substance releases. Nine states (ie, Colorado, Iowa, New Hampshire, New York, North Carolina, Oregon, Rhode Island, Washington, Wisconsin) participated in the program in 1990.

- From 1990-1992, a total of 3125 HAZMAT releases were reported. Injuries occurred in 14% of these, involving 1446 persons. Approximately 16% of the incidents resulted in evacuations.
- In 1993, 11 states reported a total of 3945 HAZMAT releases involving 2269 exposures and 16 deaths.
- In 1995, 14 states reported 5351 incidents involving 1689 exposures and 14 deaths.

Approximately 80% of the incidents occurred at fixed facilities involved with the production, use, or storage of HAZMATs. The other 20% occurred during transportation. Because most incidents occurred at industrial sites, most persons exposed to HAZMAT were employees (57%). Approximately 64% of individuals exposed to HAZMAT were transported to the hospital; in 1993, 15% were admitted, and in 1995, 9% were admitted. Most of the first responders (40%) did not wear any personal protective equipment (PPE), and only 18% of the injuries were treated at the scene. Most of the injuries involved the respiratory system (42%); however, 13% also involved trauma. The most common substances involved were volatile organic compounds, pesticides, ammonia, chlorine, petroleum products, and acids.

Several important points can be drawn from the above statistics. Most importantly, this is not a rare problem, and hospitals should have a plan that addresses HAZMAT situations. Several thousand patients were transported to hospitals in only a few states, and many had serious injuries.

Because most incidents occur at fixed sites, knowing the industries that operate in the catchment area of a

hospital and the chemicals used or stored at those sites is imperative. Trauma centers need to have a plan to attend to trauma patients who are contaminated, because 20% of the incidents occurred during transportation to the centers; 13% involved trauma in addition to chemical injury.

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Hospital and Community Planning for Hazmat Incidents

Most hospitals are unaware of the requirement by several different federal agencies, as well as the Joint Commission for Accreditation of Healthcare Organizations (JCAHO), to participate in community planning for HAZMAT incidents. The most important federal statute that hospitals must be familiar with is SARA Title III, a portion of the Superfund Amendments and Reauthorization Act, otherwise known as the Emergency Planning and Community Right to Know Law.

SARA Title III states that facilities manufacturing or storing hazardous chemicals must report inventory and every HAZMAT release to public officials and emergency health agencies. This act also requires the establishment of state emergency response commissions (SERC) and local emergency planning committees (LEPC). The LEPC includes local officials, police, fire, and public health authorities in addition to representatives of local hospitals, media, and the community.

Emergency response plans

The primary responsibility of the LEPC is to develop emergency response plans (ERPs) to do the following:

- Identify local facilities using hazardous substances
- Designate community and industrial coordinators
- Establish mechanisms of emergency notification
- Establish procedures for determining the occurrence of a release and estimation of the affected population
- Identify community emergency equipment facilities
- Establish evacuation plans

- Establish and schedule training programs for emergency personnel

Hospitals are required to be an integral part of the ERP. Additionally, emergency medical services (EMS) units and coordinators have critical roles in the planning and execution of an emergency response. The plan for each community varies depending on the types of industries involved, chemicals used, and resources available. For example, many fire departments in metropolitan areas have developed specialized HAZMAT teams to respond to these situations. These teams are responsible for containing releases and for decontaminating persons exposed to HAZMAT. In these cases, decontaminated patients can be transported safely and treated in the hospital with minimal precautions.

Conversely, in communities where a HAZMAT team is not available, the ERP must consider how persons exposed to HAZMAT will be decontaminated and transported. Hospitals must be capable of caring for severely contaminated patients under the ERP guidelines. Because most hospitals are poorly prepared to attend to a severely contaminated patient, early involvement of hospital representatives in the planning process is critical. Similarly, EMS coordinators must train emergency medical personnel to attend to contaminated patients and to establish contingency plans for their transport and care.

Joint Commission for Accreditation of Healthcare Organizations guidelines

Several JCAHO HAZMAT requirements affect hospitals. Previously, JCAHO required each hospital to have a plan to address radiation emergencies. This requirement was dropped, but additional requirements concerning more likely HAZMAT incidents have been added.

Some of the more specific JCAHO guidelines are as follows:

- EC 1.2.4: Hospital emergency preparedness plans should be integrated into the community-wide plan and should identify the available facilities for radioactive and chemical isolation and decontamination.
- EC 1.4.4: Hospitals must have emergency procedures and precautions for HAZMAT as well as protective equipment for spills and exposures.
- EC 1.3.3: Hospitals must have an orientation and education that address emergency procedures during a hazardous waste spill, incident, or personnel exposure.
- EC 1.5.4: Hospitals must develop performance standards for staff to respond effectively to disasters and emergencies that occur.
- EC 1.2.3: Hospitals must have a management plan to educate and monitor personnel who regularly come into contact with HAZMAT.
- EC 1.2.1: Hospitals must provide a physical environment free of hazards.

Occupational Safety and Health Administration regulations

The Occupational Safety and Health Administration (OSHA) has issued several regulations that pertain to any hospital employee who may come into contact with a HAZMAT. The [OSHA Hazard Communication](#) standard states that every worker has the right to a hazard-free work environment. Other standards (ie, [29 CFR 1910.132](#), 1988) regulate the safety and health of any employee who responds to hazardous substances emergencies. If employees are expected to be in contact with hazardous substances, they must be educated adequately and supplied with appropriate protective equipment.

EMS coordinators should be aware that OSHA also has developed minimal training standards for emergency personnel who respond to a HAZMAT incident. Under the [Hazardous Waste Operations and Emergency Response](#) (HAZWOPER) standard, an emergency response team is defined as an individual or group who responds to a release of a HAZMAT, no matter where it occurs. This regulation initially was intended for hazardous waste operators and emergency response personnel at hazardous waste facilities; however, in the case of a patient who has been contaminated, hospital and EMS personnel also may be included.

The application of HAZWOPER standards to hospitals has been questioned. It states that all ED personnel must be trained at a minimum of first responder awareness level (level 1), and any personnel involved in patient decontamination must be trained to first responder operation level (level 2). Planning the roles of HAZMAT and EMS workers requires familiarity with the definitions and training requirements (described below) of individuals who may respond to a HAZMAT incident as defined by the HAZWOPER standards. If emergency medical transport personnel are expected to transport contaminated individuals or to provide medical care in the field prior to decontamination, they at least should have the appropriate level of training. The Code of Federal Regulations outlines levels of training (ie, [29,1910.120](#), 1989) as follows:

- **Level 1: First responder awareness:** The level 1 first responder is the person who witnesses or discovers the release of a HAZMAT and notifies the proper authorities. Training includes recognition and identification of HAZMATs, notification procedures, and the employee's role in the emergency release plan.
- **Level 2: First responder operations:** The level 2 first responder is the person who responds to the release of hazardous substances without trying to stop the release. This level requires level 1 competency and 8 hours of additional training in basic hazard and risk assessment, PPE selection, containment and control procedures, decontamination, and the ERP.
- **Level 3: HAZMAT technician:** The level 3 technician responds aggressively to stop a release. This level requires 24 hours of level 2 training and competencies in detailed risk assessment, toxicology, PPE selection, advanced control, containment and decontamination procedures, air-monitoring equipment, and the incident command system (ICS).

- **Level 4: HAZMAT specialist:** The level 4 specialist has advanced knowledge of HAZMAT and responds with and provides support to HAZMAT technicians. This level requires 24 hours of level 3 training and proven competencies, along with advanced instruction on all specific HAZMAT topics.
- **Level 5: On-site incident commander:** The level 5 commander assumes control of the incident. Level 5 requires 24 hours of training equivalent to level 2 with competencies in the ICS and ERP, hazard and risk assessment, and decontamination procedures.

Hospital and community planning

Hospitals must have adequate plans for addressing HAZMAT incidents. Incorporate these plans into community ERPs. The responsibility of hospitals cannot stop at the planning stage. Adequate PPE and training to use the equipment must be supplied. Incidents of hospital workers becoming ill as a result of chemical exposure when caring for a contaminated patient have been reported. If this occurs, the legal position of the hospital is tenuous.

Providing universal guidelines for all communities is difficult. In formulating a hospital and community response plan, the most critical aspects to consider are location of and responsibility for decontamination. Ideally, decontamination takes place in the field and is performed by specially trained HAZMAT teams. In this case, subsequent prehospital and hospital care can be performed with little change in the usual routine and with minimal risk to health care providers. In situations where several hospitals are located in a given area, it is not financially feasible for all hospitals to have good decontamination facilities. Choose one hospital as the receiving facility. Base the choice of hospitals on the availability of decontamination facilities, intensive care facilities, training of ED personnel, and staff trained in medical toxicology.

Regardless of whether a hospital is a receiving facility or if it is in an area where a trained HAZMAT team is located, situations always occur when contaminated patients present to other prehospital or hospital systems. Consequently, all hospitals should have a plan and appropriate employee training for attending to the contaminated patient.

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Hazmat Incident Management

Chain of command

The HAZMAT disaster plan for a community clearly should define who is in charge of a situation. This person ultimately is responsible for protecting public health and the environment and ideally should be a specially trained individual representing either the HAZMAT team or the LEPC. The ERP clearly should delineate the authority of this person, even on private property or private facilities.

The ERP must address many aspects other than medical care. The plan should stipulate the reasons for evacuation as well as local evacuation centers. The incident commander must consider public and rescuer hazards from toxic and corrosive materials as well as those from explosive or flammable materials. Finally, the plan must stipulate at what point EMS personnel not trained to attend to HAZMAT issues will interact in patient care.

The community disaster response plan delineates the line of command for a situation and specifies how the EMS system will interact with the HAZMAT team. Defining the point at which the EMS system gets involved with injured persons is not an easy task. Ideally, specially trained HAZMAT workers decontaminate all exposed individuals prior to turning them over to the EMS system. However, in any individual situation, severity of the patient's injuries and degree and type of contamination must be weighed against the medical training of the HAZMAT worker and the EMS system.

The ideal situation is that of Los Angeles County, where the HAZMAT team is made up of specially trained members of the fire department who also are cross-trained as paramedics or Emergency Medical Technicians (EMTs). However, smaller communities commonly do not have this luxury. Especially in situations in which the HAZMAT team is not trained in prehospital care, involving medical control physicians and poison control centers in patient care decisions is essential.

Goals in managing a HAZMAT incident

The goals of managing a HAZMAT incident include the following:

- Recognition of the situation that may involve HAZMATs and notification
- Establishment of a command center
- Protection of site and emergency workers from any further exposure
- Identification of the HAZMATs involved
- Rescuing any HAZMAT-exposed persons on-site
- Decontamination and initial medical care of persons exposed to HAZMAT
- Containment of the HAZMAT
- Evaluation of further public exposure and evacuation where necessary

Obviously, many of these functions are performed concurrently. Recognition of the danger may seem a

simple matter, but this usually depends on local workers or first responders. Most industrial site workers should be familiar with the site's ERP, including contact information in case of emergency and what to report. Potentially disastrous situations occur with motor vehicle or agricultural accidents in which the first responders are not aware of HAZMAT dangers. In 1 series of HAZMAT incidents, 14% of exposed individuals were first responders. This emphasizes the importance of emergency medical personnel being trained, at a minimum, to the level of first responder awareness.

Site command center

One of the earliest priorities is the establishment of a site command center. Ideally, locate this command center near the incident but far enough away to avoid further exposure. The command center should be located upwind and uphill to avoid further contact and should have a wind monitor and alarm system to warn of any shifting wind currents that may carry HAZMATs toward the command center. The command center should have a rapidly deployable communications system, which is critical to maintain contact with on-site workers and off-site emergency management and medical personnel to access information on the hazardous substances involved and necessary containment and safety procedures.

The site should be divided and managed within 3 zones as follows:

- The contaminated area is known as the hot zone. Only allow individuals with appropriate PPE and specialized training into this zone.
- The intermediate zone, also known the decontamination zone, is where patient decontamination should take place. A degree of contamination still is found in this zone; thus, some PPE still is required, although it is usually of a lesser degree than that required for the hot zone.
- The command zone is located outside the decontamination zone. Decontaminate all HAZMAT-exposed individuals and equipment from the hot zone and decontamination zone before entering the command zone. Access to all zones must be controlled. Keeping the media and onlookers well away from the site is critical.

Untrained medical personnel should pick up patients in the command zone. Allow only trained individuals wearing necessary PPE into the decontamination zone. This produces a dilemma when persons exposed to HAZMAT require immediate medical attention. If this is the case, the ideal situation is for some EMS personnel to have the appropriate level of training to work in PPE. If this is not possible, the medical control physician and the site commander make decisions on an individual basis.

Transportation

Discourage placing a contaminated patient in an ambulance. This is a closed environment and presents increased risk to those in the ambulance. This action also results in the contamination of the ambulance and its equipment. No further use of the ambulance is allowed until it can be decontaminated

appropriately. Ambulances usually are resources that most communities cannot spare. Transportation prior to decontamination increases the amount of time the HAZMAT is in contact with the patient.

Some authors have recommended that patients be transported in the back of open trucks. These patients are not medically monitored or treated while being transported in these cases. Consider transportation in the back of an open truck only in those situations where no decontamination options exist at the scene and the hospital is prepared for decontamination.

HAZMAT identification

Identification of the HAZMATs involved is critical to all aspects of the rescue operation. As part of the SARA legislation, industrial sites are required to report all HAZMATs at their facility to the local emergency-planning agency. In most instances, this information is maintained by the fire department. Industries also are required to post this information in a location external to the site, usually in an external electrical box or fire safety location. This assumes that the information contained in the external location or by the fire department is current. Problems may arise when new chemicals are added to an inventory and the lists are not updated.

The DOT requires all vehicles carrying HAZMATs to display placards identifying them. Generally, these are diamond-shaped signs that have specific colors and numbers that define the class of HAZMAT that is present.

The DOT classes and defining colors of HAZMATs include the following:

- Explosives (solid orange color)
- Nonflammable gases (solid green color)
- Flammable liquids (solid red color)
- Flammable solids (white and red stripes)
- Oxidizers and peroxides (solid yellow color)
- Poisons and biohazards (solid white color)
- Radioactive materials (half white, half yellow with black radiation symbol)
- Corrosives (half white, half black)
- Other (usually white)

Each placard usually contains a descriptive color, symbol, and number. The triple redundancy is so that, in case of an explosion, any remaining portion of it can be used to identify the type of material present. The DOT identification system only identifies the type of hazard present and does not identify materials.

Many placards also contain a 4-digit number, known as the United Nations (UN) identification number. These numbers identify individual chemicals or groups of chemicals. Because several hundred thousand chemicals are known, obviously, only a relatively few can be identified by a 4-digit classification system. For this reason, many chemicals with similar characteristics are given the same UN number.

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Personal Protective Equipment

Specialized equipment, known as PPE, is required to adequately protect rescue personnel and health care providers from secondary contamination. The type of PPE used depends entirely on the situation and the level of training of the user. HAZMAT workers required to enter the hot zone require a greater level of protection than medical personnel providing care to contaminated patients.

Levels of protection for work involving HAZMATs

The US Environmental Protection Agency (EPA) has defined 4 levels of protection for work involving hazardous chemicals. Although these levels originally were intended for work at hazardous waste disposal sites, they have been adopted widely in other situations, such as rescue work.

Level A is the maximum level of protection and usually is required only of HAZMAT personnel and others working in areas of very high concentrations of toxic agents. It consists of a fully encapsulating chemical-resistant suit, positive pressure self-contained breathing apparatus (SCBA), double layers of chemical-resistant gloves, and chemical-resistant boots. Airtight seals should be in place between the suit and the inner layer of hand, face, and foot protection.

Level B is used when full respiratory protection still is required but dangers to the skin are less. It consists of a SCBA and a chemical-resistant suit with resistant gloves and boots. No airtight seals on the face, hands, and feet are necessary.

Level C is required when air concentrations are expected to be much lower and less likelihood of skin exposure exists. It consists of a full-face air purification device and a nonencapsulating chemical-resistant suit with gloves and boots.

Use Level D only when no danger of chemical exposure exists. It consists of standard work clothes and no respiratory protection.

Most HAZMAT rescue workers require Level A protection, which is very expensive, bulky, and requires specialized training. The typical Level A HAZMAT suit costs several thousand dollars and must be cleaned between uses. Manual dexterity is poor and the suits are very hot, limiting the amount of time that they can be worn.

HAZMAT teams usually use Level B or C PPE for decontamination. This takes place away from the hot zone and when the amount of chemical present on a patient is significantly less than in the hot zone. Also, sufficient quantities of chemicals to present physical hazards, such as explosions, should not be present on a patient. EMS workers and other health care providers require less protection than HAZMAT workers, but they still must be protected adequately when attending to contaminated patients. The level of protection required is usually C or B. This includes a chemically resistant suit, gloves, and boots; respiratory protection; face protection; and disposable boots. Current recommendations for PPE to be used in situations of radiation contamination suggest only Level D-type protection plus a dust filter for respiratory protection.

For medical applications, inexpensive (\$50-100) and disposable chemical-resistant multilayer polymer suits are available. Suits much more expensive than this commonly are used for surgery involving patients with HIV or other infectious diseases.

One common misconception is that Tyvek suits, which are very inexpensive and readily available around hospitals and laboratories, are suitable for decontamination work. This material provides no chemical protection, and most chemicals can penetrate this material immediately, although it suffices for work with dusts, including radioactive dusts and biological agents. These suits are recommended for training exercises but should not be relied upon for chemical protection.

Glove material also is an important consideration, because the hands have the most contact with the patient. Unfortunately, no single glove material provides adequate protection against all chemicals. To counter this situation, most HAZMAT workers use several gloves of different materials. An ideal combination is nitrile and Viton. However, this is quite bulky and markedly limits manual dexterity. Because patients should be washed immediately with large quantities of water during decontamination, actual contact with pure chemicals generally is minimized. Typical latex gloves used in most hospitals offer little chemical protection. Nitrile has much better chemical resistance than latex and is now available in a thin, flexible, disposable glove that permits good manual dexterity. This is presently the ideal glove material for use when providing medical care to a patient who has suffered chemical contamination.

Aldehydes, halogenated hydrocarbons, ketones, aromatic hydrocarbons, nitroorganic compounds, and carbon disulfide rapidly can permeate nitrile. Unfortunately, most common solvents consist of chemicals within these classes. If these are encountered, use a thicker overglove, preferably made of Viton, until the patient at least is decontaminated partially. Once no chance exists of coming into contact with large quantities of pure chemical, such as during removal of the patient's clothes, disposable nitrile gloves

should be sufficient.

Boots should be worn, since the feet are in constant contact with contaminated water during patient decontamination. Because chemicals are diluted, inexpensive disposable boots should suffice. Boots also provide slip resistance on wet floors. Avoid leather and cloth footings since these materials may wick up contaminants and are impossible to clean.

Respiratory exposure to vapors is an additional risk to the health care worker. The small quantity of materials present on a patient makes generation of toxic concentrations of vapors unlikely. Respiratory protection especially is important when working in enclosed spaces, such as transport vehicles or medical care rooms. While inhalation of toxic fumes and vapors can be prevented, it does require some degree of advance planning and training to provide adequate protection.

Available types of respiratory protection - Cartridge respirators and supplied air respirators

Cartridge respirators function by allowing the wearer to inhale air through a canister filled with a special sorbent material that binds chemical vapors. Cartridge respirators are inexpensive, portable, and easy to use and store. However, drawbacks exist to their general use. The cartridges are interchangeable, but the type of cartridge used must match the chemical vapor in question. Different cartridges must be used to protect from organic vapors, acid gases, chlorine, ammonia, and methylamine. The sorbent materials also have a breakthrough phenomenon, in which chemicals elude off the sorbent after a period of use and then expose the user. In general, this limits cartridge respirators to short-term use and to low concentrations of chemicals in the air. This is the situation that exists when patients require decontamination.

Cartridge respirators depend on an airtight seal against the face. They require a good fit and cannot be used with facial hair. A moderate amount of work is involved when inhaling across the pressure resistance of the cartridge. All of this requires that any individual using this type of respirator be fitted properly and trained in its use. Cartridge respirators are very versatile for short-term use. They require adequate training of all personnel who may be expected to use them and require someone available at all times to decide which type of cartridge to use. Cartridge respirators are ideal for performing decontamination outside the ED.

Supplied air respirators provide a source of clean breathing air through a hose and an external supply. The external supply can be provided from a pump or compressed air. Two types of masks are available: one with a pressure-actuated valve and one that continuously blows fresh air across the face. The second type uses much more air than the first. The fit is less critical since any leaks always have air flowing from inside to out. Although continuous fresh air type masks are not recommended for use with facial hair, they are the best choice if faced with this situation. The supplied air respirators can be used in all situations and for any length of time without worry about choice of the proper cartridge and breakthrough.

Use of supplied air respirators also requires training, although proper fit is less critical, at least with the

continuous flow type. Due to the necessary air supply and hoses, generally supplied air respirators are impractical for use with outside decontamination. Some HAZMAT teams use this method for personnel providing decontamination close to a supply vehicle that can pump the necessary air. If a decontamination room is to be established inside a hospital, supplied air respirators are the ideal choice.

Most respiratory protection can be obtained using a half-face design, which covers the nose and mouth, or a full-face design that also covers the eyes. If the half-face design is used, goggles also must be worn to protect the eyes from splashes. However, the eyes are still exposed to vapors that can be irritating or toxic. If respiratory protection is to be used, choosing the full-face version to protect the eyes and entire face makes much more sense.

Protection of health care workers from HAZMAT exposures can be achieved with some degree of advance planning and training. Chemically protective suits that are inexpensive and disposable are available. Respiratory protection also can be obtained without significant expense; however, the least expensive type, cartridge respirators, requires some additional training.

The recommended PPE for decontamination of persons who have radiation exposure, usually consisting of a filter-type dust mask and Tyvek or surgical scrub suit, was intended to protect the health care provider from radioactive dust particles. Unfortunately, this PPE is completely inadequate to protect from chemicals in the liquid or vapor states. Alternatively, PPE designed for chemical protection is more than adequate to provide protection in the case of a patient exposed to radiation. To avoid confusion and simplify the protocol, only one type of PPE is recommended. This may be more elaborate and expensive than that needed for a radiation protocol; however, it can be used in all situations involving persons exposed to HAZMATs.

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Decontamination

Decontamination is the process of removing or neutralizing HAZMATs on people or equipment. Removal of chemicals on skin is important for the following 2 reasons:

- To prevent any further absorption and subsequent toxicity since many substances disrupt the integrity of the skin and then become systemic toxins following absorption
- To prevent other persons or equipment from becoming contaminated with substances on the patient's clothes or skin (secondary contamination)

The type of decontamination procedure used depends on the situation. Removing all clothes removes 70-85% of the contaminants. Most decontamination can be accomplished by simple high-volume dilution

with water. Occasionally, mild soaps are required to remove oily or greasy substances.

Avoid water in the presence of metallic sodium, potassium, lithium, cesium, and rubidium, because these react on contact with water. Dusts of pure magnesium, white phosphorus, sulfur, strontium, titanium, uranium, yttrium, zinc, and zirconium ignite on contact with air. If burning, many of these explode if exposed to water. If these substances are suspected, remove residual metal with forceps and store it in a container of mineral oil. If radioactive particles are on or embedded in the skin, remove them by forceps; the radiation safety officer should dispose of them. Some HAZMAT teams use 4 special solutions recommended by the National Fire Protection Association for patient decontamination. No evidence exists that these solutions are more effective at decontaminating human skin than water alone. In addition, none of the solutions can be used on open skin, mucous membranes, or eyes.

Collect the water runoff from the decontamination and do not allow it to enter parking lots or storm drains. However, if a drain is readily available and arranging a collection system will require considerable time, decontaminating the patient and allowing the water to enter the drain may be prudent. Although this action theoretically can result in a fine from the EPA for an unscheduled discharge, this situation has not happened to date.

After collection, consider water to be contaminated with a hazardous substance. If it is allowed to spread into an open area, it likely will be tracked off-site into private vehicles and homes. Collection of the decontamination runoff is accomplished using a series of collection pools, which can be specially designed devices or can be as simple as inflatable children's pools. For ambulatory patients, a series of 3 collection pools usually is used, with contaminated patients or workers always starting in the most contaminated pool and finishing in the least contaminated pool. For nonambulatory patients, specialized runoff collection litters are available.

Remove clothes and place them in a plastic bag marked as contaminated. Give priority to decontaminate the eyes, mucous membranes, and severely affected areas of skin. Take care not to wash contaminants onto unaffected areas of skin. Thoroughly irrigate areas of skin where disease processes or the HAZMAT has broken down the surface structure. Avoid abrasive cleansing.

For radioactive materials, a Geiger counter can be used to detect any residual contamination. Unfortunately, no simple instrument is available for the wide range of chemical contaminants. Clinical judgment must suffice to determine when a sufficient amount of irrigation has been performed. Copious irrigation is the standard rule; however, this should not be to the point of irritating or denuding the skin.

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Hospital Protocol for Hazmat Incidents

Hospital care for a person exposed to HAZMAT should begin with adequate planning well before the incident occurs. Failure to have an adequate plan to attend to a HAZMAT situation can result in injury to hospital employees and subsequent liability to the hospital for not meeting OSHA standards. More importantly, most hospital EDs serve integral functions to the hospital and to the community. Closure of an ED due to contamination can have dire effects on the hospital and the community.

Decontamination plan

Most hospitals are poorly prepared to care for a contaminated patient and have little protection for hospital employees who are involved. This is even more reason to be involved in the community planning process. If hospitals are unprepared to decontaminate persons exposed to HAZMAT, every effort should be made to decontaminate patients in the field. Even if this is the case, hospitals should have some plan to decontaminate those exposed to HAZMAT, because, in many cases, they arrive by private vehicle or by an inadvertent ambulance transport. In the worst-case scenario, hold the contaminated patient outside the ED until the community HAZMAT team can assist with the decontamination. Whatever the situation, it is essential that the patient be decontaminated prior to entering the ED.

The first decision to be made in formulating a HAZMAT plan for the ED is where decontamination is to take place. An inside decontamination area is ideal, although it often may not be practical or possible. Locate an inside decontamination room so that it can be accessed without entering the hospital or the ED. The room can be used for other purposes, although anything inside the room should be easy to remove. An advantage of an inside area is proximity to ED personnel, supplies, and electrical and water sources. Monitoring patients and providing critical medical care obviously is easier in a room adjacent to or in the ED. Controlling access to a single room also is much easier. However, after the room is used, provisions need to be made to clean the room and remove wastes. Performing decontamination outside the ED is probably more practical in most situations, although it detracts from the care of the patient who is critically ill.

If the decontamination area is located inside, it should be adjacent to an entrance, preferably the ambulance entrance. Gaseous, liquid, and solid wastes must be collected. The room should be under negative pressure and have a separate ventilation system that discharges to the outside. Contaminated irrigation water should not be discharged into the general hospital drain system but should be collected in a specialized holding tank under the decontamination room. The expense of installing a tank for this purpose is difficult to justify unless a new room is being constructed specifically for decontamination. Alternatively, liquid wastes can be collected in inflatable plastic children's swimming pools or expandable plastic containers designed for this purpose. For patients who are not ambulatory, specialized litters are available to collect water runoff in special collection barrels. Plans need to be made for eventual removal and disposal of collected wastewater.

Planning for decontamination outside is simpler and less expensive than equipping an indoor decontamination room. Ventilation is not a significant problem outside, although the decontamination

team should wear respiratory protection if indicated by the type of HAZMAT. The prospective site must have a water hose for decontamination. The wastewater still must be collected and not allowed to run over lawns or pavement into sewer drains. Portable pools can be used for collection, but provisions must be made to prevent access to these prior to removal. Similarly, solids must be collected and properly stored prior to disposal. Two of the drawbacks to outside decontamination are patient privacy and weather. These problems can be handled by using tarps and portable heaters. Portable showers that can be assembled easily are now available commercially.

Providing medical care to patients who are critically ill is more difficult if decontamination is performed outside. If medically indicated, portable monitors and portable oxygen may be needed. This equipment must be cleaned thoroughly or discarded after use. A special portable communication system may be needed if the decontamination team is using respiratory protection. Additional personnel are needed to obtain necessary medications and supplies from inside the hospital. Finally, if the decontamination is to be performed outside, the area must be secured properly. Encircle the area by rope or tape; security personnel are required to prevent unauthorized entry. The area may need to be secured for several days until all wastes are removed.

The goal of decontamination is to remove enough of the contaminating material so any danger of secondary contamination to those providing medical care or to the patient no longer exists. Ideally, decontamination should require only 15-30 minutes, although patient stabilization may prolong this period. After decontamination, the patient can be moved into the general treatment area and treated as any other patient. The authors recommend limiting treatment to only basic life support measures and life-saving procedures within the decontamination area.

Procedures within the decontamination area

The choice of which procedures will be performed in the decontamination area must be made on an individual basis. Place all patients on a cardiac monitor and secure intravenous access. If indicated, also place patients on oxygen. Provide medical therapy to treat cardiac dysrhythmias or to initiate toxin-specific treatment. While conditions such as tension pneumothorax or respiratory distress should be treated immediately, most conditions can be treated with basic stabilization until the patient has been decontaminated. Consider any item used in the decontamination area contaminated until it has been cleaned thoroughly. Postpone the acquisition of radiographs, ECGs, and routine blood work until the patient is moved out of the decontamination area. In the case of a contaminated trauma patient, sending blood for hemoglobin determination and crossmatch may be necessary.

Some published radiation protocols recommend long lists of supplies to be maintained in the decontamination area. This should not be a problem if the decontamination area is in or adjacent to the ED, where all necessary supplies should be readily available. It is much more efficient to use a runner for needed items.

Include guidelines in the hospital protocol that outline who is to perform decontamination. Although

having a single team to train is easier, training at least 1-2 members of each shift is more practical. HAZMAT situations occur spontaneously and with no advance notice. The delay of calling a special decontamination team from home is not practical.

In the protocol and training, specify how team members are to remove PPE, disposition of contaminated material until it can be removed, and who is responsible to remove contaminated material.

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Conclusions

- Hospitals are required to participate in community disaster planning for HAZMAT incidents according to SARA Title III.
- Hospitals are required by JCAHO and OSHA to protect their employees from HAZMAT exposures, including exposure that may occur as a result of patient care.
- Accomplish decontamination of patients exposed to HAZMAT in the field.
- Unprepared EMS units should not transport contaminated patients.
- Adequate PPE for health care providers includes a chemical-resistant suit (Tyvek is not sufficient), nitrile gloves, disposable boots, and full-face cartridge or supplied air respirator. This equipment is less expensive than some operating suits.
- Health care providers need training prior to using PPE.
- Hospitals should have plans to attend to patients contaminated by HAZMAT.
- Decontamination can be accomplished safely outside the ED or in specially prepared rooms indoors. Contaminated patients should not enter the main areas of the ED or hospital.
- If a hospital is unprepared to handle a contaminated patient, hold the patient outside the ED until a HAZMAT team safely can decontaminate the patient.

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Additional Information

This article is an overview of issues attending to individuals who have been exposed to HAZMAT. Because entire books have been written on this subject, all aspects can not be covered here.

Listed below are additional sources of information on HAZMAT exposures.

Local resources

- Regional poison control centers provide information 24 hours per day.
- LEPC provides information about chemicals in the community.
- Fire department and/or HAZMAT team assists with response and planning and chemicals in the community.

National resources

- CHEMTREC (800-424-9300) provides 24-hour information regarding manufacturers' product information (see [CHEMTREC](#)).
- Agency for Toxic Substances and Disease Registry (404-639-0615) provides 24-hour emergency assistance for hazardous chemical health-related issues (see [Agency for Toxic Substances and Disease Registry](#)).
- EPA's regional offices offer technical assistance for environmental issues (see [EPA](#)).
- National Response Center (800-424-8802) provides 24-hour assistance for identifying chemicals and planning a response (see [National Response Center](#)).
- The National Pesticide Telecommunications Network (800-858-7378) provides 24-hour assistance to physicians and emergency responders regarding pesticide accidents (see [National Pesticide Telecommunications Network](#)).
- Radiation Emergency Assistance Center/Training Site (REAC/TS) (615-481-1000) provides emergency consultation for accidents involving radioactive materials (see [REAC/TS](#)).
- The Nuclear Regulatory Commission (301-951-0550) provides assistance to emergency responders attending to radiation accidents (see [Nuclear Regulatory Commission](#)).

Computer-based resources

- Chemical Hazard Response Information System (CHRIS; 800-247-8737) contains general and health hazard information (see [CHRIS](#)).
- The MICROMEDEX (800-525-9083) CD-ROM (expensive) includes the following databases:
 - POISINDEX contains comprehensive acute and chronic toxicity information and medical management guidelines (see [POISINDEX](#)).
 - The TOMES Plus CD-ROM (expensive) compiles hazard and toxicity information from the National Library of Medicine Databases, CHRIS, and several other sources (see [TOMES System](#)).

Internet resources

Toxicology Data Network (TOXNET; see [TOXNET](#)) is a new online addition to the National Library of Medicine. TOXNET is a comprehensive data bank for health effects of industrial and environmental exposures. TOXNET contains the following databases:

- Hazardous Substances Data Bank (HSDB) provides toxicology, emergency handling, and regulatory requirements on more than 4500 hazardous substances.
 - Toxline maintains a large bibliographic database covering the toxicologic, pharmacologic, physiologic, or biochemical effects of drugs or chemical substances.
 - Chemical Carcinogenesis Research Information System (CCRIS) contains carcinogenicity and mutagenicity data on more than 7000 chemicals and is maintained by the National Cancer Institute.
 - Integrated Risk Information System (IRIS), maintained by EPA, provides health risks and regulations on more than 700 chemicals. IRIS also contains health advisories.
 - GENE-TOX provides genetic toxicology on more than 3000 chemicals.
 - Environmental Mutagen Information Center-Front and Back Files (EMIC/EMICBACK) maintains bibliographic files on chemical, biological, and physical agents and is maintained by Oak Ridge National Laboratory.
 - Developmental and Reproductive Toxicology Database/Environmental Teratology Information Center Backfile (DART/ETICBACK) maintains bibliographic databases on teratology and developmental toxicology. DART/ETICBACK is maintained by EPA and NIEHS.
 - Toxic Chemical Release Inventory (TRI), mandated by SARA III, covers releases of hazardous chemicals in local communities.
 - Toxic Chemical Release Inventory Facts (TRIFACTS) supplements TRI with summary information on health effects.
-
- Hazardous Materials Fact Sheet, maintained by EPA, provides detailed information on hundreds of toxic chemicals.
 - OSHA provides information on worker health standards and regulations.

- Federal Emergency Management Agency (FEMA) provides information on disaster preparedness plans and frequently asked questions (see [FEMA](#)).
- The EPA Web site (see [EPA](#)) is another source of information.
- EPA's Computer-Aided Management of Emergency Operations (CAMEO) is a resource (see [CAMEO](#)).
- Comprehensive Environmental Response Compensation and Liability Information System (CERCLIS) provides information on Superfund sites, chemicals involved, and cleanup status (see [Superfund Overview](#)).
- DOT's HAZMAT (see [DOT's Office of Hazardous Materials Safety](#)) provides further information.
- Hazardous Chemical Database is another resource.

Printed resources

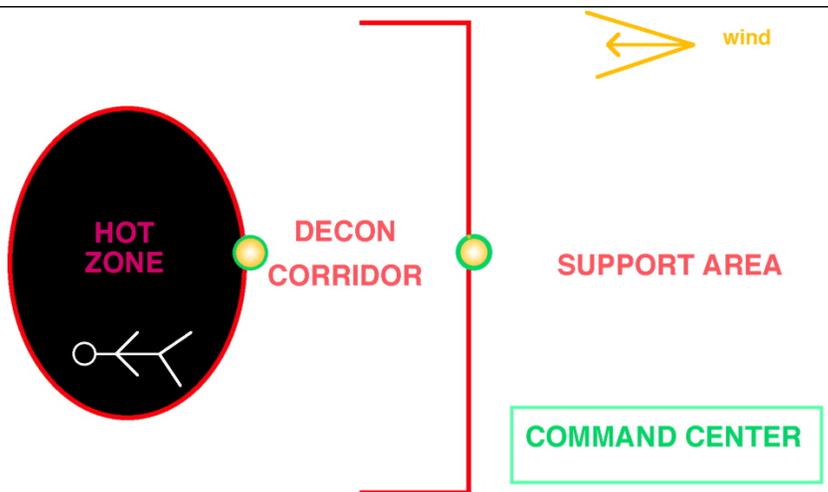
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Pictures



Picture 1: Zone control of the hazardous materials site

Picture type: Photo

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Medical Control

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Overview

Medical direction is the process by which a physician or, occasionally, group of physicians guide and oversee the patient care provided by an emergency medical services (EMS) system.

Law requires medical direction for all advanced life support (ALS) service providers. Most states require basic life support (BLS) agencies to have a medical director as well. Medical direction from a physician is recommended for all EMS activities.

EMS Medical Director/Administrators

The role of an EMS medical director is far greater than merely lending the physician's name and license number to satisfy a legal requirement. The medical director is responsible for all aspects of care provided in the EMS system. In addition to writing protocols for prehospital care, the EMS medical director offers continuing education for EMS personnel, contributes expertise to the process of system planning and dispatch, reviews quality of care, supervises individuals providing on-line medical care, and solves problems.

In most cases, the administrative chain of command in an EMS system is separate from the position of medical director. The EMS system administrators hire, fire, schedule, and promote employees; purchase and maintain equipment and supplies; and perform the hundreds of other tasks required to operate the

EMS system. The EMS medical director must have a good working relationship with EMS system administrators. Ultimately, the medical director has the responsibility to certify and decertify EMS medical providers and decide what forms of prehospital care will be provided.

Any physician who is seriously considering becoming an EMS medical director should, at minimum, read the American College of Emergency Physicians (ACEP) publication, "Medical Direction of Emergency Medical Services." This concise 80-page guide outlines the standards of responsibility and reviews national policies on EMS medical direction in more detail than is possible in this article.

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Definition of Medical Control

Off-line medical control refers to all physician activities that prospectively and retrospectively are performed to improve quality of care in an EMS system. On-line medical control is the process through which an emergency physician guides prehospital care while it occurs.

When most people think of medical control, they envision on-line medical control (eg, paramedics communicating with and receiving instructions from a physician in the ED). However, in order to maintain a quality EMS system, several hours of off-line medical control activity are necessary for every minute of on-line control.

Off-Line Medical Control

The medical director of an EMS system is responsible for off-line medical control actions, including the following:

- Development and implementation of protocols and standing orders
- Supervision of any initial and recertification training programs provided by the EMS agency
- Retrospective review of the care delivered (to ensure compliance with patient care standards)
- Liaison of activities between EMS professionals and others, including other physicians; ED personnel; and regional, state, and local EMS authorities
- Providing input on dispatch, mutual aid, disaster planning, and hazardous materials response activities
- General supervision of physicians who provide on-line medical control
- Acquiring and maintaining up-to-date knowledge of EMS issues
- Support of EMS research, where practical
- Problem solving

Generally, an EMS system has one overall medical director for off-line activities and a group of

physicians designated as the source of on-line medical control.

On-Line Medical Control

On-line medical control involves directing the care of a single patient. The on-line medical control physician evaluates information given by medics, makes decisions regarding immediate patient care, and gives appropriate orders. Medics and their patients benefit from having immediate access to an emergency physician for advice in difficult or unusual situations.

In addition, the EMS medical director may use mandatory on-line physician authorization to maintain tight control of certain potentially dangerous prehospital treatment options (eg, prehospital thrombolytic administration).

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the Ems Medical Director

Qualifications of a Medical Director

An EMS medical director must be a licensed physician with interest, experience, and knowledge in emergency medicine and prehospital care. It is extremely helpful if the medical director is a full-time, practicing, emergency physician at the lead hospital for the EMS system, with additional training and experience in EMS.

When looking for a medical director, many emergency medicine physician groups may find a former paramedic and who maintains an enthusiasm for EMS. Although these are helpful qualifications, a medical director also must be knowledgeable in medical, administrative, and legal issues. However, this information is not taught in medical school or by direct EMS experience and may only partially be covered in an emergency medicine residency training program.

Medical director courses, which are available through ACEP and the National Association of EMS Physicians, are a valuable experience. For those with a career commitment to EMS, 1-year fellowships are available.

Duties of the Medical Director to the EMS System

The medical director is responsible to the EMS system and the community for ensuring that prehospital care providers function at the highest possible level, given their available resources.

Responsibilities of the medical director include the following:

- To actively participate in system design, personnel training and retraining, supervision, and quality improvement
- To ensure that appropriate standing orders and protocols are in place for prehospital care of common and foreseeable medical conditions, interfacility transfer, disaster response, and hazardous materials response
- To be responsible for general protocol to cover the unforeseen situations that inevitably arise
- Above all, to serve as a patient advocate

Duties of the EMS System to the Medical Director

Responsibilities of the EMS system include the following:

- To provide the medical director with the authority to enforce standards of care; this includes the authority to decertify individual EMS providers who fail to maintain training, patient care, or communications standards
- To provide administrative help because, without it, the medical director's role would be impossible owing to the amount of time this responsibility requires.
- To provide liability insurance for the physician's actions as the medical director
- Possibly, to compensate the physician for time spent working as medical director

The medical director's duties, responsibilities, and authorities, as well as the EMS system's obligations, should be recorded in a written agreement.

Individual Medical Director Versus the EMS Council

Some EMS systems choose to provide off-line medical control through an EMS council or physician advisory board. This body usually consists of representatives from each hospital within the EMS system's coverage area.

It often includes a representative from each EMS agency within the system. In this case, the chairman of the EMS council becomes the overall medical director for legal purposes; however, decisions about system design, protocols, and certification and decertification of EMS personnel are made by the entire committee.

Advantages of an EMS Council

By definition, an EMS council is less autocratic than an EMS system with a single medical director. Occasionally, an EMS council can buffer the EMS system from the politics that are inherent in competition between hospitals and companies. A system design decision (eg, designating a preferred

receiving center for trauma, pediatrics, or burns) that seems to favor the single medical director's hospital may be perceived by personnel of other hospitals as being motivated by the medical director's proprietary interest. The same decision by an EMS council does not draw such criticism.

Discipline of individual medics or agencies by a council may not seem as personally vindictive as when the same actions are taken by an individual medical director; therefore, the discipline may be better accepted when it is imposed by an EMS council.

- By distributing responsibilities among more people, an EMS council can sometimes accomplish more without overburdening any one individual, and a council may be able to devote more energy to planning improvements.
- If the EMS council includes senior paramedics, then a practical, experience-based standpoint is brought into each decision; however, such a point of view may be lacking in the decisions of a single medical director.
- Often, a given paramedic (ie, medic) or emergency medical technician (EMT) will work for 2 or more different EMS agencies. In these situations, it becomes difficult for the medics to follow one set of protocols. Regional protocols, as designed by regional EMS councils, are used to avoid this problem.

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Prehospital Care Protocols

EMS medical directors write prehospital care protocols (ie, standing orders) to instruct medics of the kind of care needed for patients in a wide variety of situations. This document provides the framework for all prehospital care, including assessment and management instructions for a variety of patient conditions, dosing and indications for medications, and specific instructions for occasions when the paramedic or EMT must call for advice and authorization from on-line medical control.

While writing or revising prehospital care protocols, consider the following:

Use previously established protocols

Contact the medical directors of EMS systems in the area and request permission to review and copy their protocols. Read several different protocols and look for the subtle, but important, differences among them.

Do not fix what is not broken

If the EMS system already has an effective protocol in place, only incorporate changes when the benefit is clear. Avoid changing the same part of the protocol several times. Examples of this are as follows:

- The current protocol may still include the use of military antishock trousers (MAST) for hemorrhagic shock. Currently, solid evidence indicates that MAST are harmful, and no research supports a likely change to this conclusion. Therefore, the use of MAST should be removed from prehospital protocols.
- Based on active research, recommendations for fluid resuscitation in trauma are rapidly changing. Rather than modifying the fluid resuscitation protocol several times, it is usually wise to leave an acceptable current protocol in place until definite changes can be made.

Write protocols for each level of certification

If the EMS system includes providers with different levels of training, a protocol for each level of medic needs to be written. For example, the cardiac arrest protocol for different levels of medics should be written as follows:

- An EMT-Basic (EMT-B) should perform cardiopulmonary resuscitation (CPR), placement of oropharyngeal airway, and bag-valve-mask resuscitation.
- An EMT-Defibrillation (EMT-D) should perform the above protocol and apply a semiautomatic external defibrillator.
- An EMT-Intermediate (EMT-I) should perform the above protocol, start an IV line, and, perhaps, intubate or place a Combi-tube.
- An EMT-Paramedic (EMT-P) should perform the above protocol, intubate, and administer epinephrine and IV antiarrhythmics per advanced cardiac life support (ACLS) protocol.

What works in the city may not work in the country

Protocols that work well in urban areas often fail in rural areas (and vice versa).

- Volunteers staff many rural EMS systems; therefore, their time available for training often is less than if the staff was comprised of full-time medics.
- Lower call volumes offer rural medics fewer opportunities to apply their skills; thus, skill atrophy is a problem.
- Rural EMS systems have longer average transport times than urban EMS systems; this influences the effect of prehospital care.

Comparison of rural and urban EMS protocols may create a paradox. For example, consider the following factors for whether to train EMT-Bs to intubate:

- In an urban area, paramedics may be full-time employees who transport up to 2500 emergency patients per year and who have access to full-time training personnel. These paramedics have

many opportunities to intubate and have access to frequent refresher programs.

- In contrast, a rural volunteer EMT may transport an average of 100 patients per year and have few opportunities to maintain skills, which often decay when not frequently practiced.
- ALS in urban areas is often readily available and transport times are likely to be 10 minutes or less. However, rural EMTs may face average transport times of 30 minutes with no ALS back up. Intubation by an EMT is potentially advantageous over prolonged bag-valve-mask ventilation.

Target your protocols to the skill of the medics involved

When writing protocols, take into account the average and the lowest skill level of the medics in the EMS system. Provide medics with enough information about drugs, devices, and latitude to do their jobs effectively, however, do not allow potentially dangerous modalities unless all medics in the system can safely handle them. Resist expanding protocols until the entire group is ready to advance to the necessary level of competency.

Avoid protocol sprawl

Every skill, drug, or device in an EMS protocol requires initial training, memorization, and continuing training for EMS caregivers to remain proficient. In addition, purchase and resupply of equipment and medications costs money. Whenever possible, remove treatment modalities that are out-of-date or no longer necessary from the protocol. Do not expand protocols beyond the level of a capable medic's competence.

Query the receiving physicians about the patients' needs

Solicit feedback from the emergency physicians who treat the patients being transported by the EMS agency.

- Determine prehospital conditions that the medics inappropriately are managing because of current protocols.
- Determine what the medics are doing that may make receiving physicians uncomfortable.
- Determine if medics are spending time on prehospital tasks that easily could wait until the patient's arrival.

Involve EMS personnel in protocol development

Ask medics for suggestions when writing the protocol. If the medical director is uncertain whether a protocol change is worth the required amount of time and money, a mock protocol should be written. After the medics have reviewed the mock protocol, have them make a copy of the patient's chart for whom they feel would have benefitted from the proposed treatment. This process gives the medical director an idea of how many times a new treatment will likely be used. It also acts as a check to evaluate if the medics are likely to inappropriately apply the treatment.

Make on-line medical control mandatory in certain situations

If a particular treatment decision has particularly high medical or medicolegal risk (eg, administration of prehospital thrombolytics, termination of resuscitative efforts in the field), consider requiring approval of the on-line medical control physician. In addition to providing guidance, oversight, and experience, this relieves the medics of some medicolegal responsibility.

The process of establishing on-line medical control slows the delivery of care, prolongs scene time, and occupies a busy emergency physician for several minutes. Limit mandatory on-line medical control to those situations in which a physician's judgment is necessary.

Decide in advance how to handle "Do not resuscitate orders"

Situations in which medics are called to care for a patient who is dying of an incurable condition are volatile and emotionally charged. Morally, medics are bound to respect a patient's desires not to be resuscitated. Several states have adopted statutes that govern prehospital orders not to resuscitate. Incorporate the state statute into the protocol, if possible.

- If the state EMS codes do not address this situation, the medics need to be instructed through the protocols.
- A 1988 ACEP position statement provides guidance in this area. In general, if on-line medical control is available, instruct the medics to discuss each potential do-not-resuscitate (DNR) situation with the on-line physician.
- It often is helpful for the medics and/or the on-line medical director to discuss the situation with the patient's personal physician and family if this quickly can be accomplished.
- Written DNR orders generally should be honored as long as the identity of the patient is not in question and all involved agree.
- The decision not to attempt resuscitation does not include withholding fluids and oxygen when they are needed.

Allow for variations from protocol

No set of standing orders will provide the correct instructions for every situation that medics encounter. The simplest way to authorize medics to deviate from their protocols is to require the medic to contact the on-line medical control physician to discuss the situation and receive appropriate orders. The protocol also should permit medics to perform to their level of training in the event of communications failure.

Include procedures for interfacility transfer

Interfacility transfer requires different skills and behavior than prehospital emergency care. If the EMS agency provides this service, standing orders must address the important differences.

In general, EMT-Bs can transport medically stable and spontaneously breathing patients who are on oxygen, including patients with tracheostomies. With minimal additional training, EMTs can be taught to maintain noncritical IV lines. The protocol clearly should state what types of IV the EMTs are allowed to supervise. Because EMT-Bs are not trained to restart IVs if pulled out, they should not accept a patient with life-sustaining IV fluids or medications unless accompanied by a paramedic or registered nurse (RN).

It is suitable for an appropriately trained EMT-Ambulance (EMT-A) to transport patients who are on maintenance crystalloid with no more than 20 mEq/L potassium chloride (KCl). The addition of benign additives (eg, vitamins) does not pose a problem.

Paramedics are capable of performing the wide range of ACLS skills, can titrate and restart IVs, and can manage much more unstable patients during interfacility transfer than EMTs. If many unstable cardiac patients are transported, the paramedics need to know how to run IV infusion pumps and should have additional training on drugs (eg, IV heparin, nitroglycerin, common vasopressors, paralytic medications).

Occasionally, critically unstable patients need to be accompanied by an RN during transfer from a hospital. Under the federal Emergency Medical Treatment and Active Labor Act (EMTALA), the transferring hospital is legally responsible for providing an RN, respiratory therapist, or medical doctor to accompany the patient during transport in case an advanced level of care is needed. Legal considerations aside, this is expensive and inconvenient for the transferring hospital. Trusting the paramedics to effectively transfer the patient is highly tempting, even when the patient's needs are more complex than those the paramedics are trained to handle. The system's protocols should specify the situations in which paramedics may transport an unaccompanied patient and the situations that mandate a nurse to accompany the patient during transport.

Patients who at least require an RN-paramedic team to assist in transport include the following:

- Patients with unstable vital signs at the time of transfer, with the possible exception of patients presenting with fresh trauma who have not yet been to the operating room
- Patients in advanced or preterm labor
- Seriously ill children younger than 6 years
- Patients likely to require intubation en route

The medical director or on-line medical control physician should become involved in difficult cases. Paramedics are professionals and their judgment should be trusted; when paramedics are uncomfortable accepting a patient, that admission should be respected, even if it inconveniences the transferring hospital.

Specify the procedure for medical control when a physician is on the scene

Prehospital care protocols need to provide guidelines for the medic's actions when a physician is on the scene. Sometimes, on-scene physicians tend to interfere more than help. These situations must be handled with tact and diplomacy, but must not delay patient care.

ACEP has chosen a clear and rational position on this issue. It is summarized in a 1984 position that states, "When an ALS squad, under medical direction, is requested...a doctor/patient relationship has been established between the patient and the physician providing medical direction. The paramedic is responsible for management of the patient and acts as an agent of medical direction unless the patient's physician is present (as would occur in a doctor's office)."

If the patient's private physician is present and assumes responsibility for treatment, medics should defer to that private physician. If an intervening physician is present and on-line medical direction is possible, the intervening physician should speak directly with the on-line medical control physician to discuss the situation. The on-line physician has the option of entirely managing the case, working with the intervening physician, or allowing the intervening physician to assume responsibility. If an intervening physician is present and on-line medical control is not possible, ACEP states, "A paramedic...should relinquish responsibility for patient management, but if the treatment...differs from that outlined in local protocol, the physician should agree in advance to accompany the patient to the hospital...."

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Quality Assurance and Problem Solving

Medical directors have the responsibility to ensure that all care provided under their authority is of the best quality possible. Several actions are required to accomplish this goal, including the following:

Routine Run Review by EMS Agency Leadership

Someone within the EMS organization routinely should review all EMS run records for completeness. This person, usually the chief medic for the service, should perform the following:

- Maintain statistics on factors (eg, scene times, number of attempted IVs and intubations, number completed IVs and intubations, number of cardiac arrests managed for each medic in the system)
- Collect data to determine necessary areas of improvement for the entire service (ie, continuous quality improvement [CQI] data)
- Screen for problems that might otherwise be undetected (ie, quality assurance [QA])

Random and Focused Run Review by the Medical Director

The medical director should review a reasonable percentage of EMS runs for the preceding month.

- If all runs cannot be reviewed, critical runs should be targeted (eg, runs in which ACLS drugs and advanced airway modalities were used, runs by problem medics). Additional runs for review should be selected by random sampling of those remaining.
- Provide written feedback for individual medics and identify patterns of behavior that need individual or group retraining.
- Maintain a file of each paramedic's feedback for future review, if needed.

Soliciting Feedback from Receiving Physicians

Emergency physicians (EPs) and administrators at all receiving hospitals in the EMS system's coverage area need to know whom to call with problems or questions about the service. The medical director should be introduced to colleagues at other hospitals, preferably during an ED or EP group meeting. This process should periodically be repeated to encourage open communication and feedback, which can prevent many interinstitutional problems and may quickly help identify problems with specific medics or situations. The medical director should meet with the administrative directors of hospitals in the area where the EMS system regularly operates (eg, coronary care staff for frequent interfacility transfers).

Handling Problems

No organization or person does the right thing every time. By its nature, EMS requires medics with incomplete knowledge of the situation to make complex life-or-death decisions very rapidly and during continuous distraction. It is a tribute to EMS professionals that, through dedication and training, they get things right the vast majority of the time.

When mistakes happen, the challenge of the medical director is to decide the following:

- If, given the situation faced by the medics, the mistake is understandable
- If the mistake at hand indicates a need for further training
- If the mistake was simply a fluke
- If a specific medic or the entire service needs to be addressed about the mistake

All possible information should be gathered before action is taken to handle a potential problem situation. Feedback should be collected from everyone involved, including the medics, physicians, and nurses at the receiving hospital and, perhaps, the patients and their families. Obtain run records, ED reports, and, when relevant, inpatient care records and autopsy records. Often, what initially appeared to be a dangerous mistake will turn out to be a reasonable decision based on the information available to the medic at the time.

Situations That Always Require Intervention

Unrecognized esophageal intubation

Although an endotracheal tube (ETT) can become dislodged when the patient becomes agitated, it should not become displaced with simple movement of the patient. This occurs more often than many would imagine, and a response to this problem should be consistent. The director should meet with the paramedic involved to discuss the situation. If a pattern develops, refresher training for the individual paramedic or, perhaps, all paramedics in the service should be required.

Colorimetric carbon dioxide detectors and esophageal intubation detectors are commercially available, inexpensive, and reasonably reliable. These could be added to the equipment list and protocols. If a specific paramedic has recurrent problems with unrecognized esophageal intubations, the paramedic should be decertified until the problem has been corrected.

Inappropriate medical care and inappropriate withholding of medical care

The medical director should conduct a thorough investigation. Policies should be revised, refresher training should be provided, or discipline of medics should be carried out, as appropriate.

Injuries to patients

Patient injuries usually occur because of the following 2 situations:

- Patients are dropped from the ambulance stretcher.
- Patients are injured during the application of restraints.

These situations can typically be corrected with refresher training and written policies.

Many ambulance stretchers are not designed to be in the up position when patients are rolled long distances. The EMS service may need to invest in new stretchers or require that stretchers are in a down position when rolling patients; the latter policy eventually will lead to back injuries among medics.

Storm drain covers with inch-wide rectangular vents may potentially cause accidents. To reduce the chance that the EMS service and facility are sued over a patient injury, these storm drain covers need to be replaced with ones that will not trap stretcher wheels.

Allegations of theft from patients

This is a personnel matter, not a medical issue, and it should be handled by the administrative chain of command of the EMS system.

Substance abuse among medics

This problem requires action by the EMS system chain of command and medical director. The EMS agency must have personnel policies and procedures to address this situation. However, the medical director is responsible for appropriate use of all narcotic medications purchased by the EMS service under the drug enforcement agency (DEA) number of the medical director.

The medical director's responsibilities in these situations include the following:

- Act as a patient advocate by protecting patients from the risk of a medic acting under an extension of the director's physician license while intoxicated.
- Act in the best interest of the patients and medic involved.
- Comply with legal requirements to report impaired medics to the state EMS licensing authority.
- Work with the EMS agency's attorney during every step to ensure that the medic is treated fairly and to protect the director and EMS agency from lawsuit.
- Realize that the potentially impaired health care professional is handled best by consulting with an expert in the areas of addiction to medicine and substance abuse.
- Carefully handle the process of confrontation.
- Be aware that most state medical societies are a valuable resource in these matters.

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Training

An EMS medical director should actively be involved in initial and recertification courses that are conducted by the agency. Generally, a training officer or course director will run the day-to-day mechanics of the program. The EMS medical director serves as the medical director for the course, helps arrange for lecturers, provides some direct instruction, and supervises testing. Many states require the medical director to personally certify, in writing, that each graduate of the training program clinically is prepared to practice prehospital care at the appropriate level of certification before the applicant is permitted to take the state EMT or paramedic examination.

The EMS medical director also must coordinate a regular, ongoing, training program for medics. It is effective to combine programs of general interest with refresher training that is targeted at areas of potential improvement. The medical director should do some teaching, occasionally recruit outside instructors, and encourage medics within the service to develop areas of expertise for peer instruction.

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on-Line Medical Control

In an ALS, paramedics and other EMT-As make 99% of the requests for advice and orders; however, EMT-Bs also require the advice of a physician. Therefore, providers at all levels of certification must have access to on-line medical control. On-line medical control must be available 24 hours a day, 365 days a year.

The medical director designates a group of physicians to perform on-line medical control. Three basic ways to provide this service are as follows.

- The on-duty emergency physician at the lead hospital for the EMS service (ie, the resource hospital) can serve as on-line medical control.
- Alternatively, the on-duty emergency physician at the hospital that will receive the patient can be responsible for on-line medical control.
- Finally, medical control can be provided by a small group of physicians at a centralized location.

In the third scenario, the medical control physician dedicates his full attention to this task and is not simultaneously responsible for patient care. This is practical in large EMS systems, and it is often coordinated with an emergency medicine residency program, such as exists in Pittsburgh and Milwaukee.

Advantages and Disadvantages

Advantages and disadvantages exist for each system. The first and third options of providing on-line medical control (mentioned above) use a smaller number of physicians, usually partners or residents of the medical director, which improves consistency and communication between the physicians. The second option, which is probably the most common nationwide, sacrifices consistency of medical control and makes it difficult to reach every medical control physician and disseminate changes to protocols.

Paramedics may dislike the inconsistency of the orders they receive when service is provided according to option 2. However, the second method has advantages. Paramedics and emergency physicians receive immediate feedback since the medic, physician, and patient arrive at the same place at the same time. Also, with option 2, the receiving physician can hardly be dissatisfied with the orders given, and may change those orders based on the situation at the receiving hospital (eg, withholding an order for prehospital thrombolytics if the cardiologist and catheterization lab team already are in the hospital). Overall, the second method is the easiest to set up but the hardest to do well.

Personnel Providing On-Line Medical control

At a minimum, any physician providing on-line medical control must be skilled in emergency medicine

and familiar with the equipment and capabilities of the EMS system and the paramedics' training and protocols. A short base station training course for physicians, especially those without prior EMS experience, will greatly improve the quality of medical control. However, it often is hard to get physicians to attend. One possible solution is to incorporate a brief training program into a regular ED or physician group meeting.

Sometimes, nurses from the ED, physicians' assistants, or specially trained paramedics are permitted to provide on-line medical control. This is convenient in a busy ED, but medics prefer and patients may benefit from the physician's direct input. Of course, the designated on-line medical control physician is legally responsible for the orders given by others who are under his supervision.

Communication with Physician Verses Other ED staff

If the on-line medical control physicians feel that their time is taken unnecessarily by EMS calls, the medics may be speaking with the physician when physician guidance is not necessary. The first step to correct this problem is to ask medics to begin their call to the medical control hospital with the introduction of "report only" or "physician needed for orders." Any qualified ED staff member can take a report about a patient coming in. The physician is summoned only if the medic has a question or is requesting advice or orders for specific treatment changes. Emergency physicians at the receiving hospitals may be uncomfortable with this system at first but, in most cases, eventually prefer it.

If this is already part of the system and physicians still feel that too much of their time is occupied with by EMS calls, one of two things is happening; either the medics lack the confidence to independently carry out protocol orders or the protocols and standing orders have been structured to require an on-line physician order for tasks that the paramedics should be able to independently initiate. These problems should be recognized and addressed by the off-line medical director.

Forms of Communication

Although most medical control conversations use EMS radios, telephones (ie, land-based, cellular) possess advantages in clarity and confidentiality. EMS professionals should be encouraged to use the phone whenever practical and use the EMS radio only when the telephone is not available.

In the early days of EMS, medics in the field commonly transmitted cardiac rhythm strips via VHF radio for physician interpretation. This is time-consuming and the equipment often does not produce a readable strip at the receiving facility. Paramedics are trained to recognize and treat arrhythmias.

Today, rhythm strips rarely are transmitted for off-site interpretation. However, obtaining a 12-lead prehospital ECG to transmit over cellular phone lines to a receiver in the ED has some advantages. Medical directors and EMS system administrators should carefully consider the cost (eg, equipment, airtime, training, increased scene time) and benefits (eg, more rapid administration of thrombolytics in

the receiving hospital, possible prehospital thrombolytics) of such a system.

Occasionally, questions arise about what was said and ordered during on-line medical control conversations. An accurate record of these conversations is useful in training, for quality assurance, and for medicolegal purposes. Tape recording, written record keeping, or both are used for this purpose.

Tape recording is more accurate but more expensive than keeping a written record. However, tape recording medical control conversations can inexpensively be accomplished through the dispatch center, which may already have a multichannel tape recorder in use to record incoming calls and radio traffic. If this technology is not available, recording devices must be attached to all radio and telephone lines at the medical control hospital(s) that are used for this purpose. The EMS system also must establish a procedure to securely catalog and store tapes.

Printing and distributing forms for written documentation of medical control conversations is easy, but this system has several shortfalls. It is difficult to simultaneously listen, analyze, and write down everything the medic says. On-line physicians tend to write less and think more. The result can be a very fragmented and incomplete record. Also, because of patient confidentiality concerns, the patient's name usually is not broadcasted over EMS communications systems. This creates a problem when trying to match the medical control record with a specific patient.

As digital cellular technology gradually replaces VHF radio, concerns about eavesdropping should lessen. Cataloging and storing on-line medical control reports is a challenge, especially when the patient is not transported to the hospital or when the medical control facility and receiving hospital are not the same.

Finally, many disputes and problems in on-line medical control arise from a misunderstanding between what the medic "knows" was said and what the physician "knows" was heard. A hastily scribbled record by the medical control physician almost never contains enough information to sort out the truth in these situations.

In general, the tape-recorded systems are the better choice for most EMS systems. The start-up costs are small, although not negligible, when compared to the cost of gasoline, insurance, salary, and other operating costs of an EMS system. Although setting up a tape-recording system initially requires more work than printing and distributing forms, the amount of work saved in the long run far exceeds the amount invested.

The off-line medical director may choose to review medical control records only when problems arise. However, random or focused reviews of medical control conversations are very useful for quality improvement and training purposes and a productive use of the medical director's time.

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Prehospital Airway Devices

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Synonyms, Key Words, and Related Terms

airway adjuncts, airway control, airway management, out-of-hospital airway devices, EMS airway devices

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Introduction

The establishment and maintenance of a patent airway is the primary task of the prehospital emergency care provider. With few exceptions, the airway devices available in the field are the same as those used in emergency departments (EDs).

Just as the types and sizes of airway devices available in any given ED vary tremendously, so does the stocking of emergency medical service (EMS) units (see [Picture 1](#), [Picture 2](#), [Picture 3](#), [Picture 4](#), [Picture 5](#), [Picture 6](#)). Furthermore, just as the training, experience, and competence vary among EDs and on-duty physicians, they vary similarly among EMS units and providers. Thus, ED personnel must be familiar with the training, experience, and equipment of the EMS providers and units in their area.

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Training

Two broad levels of training in EMS exist, with great variation from state to state in the scope of practice of these levels.

Basic life support (BLS) providers generally are classified as emergency medical technicians (EMTs) - basic (EMT-B) or -ambulance (EMT-A). Advanced life support (ALS) providers generally are classified as EMT-P (paramedic). Various intermediate classification suffixes exist, such as EMT-CT (cardiac technician), -I (intermediate), -E (epinephrine), and -D (defibrillation). Providers in mobile intensive care units (MICUs) also may obtain CCEMT-P (critical care) certification.

More information about the EMS Practice Act that applies to a specific area may be obtained from the appropriate state or local agency. A summary of this act should be posted near the medical control radio, along with the standing orders, protocols, and drug lists for each EMS unit a hospital serves.

ED physicians may get an idea of the level of training of prehospital personnel to whom they are providing medical control or from whom they are receiving a patient by determining their level of certification. In most EMS training programs, EMTs receive training in the use of the airway devices described in the following sections.

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Basic Airway Devices

Basic airway devices include the following:

- Oropharyngeal airway (OPA): This is a plastic or rubber device inserted in the oropharynx to create an airway between the tongue and the palate. This device should not be used on a patient with an intact gag reflex.
- Nasopharyngeal airway (NPA): This is a rubber tube inserted into the nare and extending into the oropharynx (see [Picture 7](#)); it is designed to create an airway between the tongue and palate. It may be used in a semiconscious patient. It also may be used to supplement positioning in maintaining an open airway.

- **Pocket mask:** This plastic and rubber mask is designed to protect the rescuer during rescue breathing. It may have a one-way valve to isolate patient secretions and an oxygen port to supplement the rescuer's exhaled oxygen. It is used primarily by first responders when a bag valve mask is not available.
- **Bag valve masks (BVM):** This is a combination of face mask and self-inflating resuscitation bag. It also should have an oxygen reservoir and tubing. It is used in conjunction with positioning, NPA, OPA, and endotracheal tubes to provide oxygenation and ventilation to the apneic or hypoventilating patient.
- **Suction devices:** These are used to remove secretions from the oropharynx or endotracheal tube. Many types of suction units are used in EMS, including portable hand-powered and battery-powered units as well as wall-mounted units (see [Picture 8](#)).
- **Demand valve mask:** These oxygen-powered resuscitator-mask combinations are activated by the rescuer pushing a button. Although easy to use, these units do not allow the operator to feel the effectiveness of ventilation or lung compliance. Because of this drawback, they no longer are recommended.
- **Esophageal obturator airway (EOA):** This device consists of a face mask that snaps onto a cuffed tube with a closed end that is inserted blindly into the oropharynx with the head in a neutral position. It is designed to enter and occlude the esophagus, preventing regurgitation. A bag attached to the mask provides ventilation. This device has fallen out of favor over the past 10 years with the increasing acceptance of EMS intubation, the development of superior devices, such as the PTL Airway and Combitube, and the risk of unrecognized tracheal obturation by the basic personnel for whom it was designed originally.
- **Esophageal gastric tube airway (EGTA):** This is similar to the EOA but has an opening in the esophageal tube for inserting an orogastric tube for stomach decompression.
- **Pulmonary resuscitator:** This unusual device is used primarily in rescue situations when available personnel are limited. It consists of a mask attached to a 3-foot length of corrugated tubing with a mouthpiece at the distal end of the tubing and a one-way valve. The rescuer can use 2 hands to secure the mask to the patient's face and provide mouth-to-mask ventilation through the tube. Supplemental oxygen can be introduced into the mask through an inlet port. The mask also can be secured to the patient's face with straps, freeing the rescuer's hands for other tasks, such as cardiopulmonary resuscitation (CPR). An example of the use of this device would be in a ski rescue where one rescuer has to continue CPR while straddling a patient on a sled.

Note: Most EMS agencies and organizations no longer recommend the use of the EOA and EGTA.

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Advanced Airway Devices

These devices are divided here into commonly and less commonly used devices. A description of some of the devices with which physicians may not be familiar has been included.

Commonly used devices

Laryngoscope: Consisting of a handle containing batteries and interchangeable blades of various shapes and sizes containing a bulb or fiberoptic light, the laryngoscope is used to lift the tongue, mandible, and epiglottis and visualize the glottic opening, both for removing foreign bodies and inserting endotracheal tubes. Blades commonly are available in 5 sizes, from 0 for infants to 4 for large adults. The 2 main styles are straight (Miller, Wisconsin, or Flagg blade) and curved (Macintosh). The straight blade is designed to lift the epiglottis directly. The curved fits into the vallecula. Both reusable and disposable laryngoscope handles and blades are available.

Endotracheal tubes (ETTs): Used to establish a definitive airway, ETTs are inserted through the nose or mouth into the trachea. The open tip should lie distal to the vocal cords and proximal to the bifurcation of the mainstem bronchi and carina. ETTs are available cuffed (for adults) and uncuffed (for children) and in various sizes from 2.5 for premature infants to 10 for very large adults. Most have a standard 15-mm fitting to attach to ventilation devices.

Endotrol ETT: The Endotrol is an ETT with a thin nylon line attached above the cuff that runs through a sleeve and ends in a loop (see [Picture 9](#)). As the tube is grasped with the right hand prior to intubation, the pointer finger of the grasping hand is slipped through the loop, and the thumb is placed over the 15-mm adapter for leverage. By pulling up on the loop, the tip of the tube can be flexed several centimeters. This is especially helpful in maneuvering the tube in a patient with an anterior glottic opening during endotracheal intubation and also greatly facilitates blind intubations.

End-tidal CO₂ detectors: These are devices containing litmus paper to measure the level of CO₂ exiting the lungs during ventilation (see [Picture 10](#)). They are a good indicator of whether the ETT has been placed in the trachea or the esophagus, unless the patient is in cardiopulmonary arrest. They are available as "caps" that are placed between the ETT/face mask and the BVM, or they come integrated into the BVM. They are available in adult and child sizes. Careful attention must be given to the expiration date and the integrity of the airtight packaging to insure accuracy.

Esophageal intubation detector: This ingenious device is startling in its simplicity. It is a large syringe with a 15-mm adapter at the open end and either a standard plunger or a rubber bulb at the other. After ETT placement, but before administering any ventilations, the device is placed on the 15-mm adapter of the ETT and air is drawn into the syringe. If the ETT is in the esophagus, drawing air out is difficult, as the surrounding esophageal tissue occludes the ETT tip. If the ETT is in the trachea, drawing air out is easy. Also known as the "turkey baster," this device has been shown in recent literature to be useful in

the field as well as the hospital.

Pharyngotracheal lumen airway (PTL) and esophageal tracheal Combitube (ETC): These devices are designed for blind insertion (see [Picture 11](#)). Regardless of whether the tube is inserted into the esophagus or trachea, the operator determines placement by examination and auscultation. He/she inflates the proper cuff and accesses the proper ventilation port according to tube placement. If the trachea is intubated, the tube cuff is inflated and the tracheal tube accessed. If the esophagus is intubated, the tube cuff and the large oropharyngeal cuff are inflated, and the oropharyngeal port ventilated through holes in the upper end of the tube and occluding the esophagus. These devices are in limited use; depending on local protocol, EMT-Bs and EMT-Ps have been trained in their use.

McGill forceps: Available in adult and child sizes, these forceps are designed to be used in conjunction with direct laryngoscopy in removing foreign bodies from the airway.

Less commonly used devices

Cricothyrotomy equipment: Numerous kits have been developed by various manufacturers to aid in rapidly establishing an airway when endotracheal intubation fails (see [Picture 12](#)). As these devices are seldom used, selection should be based primarily on ease of use and retention of procedure for placement.

Laryngeal mask airway (LMA): The LMA was introduced into clinical practice in 1988. Since that time, the LMA has fundamentally changed the airway management of patients undergoing routine general anesthesia, especially in Europe and Australia. At the present time in the United Kingdom, the LMA is used in more than 50% of surgical procedures when an ETT formerly would have been used. The LMA is not used widely in the US in the prehospital setting, but it has been used prehospital in Australia by basic and intermediate level providers with good results. When compared to BVM, the LMA is thought to be easier to insert (with only minimal training) and often produces better ventilation with less gastric insufflation. Gastric insufflation is minimized because excess pressure is vented upwards around the LMA rather than forced down the esophagus as in the case of BVM ventilation.

The LMA consists of an inflatable pink silicon laryngeal mask attached to a wide-bore tube that connects to the patient tubing via a standard 15-mm connection. The opening in the middle of the mask has 3 vertical slits and prevents obstruction by preventing the tip of the epiglottis from flopping up into the lumen of the wide-bore tube. It comes in the following various sizes:

- #1 (neonatal)
- #2 (child)
- #2.5, #3 (larger children and small adults, especially women)

- #4 (standard adult)
- #5 (large adult)

The LMA is inserted into the pharynx with the pointed end in the esophagus and the mask outlet sitting over the laryngeal inlet. The mask is then inflated (volume according to mask size [ie, 15-20 mL for the #3, 25-30 mL for the #4]), sealing itself around the laryngeal inlet. The LMA is not a definitive airway and provides only limited protection from aspiration of blood (from above) and little, if any, protection from aspiration of stomach contents from below.

Currently, the LMA is used in the emergency setting by providers not trained in tracheal intubation and by higher level providers as an option in the management of a difficult airway in which intubation is unsuccessful.

Nasoscope: The nasoscope looks like a stethoscope, but instead of a bell at the end, it has an adapter that fits into the 15-mm adapter at the end of an ETT. It is used to listen for proper placement in a noisy environment when performing blind nasotracheal intubation.

Retrograde intubation equipment: Not commonly used in the field because of its difficulty of use, especially in the uncontrolled setting, retrograde intubation equipment and training have been introduced by some EMS services to aid in very difficult intubations. Equipment consists of a large bore over the needle catheter that is introduced into the trachea via the cricothyroid membrane and a guidewire that is introduced through the catheter cephalad into the trachea to the oropharynx. The ETT is then slid over the guidewire and into the trachea, and the wire removed.

Tracheal tube introducer: This is a long stylette with a very smooth surface. The last couple of inches of the tip are angled a few degrees. In a difficult intubation, especially where the vocal cords and tracheal opening are anterior and difficult to visualize, this stylette is introduced first; it can often get around the corner that an ETT cannot. The tube then is slipped over the stylette and into the trachea, and the stylette is withdrawn. One director of anesthesiology reports that this is the primary device used in difficult intubations in his department, with a 90% success rate.

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Protocols and Standing Orders

Regardless of training, ALS units carry the devices and abide by the protocols for their use determined by their prehospital medical director. Paramedics practice under the license of the EMS medical director under the principle of delegated practice. BLS units in most states are not required to have a medical director and operate under state statutes.

Protocols determine the unit's scope of practice. Standing orders are orders that are to be carried out before contact with medical control and are a subset of protocols. For example, a standing order may be in place that, in the event of cardiac arrest, the patient is to be intubated.

Surgical cricothyroidotomy may be included in the protocols, but it requires an order from medical control, and, therefore, is not a standing order. ED personnel must be familiar with the protocols and standing orders of the units they serve and have them readily available for consultation near the medical control radio.

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Experience and Competence

Personnel providing medical control must be aware of the level of experience and competence of the various EMS services and providers in their area.

Medical control personnel should get to know EMS personnel by name and service so that they immediately can recognize the level of competence of each provider and the equipment available. In the same way that ED physicians can anticipate the level of care provided by physicians referring to their ED, they can anticipate the level of care patients receive in the field by various EMS units. The best way to do this is to take an active role in the education, training, and medical direction of local EMS agencies.

The medical director of each service must insist on the highest level of training for prehospital personnel and work with the hospital to provide opportunities for ongoing continuing education, including regular operating room rotations for intubation and tracheotomy practice. Currency of all personnel, physicians, and paramedics in advanced cardiac life support (ACLS), basic trauma life support (BTLS), pediatric advanced life support (PALS), neonatal advanced life support (NALS), and advanced trauma life support (ATLS) is highly desirable (nonphysicians may audit the latter).

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Conclusions

Without the ability to control the airway adequately, all other interventions are futile. EMS can make its greatest contribution to minimizing morbidity and mortality in its ability to rapidly establish and maintain airway patency. For this reason, continuing research, training, and standardization of advanced techniques, such as transillumination, retrograde intubation, and rapid sequence intubation (RSI), should be the next priority in prehospital airway management.

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Web Sites

Tom Trimble, RN. [Action-Plan for Airway Problems from Hell!](#) This site lists an incredible collection of relevant web sites. It was updated and expanded in March 1999.

Pictures



Picture 1: Prehospital airway bag (Courtesy of Bloomington Hospital Ambulance Service, Bloomington, Ind)

Picture type: Photo



Picture 2: Prehospital airway bag, open (Courtesy of Bloomington Hospital Ambulance Service, Bloomington, Ind)

Picture type: Photo



Picture 3: Prehospital airway bag contents (Courtesy of Bloomington Hospital Ambulance Service, Bloomington, Ind)

Picture type: Photo



Picture 4: Intubation bag (Courtesy of Bloomington Hospital Ambulance Service, Bloomington, Ind)

Picture type: Photo



Picture 5: Continuous positive airway pressure (CPAP) equipment (Courtesy of Bloomington Hospital Ambulance Service, Bloomington, Ind)

Picture type: Photo



Picture 6: Continuous positive airway pressure (CPAP) kit (Courtesy of Bloomington Hospital Ambulance Service, Bloomington, Ind)

Picture type: Photo



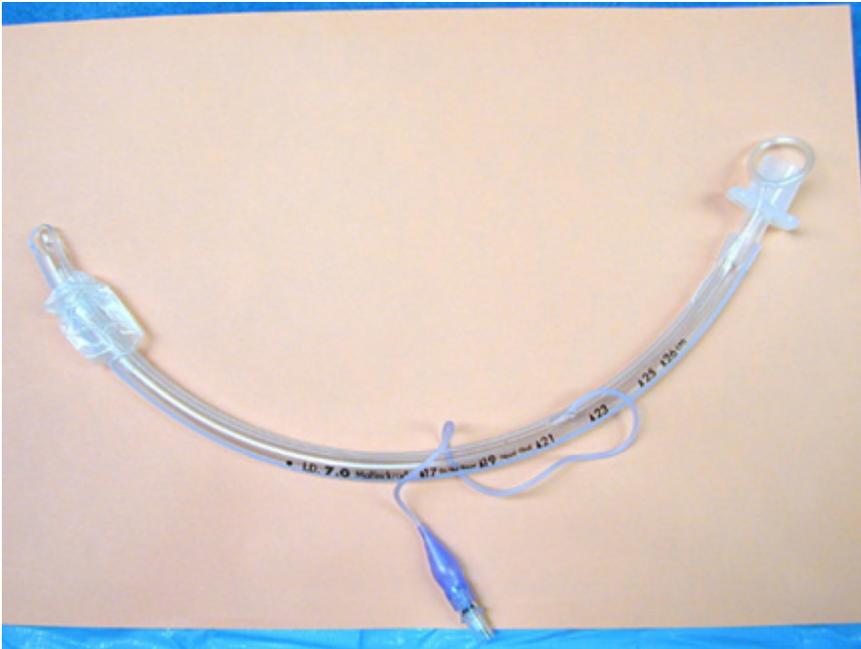
Picture 7: Nasopharyngeal airways (Courtesy of Bloomington Hospital Ambulance Service, Bloomington, Ind)

Picture type: Photo



Picture 8: Suction (Courtesy of Bloomington Hospital Ambulance Service, Bloomington, Ind)

Picture type: Photo



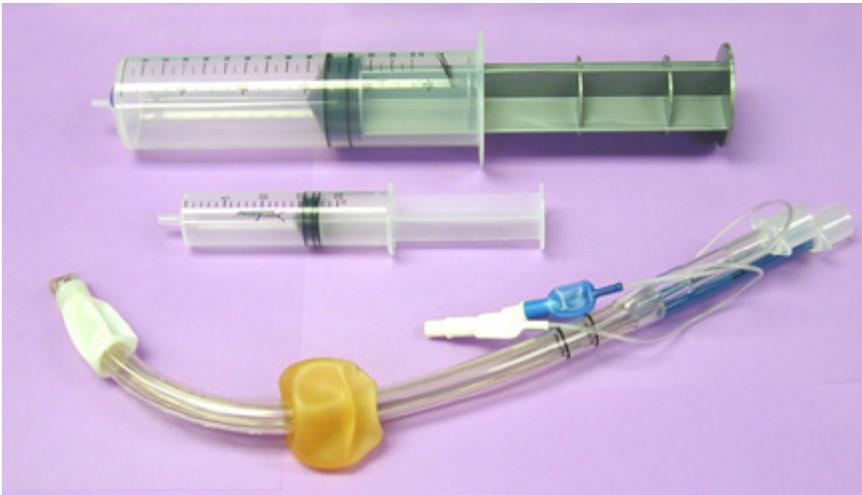
Picture 9: Endotrol endotracheal tube (Courtesy of Bloomington Hospital Ambulance Service, Bloomington, Ind)

Picture type: Photo



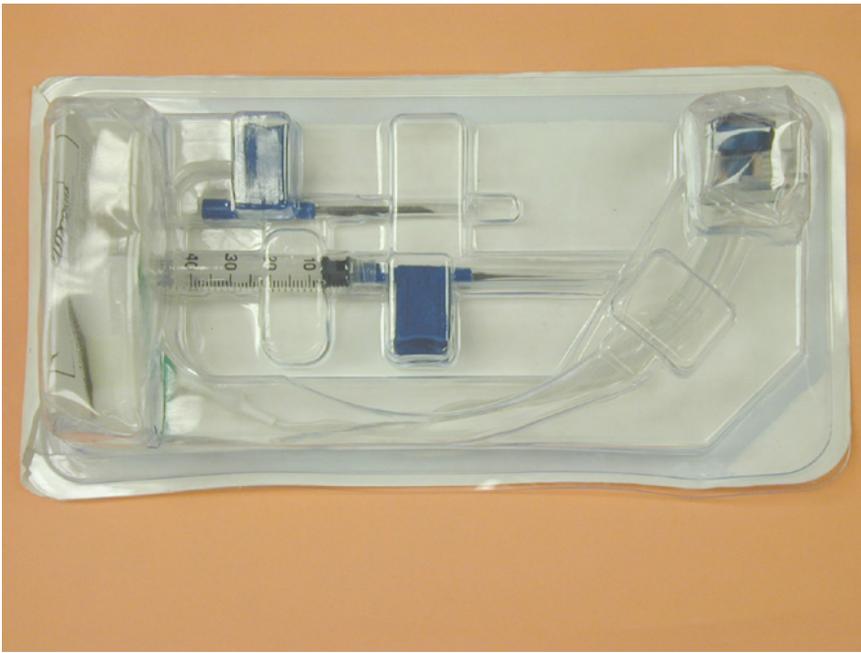
Picture 10: Bag valve mask with built-in carbon dioxide detector (Courtesy of Bloomington Hospital Ambulance Service, Bloomington, Ind)

Picture type: Photo



Picture 11: Combitube (Courtesy of Bloomington Hospital Ambulance Service, Bloomington, Ind)

Picture type: Photo



Picture 12: Pertrach kit (Courtesy of Bloomington Hospital Ambulance Service, Bloomington, Ind)

Picture type: Photo

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Abdominal Trauma, Blunt

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Introduction

Background

Blunt abdominal trauma is a leading cause of morbidity and mortality among all age groups. Identification of serious intraabdominal pathology often is challenging. Many injuries may not manifest during the initial assessment and treatment period. Mechanisms of injury often result in other associated injuries that may divert the physician's attention from potentially life-threatening intraabdominal pathology.

Pathophysiology

Injury to intraabdominal structures can be classified into 2 primary mechanisms of injury—compression forces and deceleration forces.

Compression or concussive forces may result from direct blows or external compression against a fixed object (eg, lap belt, spinal column). Most commonly, these crushing forces cause tears and subcapsular hematomas to the solid viscera. These forces also may deform hollow organs and transiently increase intraluminal pressure, resulting in rupture. This transient pressure increase is a common mechanism of

blunt trauma to the small bowel.

Deceleration forces cause stretching and linear shearing between relatively fixed and free objects. These longitudinal shearing forces tend to rupture supporting structures at the junction between free and fixed segments. Classic deceleration injuries include hepatic tear along the ligamentum teres and intimal injuries to the renal arteries. As bowel loops travel from their mesenteric attachments, thrombosis and mesenteric tears, with resultant splanchnic vessel injuries, can result.

The liver and spleen seem the most frequently injured organs, although reports vary. Small and large intestines are the next most injured organs, respectively. Recent studies show an increased number of hepatic injuries, perhaps reflecting increased use of CT scanning and concomitant identification of more injuries.

Frequency

- **In the US:** True frequency is unknown. Data collected from trauma centers reflect patients who are transported to or seek care at these centers. These data may not reflect patients presenting to other facilities. Incidence of out-of-hospital deaths is unknown.
 - One review from the National Pediatric Trauma Registry by Cooper et al reported that 8% of patients (total=25,310) had abdominal injuries. Eighty-three percent of those injuries were from blunt mechanisms. Automobile-related injuries accounted for 59% of those injuries.
 - Similar reviews from adult trauma databases reflect that blunt trauma is the leading cause of intraabdominal injury and that motor-vehicle collisions are the leading mode of injury. Blunt injuries account for approximately two thirds of all injuries.
 - Hollow viscus trauma is more frequent in the presence of an associated, severe, solid organ injury, particularly to the pancreas. Approximately two thirds of patients with hollow viscus trauma are injured in motor-vehicle collisions.

Mortality/Morbidity

- The National Pediatric Trauma Registry reported that 9% of pediatric patients with blunt abdominal trauma died. Of these, only 22% were reported as having intraabdominal injuries as the likely cause of death.
- A review from Australia of intestinal injuries in blunt trauma reported that 85% of injuries occurred from vehicular accidents. Mortality rate was 6%.
- In a large review of operating room deaths in which blunt trauma accounted for 61% of all injuries, abdominal trauma was the primary identified cause of death in 53.4% of cases.

Sex

Male-to-female ratio is 60:40, according to national and international data.

Age

Most studies indicate peak incidence occurs in persons aged 14-30 years. A review of 19,261 patients with blunt abdominal trauma revealed equal incidence of hollow viscus injuries in both children (ie, ≤ 14 y) and adults.

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Clinical

History

- Initially, evaluation and resuscitation simultaneously occur.
- In general, do not obtain a detailed history until life-threatening injuries have been identified and therapy has been initiated. However, to better predict injury patterns and to identify potential pitfalls, ascertain the mechanism of injury from bystanders, paramedics, or police.
- **AMPLE** is often useful as a mnemonic for remembering key elements of the history.
 - Allergies
 - Medications
 - Past medical history
 - Last meal or other intake
 - Events leading to presentation

Physical

- Initial examination
 - After appropriate primary survey and initiation of resuscitation, focus attention on secondary survey of the abdomen.
 - For life-threatening injuries requiring emergent surgery, delay comprehensive secondary survey until the patient has been stabilized.
 - At the other end of the spectrum are victims of blunt trauma who have a benign abdomen upon initial presentation. Many injuries initially are occult and manifest over time. Frequent serial examinations, in conjunction with the appropriate diagnostic studies, are essential in any patient with significant mechanism of injury.
- Auscultation
 - Abdominal bruit may indicate underlying vascular disease or traumatic arteriovenous fistula.
 - During auscultation, gently palpate the abdomen while noting the patient's reactions.
- Percussion

- Percussion tenderness constitutes a peritoneal sign.
- Tenderness mandates further evaluation and probably surgical consultation.

Causes

- Most common causes of blunt abdominal trauma are from motor vehicle accidents and automobile-pedestrian accidents.
- Other common etiologies include falls and industrial or recreational accidents.

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Differentials

Abdominal Trauma, Penetrating
Domestic Violence
Pregnancy, Trauma
Shock, Hemorrhagic
Shock, Hypovolemic
Trauma, Lower Genitourinary
Trauma, Upper Genitourinary

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Workup

Lab Studies

- In recent years, laboratory evaluation of trauma victims has been a matter of significant discussion. Commonly recommended studies include serum glucose, CBC, serum chemistries, serum amylase, urinalysis, coagulation studies, blood type and match, ABG, blood ethanol, urine drug screens, and a urine pregnancy test (for females of childbearing age).
- Complete blood count
 - Normal hemoglobin and hematocrit results do not rule out significant hemorrhage. Patients bleed whole blood. Until blood volume is replaced with crystalloid solution or hormonal effects (eg, adrenocorticotrophic hormone [ACTH], aldosterone, antidiuretic hormone [ADH]) and transcapillary refill occurs, anemia may not develop. Do not withhold transfusion in patients who have relatively normal hematocrit results (ie, >30%) but have evidence of clinical shock, serious injuries (eg, open-book pelvic fracture), or significant

- ongoing blood loss.
- Use platelet transfusions to treat patients with thrombocytopenia (ie, platelet count <50,000/mL) and ongoing hemorrhage.
- Bedside diagnostic testing with rapid hemoglobin or hematocrit machines quickly may identify patients who have significant volume deficits and hemodilution. Reported hemoglobin from ABGs also may be useful in identifying anemia.
- Some studies have correlated a low initial hematocrit (ie, <30%) with significant injuries.
- Liver function studies
 - LFTs may be useful in the patient with blunt abdominal trauma; however, test findings may be elevated for several reasons (eg, alcohol abuse).
 - One study has shown that an aspartate aminotransferase (AST) or alanine aminotransferase (ALT) level more than 130 U corresponds with significant hepatic injury.
 - Lactate dehydrogenase (LDH) and bilirubin are not specific indicators of hepatic trauma.
- Urinalysis
 - Indications for diagnostic urinalysis include gross hematuria, microscopic hematuria in the setting of hypotension, and a significant deceleration mechanism.
 - Obtain a contrast nephrogram by utilizing IV pyelography (IVP) or CT scanning with IV contrast.
 - Gross hematuria indicates a workup that includes a cystogram and IVP or CT scanning of the abdomen with contrast.
 - Obtain serum or urine pregnancy test on all females of childbearing age.
- Blood type, screen, and crossmatch
 - Screen and type blood from all trauma patients with suspected blunt abdominal injury. If an injury is identified, this practice greatly reduces the time required for crossmatch.
 - Perform an initial crossmatch on a minimum of 4-6 units for those patients with clear evidence of abdominal injury and hemodynamic instability.
 - Until crossmatched blood is available, utilize O-negative or type-specific blood.
- Drug and alcohol screens
 - Perform drug and alcohol screens on trauma patients who have alterations in their level of consciousness.
 - Breath or blood testing may quantify alcohol level.

Imaging Studies

- Focused abdominal sonogram for trauma
 - Bedside ultrasound in the form of focused abdominal sonogram for trauma (FAST) has been used in the evaluation of trauma patients in Europe for more than 10 years. FAST's diagnostic accuracy generally is equal to that of diagnostic peritoneal lavage (DPL). Studies in the US over the last few years have demonstrated the value of bedside sonography as a noninvasive approach for rapid evaluation of hemoperitoneum. The studies demonstrate a degree of operator dependence; however, some studies have shown that with a structured learning session, even novice operators can identify free intraabdominal fluid.
 - In the patient with isolated blunt abdominal trauma and multisystem injuries, a bedside

ultrasound performed by an experienced sonographer rapidly can identify free intraperitoneal fluid. The sensitivity for solid organ encapsulated injury is moderate in most studies. Hollow viscus injury rarely is identified; however, free fluid may be visualized in these cases. For those patients with persistent pain or tenderness or for those developing peritoneal signs, consider FAST as a complementary measure to CT scan, DPL, or exploration.

- FAST evaluation of the abdomen consists of visualization of the pericardium (from a subxiphoid view), the splenorenal and the hepatorenal spaces (ie, Morison pouch), the paracolic gutters, and the pouch of Douglas in the pelvis. The Morison pouch view has been shown the most sensitive, regardless of the etiology of the fluid.
- Free fluid, generally assumed to be blood in the setting of abdominal trauma, appears as a black stripe. Free fluid in a hemodynamically unstable patient indicates exigent laparotomy; however, CT scan may further evaluate the stable patient with free fluid.
- Sensitivity and specificity of these studies range from 85-95%.

Procedures

- Diagnostic peritoneal lavage
 - DPL is used as a method of rapidly determining the presence of intraperitoneal blood. DPL is particularly useful if the history and abdominal examination of a patient who is unstable and has multisystem injuries is either unreliable (eg, head injury, alcohol, drug intoxication) or equivocal (eg, lower rib fractures, pelvic fractures, confounding clinical examination). DPL also is useful for those patients in whom serial abdominal examinations cannot be performed (eg, those in an angiographic suite or operating room during emergent orthopedic or neurosurgical procedures).
 - The preferred method involves an open or semiopen technique that is performed in an infraumbilical location. In pregnant patients or in those patients with particular risk for potential pelvic hematoma, perform the DPL superior to the umbilicus.
 - Following insertion of the catheter into the peritoneum, attempt to aspirate free intraperitoneal blood (at least 15-20 mL). Abdominal exploration always is indicated if approximately 10 mL of blood is aspirated upon insertion of the peritoneal catheter (grossly positive) in the unstable patient. If findings are negative, infuse 1 L of crystalloid solution (eg, lactated Ringer solution) into the peritoneum. Then, allow this fluid to drain by gravity, and ensure laboratory analysis is performed.
 - Presence of more than 100,000 RBC/mm³ or more than 500 WBC/mm³ is considered a positive finding.
 - Other results from DPL fluid that indicate the need for exploration include the presence of bile or abnormally high amylase (indicative of intestinal perforation), food fibers, or bacteria noted on microscopic examination.
 - In some contexts, DPL may be complemented with a CT scan if the patient has positive lavage results but stabilizes.
 - The only absolute contraindication for a DPL is the patient who will undergo laparotomy regardless of the findings.

- Complications of DPL include bleeding from the incision and catheter insertion, infection (ie, wound, peritoneal), and injury to intraabdominal structures (eg, urinary bladder, small bowel, uterus). These complications may increase the possibility of false-positive studies. Additionally, infection of the incision, peritonitis from the catheter placement, laceration of the urinary bladder, or injury to other intraabdominal organs can occur.
- Bleeding from the incision, dissection, or catheter insertion can cause false-positive results that may lead to unnecessary laparotomy. Achieve appropriate hemostasis prior to entering the peritoneum and placing the catheter.

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Treatment

Prehospital Care

- Focus prehospital care on rapidly evaluating life-threatening problems, initiating resuscitative measures, and initiating prompt transport to the closest appropriate hospital, which typically is a trauma center.
- With endotracheal intubation, secure airways of patients who are unable to maintain the airway and who have potential airway threats. Secure the airway in conjunction with in-line cervical immobilization. Support patients exhibiting compromised breathing respirations with artificial ventilation using a high fraction of inspired oxygen (FIO₂). Maintain oxygenation at more than 90-92% saturation.
- External hemorrhage rarely is associated with blunt abdominal trauma. If present, control the hemorrhage with direct pressure. Note any signs of inadequate systemic perfusion. Consider intraperitoneal hemorrhage whenever evidence of hemorrhagic shock is found in the absence of external hemorrhage. Initiate volume resuscitation with crystalloid solution; however, never delay patient transport while IV lines are inserted. En route, administer a fluid bolus of lactated Ringer or normal saline solution to patients with evidence of shock.
- Titrate IV fluid therapy to the patient's clinical response. As overaggressive volume resuscitation may lead to recurrent or increased hemorrhage, titrate IV fluids to a systolic blood pressure of 90-100 mm Hg. This practice should provide the mean blood pressure necessary to maintain perfusion of the vital organs.
- Acquire expeditious and complete spinal immobilization on patients with multisystem injuries and on patients with a mechanism of injury that has potential for spinal cord trauma. In the rural setting, the pneumatic antishock garment may have a role for treating shock resulting from a severe pelvic fracture. In addition, transport patients meeting physiologic or anatomic criteria to the closest trauma center. Promptly notify the destination hospital in order for that facility to activate its trauma team and prepare for the patient.

Emergency Department Care

- Perform a rapid primary survey to identify immediate life-threatening problems. Focus close attention on whether the patient can maintain the airway or if a potential threat is present. Secure airways by orotracheal intubation, which is performed with concurrent in-line manual immobilization of the cervical spine. If intubation is required, perform and record a brief neurological examination prior to neuromuscular blockade and intubation, if possible.
- Patients who display apnea or hypoventilation require respiratory support, as do those patients with tachypnea. Provide all patients with supplemental oxygen from a device capable of delivering a high FIO₂ (eg, nonrebreather mask). Decreased or absent breath sounds raise the possibility of hemothorax or pneumothorax; therefore, consider needle decompression or tube thoracostomy.
- Identification of hypovolemia and signs of shock necessitate vigorous resuscitation and attempts to identify the source of blood loss. Initiate at least 2 large-bore peripheral IV lines. Use central lines (preferably femoral) and cutdowns (eg, saphenous, brachial) for patients in whom percutaneous peripheral access cannot be established. Administer a rapid bolus of crystalloid.
- Perform physical examination that consists of a complete head-to-toe secondary survey, with attention paid to evidence of the mechanism of injury and potentially injured areas. Before the placement of a nasogastric tube and Foley catheter, perform appropriate head, neck, pelvic, perineum, and rectal examinations.
- Based on mechanism and physical examination, order initial trauma radiographic studies. In general, trauma suite views include a cervical spine, chest, and pelvis radiograph. In-line spinal immobilization must be continued until spinal fractures have been ruled out. Additional radiographs are indicated for other findings in the secondary survey.
- After the primary survey and initial resuscitation have begun, complete the secondary survey to identify all potential and present injuries. Log roll the patient, so an examination of the back and palpation of the entire spinal column can be performed. Investigate any signs of injury. Perform a rectal examination.
- If signs of shock persist after an initial 2-3 liters of crystalloid infusion, administer blood products. Type O Rh-negative blood typically is given to women of childbearing age. Type O-positive blood may be given safely to all other patients. As soon as available, use type-specific or crossmatched blood.
- A bedside ultrasound using a trauma examination protocol (eg, FAST) can be used to determine the presence of intraperitoneal hemorrhage (see [Pictures 1-2](#)). If findings are negative or equivocal, a DPL may be performed in unstable patients.
- Based on stability, mechanism, and suspicion of intraabdominal injury, further investigation may be warranted for patients who are hemodynamically stable after the initial assessment and resuscitation and who have negative or equivocal bedside ultrasound and/or DPL results. Further investigation includes contrast-enhanced CT scans of the abdomen and pelvis or serial examinations and ultrasound.

Consultations

- The best outcomes from trauma are obtained by involving consultants who possess specific expertise and/or training in managing trauma patients. Consider evaluation by a trauma surgeon for all patients with evidence of blunt abdominal trauma. Clearly, patients who have hemodynamic instability or significant abnormalities found during physical examinations and diagnostic procedures require involvement of a trauma surgeon.
- Specific physical examination findings indicate timely surgical evaluation as follows:
 - History of blunt abdominal trauma, shock, or abnormal vital signs (eg, tachycardia, hypotension)
 - Evidence of shock without obvious external blood loss
 - Evidence of peritonitis (eg, marked tenderness, involuntary guarding, percussion tenderness)
 - Findings consistent with potential intraabdominal injury (eg, lap belt signs, lower rib fractures, lumbar spine fractures)
 - Altered levels of consciousness or sensation, whether due to drugs, alcohol, or head/spinal injury
 - Patients requiring other prolonged operative intervention (eg, orthopedic procedures)

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Follow-up

Further Inpatient Care

- Serial examinations
 - Serial ultrasounds may play a role in identifying occult injuries.
 - Any change in the physical examination indicating peritoneal irritation warrants additional studies and/or laparotomy.

Further Outpatient Care

- Before discharge, provide patients with detailed instructions that describe signs of undiagnosed injury.
 - Increased abdominal pain, distention, nausea and/or vomiting, weakness or fainting, or new bleeding in urine or feces mandates immediate return and further evaluation.
 - Ensure close follow-up and repeat examinations are available for all patients.

in/Out Patient Meds

- Judiciously prescribe pain medications to patients who are discharged.

- To prevent masked or delayed presentations, ensure that a close follow-up for reevaluation is available to all patients who are provided pain medications.
- With the potential for hemorrhage, nonsteroidal anti-inflammatory drugs (NSAIDs) probably should be avoided.
- Acetaminophen with or without small quantities of mild narcotic analgesics may be all that should be prescribed initially.
- Minimize use of analgesics in patients who are admitted for observation.
- Patients with repaired hollow organ injury may require additional antibiotics.

Transfer

- If expertise in managing blunt abdominal injuries is unavailable, arrange patient transfer to the nearest appropriate trauma center as soon as injury is identified.
 - Lengthy diagnostic workup is counterproductive, once it is recognized that a patient cannot be managed at the initial facility.
 - Physician-to-physician consultation must occur before transport to ensure that the receiving facility has the resources necessary to care for the patient.

Complications

- Complications can arise for identified and unidentified injuries.
- Intraabdominal hemorrhage, infection, sepsis, and death can occur.
- Delayed rupture or hemorrhage from solid organs, particularly the spleen, has been described.
- In patients that undergo laparotomy and repair, complications are similar to other conditions requiring operative intervention.

Prognosis

- Overall prognosis for patients who sustain blunt abdominal trauma is favorable.
 - Without statistics indicating the number of out-of-hospital deaths and the total number of patients with blunt trauma to the abdomen, description of the specific prognosis for patients with intraabdominal injuries is difficult.
 - Mortality rates for hospitalized patients are approximately 5-10%.

Patient Education

- Proper adjustment of restraints in motor vehicles is an important aspect of patient education.
 - Wear lap belts in conjunction with shoulder restraints.
 - Snug and place lap belts across the lower abdomen and below the iliac crests.
 - Wear restraints even in vehicles equipped with supplemental vehicle restraints (eg, airbags).
 - Adjust seats and steering wheels to maximize the distance between the abdominal wall and

steering wheel, while still allowing proper control of the vehicle.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to suspect intraabdominal injury from appropriate mechanisms
 - Failure to evaluate abdominal/flank/costal margin pain after blunt abdominal injury
 - Failure to obtain timely surgical consultation and operative intervention
 - Failure to recognize intraabdominal hemorrhage and delay operation for additional diagnostic testing in the face of hemodynamic compromise
-

Pictures



Picture 1: Ultrasound image of right flank. A clear hypoechoic stripe exists between the right kidney and the liver in the Morison pouch.

Picture type: Photo



Picture 2: Ultrasound image of the left flank in the same patient, with a thin hypoechoic stripe above the spleen and a wider hypoechoic stripe in the splenorenal recess.

Picture type: Photo

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Abdominal Trauma, Penetrating

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Synonyms, Key Words, and Related Terms

gunshot wounds, GSWs, stab wounds, SW, impalement

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Introduction

Background

Reports of penetrating abdominal trauma appeared in literature as early as Homer's *Iliad*, in which King Menelaus was pierced by an arrow in his abdomen. Early penetrating trauma took the form of impalements upon wood or crude metal, while lacerations frequently stemmed from animal attacks sustained during hunting and, later, injuries from crude wood- or metal-based implements.

As technology advanced through the metal ages, weaponry designed for hunting was used for local defense and, eventually, conquest. Greek literature brims with descriptions of penetrating abdominal trauma as an ingenious and effective method of penance and torture. Prometheus, for example, lay bound upon a rock, while a fierce eagle devoured his liver as a penalty for presenting humans with fire. True to

form, Prometheus's liver regenerated, albeit in accelerated fashion, to meet each morning whole again.

As humans became more technologically advanced, the incessant desire for land, riches, and power spurred development of increasingly lethal weaponry. This quest has culminated with the weapons of mass destruction that ring the globe today.

Throughout history, humans created easily concealed personal weaponry designed initially for self-defense. Currently, personal weapons too often are used as a means to steal, murder, and create mayhem. The ubiquity of personal handguns, spring-loaded stiletto blades, and similar weaponry has created an epidemic of violence that is spreading rapidly into all walks of life, affecting people of all ages.

This situation has created intense pressure to establish regional resource trauma facilities to coordinate focused high-quality care of trauma patients. The success of these programs is evident in the internationally recognized care and research endeavors of centers such as the [R Adams Cowley Shock Trauma Center](#) in Baltimore, Md.

Pathophysiology

Penetrating injuries or stab wounds (SWs) to the abdomen are caused by a wide variety of instruments, including but not limited to knives, high- and low-velocity projectiles, ice picks, and industrial implements. Each class of instrument is associated with a different injury pattern. The most commonly injured organs associated with penetrating injuries are the small intestine (29%), liver (28%), and colon (23%).

In general, SWs cause injury in the direct path of the offending implement, although only 33% of SWs to the abdomen penetrate the peritoneal envelope. Only 50% of peritoneal violations from SWs require surgical intervention. Anatomic site, number of wounds, type and size of weapon, and the wound angle are important considerations. Determination of the likely implement path and whether the peritoneum was violated is crucial.

Tissue is lacerated by the passage of the wounding implement. If the structure is a vein, surrounding tissue may tamponade bleeding. Partially transected arterial walls probably will continue to bleed as the elastic tissue of the media contracts and further widens the wound. Completely divided arteries may contract enough to arrest hemorrhage. Intraperitoneal blood can induce severe local irritation and pain, which usually is accompanied by a sympathetic discharge resulting in tachycardia. Intraperitoneal blood also can induce a seemingly paradoxical vagal response and an associated bradycardia by unclear mechanisms when the blood loss is small. A large-volume hemorrhage may present with bradycardia as a preterminal event, signifying a severe reduction in coronary perfusion pressure and sinoatrial and atrioventricular node ischemia.

Lacerated hollow or solid organs result in hemorrhage and leakage of contained fluids (eg, succus entericus, bile) into the peritoneal and/or retroperitoneal space. Irritated nerve endings at the skin and

facial levels result in local wound pain. The peritoneal or retroperitoneal blood and/or organ contents inflame deeper nerve endings (visceral afferent pain fibers) and result in poorly defined and localized somatic pain. The back or shoulder distribution of pain may provide an excellent clue to the damaged organ (eg, shoulder pain from a damaged spleen with subphrenic blood).

Peritoneal signs develop when the peritoneal envelope and the posterior aspect of the anterior abdominal wall are both inflamed. Impaling objects may tamponade otherwise uncontrolled hemorrhage if the object resides within or crosses a major vessel or solid organ such as the portal vein or liver. Therefore, penetrative objects should not be removed except within an operating room (OR).

Gunshot wounds (GSWs) have a much broader injury pattern due to several mechanisms. First, any structure directly in the path of the missile suffers a loss of integrity. Approximately 85% of abdominal wall GSWs penetrate the abdominal cavity, and 95% require a surgical procedure for correction. Injury also is caused by fragmentation of the bullet on impact.

The transfer of kinetic energy from the missile to surrounding tissue as it traverses its path creates injury in several unique fashions. Kinetic energy is directly proportional to the mass and the square of the missile velocity; impact velocity directly relates to the wounding potential of a projectile. Missiles are categorized by their velocity profile into low-, medium-, and high-velocity projectiles. The impact speed is affected by several factors, including target distance, missile velocity, missile mass, missile shape, and drag. A missile creates longitudinal and horizontal shock and shear waves as it traverses different media. These, in turn, lead to cavitation of surrounding structures. The temporary cavity formed can contuse and lacerate tissues, fracture vascular intima, and rupture large vascular conduits. Dependent upon the energy imparted by the missile, these injuries may extend a significant distance from the bullet track.

The borders of the abdomen are circumscribed anteriorly from the costal margins to the groin and laterally to the anterior axillary lines. The flank is that region extending from the tip of the scapula to the iliac crest vertically and as far anteriorly as the anterior axillary line. Other authors limit the flank to that region between the anterior and posterior axillary lines. The back has the same superior/inferior border as the flank and extends between the posterior axillary lines.

Remember that peritoneal injury can result from penetrating wounds to the low chest and back, as the diaphragm ascends during expiration to the level of the fourth intercostal space anteriorly and sixth to seventh posteriorly. Knowledge of the anatomic boundaries of the abdomen is crucial to discussion of injury paradigms such as loss of abdominal wall integrity, hollow viscus injury, vascular injury, solid organ injury, and retroperitoneal injury.

Regardless of the mode of injury, absolute indications for celiotomy are virtually identical: hemodynamic instability, major vascular injury (including devascularized solid organs), evisceration, peritoneal signs, pneumoperitoneum, evidence of diaphragmatic injury, neurologic injury with cord compromise, significant intraperitoneal blood (eg, positive diagnostic peritoneal lavage [DPL] findings), and evidence of hollow viscus perforation.

Remember that intraperitoneal structures may demonstrate a different presentation complex compared to retroperitoneal or extraperitoneal structures. All structures are intraperitoneal except the bladder, ureters, kidneys, ascending and descending colon, rectum, pancreas, duodenum, aorta, iliac arteries and veins, and the vena cava.

Retroperitoneal injury may not present with classic peritoneal findings since an intact peritoneal envelope (as in a flank injury) contains the retroperitoneal extravasation of blood or organ contents. This is not to suggest that patients do not demonstrate pain on deep palpation—they surely do. Patients, however, may have a large left colonic rent from a flank SW but only mild anterior abdominal findings. Therefore, visualize flank and retroperitoneal injury complexes with a study modality that specifically targets structures in retroperitoneal and extraperitoneal spaces.

Frequency

- **In the US:** Tracking trauma is the purview of the [National Center for Injury Prevention and Control \(NCICP\)](#). Data collected by this organization suggest that traumatic injury is the third overall leading cause of death and the number one cause of death in those aged 1-44 years. Penetrating abdominal trauma affects approximately 35% of those patients admitted to urban trauma centers and 1-12% of those admitted to suburban or rural centers.
- The mechanism that underlies the penetrating trauma (eg, GSW, SW, impalement) relates to the mode of injury (eg, accidental or intentional injury, homicide or suicide). Homicide or intentional injury clearly is the predominant mode of injury in this patient population. Accidental injury is most common in pediatric home firearm injury but is uncommon by comparison with homicide and intentional injury. Suicide via penetrating abdominal trauma is distinctly uncommon.
- **Internationally:** The frequency of penetrating abdominal injury across the globe relates to the industrialization of developing nations and, significantly, to the presence of military conflicts. Therefore, frequency varies. The Global Burden of Disease Study, sponsored by the World Health Organization (WHO), identified injury as responsible for 10.1% of the global deaths in 1990 and listed injury as a consistent health problem throughout all parts of the world. Also, injury was responsible for 15.2% of the disability-adjusted life years lost in 1990. The WHO study projected a steady increase in violent deaths by the year 2020. At that time, the prevalence of violent deaths will equal that of communicable and infectious diseases (the second leading cause of death).

Mortality/Morbidity

- The death rate from penetrating abdominal trauma spans the entire spectrum (0-100%), depending on the extent of injury. Patients with violation of anterior abdominal wall fascia without peritoneal injury have a 0% mortality and morbidity rate, while those with multiorgan injury complexes presenting with hypotension, base deficit less than -15 mEq/L HCO_3^- , lactate more than 20 mmol/L, and near exsanguination have a nearly 100% mortality rate.
- An average mortality rate for all patients with penetrating abdominal trauma is approximately 5%

in most level 1 trauma centers, but this population is necessarily preselected, thus skewing the data.

- Abundant documentation reveals that the most common morbidities following penetrating abdominal trauma are wound infection (2-8%) and intra-abdominal abscess with or without sepsis (10-80%, depending on presence or absence of bowel injury in combination with major vascular injury).

Race

- Race distribution in patients with penetrating abdominal trauma depends significantly on the location of the receiving hospital. Urban centers predominantly receive young African American and Hispanic males more frequently than young white males. A similar distribution occurs for their female counterparts.
- Although it is difficult to quantify the death rate for penetrating abdominal trauma by race, the relative risk of death for penetrating injury in general is known. African American males have a 3-fold increase in relative risk of death compared to their white male counterparts. African American females have a 2.5-fold increase in relative risk of death compared to their white female counterparts. Suburban centers, however, tend to receive a greater proportion of youthful to middle-aged white males as their predominant patient population because of regional demographics.
- Obviously, persons from all racial and ethnic backgrounds may be affected by the rising incidence of penetrating trauma. The particular patient mix treated at a given center relates to the surrounding demographics rather than any unique characteristic(s) ascribed to a particular patient population.

Sex

- Sex as a predictor of injury and death is virtually inseparable from a discussion of race and is included in the above discussion.
- Males comprise the majority of patients with penetrating trauma injuries across the US and the world.

Age

- Trauma is the leading cause of death in patients aged 1-44 years.
- Incidence of elderly patients is on the rise as the population ages and the incidence of violent crime rises. Older people are increasingly visible and are presumably easy targets for a multitude of reasons. The impact of elderly trauma patients on resource use and the attention that must be paid to comorbid illnesses cannot be overstated. Age-adjusted elderly (>75 y) death rates by urbanization strata indicate approximately 20 cases per 100,000 deaths, an increase of approximately 5 cases per 100,000 over an 11-year period.
- By comparison, the age-adjusted death rate for males aged 15-34 years is approximately 40 cases

per 100,000 deaths, a rate that has remained relatively stable except in males aged 15-19 years. This subgroup has increased its death rate from 15-18% to its current level over an 11-year period.

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Clinical

History

The history surrounding a patient with penetrating trauma is important, as it provides clues to the likely injury complex and to potential therapeutic priorities. Much of this information may be obtained by seeking an AMPLE history as described by Freark and Baker at the Cook County Hospital in Chicago:

- Allergies
- Medications
- Prior illnesses and operations
- Last meal
- Events and environment surrounding injury
- Clues to the likely injury complex are gleaned from the injury location and from determination of the associated weapon (eg, gun, knife) or injury-causing object. The number of gunshots heard, times stabbed, and position of the patient at the time of injury help describe the trajectory and path of the injuring object.
- Range also is important when assessing GSWs. Close-range injuries transfer more kinetic energy than those sustained at a distance.
- A careful history that assesses secondary and multi-cavitary injuries is vital, as many victims sustain a blunt assault or fall from various heights after sustaining a penetrating trauma.
- Blood loss at the scene should be quantified as accurately as possible to help determine transfusion needs. The character of the bleeding (eg, arterial pumping, venous flow) helps determine whether major vascular injury has occurred.
- The initial level of consciousness or, for moribund patients, the presence of any signs of life at the scene (eg, pupillary response, respiratory efforts, heart rate or tones) is vital to determine the prognosis and to guide resuscitative efforts. Particularly important is the patient's response to therapy en route to the ED.
- Ongoing hypotension from acute intravascular volume loss despite 2 liters of IV fluids suggests blood component therapy in addition to IV fluids.
- Transport time and mode (ie, private vs emergency medical service [EMS]) affects survival. Patients arriving by private vehicle have a survival advantage due to shorter transport time.

Physical

- **Initial examination**
 - The initial physical examination begins with visual assessment of the patient during transport into the ED. Rapid determinations regarding perfusion, external hemorrhage, and consciousness level are made easily.
 - Once the patient is transferred to the ED stretcher, proceed with a proper physical examination as the primary survey of trauma resuscitation, with attention directed to the ABCs.
 - Once the primary survey is complete, perform the complete head-to-toe physical examination as an integral part of the secondary survey, including digital rectal and genital examinations. This detailed examination may need to be delayed until after operative therapy has corrected obvious life-threatening injury.
 - Initial vital signs may be used as a discriminator of injury severity. The author successfully has used vital sign abnormality or the need for acute airway control as a trigger to mobilize the complete trauma team. Mechanism of injury alone engenders a more limited team response. This stratified response is driven by the ED staff evaluation of trauma victims upon arrival, instead of reliance on field evaluation. Stratified response has achieved substantial reductions in trauma care costs and has reduced resource use without increases in complications or missed injuries.

Causes

- **Urban violence**
 - Urban violence is increasing steadily across the nation with a few notable exceptions such as the city of New York. The factors responsible for promulgating urban violence include social, economic, and environmental variables.
 - Social and environmental factors include such diverse elements as overcrowded living quarters and population density. Limited jail capacity and inability to incarcerate all convicted criminals reduces the penalty for violent actions and allows a larger criminal element to roam free. Local and national gangs often promote violence as a means to improve personal status within the gang hierarchy.
 - The influence of illegal, drug-induced violent behavior cannot be overstated. This category includes anabolic steroid-induced behaviors and the well-publicized steroid rage.
 - It has been well documented that many urban youth and young adults lead lives as if already dead. This peculiar notion arises from the fact that average life expectancy of young males in ghetto areas of major inner cities is less than 30 years. This awareness of relatively imminent and expected mortality may lead to socially unacceptable behavior patterns, since little hope for the future exists and little penalty can be exacted. Incarceration often increases longevity, albeit in an undesirable social system.
- **Low income**
 - Low income combined with deficient educational systems provides a milieu that engenders dependency, anger, and violence as almost expected outcomes. Inner-city streets and shelters burgeon with young adults of employable age and physical condition who lack the

basic skills to gain secure employment.

- Moreover, the welfare system frequently provides a larger paycheck than that accrued from labor at minimum wage. Readily available money and an excess of time in a hostile environment spells disaster for urban peace.

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Differentials

Abdominal Trauma, Penetrating
Acute Respiratory Distress Syndrome
Alcohol and Substance Abuse Evaluation
Anemia, Acute
Blast Injuries
Lactic Acidosis
Mesenteric Ischemia
Metabolic Acidosis
Necrotizing Fasciitis
Pediatrics, Child Abuse
Pediatrics, Dehydration
Pregnancy, Trauma
Shock, Hemorrhagic
Shock, Hypovolemic
Shock, Septic
Spinal Cord Injuries
Ultrasonography, Abdominal

Other Problems to be Considered

Coagulopathy
Comorbid medical conditions
Concomitant closed head injury
Concomitant penetrating chest injury

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Workup

Lab Studies

- Patients with penetrating abdominal trauma requiring surgical and blood component therapy and patients with evidence of hypoperfusion on admission should have complete laboratory profiles to include the following:
 - Blood type and crossmatch
 - Complete blood count (CBC)
 - Electrolytes
 - BUN
 - Creatinine
 - Glucose
 - Prothrombin time (PT)/activated partial thromboplastin time (aPTT)
 - Arterial lactate
 - Arterial blood gas (ABG)
 - Calcium, magnesium, and phosphate
 - Urinalysis
 - Urine dip for blood
 - Serum and urine toxicology screen
- Stable patients
 - Laboratory evaluation may be streamlined to include type and screen, CBC, electrolytes, BUN, creatinine, glucose, and urine dip for blood. Toxicology screens may be reserved for at-risk patients (eg, driver in an MVC).
 - Additional studies may be obtained if the patient's condition deteriorates. Individual laboratory values may be obtained if the hospital facility does not perform batch testing as a cost-saving measure.

Imaging Studies

- The imaging needs of each patient differ, depending on hemodynamic stability and associated injuries.
- Obtain chest x-ray (CXR) for all patients with penetrating abdominal trauma to ascertain presence of thoracic injury and to help place intrathoracic devices (eg, endotracheal tube).
- Obtain an abdominal flat-plate film in stable patients to seek retained foreign bodies, fractures, or abnormal shadows (eg, absent psoas, flank stripes). If fascial penetration is confirmed or suspected, these patients also may require additional imaging tests to identify the extent of injury.
- Explore virtually all GSWs to the abdomen proper (not flank). GSWs to the flank with tangential trajectories and SWs anywhere in a hemodynamically stable patient may be observed selectively. The observation population undergoes additional imaging.
- Additional imaging may take the form of oblique views to confirm an extraperitoneal location of a

missile, noncontrast CT scan to confirm an extraperitoneal path, or triple-phase (ie, PO, IV, PR contrast) CT scan of the abdomen and pelvis to determine the injury complex, with attention focused on the retroperitoneal colon and/or rectum.

- Many of these patients now undergo concomitant abdominal ultrasound to detect free fluid as a focused abdominal sonography for trauma (FAST) administered by emergency medicine or trauma surgery staff. Ultrasound does not indicate the source of the fluid or its character (eg, blood, urine, intestinal contents); it merely identifies the fluid's presence.
- CT scan may be needed to identify the source of the fluid and to determine the Hounsfield units, which indicate the nature of the fluid based on electron density.
- Future modalities
 - Open MRI with real-time images and continuous patient access and treatment is on the horizon.
 - MRI clearly is not an option for patients with bullets or bullet fragments within their body cavities due to the strong magnetic fields required for such imaging.
 - All of the modalities of MRI data acquisition (eg, MR angiography, venography, cholangiography) are unavailable to patients with retained metallic fragments. Portable CT scanning in the ED is available.
 - Gasless exploratory laparoscopy with a 2-mm umbilical laparoscope has been described and is under trial in a number of centers across the US for the evaluation and treatment of intra-abdominal injuries.
 - The major drawbacks of ED laparoscopy include the need for a cooperative patient or intubation and general anesthesia.

Other Tests

- Almost all other tests relate to (1) a skeletal survey for associated fractures, (2) a CT scan of the brain for coincident head injuries, or (3) a retrograde urethrogram or cystogram in a stable patient who has a displaced prostate, symphysis pubis diastasis, or blood at the urethral meatus.
 - Unstable patients with these findings usually undergo exploration if they have injuries other than an isolated pelvic fracture and receive an on-table cystourethrogram or a suprapubic bladder catheter and subsequent antegrade or retrograde studies.
 - Nuclear medicine studies have no role in the acutely injured abdominal trauma patient.

Procedures

- Local wound exploration
 - If it is unclear that a laceration is superficial, local wound exploration may be performed using anesthesia similar to that for wound repair (see [Emergency Department Care](#)). This exploration is a sterile procedure that requires gowns, hats, masks, gloves, and skin preparation as would a standard operation. Excellent lighting is essential; the operator may require a headlamp. This procedure is most useful for lacerations overlying the rectus abdominus musculature because the bundles are oriented along the long axis of the body,

making wound tracking fairly straightforward. Flank lacerations, however, are problematic due to the multiple muscle bundles that are oriented at differing angles and planes. For these injuries, wound tracking is virtually impossible, and local exploration is not recommended.

- Once the area is prepared with a povidone-iodine solution (or parachlorometaxyxylenol if the patient is iodine sensitive or allergic), draped, and anesthetized, the wound may be widened with gentle retraction and gently probed with a hemostat to determine if a tract exists. If the wound is small, extending it to aid visualization is accomplished with a No. 10 blade scalpel. The rectus fibers may be separated by spreading in their direction using a hemostat or Kelly clamp. The posterior rectus sheath is easily identifiable as a white layer directly underlying the rectus musculature. If yellow fat is identified, preperitoneal fat may have herniated through a posterior fascial rent. Alternatively, especially in thin patients with limited adipose tissue, the fat may be the omental drape protruding through a rent in the posterior rectus sheath. Either case establishes fascial violation, which requires formal surgical exploration.
- Proctosigmoidoscopy
 - This procedure is indicated for evaluation of suspected rectal or sigmoid injury. The patient is placed in the left lateral decubitus position in a knee-chest manner (provided the spine is clear). If the spine is not clear, a flexible colonoscope may be used with the patient remaining supine, although this is more difficult. A digital rectal examination is performed as the initial maneuver; the sigmoidoscope is then introduced into the anal canal and directed toward the patient's umbilicus. The anorectal lumen is insufflated with air as needed to guide the scope within the center of the lumen at all times.
 - Frequent swabbing and suctioning to clear stool is usually necessary, since this is an unprepared examination. Typically, sedation is not required for this procedure. Identification of rectal or low sigmoid colon full-thickness rents, intramural hematoma, or luminal arterial blood loss requires prompt surgical exploration.

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Treatment

Prehospital Care

- One of the main goals of prehospital care is rapid transport to the closest appropriate facility following expeditious evaluation of the ABCs. This concept is known as "scoop and run." This is in contrast to patients with primarily cardiac illness who obtain benefit from a more detailed prehospital care regimen, known as "stay and play."
- Along with assessing ABCs, place the patient on high-flow oxygen by face mask. Controversy continues concerning the extent of appropriate fluid resuscitation in the hypotensive penetrating

trauma patient as described initially by Ken Mattox at Baylor. This controversy stems from the observation that rapid fluid resuscitation with a subsequent increase in systolic and mean blood pressures can promote bleeding from uncontrolled major vascular injuries. Therefore, some centers have adopted a policy of resuscitation to a systolic blood pressure (SBP) of 70 mm Hg for the pre-OR phase of care. Other centers transport their unstable trauma patients directly to the OR and pursue no fluid resuscitation prior to exploration.

- No consensus has been reached as to the most beneficial strategy in terms of survival advantage. More likely, an individualized strategy focusing on patients whose injury complex and mechanism describe a vascular injury is most beneficial.
- Maintenance of circulation and external hemorrhage control are essential parts of prehospital treatment. For obvious bleeding, direct pressure must be applied to prevent further blood loss, worsening hemodynamic instability and exsanguination.
- Current basic trauma life support (BTLS) indications for the use of medical antishock trousers (MASTs) or pneumatic antishock garments (PASGs) do not include penetrating injuries of the torso.
- Prehospital personnel must be well trained in properly assessing patients and in transporting them to the closest appropriate facility or trauma center. The receiving hospital should be notified as soon as possible by radio or ground line to give the ED time to prepare and to alert the appropriate staff. The mode of transportation also must be considered. In an urban setting with multiple appropriate facilities and few areas to land a helicopter, the fastest and most appropriate transport is probably by ground. In the rural setting, where the closest facility (appropriate or not) is 25 minutes or more away, trauma patients' only chance for survival may be transport by air ambulance.

Emergency Department Care

- Resuscitation of trauma patients with penetrating abdominal injury is based on advanced trauma life support (ATLS) standards regardless of the type of receiving center. The manner in which the assessment and treatment are implemented depends on resources. As with all trauma patients, the team leader (resuscitation physician) rapidly assesses ABCDEs as follows:
 - A - Airway with cervical spine (C-spine) control as needed
 - B - Breathing
 - C - Circulation with hemorrhage control
 - D - Disability (pupils and AVPU - alert, voice responsive, pain responsive, unresponsive)
 - E - Exposure/environment control
- General wound repair
 - Procedures performed in the ED for victims with penetrating abdominal trauma are aimed at repairing superficial wounds and at identifying fascial violation, intraperitoneal fluid, and colorectal injury as well as arresting hemorrhage and ensuring cardiopulmonary cerebral resuscitation.
 - Superficial lacerations that do not result in peritoneal violation may be anesthetized, irrigated with a high-pressure irrigation device, debrided, and primarily repaired if the wound is evaluated and treated within 6 hours of injury.

- A 50:50 mixture of 1% lidocaine and 0.25% bupivacaine with 1:100,000 epinephrine solution is excellent for local anesthesia, as it combines a rapidly acting agent with a long-acting substance. The addition of sodium bicarbonate to the anesthetic mixture raises the pH, reduces local pain on injection, and enhances anesthetic activity. In addition, large volumes may be used to cover multiple areas or a single large laceration with minimal risk of toxicity.
- Saphenous vein cutdown
 - The saphenous vein may be approached at the groin or ankle. The ankle is the easiest site for the novice. The vein runs anterior to the medial malleolus. A transverse incision just through the skin over the expected vein course is followed with a curved hemostat anterior to the vein and scraped off the periosteum of the medial malleolus. The vein should be above the hemostat at this point. A 3-0 tie is placed distally on the vein for traction. Either a 14 or 16G catheter over the needle assembly may be introduced through the skin and into the vein or directly inserted into the vein. The catheter is advanced and the needle withdrawn. The catheter is secured to the vein and the skin with separate sutures.
 - The skin is closed with a 3-0 nylon suture or skin clips. A normal saphenous vein can accommodate a 7.5 French sheath introducer placed in a Seldinger fashion (ie, insert the guidewire through a transverse venotomy with a No. 11 blade scalpel and the sheath-dilator over the guidewire). IV macro-tubing may be inserted directly through a transverse venotomy (instead of a sheath or catheter over the needle assembly).
 - A groin saphenous vein cutdown is performed easily by externally rotating the lower extremity and placing a slightly oblique incision from the midinguinal point (the anatomic landmark for the common femoral artery) to the medial thigh. Once the incision punctures the skin and superficial adipose tissue, the operator's fingers grasp each side of the wound and pull in opposite directions (ie, one hand pulls toward the head and the other pulls toward the feet). The only structure in the depths of this wound is the greater saphenous vein. The vein may be cannulated in a fashion identical to that at the ankle. A sheath is placed at this level because the vein usually is generous as it approaches the fossa ovalis to join the common femoral vein.
 - Peripheral cutdowns of the saphenous vein are preferable to central lines for the same reason. The choice of site for a saphenous vein cutdown is institution and operator dependent. Remember that a groin cutdown requires a large incision to allow for rapid identification of the saphenous vein just proximal to the saphenofemoral junction. Internal jugular and subclavian vessels are not used routinely during initial treatment and evaluation because they obstruct airway control and C-spine protection efforts.
 - Moreover, iatrogenic injury, such as the development of a visceral pleural laceration and a subsequent simple pneumothorax that progresses to a tension pneumothorax, is disastrous in a critically ill patient. In pediatric patients (<6 y), an intraosseous line may be the easiest and fastest way to obtain vascular access.
- The trauma/surgical specialist should be notified as soon as possible. Depending on the facility, the trauma consultant may be a general surgical intern (R-1) or a full trauma team led by an attending surgeon with American Board of Surgery credentials. The surgeons must be involved at the beginning of the case because they are the only practitioners who can offer definitive therapy.

Additional consultants should be involved as the patient's injury complex dictates. Frequently consulted services include orthopedic surgery and neurosurgery.

- Medications aimed at pain control, airway control, sedation, tetanus prophylaxis, and antimicrobial coverage are covered in detail in a later section.
- Patient disposition relates to the type of facility and to the mechanism of and potential for injury. The most common post-ED disposition for patients with penetrating abdominal trauma is to the OR. Any patient with an obvious reason for laparotomy (eg, evisceration, rigid abdomen, hypotension) should be taken directly to the OR following initial evaluation and therapy in the ED. Some facilities do not have a surgical or OR team available 24 hours a day. In this case, these patients must be transferred to an appropriate facility. Similar concerns occur if the patient's injury complex overwhelms the available resources at the receiving facility. Decisions as to the mode of transportation, as detailed above, must reflect the transport time, patient's injury complex, potential for untoward events during transport, and available personnel to accompany the patient during transport. In many cases, air ambulance transport couples a skilled transport team with the most rapid transport available.

Consultations

- As indicated above, the initial trauma consultant may be a general or trauma surgeon. Depending on the type of center, the trauma surgeon may obviate all other consultation except an orthopedic surgeon and a neurosurgeon.
- At other centers, consultants may be involved as individual injuries are identified. In this scheme, a vascular surgeon repairs major arterial and venous injuries, a urologist addresses injuries to the bladder, kidneys, and ureters, while the trauma surgeon coordinates the patient's care and repairs injured intra-abdominal viscera.

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Medication

In general, medications used to treat victims of penetrating abdominal trauma fall into discrete categories. Analgesics, anxiolytics, antimicrobials (skin and enteric flora), immune boosters (tetanus booster), and neuromuscular blockers comprise the major classes of pharmacotherapeutic agents used for these patients.

Analgesics

Pain control is essential to quality patient care, and it ensures patient comfort, promotes pulmonary toilet, and enables physical therapy regimens. Most analgesics have sedating properties, which are beneficial for

patients who have sustained traumatic injuries.

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| Drug Name | Morphine sulfate (MS Contin, Oramorph SR, Duramorph)- DOC for analgesia due to reliable and predictable effects, safety profile, and ease of reversibility with naloxone. Various IV doses are used; commonly titrated until desired effect is obtained. |
| Adult Dose | Initial dose: 0.1 mg/kg IV/IM Maintenance dose: 5-20 mg/70 kg IV/IM q4h Relatively hypovolemic patients: Start with 2 mg IV/IM/; reassess the hemodynamic effects of the dose |
| Pediatric Dose | Neonates: 0.05-0.2 mg/kg/dose IV prn Children: 0.1-0.2 mg/kg/dose IV/IM q2-4h prn |
| Contraindications | Documented hypersensitivity; hypotension; respiratory depression; potentially compromised airway in which establishing rapid airway control would be difficult; nausea; emesis; constipation; urinary retention |
| Interactions | Phenothiazines may antagonize analgesic effects of opiate agonists; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

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| Drug Name | Fentanyl citrate (Duragesic, Sublimaze)- Potent narcotic analgesic with much shorter half-life than morphine sulfate. DOC for conscious sedation analgesia. Ideal for analgesic action of short duration during anesthesia and immediate postoperative period. Excellent choice for pain management and sedation with short duration (30-60 min) and easy to titrate. Easily and quickly reversed by naloxone. After initial dose, subsequent doses should not be titrated more frequently than q3h or q6h thereafter. Most patients are controlled with 72-h dosing intervals when using transdermal dosage form, although some patients require 48-h dosing intervals. |
| Adult Dose | 0.5-1 mcg/kg/dose IV/IM q30-60min Alternatively, apply a 25 mcg/h transdermal system q48-72h |
| Pediatric Dose | <2 years: 2-3 mcg/kg/dose IV/IM q30-60min 2-12 years: 1-2 mcg/kg/dose qh >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which it would be difficult to establish rapid airway control |

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| Interactions | Phenothiazines may antagonize analgesic effects of opiate agonists; tricyclic antidepressants may potentiate adverse effects of fentanyl when both drugs are used concurrently |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hypotension, respiratory depression, constipation, nausea, emesis, and urinary retention; idiosyncratic reaction, known as chest wall rigidity syndrome, may require neuromuscular blockade to increase ventilation |

Anxiolytics

Patients with painful injuries usually experience significant anxiety. Anxiolytics allow the clinician to administer a smaller analgesic dose to achieve the same effect.

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| Drug Name | Lorazepam (Ativan)- Sedative hypnotic with short onset of effects and relatively long half-life. By increasing the action of GABA, a major inhibitory neurotransmitter in the brain, may depress all levels of CNS, including limbic and reticular formation. Excellent choice when patient must be sedated for longer than 24 h. |
| Adult Dose | Initial dose: 2 mg IV total or 0.044 mg/kg IV, whichever is smaller For greater lack of recall: 0.05 mg/kg IV q4-8h; not to exceed 4 mg/dose |
| Pediatric Dose | 0.05-0.1 mg/kg IV slowly over 2-5 min; may repeat a dose of 0.5 mg/kg IV slowly |
| Contraindications | Documented hypersensitivity; preexisting CNS depression; hypotension; narrow-angle glaucoma |
| Interactions | Toxicity of benzodiazepines in CNS increases when used concurrently with alcohol, phenothiazines, barbiturates, and MAOIs |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in renal or hepatic impairment, myasthenia gravis, organic brain syndrome, or Parkinson disease |

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| Drug Name | Midazolam hydrochloride (Versed)- Shorter-acting benzodiazepine sedative-hypnotic useful in patients requiring immediate and/or short-term sedation. Also useful for its amnestic effects. |
| Adult Dose | Conscious sedation: Loading dose: 0.05-0.2 mg IV over 2 min Maintenance dose: Infuse 1-2 mcg/kg/min IV titrated to the desired effect Dosing range: 0.4-6 mcg/kg/min Alternatively, 0.07-0.08 mg/kg IM Average total dose is 5 mg administered up to 1 h before surgery |

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| Pediatric Dose | Sedation, anxiolysis, or amnesia: 0.1-0.15 mg/kg IV over 2 to 3 min; may use up to 0.5 mg/kg in very anxious patients Intranasal route may be used for pediatric sedation (to age 2 y); doses are 1-2 mg and are limited by volume delivered |
| Contraindications | Documented hypersensitivity; preexisting hypotension; narrow-angle glaucoma; sensitivity to propylene glycol (the diluent) |
| Interactions | Sedative effects may be antagonized by theophyllines; narcotics and erythromycin may accentuate sedative effects due to decreased clearance |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in congestive heart failure, pulmonary disease, renal impairment, and hepatic failure |

Antibiotics

Penetrating abdominal trauma that requires operative therapy usually entails a hollow or solid viscus or vascular injury. Perioperative antimicrobial coverage directed against skin and enteric flora is indicated to decrease the incidence of postoperative wound infection and intra-abdominal sepsis.

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| Drug Name | Cefazolin sodium (Ancef, Zolicef)- First-generation semisynthetic cephalosporin that arrests bacterial cell wall synthesis, thus inhibiting bacterial growth. Primarily active against skin flora, including <i>Staphylococcus aureus</i> . Typically used alone for skin and skin-structure coverage. IV and IM dosing regimens are similar. |
| Adult Dose | 250 mg to 2 g IV/IM q6-12h depending on severity of infection; not to exceed 12 g/d |
| Pediatric Dose | 25-100 mg/kg/d IV/IM divided q6-8h depending on severity of infection; not to exceed 6 g/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid prolongs effect; coadministration with aminoglycosides may increase renal toxicity; may yield false-positive urine dip test for glucose |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal impairment; superinfections and promotion of nonsusceptible organisms may occur with prolonged use or repeated therapy |

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| Drug Name | Metronidazole hydrochloride (Flagyl)- Imidazole ring-based antibiotic active against various anaerobic bacteria and protozoa. Used in combination with other antimicrobial agents (except for <i>Clostridium difficile</i> enterocolitis). |
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| Adult Dose | Loading dose: Infuse 15 mg/kg IV over 1 h or 1 g for a 70-kg adult Maintenance dose: 6 h following the loading dose infuse 7.5 mg/kg IV over 1 h q6-8h or 500 mg for a 70-kg adult; not to exceed 4 g/d |
| Pediatric Dose | Administer as in adults (using body weight) 15-30 mg/kg/d IV divided bid/tid for 7 d or 40 mg/kg PO once; not to exceed 2 g/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Cimetidine may increase toxicity; may increase effects of anticoagulants; may increase toxicity of lithium and phenytoin; disulfiramlike reaction may occur with orally ingested ethanol |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in hepatic disease; monitor for seizures and development of peripheral neuropathy |

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| Drug Name | Gentamicin sulfate (Garamycin, Gentacidin)- Aminoglycoside antibiotic for gram-negative coverage. Used in combination with an agent against gram-positive organisms and one that covers anaerobes. Not DOC. Consider if penicillins or other less toxic drugs are contraindicated, when clinically indicated, and in mixed infections caused by susceptible staphylococci and gram-negative organisms. Dosing regimens are numerous; adjust dose based on CrCl and changes in volume of distribution. May be administered IV/IM. |
| Adult Dose | Serious infections and normal renal function: 3 mg/kg/d IV q8h Loading dose and maintenance dose: 1-2.5 mg/kg IV and 1-1.5 mg/kg IV q8h, respectively Extended dosing regimen for life-threatening infections: 5 mg/kg/d IV/IM q6-8h Follow each regimen by at least a trough level drawn on the third or fourth dose (0.5 h before dosing); may draw a peak level 0.5 h after 30-min infusion |
| Pediatric Dose | <5 years: 2.5 mg/kg/dose IV/IM q8h >5 years: 1.5-2.5 mg/kg/dose IV/IM q8h or 6-7.5 mg/kg/d IV divided q8h; not to exceed 300 mg/d; monitor as in adults |
| Contraindications | Documented hypersensitivity; non-hemodialysis-dependent renal insufficiency |
| Interactions | Coadministration with other aminoglycosides, cephalosporins, penicillins, and amphotericin B may increase nephrotoxicity; aminoglycosides enhance effects of neuromuscular blocking agents (prolonged respiratory depression may occur); coadministration with loop diuretics may increase auditory toxicity of aminoglycosides; possible irreversible hearing loss of varying degrees may occur (monitor regularly) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

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| Precautions | Narrow therapeutic index (not intended for long-term therapy); caution in renal failure (not on dialysis), myasthenia gravis, hypocalcemia, and conditions that depress neuromuscular transmission; adjust dose in renal impairment |
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| Drug Name | <p>Vancomycin hydrochloride (Vancocin, Lyphocin)- Potent antibiotic directed against gram-positive organisms and active against enterococcal species. Useful in the treatment of septicemia and skin-structure infections. Indicated for patients who cannot receive or who are unresponsive to penicillins and cephalosporins or who have infections with resistant staphylococci. For penetrating abdominal injuries, it is combined with an agent active against enteric flora and/or anaerobes.</p> <p>To avoid toxicity, current recommendation is to assay trough levels after third dose drawn 0.5 h prior to next dosing. Use CrCl to adjust dose in patients diagnosed with renal impairment.</p> <p>Used in conjunction with gentamicin for prophylaxis in penicillin-allergic patients undergoing GI or GU procedures.</p> |
| Adult Dose | 500 mg to 2 g/d IV divided tid/qid for 7-10 d |
| Pediatric Dose | 40 mg/kg/d IV divided tid/qid for 7-10 d |
| Contraindications | Documented hypersensitivity |
| Interactions | Erythema, histamine-like flushing, and anaphylactic reactions may occur when administered with anesthetic agents; taken concurrently with aminoglycosides, risk of nephrotoxicity may increase above that with aminoglycoside monotherapy; effects in neuromuscular blockade may be enhanced when coadministered with nondepolarizing muscle relaxants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in renal failure, neutropenia; "red man" syndrome (not an allergic reaction) is caused by too-rapid IV infusion (dose administered over a few minutes), but this rarely occurs when dose is administered over 2-h period or by PO or IP routes |

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| Drug Name | Ampicillin sodium-sulbactam sodium (Unasyn)- Drug combination of beta-lactamase inhibitor with ampicillin. Covers skin, enteric flora, and anaerobes. Not ideal for nosocomial pathogens. |
| Adult Dose | 1.5 (1 g ampicillin + 0.5 g sulbactam) to 3 g (2 g ampicillin + 1 g sulbactam) IV/IM q6-8h; not to exceed 4 g/d sulbactam or 8 g/d ampicillin |
| Pediatric Dose | <p>3 months to 12 years: 100-200 mg ampicillin/kg/d (150-300 mg Unasyn) IV divided q6h</p> <p>>12-years: Administer as in adults</p> |
| Contraindications | Documented hypersensitivity |

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| Interactions | Probenecid and disulfiram elevate ampicillin levels; allopurinol decreases ampicillin effects and has additive effects on ampicillin rash; may decrease effects of PO contraceptives |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal failure; evaluate rash and differentiate from hypersensitivity reaction |

Neuromuscular Blocking Agents

Many patients with penetrating abdominal trauma require urgent airway control. A working knowledge of paralytic agents is essential for the ED practitioner who initially evaluates and resuscitates victims of penetrating abdominal trauma.

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| Drug Name | Succinylcholine chloride (Anectine Chloride, Anectine Flo-Pack)- Prototypical depolarizing neuromuscular blocker that is ultra-short-acting and predictable in onset (<1 min) and duration (4-6 min). Highly ionized, relatively fat-insoluble; does not readily cross placenta. Pediatric patients must be pretreated with atropine to avoid bradycardia and cardiac arrest. May also occur in adults but more commonly is associated with administration of either a higher or a second dose. |
| Adult Dose | Intubation: 0.6 mg/kg IV Dose should be individualized and may range from 0.3-1.1 mg/kg |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; malignant hyperthermia; myopathies associated with elevated serum creatine phosphokinase values; narrow-angle glaucoma; hyperkalemia; malignant hyperthermia; penetrating eye injuries |
| Interactions | Concomitant administration with nondepolarizing muscle relaxants may enhance neuromuscular blocking action; activity is prolonged when concurrently administered with oxytocin, quinidine, beta-blockers, and procainamide |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in paraplegia, hyperkalemia, severe burns, and deficiencies in plasma cholinesterase; bolus administration in infants and children has been associated with malignant arrhythmias and hyperkalemic rhabdomyolysis |

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| Drug Name | Vecuronium bromide (Norcuron)- Prototypic, nondepolarizing, neuromuscular blocking agent that reliably results in muscular paralysis. For intubation and maintenance of paralysis, a continuous infusion may be used. Infants are more sensitive to neuromuscular blockade activity; recovery is prolonged by 50%, although the same dose is used. Not recommended for use in neonates. |
| Adult Dose | 0.08-0.1 mg/kg IV Dose may be reduced to 0.05 mg/kg if patient has been treated with succinylcholine Maintenance dose for paralysis: 0.025-0.1 mg/kg/h IV; can be titrated to desired train-of-four response (commonly 2 of 4 twitches) |
| Pediatric Dose | Neonates: Not recommended 7 weeks to 1 year: 0.08-0.1 mg/kg/dose followed by maintenance dose of 0.05-0.1 mg/kg q1h prn 1-10 years: May require higher initial dose and more frequent supplementation >10 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; myasthenia gravis or related syndromes |
| Interactions | Neuromuscular blockade is enhanced when used concurrently with inhalational anesthetics; renal or hepatic failure, as well as concomitant administration of steroids, may result in prolonged blockade despite withdrawal of the agent |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Small doses may have profound effects in myasthenia gravis or myasthenic syndrome |

Immune Enhancement

For penetrating abdominal trauma resulting in wounds contaminated with either dirt or debris or for wounds caused by metallic objects carrying a risk of *Clostridium tetani* infection. Tetanus results from elaboration of an exotoxin from *C tetani*. A booster injection in previously immunized individuals is recommended to prevent this potentially lethal syndrome. Patients who may not have been immunized against *C tetani* products (eg, immigrants, older African American women from the southern US) should receive tetanus immune globulin (Hyper-Tet).

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| Drug Name | Tetanus toxoid aluminum phosphate- Induces active immunity against tetanus in selected patients. The immunizing agents of choice for most adults and children >7 y are tetanus and diphtheria toxoids. Necessary to administer booster doses to maintain tetanus immunity throughout life. Pregnant patients should receive only tetanus toxoid, not a diphtheria antigen-containing product. In children and adults, may administer into deltoid or midlateral thigh muscles. In infants, preferred site of administration is the mid thigh laterally. |
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| Adult Dose | Primary immunization: 0.5 mL IM; administer 2 injections 4-8 wk apart and a third dose 6-12 mo after second injection Booster dose: 0.5 mL q10y |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; history of any type of neurological symptoms or signs following administration of this product; FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are an important cause of provocative poliomyelitis |
| Interactions | Due to poor immune response, patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent use of medication with systemic chloramphenicol, since it may impair amnestic response to tetanus toxoid; concurrent use of tetanus immune globulin may delay development of active immunity by several days (interaction is nevertheless clinically insignificant and does not preclude its concurrent use) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Do not use to treat actual tetanus infections or for immediate prophylaxis of unimmunized individuals (instead use tetanus antitoxin, preferably human tetanus immune globulin); diminished antibody response to active immunization may be seen in patients receiving immunosuppressive therapy; better to defer primary diphtheria immunization until immunosuppressive therapy is discontinued; routine immunization of symptomatic and asymptomatic persons infected with HIV is recommended |

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Follow-up

Further Inpatient Care

- In the ICU, assessment and management continues. Organization is usually by organ system, as follows:
 - Perfusion
 - Sepsis (especially abscess formation)
 - Lung injury
 - Renal and electrolytes (especially acute tubular necrosis)
 - GI function
 - Nutrition
 - Hematopoietics

- Analgesia
- Complication prevention (eg, deep vein thrombosis [DVT] and pulmonary embolism [PE], stress ulceration and bleeding, pressure ulceration, atelectasis, ventilator-associated pneumonia, catheter-related sepsis, ICU psychosis).
- Coordination of the activities of multiple consultants is a key task in ensuring smooth patient care.
- General ward care of this patient population focuses on wound care, fluid balance, nutrition, pain control, physical therapy, muscular reconditioning, social service issues, health insurance concerns (eg, enrollment in medical assistance programs), and rehabilitation needs.
- Disposition planning is essential for timely coordination of care and discharge. Coordinate follow-up appointments and establish an outpatient medication regimen. Initiate communication with the private medical doctors who will resume the patient's care.

Further Outpatient Care

- Outpatient priorities are distinctly different from those operative during the inpatient phase. Tracking the normal progression of wound healing and planning for abdominal wall reconstruction in patients with a planned ventral hernia (due to intra-abdominal hypertension [IAH]) are key elements of initial trauma care.
- Maintenance of normal GI function and nutritional status are also central concerns. Assessment of immune competence and vaccination in splenectomized patients is important.
- Reassess and remove tubes that were placed (eg, enteral access feeding catheters, long-term IV access lines) as indicated. Ostomies must be assessed for function, skin integrity, and planned reversal.
- Coordination of care with other consultants is a vital part of outpatient care. Return-to-work evaluations and ongoing communication with private medical physicians plays a key role in returning patients to work.

in/Out Patient Meds

- Analgesics, such as parenteral morphine, oral oxycodone with or without acetaminophen, acetaminophen alone, and ibuprofen, are essential.
 - Adequate pain control is vital to ensure adequate pulmonary toilet, which avoids atelectasis and subsequent pneumonia.
 - Pain relief also assists in early ambulation and aggressive physical therapy, which decreases muscular deconditioning and the risk of pressure ulceration.
- Employ subcutaneous heparin (unfractionated or low molecular weight) in combination with superficial venous compression stockings and sequential compression devices as standard therapy in patients at risk for DVT. Outpatient therapy is usually unnecessary.
 - High-risk patients include those who are immobilized or who have undergone emergency surgery or pelvic procedures and those who have intra-abdominal infection or inflammation.
 - Patients with a prior DVT or major venous injury are also at risk and deserve triple therapy

combined with early ambulation to decrease the risk for DVT and subsequent pulmonary embolus.

Transfer

- ATLS guidelines exist to help direct the transfer of trauma patients to a designated trauma facility; however, the mode of transportation is not specified.
- Patients who are potentially unstable and who require a long ground transport time (ie, >15-20 min) or a potentially problematic ground transport route (eg, across an inner city) may be better served by air ambulance transport. Airway control is always an important issue and must be addressed promptly.
- In general, transfer patients to a regional resource trauma facility if no OR or surgeon is available or if they have multiple or multi-cavitary injuries, requirements that cannot be provided at the initial receiving facility (eg, neurosurgery, cardiopulmonary bypass, post–solid-organ transplant), or injuries that would overwhelm the initial facility (eg, massive transfusion requirement).
- The issue of patient or family transfer requests is problematic if the patient is in a trauma facility that can meet the patient's needs. Practitioners must be cognizant of Emergency Medical Treatment and Active Labor Act (EMTALA)/Consolidated Omnibus Budget Reconciliation Act (COBRA) violations and document accordingly (see [COBRA Laws](#)). Furthermore, many facilities ask the patient's family to arrange for the accepting surgeon and to have that surgeon be intimately involved in arranging the transfer.
- The delicate arena of insurance coverage and financial responsibility for the cost of transport remains problematic if the transfer is at the request of the family or patient and is not directed by medical necessity.

Deterrence/Prevention

- US society's seemingly limited ability to deter violent crimes reaches far beyond a discussion of penetrating abdominal trauma.
- Major societal initiatives hinge upon certain key elements that include education of all socioeconomic strata and age groups, reconstruction of the legal climate and statutes concerning violent crime, economic parity for labor coupled with widely available training and educational opportunities, control of the extensive illegal drug traffic that permeates society, and strict firearm control.
- Each of these humanitarian endeavors carries a cost that is not limited to dollars and that extends to political platforms and agendas.
- Educating target populations such as school-aged children is an excellent first step toward curbing the exponential growth of violent crime in US society.

Complications

- Complications are fairly frequent in this high-risk patient population and are associated with the presence of hypotension, multi-cavitary injury, combined solid and hollow viscus injury, major vascular injury, cardiac arrest, reoperation, multiple comorbid diseases, prehospital near exsanguination, massive blood component transfusion, and steroid use. Clearly, all of these factors relate to impaired tissue perfusion, wound healing capacity, and immune surveillance. The practitioner may directly impact perfusion and bacterial load postinjury. Technical complications directly related to operative procedures and missed injuries set the stage for further injury.
- Wound failure
 - This category of complications includes such diverse elements as wound infection, fascial dehiscence, evisceration, and necrotizing fasciitis. Patients with rapidly treated (<6 h postinjury) superficial SWs infrequently develop wound infections following irrigation, debridement, and wound edge approximation. In contrast, victims of abdominal GSWs generally should not undergo wound closure due to contamination and the greater tissue destruction that accompanies the bullet tract due to cavitation and kinetic energy transfer.
 - Accordingly, after abdominal exploration for penetrating trauma in which hollow viscus injury is identified and the peritoneal space may be heavily contaminated or frankly infected, the fascia usually is closed, while the skin remains open to undergo delayed primary closure or healing by secondary intention.
 - If the fascia fails to heal properly due to infection, strangulation, or poor surgical technique, the fascial edges separate as the sutures pull through the fascia. The result is a fascial dehiscence, which occurs in approximately 5% of patients operated on for penetrating abdominal trauma. If the skin already was closed, a hernia results. If the skin remained open at the time of fascial separation, the patient usually is returned to the OR for irrigation, debridement, and reclosure with retention sutures. Retention sutures protect against intestinal evisceration (ie, peritoneal contents outside of the peritoneal envelope) should the fascial sutures fail again. The patient with fascial dehiscence and open skin margins is at risk for evisceration until the fascial defect is repaired. These patients should remain at bedrest and should have their abdominal contents protected with moist gauze secured with a sterile dressing.
 - Necrotizing fasciitis is a potentially devastating complication of wound closure. The condition is a multi-organism synergistic infection of the fascia (rather than the muscle) and is associated with microvascular thrombosis, tissue hypoxemia, and impaired neutrophil function (eg, diabetes). The patient's wound exudes a thin malodorous fluid rather than thick pus. The fascia easily separates from the underlying muscle. Radical excisional debridement combined with aggressive fluid resuscitation and broad-spectrum antimicrobial therapy is the treatment of choice. This complication carries a mortality rate of at least 50%. Debridement and reconstruction characteristically involve the loss of normal abdominal contour, an average of 7 operations, and an ICU length of stay exceeding 1 month.
- Malnutrition
 - Following an abdominal procedure, most patients develop a paralytic ileus. The small intestine, however, can process luminal nutrients in the absence of peristalsis. When the patient is likely to require a prolonged ICU course, it is prudent to place enteral access (eg,

nasojejunal, transgastric transpyloric jejunal, direct jejunal). Such access provides luminal nutrients to maintain host gut mucosal barrier integrity and provides metabolic substrate to fuel the cellular reparative processes essential for recovery. Luminal nutrition with an isotonic elemental or semi-elemental formula may be initiated, in most circumstances, within 6 hours following operation.

- The goal of nutritional therapy depends on whether one is simply keeping up with ongoing losses or must also correct preexisting protein or protein-calorie malnutrition. Albumin, prealbumin, retinol-binding protein, and/or transferrin levels are good markers of preexisting malnutrition. Measurement of urinary nitrogen losses (urinary urea nitrogen [UUN]) provides a straightforward measure of daily protein catabolism and losses in the urine but underestimates total losses by approximately 4 g/d (eg, hair, exfoliated GI cells, unmeasured nitrogen wastes in urine). The remainder of nonprotein calories may be estimated by a variety of formulae or may be calculated from a target nonprotein calorie-to-nitrogen ratio for the patient's stress state once a target protein intake is calculated or the actual losses (UUN + 4 g) are measured.
- Stress ulceration
 - Patients who sustain major stress are at risk for abnormal gastric mucosal blood flow with resultant loss of protective mucous. Absent or reduced mucous production allows proton-induced mucosal damage to occur. This mucosal injury may progress to submucosal involvement of the plexus of vessels and lead to stress ulceration and an upper GI hemorrhage. The ulcers may be solitary or diffuse. The best protection against stress ulceration is aggressive correction of the underlying problem and vigorous restoration of end-organ oxygen delivery.
 - Adjunctive therapies include topical antacids, histamine-2 (H₂) receptor blockade, proton pump inhibition, or topical agents that bind to exposed mucosa. Current concerns regarding the promotion of nosocomial pneumonias in intubated patients on H₂-blocking medication have spurred interest in the use of topical binding agents to combat stress ulceration because these agents do not alter gastric acidity; in fact, they require acid to become active. Stress ulceration occurs in approximately 20% of ICU patients.
 - Newly developing stress ulceration is a harbinger of an undrained infectious focus and hypoperfusion. The gastric intramucosal pH monitoring catheter is designed to provide a sensitive early warning system for intestinal hypoperfusion due to the gastric mucosa's extreme sensitivity to changes in oxygen delivery. As gastric mucosal blood flow diminishes, the intramucosal pH declines and the PCO₂ rises. The carbon dioxide equilibrates across the semipermeable membrane of the catheter and may be recorded from a sample of the equilibrated fluid. Whether such a system helps decrease incidence of stress ulceration or merely provides earlier notification of an unavoidable event is still unclear.
- Intra-abdominal infection
 - Any emergency abdominal procedure carries a risk of infection that is related to the nature of the procedure and the bacterial load at the time of operation. Risk factors for development of intra-abdominal abscess include but are not limited to the following: hollow viscus perforation (rectum > left colon > right colon > small intestine > duodenum > stomach), retained or recurrent hemoperitoneum (iron promotion of bacterial

proliferation), reoperation, missed injury, anastomotic leak, tissue ischemia, and retained and/or contaminated foreign bodies.

- With preoperative antibiotic therapy, the incidence of postoperative infection is 8.3% for hollow viscus perforations and 5% without GI tract injury. Diagnosis commonly is established by CT scan, ultrasound, or MRI. Localized collections may be drained percutaneously and, provided the collection is well drained and the patient's clinical course improves, may be definitively managed in this fashion.
- Inability to percutaneously drain the entire collection or failure of clinical improvement leads to reexploration and formal drainage. If the patient manifests signs of sepsis earlier than 5 days postoperatively, a CT scan may not localize a collection, and prompt reexploration generally is warranted.

Prognosis

● Mortality

- Victims incur an expected mortality rate of approximately 25%. The death rate clearly is influenced by prehospital hypotension, exsanguination, arrest in the field or on presentation, acidosis with an initial pH less than 7, lactate greater than 20 mmol/L, or base deficit more negative than -15 mEq HCO₃/L of HCO₃ body space.
- Deaths generally occur within the first 72 hours from hypoperfusion and its sequelae. Death also may occur 2 or more weeks later from complications related to sepsis, the systemic inflammatory response syndrome (SIRS), or multiple organ dysfunction syndrome.

● Sepsis

- Sepsis connotes a spectrum of physiologic abnormalities that occur in response to a vast array of cytokine-driven cascades in a susceptible host. This spectrum includes SIRS, compensatory anti-inflammatory response syndrome (CARS), mixed antagonist response syndrome (MARS), and multiple-organ failure syndrome (MOFS).
- Initiation of an injury may or may not be well controlled. The host responses are driven by macrophage interactions with foreign matter as well as native tissue debris and hypoperfusion. If the underlying disorder is rapidly corrected, the host recovers uneventfully; if not, ongoing inflammation leads to SIRS. SIRS is manifested by elevated cardiac performance, maldistributed flow, acute lung injury, and intense catabolism. The adaptive response is to reduce the inflammatory cascades to a manageable level once the injury has been controlled. This adaptive response is known as CARS; this response acts to restore homeostasis.
- If the anti-inflammatory cascades outweigh inflammation, MARS develops. At this stage, incipient organ failures become evident, and the patient is functionally immunoincompetent and increasingly susceptible to infection. The result of these competing immunologic processes is known as immunologic dissonance. The immune discord manifests as multiple organ dysfunction, which ultimately leads to death of the host. The risk for mortality increases as the number of organ failures increases (eg, 3

failures = 85% mortality; 4 failures = 95% mortality; 5 failures = 99% mortality).

Patient Education

- Paramount importance must be placed on patient, family, and support system education if the medical community wishes to proactively reduce the incidence of violent injury in our society. Such an educational initiative should begin with the initial evaluation in the ED.
- Ideally, information regarding support systems, financial resources, safe living facilities, self-help agencies, and employment training opportunities would be available to each individual as needed and would be supplied in a multidisciplinary fashion. The reality, however, of right-sizing the medical community has dramatically reduced resources and restructured in-hospital services.
- Many facilities have replaced social workers with cross-trained case managers who interface with the physicians, nurses, social agencies, and insurance companies. Documentation must fulfill the continually expanding billing documentation guidelines that have reduced the physician time available for counseling.
- Alternative strategies include hiring several part-time employees to serve as patient liaisons whose task is to provide another link to the physician and to educate patients and their families in all pertinent areas. Such individuals may be tasked on days or shifts that are typically high-volume periods for which the personnel are not available to fulfill the educational role.
- Another effective strategy is to involve volunteer community leadership (eg, clergy, school officials, social agency workers) on an as needed basis. Volunteers may be available by pager or phone. Furthermore, community outreach programs can be initiated with the help of a hospital-based group and can be made self-sustaining once the leadership has the necessary information and contacts.
- There is no single solution for every hospital or community—individualization is the key. Frequently, the single largest impediment to a successful educational program is funding. As reimbursements for services rendered decline, hospital-based funding for these important programs may vanish.

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Miscellaneous

Medical/Legal Pitfalls

- Documentation
 - Federal regulations for Medicare/Medicaid billing establish rigid guidelines for the documentation of billed services. Federal agencies recently reviewed a number of prominent medical centers and found them guilty of fraudulent billing practices based on

either their documentation of the services rendered or the involvement of the responsible physician in a resident-driven system.

- The time constraints placed upon physicians to directly render increasing amounts of care with increased documentation may reduce efficiency, patient flux, and performance evaluations. However, such documentation is essential in ensuring that the transfer of care from the ED is smooth and that the care rendered in the ED is completely understood by the receiving team.
- Many centers have undertaken voluntary reviews of their documentation practices and conducted mandatory billing and coding workshops. Novel charting strategies such as T-Charts also are available, which are driven by a specific diagnostic category. These charts list all items in a slash/circle/check format and provide areas for written input to substantiate different levels of service. Other facilities simply provide practitioners with a pocket card with the requirements for the different levels of service.
- Blood product transfusion in a Jehovah's Witness adherent
 - Virtually all medical centers require a consent for blood and/or blood product transfusion, including autologous products. Patients with acute blood loss (eg, trauma, GI bleeding, ruptured aortic aneurysm) who are unable to delineate their wishes may be transfused blood products as a lifesaving measure without formal or informal consent.
 - A legal nightmare arises if such a patient is a Jehovah's Witness adherent. Clearly, the patient must be informed of the transfused products and the rationale for that mode of therapy. If the patient is agreeable, a retroactive consent for the blood product transfusion may be advantageous. When the patient is distraught about the therapy, however, it is wise to engage the patient's family, clergy, social services, hospital administration, risk management, ethics committee, and legal department. A multidisciplinary approach to this problem may demonstrate to the patient and their family that the clinician appreciates the gravity of the problem and is sympathetic to their dilemma.
 - There is uniform agreement that clinicians should proceed with lifesaving measures appropriate to a patient's clinical situation, according to the standard of care in their community, including blood product transfusion unless it is known that the patient refuses that specific therapy.
- Errors of interpretation
 - Incorrect evaluation of patient data is not unique to penetrating abdominal trauma. The introduction of ultrasound examination of the trauma patient by nonradiologists using FAST examination raises the potential for interpretive errors; however, the interpretive error may not be enough to create a medicolegal problem. The subsequent care of the patient must devolve from that erroneous interpretation as well.
 - If the ultrasound is interpreted as demonstrating no fluid and the patient undergoes a CT scan that demonstrates fluid, no harm occurs. If a presumed negative ultrasound finding is positive, but the patient is discharged and suffers an untoward event, the physicians involved risk legal action. Therefore, most centers confirm FAST data with either a CT scan or a period of observation to confirm a clinically benign abdominal examination, normal GI function, and a stable hemoglobin level. Such observation is used increasingly in an emergency medicine-developed and staffed observation unit within or adjacent to the

ED.

- Failure of inquiry
 - Certain key areas that should trigger a physician inquiry include pediatric trauma at home, traumatic injury during pregnancy, and geriatric trauma at home. These areas (ie, child abuse, domestic violence, geriatric abuse/neglect) are targeted by local, state, and federal agencies as important areas for intervention. Agencies are dedicated to each patient population.
 - Mandatory reporting is required for each occurrence and statewide databases include external cause of injury codes (E-codes) for the coding and tracking of these injury mechanisms. Failing to inquire about abuse or neglect is an error of omission and one for which the physician may be held accountable.
 - Missed injury
 - Missed injuries occur most commonly in minimally or maximally injured patients (especially patients transferred from nondesignated facilities); the former due to minimal physical findings and the latter due to diversion of attention to life-threatening priorities. A team approach to caring for trauma patients coupled with algorithmically driven care plans minimize missed injuries.
 - Penetrating trauma to the abdomen that requires operative therapy typically has a vanishingly small missed-injury rate. The patients who tend to have missed injuries are those with flank or back wounds that are judged superficial. Formal imagery (eg, triple-contrast CT scan) or observation of these patients to define the extent of injury may be appropriate. If a missed injury is found, treat it expediently. The documentation and information chain should parallel that for iatrogenic injury.
 - Many missed injuries are of little consequence in terms of disability or hospital length of stay. A missed intestinal injury that presents with fulminant peritonitis and sepsis, however, carries a prohibitive cost to the patient, physician, and hospital. When in doubt, obtain a consultation from the trauma service or a reliable imaging study to rule in or out the injury about which concern exists. This practice serves to further document medical thought processes and may uncover an otherwise occult injury.

Special Concerns

- Pregnant patients:
 - Evaluating a gravid patient following penetrating abdominal trauma is a challenge. The growth of the gravid uterus displaces the intra-abdominal viscera, and the uterus then serves as a shield. This shield function results in a fetal mortality rate of 77% for preterm fetuses and 39% for those at term. The corresponding fetal-maternal death proportion ranges from 3:1 to 9:1.
 - As pregnancy proceeds, plasma volume expands and leads to a relative anemia. Pregnancy also increases the cardiac output, as the expanded volume increases the stroke volume of each cardiac cycle. The pregnant woman has a decrease in systemic precapillary arteriolar sphincter tone identified as diminished systolic and diastolic systemic blood pressure as

well as decreased pulmonary artery pressures.

- The metabolic demands of the growing fetus lead to an increase in carbon dioxide production, which must be cleared by an increase in minute ventilation. As pregnancy proceeds, the increased minute ventilation needs are increasingly met by an increase in respiratory rate rather than tidal volume; the uterine displacement of viscera limits diaphragmatic excursion. Moreover, the fetal-maternal oxyhemoglobin dissociation curve is such that the fetus preferentially unloads oxygen from the maternal blood resulting in a lower mixed venous oxygen saturation than in the nongravid state. This lower saturation is offset by the augmented cardiac output and oxygen delivery.
- There is no question regarding treatment for a pregnant victim with a hemodynamically significant injury. Clearly, the fetus's best chance of survival lies with maternal resuscitation. Certain priorities exist in resuscitating and evaluating all pregnant patients. An understanding of salient physiologic alterations that accompany pregnancy is essential in interpreting the patient's response to intervention.
- Traumatic injury to the uteroplacental unit can result in fetal-maternal hemorrhage with subsequent alloimmunization of the mother against the fetus and fetal exsanguination. Fetal-maternal hemorrhage may be detected by the Kleihauer-Betke test and examination of a peripheral smear (large-volume hemorrhage only). Should fetal-maternal hemorrhage occur, immunize women who are Rh negative with Rh-immune gamma globulin (300 mcg initially and 300 mcg for every 30 cc of estimated fetomaternal transfusion) and have blood sent for type and screen in case of serious hemolysis.
- Another serious complication of uteroplacental trauma is amniotic fluid embolus (as much as 80% maternal mortality). The liberation of amniotic fluid into the maternal circulation engenders life-threatening diffuse intravascular clotting (DIC), fibrinolysis, intense inflammation, and cardiovascular and pulmonary collapse. Aggressive hemodynamic and pulmonary support coupled with emergency cesarean section for fetal delivery, placental extraction, and uterine repair (if needed) are all indicated in an attempt to preserve fetal-maternal viability.
- Every woman who sustains penetrating abdominal (or other) trauma should be questioned specifically about domestic violence if her injuries were not sustained in an MVC. The incidence of domestic violence increases during pregnancy and is clustered during the third trimester.
- Fairly rigid criteria for perimortem cesarean section include a fetus of more than 26 weeks gestational age (fundal height >26 cm above the pubic symphysis or halfway between the xiphoid and the umbilicus) and a dead or moribund mother. In one review of predictors of fetal survival following such a procedure, more than 70% of surviving fetuses were delivered within 5 minutes of maternal death. Perimortem cesarean section is a heroic attempt at fetal preservation; the fetus has a 40-70% chance of survival without fetal handicap.
- More than a 20-minute delay between maternal death and fetal delivery usually results in fetal demise. Well-planned preparation is essential to ensure fetal survival should a postmortem cesarean section be indicated. Ideally, an obstetrician should perform the procedure; however, a trauma surgeon or the ED physician also may deliver the fetus in

this circumstance, especially when there is no obstetric support in the hospital. Consolidation of resources into specialty facilities may preclude in-house obstetric coverage at a receiving trauma facility. (See [Pregnancy, Trauma.](#))

- Pediatric patients
 - Adult trauma facilities should have the capability to triage, treat, and stabilize pediatric trauma patients. Transfer guidelines should be established delineating what types of injury complexes are appropriate for the surrounding pediatric facilities. For example, uni-system injury may be well cared for at a level 2 center, but a multisystem injury should initiate transfer to a level 1 facility.
 - Consider child abuse or neglect during the evaluation of all pediatric trauma patients with suspicious injuries or circumstances. [Childhelp USA](#) and the local Division of Child and Family Services provide information and resource support in such an event. Furthermore, all instances of child abuse or neglect require the completion of a CY-47 form by the treating physician. Social workers well versed in investigating child abuse are invaluable in this situation. The physician should never underestimate the impact of a child abuse investigation when the allegation is unfounded. The safety of the child is paramount and should be the physician's primary concern—not the social impact of a negative investigation.

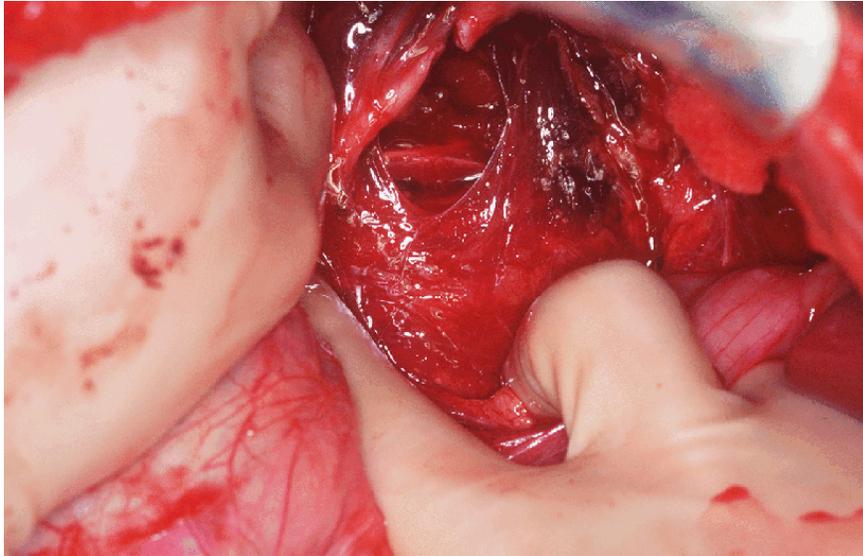
Pictures



Picture 1: Note that the resident is carefully maintaining the position of the impaled stop sign post, so as

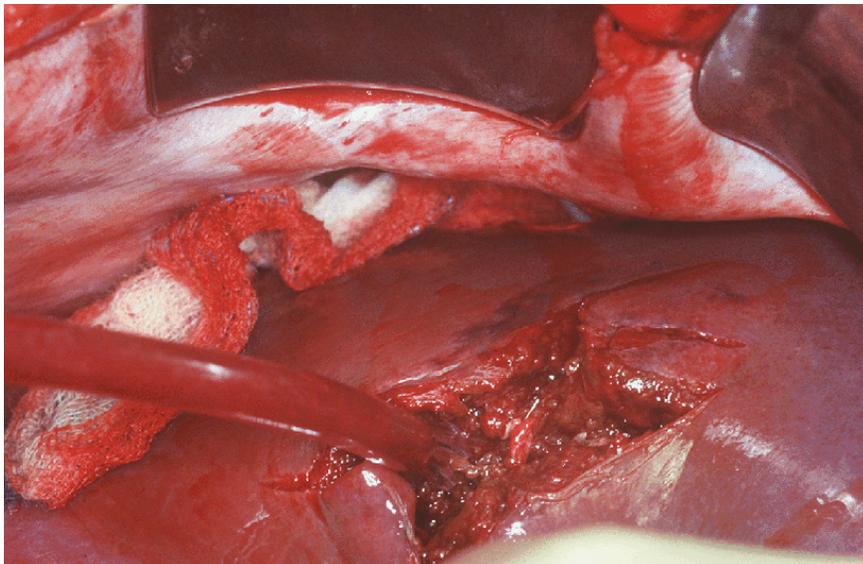
not to dislodge the shaft. The shaft was removed in the OR along with the patient's right colon.

Picture type: Photo



Picture 2: This is an operative photograph of an extremely rare injury: a midureteral transection from a gunshot wound. The patient was shot with a MAC-10 machine gun and sustained the liver injury pictured in the next slide as well as injuries to the duodenum, colon, terminal ileum, sigmoid colon, rectum, gallbladder, bladder, and left femur. He underwent a damage control operation and survived his injuries after 3 subsequent operations.

Picture type: Photo



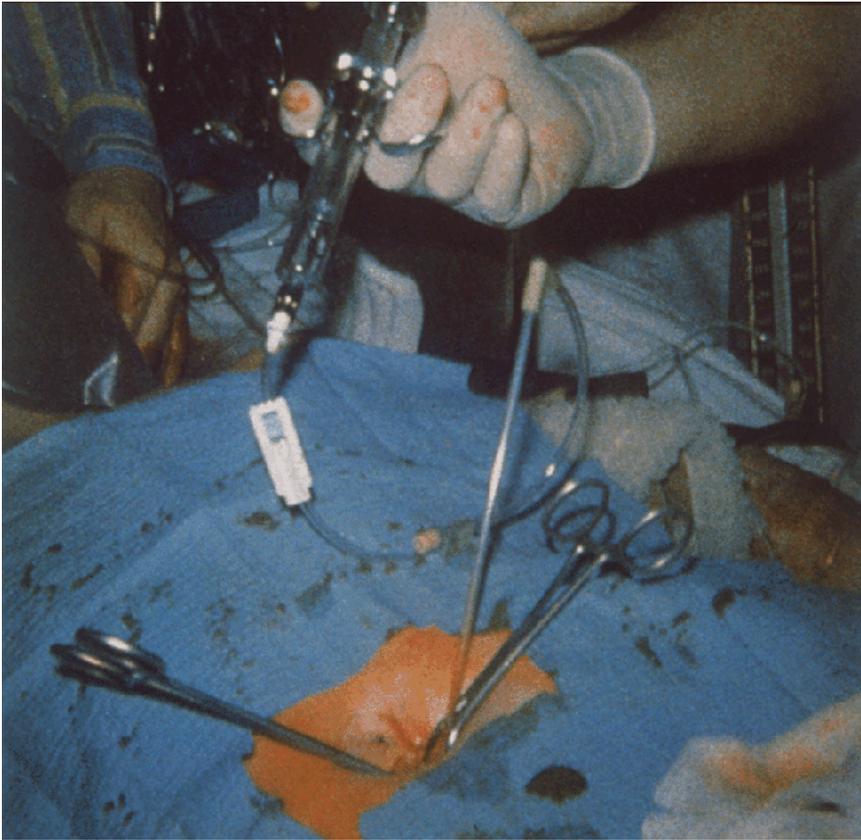
Picture 3: This is the liver injury sustained by the patient pictured in the previous slide. The injury has been opened to control bleeding branches of the portal and hepatic veins as well as the hepatic arterial radicles. Several biliary ducts were ligated and the back wall of the gallbladder can be identified in the depths of the wound. A cholecystectomy was required for management of the wound.

Picture type: Photo



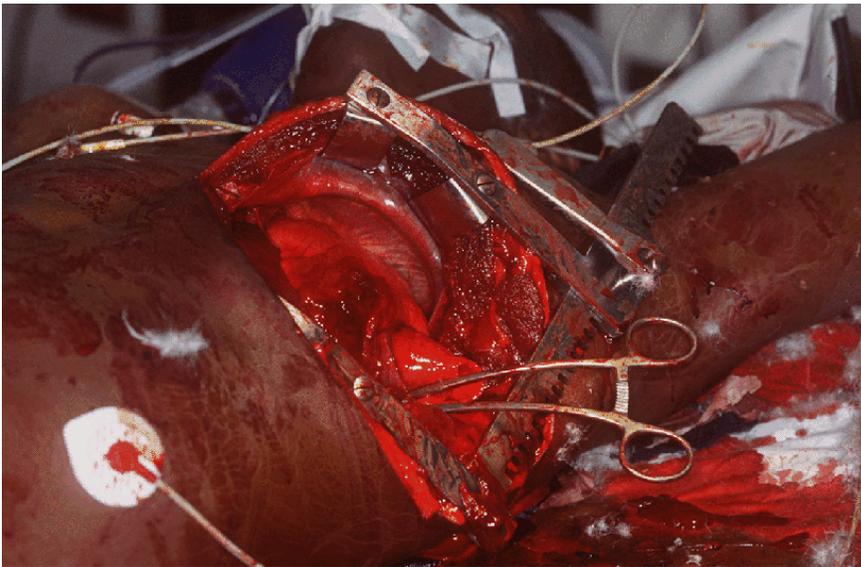
Picture 4: The patient's small intestine clearly protrudes through his anterior abdominal wall following a stab wound caused by a machete. The operative repair and recovery were uneventful.

Picture type: Photo



Picture 5: A standard diagnostic peritoneal lavage (DPL) catheter is secured in place following an open DPL. An aspirating syringe is attached to the catheter via extension tubing as the initial step in the evaluation for intraperitoneal blood.

Picture type: Photo



Picture 6: An ED thoracotomy has been performed and the aorta cross-clamped. Note the proper positioning of the ratchet mechanism of the rib spreader to allow extension of the incision to the right chest for a clamshell thoracotomy if needed. This patient arrived with a weak pulse and a systolic blood pressure of 40 mm Hg and promptly died on the ED stretcher. An ED thoracotomy was performed for cardiopulmonary-cerebral resuscitation.

Picture type: Photo



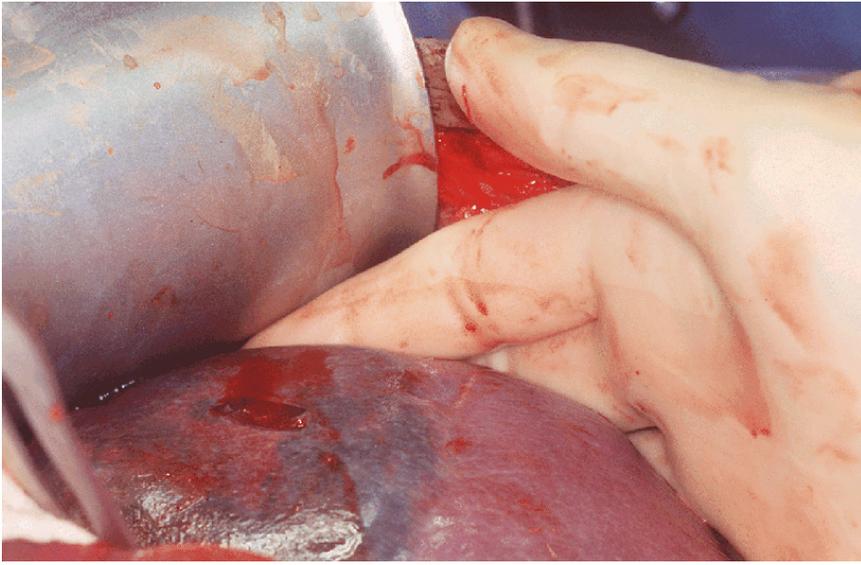
Picture 7: The patient's head is to the right, and the feet are to the left. An oblique incision has been made in the groin to expose the greater saphenous vein, which has been cannulated with a 14G catheter over a needle assembly. This patient also is imaged in the previous slide.

Picture type: Photo



Picture 8: This patient has a temporary abdominal wall closure comprised of a bowel bag and polypropylene mesh, which has been sewn to his skin to treat abdominal compartment syndrome following a gunshot wound to the abdomen. He was reexplored numerous times through the temporary closure prior to definitive repair.

Picture type: Photo



Picture 9: This 22-year-old woman sustained a gunshot wound to the left flank. At exploration, she had a through-and-through laceration of her spleen. The bleeding was arrested by finger compression of the splenic hilum while it was mobilized. A splenectomy was performed because the bullet went through the hilum.

Picture type: Photo

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Acromioclavicular Injury

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Synonyms, Key Words, and Related Terms

acromioclavicular joint, AC, acromioclavicular joint injuries, ACJ

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Introduction

Background

Acromioclavicular (AC) joint injuries most commonly occur in active or athletic young adults. Although uncommon, pediatric AC injuries are increasing because of the rising popularity of dangerous summer and winter sporting activities.

Pathophysiology

The AC joint is comprised of articular surfaces of the clavicle and the acromion, a surrounding capsule, and 2 sets of ligaments (ie, AC, coracoclavicular [CC]). AC ligaments are comprised of stronger superior and inferior ligaments as well as weaker anterior and posterior ligaments. CC ligaments are comprised of

the conoid and trapezoid ligaments, which together form a strong and heavy band that provides vertical movement stability. The AC joint has minimal mobility.

Classification of injury

The degree of clavicular displacement depends on the severity of injury to the AC and CC ligaments, the AC joint capsule, and the supporting muscles of the shoulder (ie, trapezius, deltoid) that attach to the clavicle.

Sage and Salvatore proposed a 3-grade classification; Rockwood further classified 6 types. The classification systems correlate as follows:

- Grade I - Type I
- Grade II - Type II
- Grade III - Types III-VI

The Rockwood classification is as follows:

- Type I - Minor sprain of AC ligaments, intact joint capsule, intact CC ligaments, intact deltoid and trapezius
- Type II - Rupture of AC ligaments and joint capsule, sprain of CC ligaments but CC interspace intact, minimal detachment of deltoid and trapezius
- Type III - Rupture of AC ligaments, joint capsule, and CC ligaments; clavicle elevated (as much as 100% displacement); detachment of deltoid and trapezius
- Type IV - Rupture of AC ligaments, joint capsule, and CC ligaments; clavicle displaced posteriorly into the trapezius; detachment of deltoid and trapezius
- Type V - Rupture of AC ligaments, joint capsule, and CC ligaments; clavicle elevated (more than 100% displacement); detachment of deltoid and trapezius
- Type VI (rare) - Rupture of AC ligaments, joint capsule, and CC ligaments; clavicle displaced behind the tendons of the biceps and coracobrachialis

Pediatric acromioclavicular injury

AC joint injuries in children are very uncommon, and they differ anatomically from such injuries in adults. For example, failure of an ossification center may appear to be a fracture on a child's radiograph; the clavicle is encased in a periosteal tube in younger children. As a result, the AC ligaments are injured; however, the CC ligaments remain intact because they are attached to the periosteal tube.

The pediatric Rockwood classification is as follows:

- Type I - Clavicle stable; joint radiographically normal
- Type II - Partial tear of the periosteal tube, allowing for some mobility of the distal clavicle; AC

ligaments disrupted

- Types III-VI - Larger tear through the periosteal tube, allowing for greater clavicle mobility and gross instability with clavicle positioning; CC ligaments remain attached to the clavicle periosteal tube

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Clinical

History

AC injury often involves a fall onto the apex of the shoulder, usually with the arm in adduction. Severe forces resulting from significant falls often are associated with type III-VI injuries.

Physical

- The degree of pain, at rest or elicited with movement, increases with severity of injury.
- Tenderness manifests upon palpation of the AC joint.
- Determine the presence of any step-off of the AC joint and note the position of the clavicle.
- Perform a careful neurovascular assessment of the brachial plexus motor and sensory function, as associated injuries occur on a rare basis.

Causes

Downward blunt force on the acromion results in variable injury to the AC and CC ligaments. Other injuries, depending on the force of injury, may include tears of deltoid and trapezius attachments at the clavicle and fractures of the acromion, clavicle, and coracoid (or of their cartilaginous attachments).

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Differentials

Bursitis
Dislocations, Shoulder
Fractures, Cervical Spine
Fractures, Clavicle

Fractures, Humerus
Tendonitis

Other Problems to be Considered

Septic arthritis

Erb-Duchenne injury

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Workup

Imaging Studies

- Obtain optimal AC radiographs by using adequate penetration and a slight cephalic tilt of the x-ray beam. Radiographic results according to severity of injury are as follows:
 - Type I - Normal
 - Type II - Subluxation of AC joint space less than 1 cm; normal CC space
 - Type III - Subluxation of AC joint space more than 1 cm; widening of the CC space more than 50%
 - Types IV-VI - Subluxation of AC joint space more than 1 cm, widening of the CC space more than 50%; associated displacement of the clavicle
- Assess the clavicle and scapula for associated fractures.
- For pediatric injuries, plain radiographs may reveal fractures at the base of the coracoid.

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Treatment

Prehospital Care

- Distinguishing AC injuries from other shoulder injuries (ie, clavicular fractures, shoulder dislocations, proximal humeral fractures) is difficult.
- Field management of these injuries involves immobilization of the shoulder with a sling and swath.

- Assess and immobilize the spine, if indicated.

Emergency Department Care

- Type I: Treatment should include ice, analgesics, sling immobilization, and daily range-of-motion exercises.
- Type II: Provide the same treatment as type I; however, patients with type II injuries take longer to improve. With significant instability, strap immobilization for 2-4 weeks and no heavy lifting for 6 weeks are appropriate. A Kenny-Howard shoulder harness may be used for strap immobilization, though this device frequently is uncomfortable for the patient and may not change the outcome.
- Type III: Treatment is controversial. Older or inactive patients may do well without open reduction and internal fixation (ORIF), while younger or active patients may benefit from ORIF.
- Type IV-V: ORIF
- Pediatric injuries
 - For types I-III, closed reduction can be effective, though surgical intervention for selected cases may be indicated to achieve better functional results.
 - Types IV and V commonly require ORIF.

Consultations

- Orthopedic surgery
 - Pediatric cases
 - Types III-VI

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Medication

The goals of therapy are to reduce pain and inflammation.

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

These agents are used for both anti-inflammatory and analgesic effects. Acetaminophen (with or without an opiate) is the most commonly used analgesic.

| | |
|-----------|--|
| Drug Name | Ibuprofen (Motrin, Nuprin, Midol, Advil)- In the absence of contraindications, usually the DOC for treatment of mild to moderate pain. |
|-----------|--|

| | |
|-------------------|--|
| Adult Dose | 200-400 mg q4-6h PO while symptoms persist; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

Nonopioid Analgesics

These agents are used for mild to moderate analgesic effects.

| | |
|-------------------|--|
| Drug Name | Acetaminophen (Tylenol, Aspirin Free Anacin)- DOC for pain in patients with documented hypersensitivity to aspirin or NSAIDs, in those diagnosed with upper GI disease, or in those taking PO anticoagulants. |
| Adult Dose | 325-650 mg q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; G-6-PD deficiency |
| Interactions | Rifampin can reduce analgesic effects of acetaminophen; coadministration with barbiturates, carbamazepine, hydantoin, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in persons with chronic alcoholism following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; APAP is contained in many OTC products and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

Opioid Analgesics

These agents are used for moderate-to-strong analgesic effects.

| | |
|-------------------|--|
| Drug Name | Propoxyphene and acetaminophen (Darvocet N-100)- Indicated for the treatment of mild to moderate pain. |
| Adult Dose | 1-2 tab PO q4h prn; not to exceed 600 mg/d |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | May increase serum concentrations of MAOIs, TCAs, carbamazepine, phenobarbital, and warfarin |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates (substitution may result in acute opiate withdrawal symptoms); caution in severe renal or hepatic dysfunction |

| | |
|-------------------|--|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Indicated for the relief of moderate to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn for pain |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen q4h; not to exceed 10 mg of hydrocodone bitartrate per dose; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; HACE; elevated ICP |
| Interactions | Coadministration with phenothiazines may decrease analgesic effects; toxicity increases with CNS depressants or TCAs |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tabs contain metabisulfite, which may cause hypersensitivity; caution in patients dependent on opiates, since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|--|
| Drug Name | Acetaminophen and codeine (Tylenol With Codeine)- Indicated for the treatment of mild to moderate pain. |
| Adult Dose | 30-60 mg/dose based on codeine content q4-6h or 1-2 tab q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |

| | |
|--------------|--|
| Interactions | Toxicity increases with CNS depressants or TCAs |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates, since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

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Follow-up

Further Outpatient Care

- Following discharge, orthopedic surgical follow-up is mandatory for type III-VI injuries and is recommended in pediatric injuries.

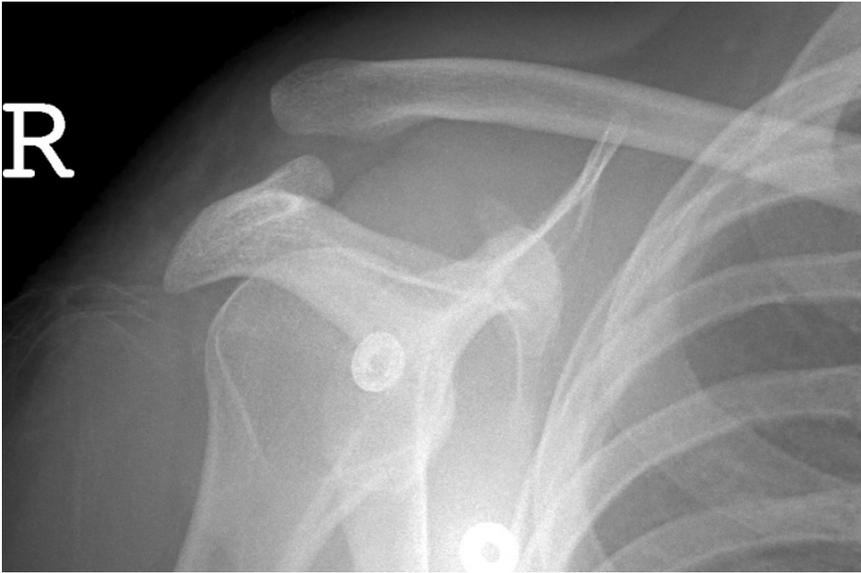
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Miscellaneous

Medical/Legal Pitfalls

- Failure to document neurological, vascular, or other associated injuries is the primary error in the assessment of AC joint injuries.

Pictures



Picture 1: Anteroposterior (AP) x-ray of right shoulder showing step-off of acromioclavicular (AC) joint

Picture type: X-RAY

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Ankle, Soft-Tissue Injuries

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Introduction

Background

Ankle injuries are the most common injuries incurred during sports and recreational activities. They are particularly common during soccer, basketball, and volleyball.

Pathophysiology

Most ankle sprains occur due to inversion during extension of the ankle. Thus, approximately 85% of injuries involve the 3 distinct lateral ligaments: anterior talofibular ligament (ATFL), calcaneofibular ligament (CFL), and posterior talofibular ligament (PTFL). Of sprains due to inversion, 65% are isolated to the ATFL. In some patients, the subtalar complex may also be injured. The CFL is rarely injured in isolation.

Isolated injury to the medial (deltoid) ligament is rare and usually involves malleolar fractures. Distal tibiofibular syndesmotoc rupture is very rare and is associated with dorsiflexion and external rotation. Recovery from this injury is significantly prolonged, unlike isolated lateral ligament sprains.

Rupture of the superior peroneal retinaculum results in subluxation or dislocation of the peroneal

tendons. The mechanism of injury is usually forced dorsiflexion with reflex contraction of the peroneal muscles. Patients complain of pain and a snapping sensation over the posterolateral ankle with weakness of eversion.

Ankle sprains are classified into 3 grades as follows:

- Grade I injuries involve a stretch of the ligament with microscopic but not macroscopic tearing. Generally, little swelling is present, with little or no functional loss and no joint instability.
- Grade II injuries stretch the ligament with partial tearing, moderate-to-severe swelling, ecchymosis, moderate functional loss, and mild-to-moderate joint instability.
- Grade III injuries involve the complete rupture of the ligament with immediate and severe swelling, ecchymosis, an inability to bear weight, and moderate-to-severe instability of the joint.

Frequency

- **In the US:** Inversion injuries occur at a rate of 1 per 10,000 people per day, which is about 27,000 injuries per day in the US. Injury to the dominant ankle is 2-3 times more likely than injury to the nondominant ankle. Ankle sprains are twice as likely in intercollegiate athletes when compared with interscholastic athletes.

Mortality/Morbidity

- Ankle sprains can cause significant morbidity. As many as 73% of athletes with an ankle sprain suffer recurrent sprains, and 59% have significant disability and impairment of athletic performance.
- Up to 50% of people who incur an ankle sprain have some type of chronic sequelae. These conditions include functional instability, mechanical instability, chronic pain, stiffness, and recurrent chronic swelling.
- Eversion injuries are more likely to result in persistent pain or chronic instability.

Sex

Women athletes are 25% more likely to sustain ankle injuries than male athletes.

Age

Ankle injuries primarily involve young people because they participate more often in physically demanding recreational activities and sports. Fractures and tendon ruptures occur more often in older adults.

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Clinical

History

- Assessment of all orthopedic injuries should include the following:
 - Mechanism of injury
 - Previous ankle injuries
 - Presence of immediate or delayed pain, swelling, and ability or inability to bear weight
 - Presence or absence of any popping-type sensations or actual noise at the time of injury

Physical

- Observe for edema, ecchymosis, or deformity.
- Palpate for tenderness, crepitant, or deformity.
- Assess active and passive range of motion as well as weight-bearing ability.
- Perform talar tilt test.
 - Place the foot in 20-30° of plantar flexion, and apply slight adduction and gentle inversion stress to the calcaneal midfoot.
 - If both the anterior talofibular and the calcaneofibular ligaments are ruptured, the examiner will detect talar tilt (ie, movement of the talus in the mortise).
- Perform and document a neurovascular examination, including checks of the dorsalis pedis and posterior tibial pulses.

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Differentials

Fractures, Ankle

Tendonitis

Tenosynovitis

Other Problems to be Considered

Achilles tendon rupture

Peroneal tendon subluxation

Septic joint

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Workup

Imaging Studies

- Radiographic studies of the ankle should include the following films:
 - An anteroposterior (AP) film with the ankle in 5-15° of adduction
 - A true lateral film
 - A 45° oblique film with the ankle in dorsiflexion (ie, Mortise view)

Other Tests

- Stress radiographs or arthrographies are not mandatory in the ED, but they may be requested by an orthopedic consultant.

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Treatment

Prehospital Care

For patient comfort, splint all ankle injuries prior to transport to the ED.

Emergency Department Care

- First-degree sprains or mild second-degree sprains
 - Rest, ice, and elevation
 - Compression dressing or commercially available air stirrup splint
 - Possible cessation of weight bearing initially
 - Early range of motion exercises
 - Wobble board training after recovery in order to reduce the number of recurrent injuries

and prevent functional instability

Consultations

- Obtain orthopedic consultation for severe sprains, suspected peroneal tendon subluxation, or associated fractures.
- Emergent orthopedic evaluation rarely is required. Office follow-up in a week usually suffices.

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Medication

The goals of therapy are to reduce pain and prevent complications.

Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)

With analgesic and anti-inflammatory properties, NSAIDs are the ideal agents for treating ankle injuries. Acetaminophen with or without an opiate analgesic may be added to NSAID therapy (or used as a substitute).

| | |
|-------------------|---|
| Drug Name | Ibuprofen (Ibuprofen, Advil, Motrin)- Usually the DOC for treatment of mild to moderate pain, if there are no contraindications. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, resulting in the inhibition of prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h while symptoms persist; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related side effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |

| | |
|-------------|---|
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |
|-------------|---|

| | |
|-------------------|---|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for the relief of mild to moderate pain and inflammation. Administer small doses initially to patients with small body size, the elderly, and those with renal or liver disease. Doses higher than 75 mg do not increase its therapeutic effects. Administer high doses with caution and closely observe patients for response. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 12 years: 0.1-1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related side effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|---|
| Drug Name | Flurbiprofen (Ansaid)- Has analgesic, antipyretic, and anti-inflammatory effects. May inhibit cyclo-oxygenase enzyme, causing the inhibition of prostaglandin biosynthesis that may in turn result in analgesic and anti-inflammatory activities. |
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related side effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |

| | |
|-------------|--|
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

| | |
|-------------------|--|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- For relief of mild to moderate pain; inhibits inflammatory reactions and pain by decreasing activity of cyclo-oxygenase, which results in a decrease of prostaglandin synthesis. |
| Adult Dose | 500 mg PO followed by 250 mg PO q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related side effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

Analgesics

Pain control is essential to quality patient care. Analgesics ensure patient comfort, promote pulmonary toilet, and enable physical therapy regimens. Many analgesics have sedating properties that are beneficial for patients who have sustained injuries.

| | |
|-------------------|--|
| Drug Name | Acetaminophen (Tylenol, Panadol, Aspirin-Free Anacin)- DOC for pain in patients with documented hypersensitivity to aspirin or NSAIDs, with upper GI disease, or who are taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1,000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg PO q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; known G-6-PD deficiency |
| Interactions | Rifampin can reduce analgesic effects of acetaminophen; coadministration with barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; acetaminophen is contained in many OTC products, and combined use with these products may result in cumulative doses exceeding recommended maximum dose |

| | |
|-------------------|--|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for the treatment of mild to moderate pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tab PO q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | Toxicity of codeine increases with CNS depressants, tricyclic antidepressants, MAO inhibitors, neuromuscular blockers, CNS depressants, phenothiazines, and narcotic analgesics Rifampin can reduce analgesic effects of acetaminophen; coadministration with barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity of acetaminophen |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|------------|--|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for moderate to severe pain. |
| Adult Dose | 1-2 tab PO q4-6h prn pain |

| | |
|-------------------|--|
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen PO q4h; not to exceed 5 doses/d; single dose should not exceed 10 mg of hydrocodone bitartrate |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema (HACE); elevated intracranial pressure (ICP) |
| Interactions | Coadministration with phenothiazines may decrease analgesic effects; toxicity increases with CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite, which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

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Follow-up

Further Outpatient Care

- Patients with grade I or mild grade II lateral sprains should have a follow-up visit with their primary care physician in 1-2 weeks.
- Consult orthopedics or sports medicine for all other injuries.

Complications

- Functional and/or mechanical instability
- Chronic pain, stiffness, and/or edema

Prognosis

- With appropriate initial treatment, referral, and physical therapy, the majority of patients have a favorable outcome.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to diagnose
 - Failure to obtain a radiograph
 - Misinterpretation of a radiograph
 - Failure to recognize ankle instability

Special Concerns

- Elderly patients may require home health visits to assess mobility and ability to perform activities of daily living (ADL).
-

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Back Pain, Mechanical

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Introduction

Background

Mechanical low back pain is one of the most common patient complaints expressed to emergency physicians in the US. Approximately 20% of the adult population is affected, making it the fourth most common complaint in ambulatory medicine and the third most expensive disorder in terms of health care dollars expended, surpassed only by cancer and heart disease.

Low back pain reportedly occurs at least once in 85% of adults younger than 50 years. Of these patients, only 20% can be given a precise pathoanatomic diagnosis. Low back pain is not a common complaint in children and, when present, is more likely to have a serious etiology such as infection or malignancy.

Pathophysiology

Many causes of mechanical low back pain exist. Discussion in this chapter is limited to musculoskeletal causes, which can be divided into nerve root syndromes, musculoskeletal pain syndromes, and skeletal causes.

Nerve root syndromes

Classic nerve root syndrome is characterized by radicular pain arising from nerve root impingement due to herniated discs. A similar syndrome can be produced by inflammation and irritation, which explains why so many people respond to conservative therapies.

Impingement pain tends to be sharp, well localized, and associated with paresthesia; irritation tends to be dull, poorly localized, and without paresthesia. Impingement is associated with a positive straight leg raising sign (ie, shooting pain down contralateral leg with leg raising), while irritation is not. Neurologic deficits and pain radiation below the knee are rarely seen in irritation alone and are most commonly found with impingement.

Causes of impingement syndrome include herniated discs, spinal stenosis, spinal degeneration, and cauda equina syndrome.

Herniated discs are produced as the spinal discs degenerate. After growing thinner, the nucleus pulposus herniates out of the central cavity against a nerve root. Intervertebral discs begin to degenerate by the third decade of life, and herniated discs are found on autopsy in one third of adults older than 20 years. Only 3% of these, however, are symptomatic. The most common locations for herniation are L4, L5, and S1.

Spinal stenosis occurs as intervertebral discs lose moisture and volume with age, which decreases the disc spaces. Even minor trauma under these circumstances can cause inflammation and nerve root impingement, which can produce classic sciatica without disc rupture.

Spinal degeneration is caused by alterations in the hydroscopic quality of the nucleus pulposus that progresses to annular degeneration. This, coupled with progressive posterior facet disease, causes spinal canal or foraminal encroachment. These retrogressive and proliferative changes in the disc anteriorly and the joints posteriorly produce clinical symptoms and radiographic findings termed 3-joint complex degeneration. Spinal degeneration has 3 distinct stages, as follows:

- Dysfunction with complaints of pain only
- Instability with advanced degeneration, pseudospondylolisthesis, and neurological abnormalities
- Stabilization with morning stiffness and with prolonged standing or walking, producing radicular pain

Cauda equina syndrome is produced by massive extrusion of nuclear material into the spinal canal, which compresses the caudal sac. The classic presentation is bilateral sciatica, with lower extremity bowel or bladder dysfunction present in 90% of patients. Perineal or perianal anesthesia is present in 60-80% of patients.

Musculoskeletal pain syndromes

Musculoskeletal pain syndromes that produce low back pain include myofascial pain syndromes and fibromyalgia.

Myofascial pain is characterized by pain and tenderness over localized areas (trigger points), loss of range of motion in the involved muscle groups, and pain radiating in a characteristic distribution but restricted to a peripheral nerve. Relief of pain is often reported when the involved muscle group is stretched.

Fibromyalgia results in pain and tenderness on palpation of 11 of 18 trigger points, one of which is the low back area, as classified by the American College of Rheumatology. Generalized stiffness, fatigue, and muscle ache are reported.

Other skeletal causes

Other skeletal causes of low back pain include osteomyelitis, sacroiliitis, and malignancy.

Osteomyelitis results from infectious processes involving the bones of the spine, while sacroiliitis results from inflammatory changes in the sacroiliac joints. This pain presents over the sacroiliac joints and radiates to the anterior and posterior thighs. This pain is usually worse at night and is exacerbated by prolonged sitting or standing.

Malignant tumors of the spine can be primary or metastatic. Most primary spinal tumors are found in patients younger than 30 years and usually involve the posterior vertebral elements. Metastatic tumors are found mostly in patients older than 50 years and tend to occur in the anterior aspects of the vertebral body.

Mortality/Morbidity

- Most etiologies of mechanical low back pain are not life threatening; however, significant morbidity is associated with chronic low back pain syndromes.
- A significant number of patients are unable to return to their normal daily routines or function in a productive work environment secondary to low back pain.
- Most cases of back pain treated in the emergency department are not true emergencies, with the exception of those due to cauda equina syndrome. These patients must undergo surgical decompression as soon as possible or face permanent neurological damage.

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Clinical

History

A thorough history and physical examination is paramount to arrive at a diagnosis.

- Patients most often complain of pain in the lumbosacral area.
 - Determining whether pain is exacerbated by movement or by prolonged sitting or standing.
 - Determine the duration of pain.
 - Establish if pain was sudden in onset or gradual over days or months.
 - Find out if the patient can identify a precipitating event such as lifting or moving furniture.
- Inquire about current medications that may produce symptomatology.
 - Chronic steroids may predispose to infection or compression fractures.
 - Anticoagulants may result in a bleed or hematoma.

Physical

- Physical examination of a patient with back pain should include range of motion and a thorough neurologic examination, including assessment of peripheral motor function, sensation, and deep tendon reflexes.
- Spinal stenosis may be present when evidence of degenerative joint disease is present on radiographic studies.
 - Patients with this disease process often complain of progressive pain down the lateral aspect of the leg during ambulation (pseudoclaudication). This pain results from neurologic compression rather than actual arterial insufficiency, which produces true claudication.
 - The stoop test helps distinguish true claudication from pseudoclaudication. Patients with true claudication sit down to rest when pain occurs, while patients with pseudoclaudication attempt to keep walking by stooping or flexing the spine to relieve the stretch on the sciatic nerve.
- Osteomyelitis may be subacute or acute.
 - Clinical findings are nonspecific, and the patient may be afebrile on presentation.
 - Classic presentation includes pain on palpation of the vertebral body, elevated sedimentation rate, and complaints of pain out of proportion to physical findings.
 - Patients particularly at risk for development of osteomyelitis include patients who have undergone recent back surgery, intravenous (IV) drug users, patients with immunosuppression, and those with a history of chronic pelvic inflammatory disease (PID).

Causes

Please refer to [Pathophysiology](#), which describes specific causes of back pain in detail. Certain clinical

clues can help differentiate between causes. Generally, impingement syndromes produce positive straight leg raising tests, while pure irritation does not. To assess for a functional disorder as the cause of low back pain, consider the following:

- Mechanical low back pain is a common complaint in patients with functional disorders. In addition, functional overlay may be present in some patients with true organic pathology. The degree of psychosocial issues affecting the patient's condition may be assessed by the following:
 - Patient may receive compensation for injury.
 - Patient has pending litigation.
 - Patient dislikes job.
 - Patient has symptoms of depression.
 - Patient caused the accident resulting in back pain.
- A particularly useful test is to have patients hold their wrists next to their hips and turn their body from side to side. This test gives the illusion that you are testing spinal rotation, but no actual stress is placed on any muscles or ligaments. Any complaint of pain during this maneuver is strongly suggestive of a functional component to the illness.

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Differentials

Vaginitis

Other Problems to be Considered

Cystitis and pyelonephritis

Perforated viscous

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Workup

Lab Studies

- Consider performing urinalysis if the problem is not musculoskeletal or an exacerbation of chronic back pain.

- Perform a complete blood count (CBC) and erythrocyte sedimentation rate (ESR) if the patient is febrile or may have an epidural or spinal abscess or osteomyelitis. While ESR has moderate specificity, sensitivity is relatively high in cases of abscess.
- Other laboratory studies are rarely needed unless a disorder other than back pain is strongly suspected.

Imaging Studies

- Radiographs
 - Lumbosacral spine series are expensive and expose the gonads to 3000 units more radiation than a chest x-ray. Annually, 7-8 million such tests are ordered, but most have little value in directing therapy, particularly among patients younger than 50 years.
 - Unless a history of traumatic injury is present, such films should be obtained only for suspicion of malignancy or infection. Malignant involvement of vertebral bodies can be evident on plain film when as little as 30% of the vertebral body has been replaced.
 - Other indications that suggest the need for radiographic imaging include chronic steroid use and acute onset of pain in older or very young patients.
 - The physician also may consider obtaining radiographs for patients whose cases involve (or potentially involve) litigation or for patients who are seeking compensation.
- Ultrasound may be useful if the differential diagnosis includes appendicitis, a pathologic pelvic process, or abdominal aneurysm.
- True emergencies that necessitate imaging include the following:
 - Patients with a history of malignancy and new evidence of nerve entrapment
 - Patients with back pain associated with paralysis or gross muscle weakness
 - Patients with bilateral neurological deficits associated with bowel or bladder function loss
 - Patients for whom an epidural hematoma or abscess is suspected

Other Tests

- Perform straight leg testing with the patient supine. Record response to raising each leg. An approximation of the test may be performed with the patient sitting and each leg straightened at the knee.
- The stoop test helps distinguish true claudication from pseudoclaudication. Patients with true claudication sit down to rest when pain occurs, while patients with pseudoclaudication attempt to keep walking by stooping or flexing the spine to relieve the stretch on the sciatic nerve.

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Treatment

Prehospital Care

- If the patient's back pain is from a traumatic injury, employ full spinal precautions.
- If no history of trauma is present, it is not necessary to utilize spinal precautions, as the patient may experience significant exacerbation of pain by lying on a rigid board. The patient may be made more comfortable by supporting the lower extremities with a pillow or blanket.

Emergency Department Care

New neurologic deficits accompanied by bowel or bladder dysfunction may be caused by cauda equina syndrome, which mandates emergency imaging and consultation.

Conservative therapy is the mainstay of treatment, as even those with true sciatica generally respond. Ultimately, only 2% of patients with sciatica and 4-6% of patients with true disc herniation require surgery. Conservative therapy traditionally includes the following:

- Bed rest, once the cornerstone of treatment, is no longer widely employed.
 - A growing body of evidence suggests that even brief bed rest is not necessary except in patients with true sciatica. In this case, the supine position decreases pressure on the spinal cord itself. Early return to work on light duty or restricted activity seems to lead to better long-term outcomes.
 - Recent studies have assigned patients to groups of those who had 4 days of bed rest versus no bed rest. No difference was found in speed of pain resolution, but those without bed rest returned to normal activities 42% sooner.
- Unless the patient is allergic to the medicine or it is otherwise contraindicated, severe low back pain in patients admitted to the ED can be improved significantly with a combination of NSAIDs, muscle relaxants, and possibly steroids.
- Use of hot or cold compresses has never been proven scientifically to speed symptom resolution, but some patients may experience brief relief.
- Gentle flexion/extension exercises are helpful.
- Spinal traction is ineffective.

Consultations

- ED consultations are necessary for patients who present with acute cauda equina syndrome, demonstrate intractable pain, or where a social situation makes hospitalization necessary.
- Whether orthopedic or neurosurgical consultation is chosen depends on local custom and resources.
- Other medical consultation may be needed if the cause of back pain is not mechanical.

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Medication

The goal of pharmacotherapy is to reduce pain and inflammation.

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

NSAIDs are most commonly used to relieve mild to moderate pain. Although the effectiveness of NSAIDs tends to be patient specific, ibuprofen is usually the DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- DOC to treat mild to moderate pain if there are no contraindications. Inhibits inflammatory reactions and pain by decreasing prostaglandin synthesis. |
| Adult Dose | 600 mg PO tid |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

| | |
|----------------|---|
| Drug Name | Flurbiprofen (Ansaid)- May inhibit cyclo-oxygenase enzyme, which in turn inhibits prostaglandin biosynthesis. These effects may result in analgesic, antipyretic, and anti-inflammatory activities. |
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |

| | |
|-------------------|--|
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related side effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

| | |
|-------------------|---|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- For relief of mild to moderate pain and inflammation. Small dosages initially are indicated in patients who are small or elderly and in those with renal or liver disease. Doses over 75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe patient for response. |
| Adult Dose | 25-50 mg q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 14 years: 0.1-1 mg/kg q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related side effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

| | |
|-----------|--|
| Drug Name | Naproxen (Anaprox, Naprelan, and Naprosyn)- For relief of mild to moderate pain; inhibits inflammatory reactions and pain by decreasing activity of cyclo-oxygenase, which results in a decrease of prostaglandin synthesis. |
|-----------|--|

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|-------------------|--|
| Adult Dose | 500 mg PO initial, followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related side effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

Muscle Relaxant

| | |
|-------------------|--|
| Drug Name | Cyclobenzaprine (Flexeril)- Skeletal muscle relaxant that acts centrally and reduces motor activity of tonic somatic origins influencing both alpha and gamma motor neurons. Structurally related to tricyclic antidepressants and thus carries some of the same liabilities. |
| Adult Dose | 10 mg PO tid with a range of 20-40 mg/d in divided doses; not to exceed 60 mg/d |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity; patients who have taken MAOIs within the last 14 d |
| Interactions | Coadministration with MAOIs and tricyclic antidepressants may increase toxicity; cyclobenzaprine may have additive effect when used concurrently with anticholinergics; effects of alcohol, CNS depressants, and barbiturates may be enhanced with cyclobenzaprine |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in angle closure glaucoma, urinary hesitance |

Analgesics

Pain control is essential to ensure patient comfort, promote pulmonary toilet, and aid physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained injuries.

| | |
|-------------------|--|
| Drug Name | Acetaminophen (Tylenol, Panadol, Aspirin Free Anacin)- DOC for pain in patients with documented hypersensitivity to aspirin or NSAIDs, with upper GI disease, or who are taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg PO q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; known G-6-PD deficiency |
| Interactions | DOC for pain in patients with documented hypersensitivity to aspirin or NSAIDs, with upper GI disease, or who are taking oral anticoagulants. |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; acetaminophen is contained in many OTC products, and combined use with these products may result in cumulative acetaminophen doses exceeding recommended maximum dose |

| | |
|-------------------|---|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- A drug combination indicated for the treatment of mild to moderate pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tabs q4h; not to exceed 12 tabs/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | Toxicity increases with CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|------------|--|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- A drug combination indicated for the relief of moderate to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |

| | |
|-------------------|--|
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen q4-6h prn; not to exceed 2.6 g/d acetaminophen >12 years: 750 mg acetaminophen q4h; not to exceed 10 mg hydrocodone bitartrate in single dose; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; elevated intracranial pressure |
| Interactions | Coadministration with phenothiazines may decrease analgesic effects; toxicity increases with CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite, which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

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Follow-up

Further Inpatient Care

- Follow-up generally is managed by the patient's private physician in patients without true sciatica or nerve root findings.
- If these findings are present, follow-up by an orthopedist or neurosurgeon is preferred. Whether an orthopedist or neurosurgeon is selected for referral depends on local resources and customs.
- Patient's primary physician should be contacted regarding the referral.

Further Outpatient Care

- Short-term physical therapy with gentle exercises may be of some benefit.
 - This has not been proven significantly more effective than self-care with instructions by the physician. However, patients appear to prefer therapy over self-care when surveyed.
 - Cost-benefit ratio should be considered prior to physical therapy referral from the ED.

in/Out Patient Meds

- Outpatient therapy generally consists of a combination of muscle relaxants and NSAIDs. In certain cases, a short course of prednisone may also be helpful.

Prognosis

- Prognosis is quite good for most patients presenting with mechanical back pain.
 - Overall, 70% of patients feel better in 1 week, 80% in 2 weeks, and 90% in 1 month.
 - Only 10% of all patients with low back pain have long-term problems.
 - Often, a significant functional overlay is present in this subgroup of patients, who also account for the majority of office visits with low back pain complaints (see [Causes](#) section).

Patient Education

- Patient education centers on prevention. This includes the following:
 - Promoting weight loss where indicated
 - Performing back strengthening exercises
 - Teaching proper lifting technique
 - Increasing overall physical conditioning

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Miscellaneous

Medical/Legal Pitfalls

- Work excuses deserve special mention
 - Most patients require some form of work excuse.
 - Prolonged work excuses often lead to functional disorders, and it is generally recommended that excuses be limited to 2-3 days following an ED visit.
 - Furthermore, it is preferable that the patient return to work immediately on light or restricted duty if possible.
 - If work excuses are needed beyond this time, ideally the patient's primary or referral physician will provide them.
 - When providing work excuses, patient confidentiality must always be protected. The only information employers are entitled to without explicit authorization by the patient is whether the work excuse is legitimate and whether employees have any medical problems that may prevent them from doing their job or pose a health risk to others.
 - Be sure to include what activities the patient may engage in and the period the restrictions are in effect.

Special Concerns

- Back pain is an uncommon complaint in children. The cause of back pain is most often infectious or malignant in these patients.
 - Suspect malignancy if back pain has lasted for more than 1 month, is unrelieved by bed rest, and occurs at night (interrupting sleep in patients older than 50 years).
-

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Blast Injuries

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Introduction

Background

The catastrophic explosions of three aircraft into three buildings on September 11, 2001 have reminded healthcare workers of the magnitude of injuries and death that can result from this mechanism. This recent terror attack adds to previous US incidents including the Oklahoma City federal building, the previous attack on the World Trade Center, the Atlanta Olympic Park scare, and explosive device attacks at schools, in the workplace and at abortion clinics. Internationally, there are many areas of the world where the use of explosive devices in war or acts of terrorism are frequent. Much of the challenge facing the care providers is the potential for the sudden creation of large numbers of patients requiring extensive medical resources. This scenario can overwhelm local EMS and hospital resources. Emergency Physicians must remain attentive to the possibility and consequences of blast injuries.

In general, most blast injuries cared for by US emergency departments tend to be accidental, including firework mishaps, unintended occupational or industrial fuel eruptions, and unforeseen mine explosions. In many parts of the world, however, the reality persists of deadly, dormant, nondetonated, military incendiary devices such as land mines and hand grenades. Such devices cause significant numbers of civilian casualties years after local hostilities cease. During wartime, injuries arising from explosions frequently outnumber those from gunshots; many victims are innocent civilians.

Once notified of a possible bombing or explosion, consider immediately activating hospital disaster and

contingency plans, including preparations to care for anywhere from a handful to hundreds of victims. Explosive devices inflict bodily harm by a variety of mechanisms, with multiple provisos ultimately determining the number of victims injured and killed. A case in point is that detonation forces deemed low by most standards can trigger the collapse of a building, crushing and maiming victims inside and nearby.

Pathophysiology

Blast injuries traditionally are divided into 4 categories: primary, secondary, tertiary, and miscellaneous injuries. A patient may be injured by more than one of these mechanisms.

Frequency

- **In the US:** Incidence is sporadic and infrequent. Cases tend to be grouped several (up to hundreds) at a time and have the ability to temporarily overwhelm a local health care system.
- **Internationally:** Incidence is sporadic. Frequency depends on the political (terrorism, military) and economic (occupational health and safety priorities) stability of the region.

Mortality/Morbidity

- Mortality rates vary widely. Injury is caused both by direct blast overpressure (primary blast injury) and by a variety of associated factors.
- Mortality is increased when explosions occur in closed or confined spaces (eg, terrorist bus bombings) or under water. Land mine injuries are associated with a high risk of below- and above-the-knee amputations. Fireworks-related injuries prompt an estimated 10,000-12,000 ED visits in the US annually, with 20-25% involving either the eye or hand.
- Presence of tympanic membrane (TM) rupture indicates that a high-pressure wave (at least 40 kilopascal [kPa], 6 psi) was present and may correlate with more dangerous organ injury. Theoretically, at an overpressure of 100 kPa (15 psi), the threshold for lung injury, TM routinely ruptures; however, a recent Israeli case series of 640 civilian victims of terrorist bombings contradicts traditional beliefs about a clear correlation between the presence of TM injury and coincidence organ damage. Of 137 patients initially diagnosed as having isolated eardrum perforation who were well enough to be discharged, none later developed manifestations of pulmonary or intestinal blast injury. Furthermore, 18 patients with pulmonary blast injuries had no eardrum perforation.

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Clinical

History

- If possible, determine the patient's location relative to the center of the explosion.
 - An explosion that occurs in an enclosed space (including a building, mine, or a relatively lightly constructed enclosed space such as a bus) or in water tends to cause more serious injury.
 - Intensity of an explosion pressure wave declines with the cubed root of the distance from the explosion. A person 3 m (10 ft) from an explosion experiences 9 times more overpressure than a person 6 m (20 ft) away. Proximity of the person to the explosion is an important factor in a primary blast injury.
 - Blast waves are reflected by solid surfaces; thus, a person standing next to a wall may suffer increased primary blast injury.
- Industrial accidents and terrorist explosions may be associated with the release of toxic and/or radioactive materials. The Federal Bureau of Investigation (FBI) is particularly concerned about the possibility that a terrorist could attach a radioactive substance (eg, a radiopharmaceutical or part of an old x-ray machine) to a conventional explosive device, causing radiation contamination of the scene and casualties.
 - Question plant managers, fire department officials, emergency medical services (EMS) personnel, and law enforcement personnel about these possibilities.
 - EMS agencies should check for radiation contamination at the scene of a deliberately caused explosion. In addition, hospital personnel should screen persons who have been exposed to deliberate explosions for radioactivity with a Geiger counter or similar radiation dosimeter. Each hospital has a radiation safety officer (usually a radiology technician) who can assist with this task.

Physical

- Examine lungs for evidence of pulmonary contusion and pneumothorax.
 - Assume that a patient's wheezing associated with a blast injury is from pulmonary contusion.
 - Other causes of wheezing in this setting may include inhalation of irritant gasses or dusts, pulmonary edema from myocardial contusion, and adult respiratory distress syndrome (ARDS).
- Abdominal injuries from explosions may be occult, and serial examinations are often required.
 - A recent large Israeli case series found that abdominal injuries occurred only as a result of massive trauma. This finding may be the result of selection bias, as all the explosions in their series occurred in open air. Air is a poor conductor of blast-wave energy, thus those who were subjected to enough energy to damage abdominal organs probably were situated near the explosive devices.
 - Other authors have reported occult injuries to both solid and hollow abdominal organs in people injured by closed-space explosions and blast injuries occurring in water.

Causes

- Primary blast injury
 - These injuries are caused solely by the direct effect of blast overpressure on tissue. Since air is easily compressible by pressure while water is not, this overpressure almost always affects air-filled structures.
 - Pulmonary barotrauma is the most common fatal primary blast injury. This includes pulmonary contusion, systemic air embolism, and free radical-associated injuries such as thrombosis, lipoyxygenation, and disseminated intravascular coagulation (DIC). ARDS may be a result of direct lung injury or of shock from other body injuries.
 - Acute gas embolism (AGE), a form of pulmonary barotrauma, requires special attention. Air emboli most commonly occlude blood vessels in the brain or spinal cord. Resulting neurologic symptoms must be differentiated from the direct effect of trauma.
 - Intestinal barotrauma is more common with underwater than air blast injuries. Although the colon usually is affected most, any portion of the GI tract may be injured.
 - The ear is the organ most susceptible to primary blast injury. Acoustic barotrauma commonly consists of TM rupture. Hemotympanum without perforation also has been reported. Ossicle fracture or dislocation may occur with very high-energy explosions.
- Tertiary blast injury
 - These injuries are caused by individuals flying through the air and striking other objects, generally from high-energy explosions.
 - Unless the explosion is of extremely high energy or focused in some way (eg, through a door or hatch), a person with tertiary blast injury usually is very close to the explosion source.
 - Together with secondary blast injuries, this category accounted for most of the pediatric casualties in Oklahoma City. There was a high incidence of skull fractures (including 17 children with open brain injuries) and long-bone injuries including traumatic amputations.

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Differentials

Abdominal Trauma, Blunt

Abdominal Trauma, Penetrating

Acute Respiratory Distress Syndrome

Burns, Chemical

Burns, Ocular

Burns, Thermal

Compartment Syndrome, Extremity

Conversion Disorder
Diaphragmatic Injuries
Disaster Planning
Disseminated Intravascular Coagulation
EMS and Mass Gatherings
EMS and Terrorism
Hazmat
Neck Trauma
Pneumothorax, Tension and Traumatic
Postconcussive Syndrome
Pregnancy, Trauma
Shock, Hemorrhagic
Shock, Hypovolemic
Spinal Cord Injuries
Toxicity, Carbon Monoxide
Trauma, Lower Genitourinary
Trauma, Upper Genitourinary
Venous Air Embolism

Other Problems to be Considered

Barotrauma

Cardiac tamponade

Compartment syndrome, abdominal

Flail chest

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Workup

Lab Studies

- Judicious use of the laboratory is essential for accurate diagnosis in the mass-casualty situation. Do not overwhelm the laboratory with screening or protocol laboratory tests of little clinical benefit.
- Most patients injured by significant explosions should have a screening urinalysis.
- If the explosion occurred in an enclosed space or was accompanied by fire, test

carboxyhemoglobin (HbCO) and electrolytes to assess acid/base status.

- Pulse oximetry readings may be misleading in cases of CO poisoning. When in doubt, apply 100% oxygen by tight-fitting face mask until CO levels can be measured.
- Exposure to CN, a product of incomplete combustion of plastics, is difficult to measure directly. CN exposure often accompanies CO poisoning. Consider CN poisoning in patients exposed to combustion in an enclosed space who have an anion gap metabolic acidosis. Treatment for CN poisoning should be started for significantly ill patients while awaiting confirmatory test results. Sodium thiosulfate (and, in Europe, hydroxocobalamin) is safe and appropriate empiric therapy.

Imaging Studies

- Perform CXRs on patients who have been exposed to high overpressure and are therefore at high risk for primary blast injury. This group of patients includes all patients with TM rupture from blast injury.
- If significant abdominal pain is present, consider an acute abdominal x-ray series (flat and upright films) or a CT scan of the abdomen to detect pneumoperitoneum from enteric rupture.
- No practical, sensitive test exists for intestinal hematoma. The best available test, abdominal CT scan, often misses this condition. Since intestinal hematoma can take 12-36 hours to develop, such symptoms as increased pain or vomiting should determine decisions about testing.

Other Tests

- If there is any question of radiation or chemical contamination, arrange to test and decontaminate patients and equipment.
 - Most fire departments' hazardous materials teams have the training and equipment to perform this task.
 - Notify the hospital's radiation safety officer (often the chief technician for the radiology department's nuclear medicine section) for assistance screening victims for radiation contamination. Contact hospital public relations to work with the press.

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Treatment

Prehospital Care

EMS personnel must aggressively seek any available information regarding possible chemical and/or radiation contamination of the scene and all those injured. EMS personnel are responsible for activating

appropriate disaster and/or hazardous material responses as early as possible.

Emergency Department Care

- Examine the lungs, abdomen, and TMs of all patients exposed to a significant explosion.
- Penetrating wounds (secondary blast injury), blunt trauma (tertiary/secondary blast injury), and burns receive standard treatment.
- Shrapnel wounds (secondary blast injury) are treated as low-velocity gunshot wounds.
- Because pulmonary contusion tends to evolve over several hours, a period of observation and repeat x-ray may be necessary.
- If abdominal pain persists or vomiting develops, seriously consider admitting the patient for observation. Intestinal hematoma may be impossible to detect in the ED.
- WP burns require unique management.
 - Initial management of WP-contaminated burns consists of copious lavage of the area, removing identifiable particles (which should be placed in water to prevent further combustion), and covering the area with saline-soaked gauze to prevent further combustion.
 - Use of a Wood lamp in a darkened resuscitation suite or operating room may help identify WP particles in the wound.
 - Definitive treatment consists of a rinse using 1% copper sulfate (CuSO_4) solution and removing the WP particles. Copper sulfate combines with phosphorous particles to create a blue-black cupric phosphide coating. This impedes further WP combustion and makes particles easier to find.
 - Rinse the contaminated burn with copper sulfate solution, remove WP particles, then use copious saline lavage to rinse off the copper sulfate.
 - Never apply copper sulfate as a dressing. Excess copper sulfate absorption can cause intravascular hemolysis and renal failure.
 - WP injury can lead to hypokalemia and hyperphosphatemia with ECG changes, cardiac arrhythmias, and death. Place the patient on a cardiac monitor and closely track serum calcium levels. Intravenous (IV) calcium may be required.
 - Moistened face masks and good ventilation help protect patients and medical personnel from the pulmonary effects of phosphorous pentoxide gas.
 - Naturally, avoid the use of flammable anesthetic agents and excessive oxygen around WP.

Consultations

- Consult a trauma surgeon, otolaryngologist, pulmonary medicine specialist, orthopedist, plastic surgeon, urologist, and toxicologist, as required.

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Medication

Research into the pathophysiology of primary blast injury continues. Blood levels of thromboxane A₂, prostacyclin (PGI₂), and leukotrienes are markedly increased in humans who incur blast injuries. Select free-radical scavengers and inhibitors of inflammatory pathways are promising in phase one animal trials.

Although animal models suggest that bradycardia and hypotension observed in primary blast injury may be vagally mediated, it would be premature to recommend atropine at this time.

Use of copper sulfate solution for management of burns contaminated with the military munition WP is described in [Emergency Department Care](#).

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Follow-up

Further Inpatient Care

- Limited data prevent establishing the optimal duration of observation. Consider the following guidelines:
 - Persons exposed to open-space explosions who have no apparent significant injury and with normal vital signs and unremarkable lung and abdominal examinations generally can be discharged after 4 hours with instructions to return to the ED if shortness of breath, abdominal pain, vomiting, or other symptoms occur.
 - Persons exposed to significant closed-space explosions, in-water explosions, and those who incur TM rupture are at higher risk of delayed complications. These patients should receive more intensive observation over a longer period. Motivated, reliable, and completely asymptomatic patients may be sent home after 4 hours of observation.
 - Admit to the hospital all patients with significant burns, suspected air embolism, radiation or WP contamination, abnormal vital signs, abnormal lung examination findings, clinical or radiographic evidence of pulmonary contusion or pneumothorax, abdominal pain, vomiting, evidence of renal contusion/hypoxia, or penetrating injuries to the thorax, abdomen, neck or cranial cavity.

Further Outpatient Care

- As symptoms of pulmonary contusion and intestinal hematoma may take 12-48 hours to develop, instruct all discharged patients to return for reevaluation if they develop breathing problems,

increasing abdominal pain, or vomiting.

- Outpatient treatments for blast-related lacerations, burns, contusions, fractures, and other injuries are the same as for these injuries from other causes.
- TM rupture
 - TM rupture by itself does not require specific treatment or hospitalization. Patients should be instructed not to put anything in the affected ear and should be referred to ENT for follow-up care. Remember that neomycin (a component of otic solutions and suspensions) is ototoxic and contraindicated in cases of TM perforation.
 - Most cases of TM perforation heal spontaneously; however, complications such as ossicle disruption, cholesteatoma formation, and development of perilymphatic fistulae are possible. About one third of patients with TM perforation have permanent hearing loss.

Deterrence/Prevention

- Research into garments designed to protect against both primary and secondary blast injuries is promising, although there is little applicability in the civilian setting.

Complications

- Various sequelae of traumatic injuries may occur.

Prognosis

- The prognosis varies based on injury.

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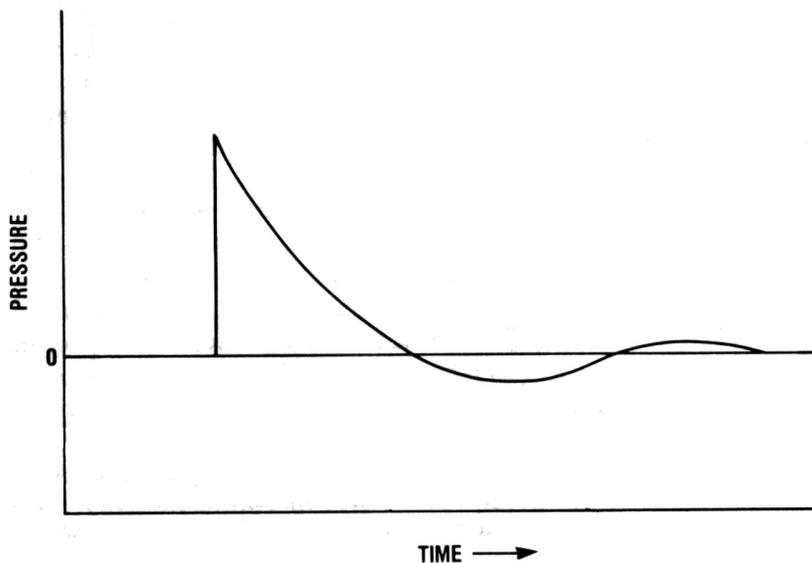
Miscellaneous

Special Concerns

- Pregnancy
 - Pregnant patients with blast injuries warrant special consideration. As the fetus is surrounded by relatively incompressible amniotic fluid, direct injury to the fetus should be uncommon. Injuries to the placenta, however, are probably more common and must be detected. After life-threatening conditions have been stabilized, patients in the second or third trimester of pregnancy who have been exposed to blast injury should be admitted to the labor and delivery area for fetal monitoring and further testing.

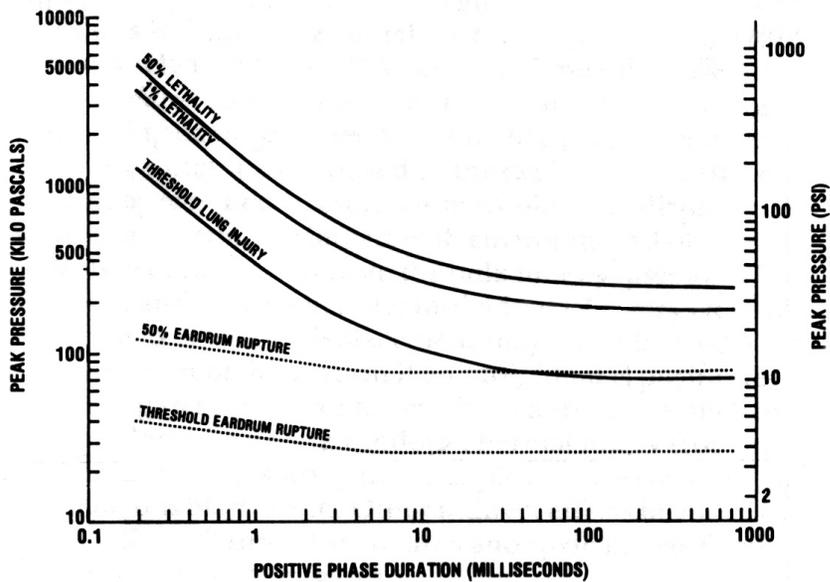
- The placental attachment is at risk for primary blast injury because of the effect of spalling, which occurs when a blast wave passing from a higher-density medium (endometrial muscle) to a lower-density medium (placenta) is partially reflected, damaging tissues at the interface. In addition, tissues of different densities are accelerated by the blast wave at different rates, causing shearing injuries and placental abruption.
- Obtain a Kleihauer-Betke assay (screening test for fetal-maternal hemorrhage) on all women in the second or third trimester of pregnancy. A positive test (detection of fetal cells in the maternal bloodstream) requires mandatory pelvic ultrasound, fetal nonstress test monitoring, and obstetrics/gynecology (OB/GYN) consultation. In addition, Rh immune globulin is administered at a dose of 300 mcg (1 vial) SC per 15 cc of calculated fetal-maternal hemorrhage if the mother's blood type is Rh negative.

Pictures



Picture 1: Idealized graph of a blast pressure wave over time (source: Bowen TE and Bellamy RF, eds, Emergency War Surgery. Washington, DC: United States Government Printing Office, 1988).

Picture type: Graph



Picture 2: Estimated human tolerances for single, sharp, rising blast waves (source: Bowen TE and Bellamy RF, eds, *Emergency War Surgery*. Washington, DC: United States Government Printing Office, 1988).

Picture type: Graph

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Cervical Strain

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Synonyms, Key Words, and Related Terms

neck strain, whiplash, whiplash neck sprain, hyperextension strain to the cervical spine

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Introduction

Background

Cervical strain is a ubiquitous disease seen routinely in the ED. Frequently the result of trauma, this disease causes much distress to patients but has few long-term sequelae. The chief diagnostic challenge is to differentiate cervical strain from other, more serious causes of neck pain.

Pathophysiology

Cervical strain is the result of stretch injury to the muscular and ligamentous elements of the cervical spine. Such injury can occur acutely, as in a motor vehicle accident (MVA), or over time. Repetitive

stress injuries to the cervical spine are common but can be difficult to differentiate from other myofascial syndromes affecting the cervical and upper thoracic region.

Frequency

- **In the US:** Cervical strain is very common.

Mortality/Morbidity

Mortality is not an issue in this musculoskeletal disease. Morbidity from long-term injury can be significant, eg, when pain leads to disuse, resulting in loss of function.

Age

Adults are affected more commonly than children. One possible explanation is that in MVAs, adults receive less head support from car seats than children. Children typically do not exceed the seat height and are often in specialized, protective seating.

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Clinical

History

- Ascertain mechanism of injury.
- Complaints may include the following:
 - Pain
 - Swelling
 - Tenderness
 - Spasm
 - Decreased range of motion
 - Radicular pain patterns

Physical

- Edema of cervical tissues
- Texture changes (eg, ropiness, tightness, increased muscle tension)
- Erythema or increased temperature over involved tissues

- Limited range of motion of the involved area
- Negative clinical examination for fracture (eg, no step-offs, no loss of sensory or motor function, absence of deformity)
- Radicular pain in nonspecific distribution

Causes

- Rapid injury
 - MVAs
 - Falls
 - Assault
 - Other trauma
- Repetitive motion injuries

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Differentials

Abdominal Trauma, Blunt
Dissection, Carotid Artery
Epidural Hematoma
Fractures, Cervical Spine
Headache, Cluster
Headache, Migraine
Headache, Tension
Meningitis
Neck Trauma
Neoplasms, Spinal Cord
Thoracic Outlet Syndrome

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Workup

Imaging Studies

- Perform cervical spine series unless patient meets the following criteria for clinical clearance:

- No altered mental status
- No midline tenderness or complaints of pain
- No significant distracting injury

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Treatment

Prehospital Care

All persons involved in MVAs who sustain neck injuries should, at a minimum, receive cervical collars prior to transport. Many emergency medical service (EMS) protocols require these patients to be placed on a backboard in full spinal precautions. Since MVAs often involve enough force to injure the cervical spine seriously, such precautions are essential to prevent further injury.

Emergency Department Care

- Apply ice to acute strain injuries.
- Administer analgesia and pain control.
- Administer muscle relaxants.
- Soft collars commonly are used but have not been proven effective. A single-blind study with 6-month follow-up conducted by Borchgrevink et al found that patients who received "usual care," with early mobilization and pain control, fared better than similarly treated patients placed in soft collars.

Consultations

Consultations rarely are required for strain injuries; however, follow-up with a physician familiar with rehabilitation therapies is essential.

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Medication

The pharmacology of cervical strain involves pain control and palliation. Nonsteroidal anti-inflammatory drugs (NSAIDs) and Tylenol are mainstays of therapy. Muscle relaxants may prove valuable when

treating severe strain injuries to reduce pain and muscle contracture.

Analgesics

For minor strain injuries, oral outpatient analgesics provide adequate pain control. OTC medications also may suffice.

| | |
|-------------------|--|
| Drug Name | Acetaminophen (Tylenol, Panadol, Aspirin Free Anacin)- Rapidly absorbed from GI tract and distributed widely to all body tissues. Serum half-life is 1-3 h but may be altered in impaired liver function. Posthepatic metabolites excreted in urine. |
| Adult Dose | 375-650 mg PO q4-6h prn or 1000 mg PO q6-8h prn |
| Pediatric Dose | 15 mg/kg/dose PO q4-6h prn |
| Contraindications | Documented hypersensitivity; known G-6-PD deficiency |
| Interactions | Rifampin can reduce analgesic effects; possible increase in hepatotoxicity with use of barbiturates, carbamazepine, hydantoins, or isoniazid |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels of acetaminophen; severe or recurrent pain or high or continued fever possible indication of serious illness |

Nonsteroidal Anti-Inflammatory Agents

NSAIDs control mild to moderate pain and decrease inflammatory reactions. This entire family of medications may ease pain in strain injuries. Tailor dosage on an individual basis.

| | |
|-------------------|---|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Rapidly absorbed orally and distributed widely through body tissues. Serum half-life is 1.8-2 h. Rapidly metabolized and excreted in urine. Complete clearance of single dose occurs in approximately 24 h. |
| Adult Dose | 400-600 mg PO q4-6h prn or 800 mg PO q8h prn |
| Pediatric Dose | Not recommended for pain control; used as antipyretic |
| Contraindications | Documented hypersensitivity; hypersensitivity to aspirin, iodides, or other NSAIDs; peptic ulcer disease, recent GI bleeding or perforation, renal insufficiency, or high risk of bleeding |

| | |
|--------------|--|
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients on anticoagulants (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal or hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy (monitor PT closely); monitor for signs of bleeding |

| | |
|-------------------|--|
| Drug Name | Ketorolac tromethamine (Toradol)- Provides effective control of moderate to severe pain, with higher potency than other NSAIDs, which results in more marked GI upset, platelet inhibition, and renal effects. |
| Adult Dose | 10 mg PO q4-6h prn; not to exceed 40 mg/d 30 mg IV q6h prn 30-60 mg IM q6h prn; repeat doses should be at the 30 mg IM level |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity; not to be administered into CNS; peptic ulcer disease, recent GI bleeding or perforation, renal insufficiency, or high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients on anticoagulants (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Acute renal insufficiency, hyperkalemia, hyponatremia, interstitial nephritis, and renal papillary necrosis possible; increases risk of acute renal failure in patients with preexisting renal disease or compromised renal perfusion; may increase risk of bleeding, monitor patient for signs of bleeding; low WBC counts (rare) usually return to normal during ongoing therapy; discontinue therapy if persistent leukopenia, granulocytopenia, or thrombocytopenia |

Muscle Relaxants

These agents provide adjunctive therapy to allow rest, control pain, and aid physical therapy for

musculoskeletal injury.

| | |
|-------------------|--|
| Drug Name | Orphenadrine citrate (Norflex)- Action not well understood, but its analgesic properties make it clinically effective for muscular injury. |
| Adult Dose | 100 mg PO bid prn 60 mg IM q12h prn |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity, GI obstruction, glaucoma, myasthenia gravis, and cardiospasm |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in cardiac arrhythmias, anxiety, hemodynamic instability, tremors, confusion, and congestive heart failure |

| | |
|-------------------|---|
| Drug Name | Cyclobenzaprine hydrochloride (Flexeril)- Centrally acting skeletal muscle relaxant structurally related to TCAs with similar liabilities. Can be useful adjunct to other therapies for acute musculoskeletal pain. |
| Adult Dose | 10 mg PO tid prn |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity; MAOIs within last 14 d; hyperthyroidism |
| Interactions | Coadministration with MAOIs or TCAs may increase toxicity; may have additive effect when used concurrently with anticholinergics; may enhance effects of alcohol, CNS depressants, and barbiturates |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Caution in angle-closure glaucoma, urinary hesitancy; may impair consciousness and ability to operate machinery |

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Follow-up

Further Outpatient Care

- Follow-up with primary care physician is recommended strongly in cervical strain to facilitate further care.

- Encourage physical therapy.
- Monitor for decreased function secondary to disuse or long-standing injury.

in/Out Patient Meds

- Outpatient medications should include acetaminophen or an NSAID.
- Muscle relaxant medications may be considered adjunctive care.

Deterrence/Prevention

- Avoid unusual postures (eg, painting overhead, sitting in front row at movies) for extended periods of time.
- Avoid chronic straining (eg, using neck to hold telephone, other malposition syndromes) of neck muscles.
- Avoid repetitive motions with neck muscles that may result in strain.

Complications

- Long-term complications of strain injuries generally are related to decreased function secondary to disuse.

Prognosis

- Patients with strain injuries who comply with therapies, including rest, ice, and physical therapy or exercise programs, often make full, rapid recoveries.

Patient Education

- Prior to discharge from ED, make the patient aware of the potential for long-term injury due to noncompliance.
- Advise patient of the benefits of ice versus heat in acute injuries.
- Teach patient basic exercises that enhance mobility and minimize discomfort during the recovery period.
- During follow-up visits, consider a referral to a physical therapist for patients with markedly decreased range of motion or long-standing injury prior to presentation.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to diagnose more severe causes of cervical spine injury
 - Failure to prevent potential injury secondary to impairments caused by medications
 - Failure to avoid medications that alter patient's sensorium, if patient is unlikely to comply during convalescence
-

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Compartment Syndrome, Extremity

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Introduction

Background

Compartment syndrome (CS) is a limb-threatening and life-threatening condition observed when perfusion pressure falls below tissue pressure in a closed anatomic space. The current body of knowledge unequivocally reflects that untreated CS leads to tissue necrosis, permanent functional impairment, and, if severe, renal failure and death.

The original description of the consequences of unchecked intracompartmental pressures is widely attributed to Richard vonVolkman. His 1872 publication documented nerve injury and subsequent contracture from CS following supracondylar fracture. That injury remains known as Volkman contracture.

While long bone fractures are a common cause of CS, vascular injury also is a common antecedent to CS. Approximately 50 years after vonVolkman, Jepson described ischemic contractures in dog hind legs resulting from limb hypertension after experimentally induced venous obstruction. In 1941, Bywaters and Beall reported on the significance of crush injury while working with victims of the London Blitz. These pioneers revealed mechanisms and consequences of CS. Almost 30 years later, in the 1970s, the importance of measuring intracompartmental pressures became apparent.

Owen et al published a series of articles describing the use of the wick catheter for pressure measurement and then documented high compartmental pressures in a variety of circumstances. Almost simultaneously, Matsen published his findings, which are the most commonly annotated group of articles in present literature.

CS has been found wherever a compartment is present: hand, forearm, upper arm, abdomen, buttock, and entire lower extremity. Almost any injury can cause this syndrome, including injury resulting from vigorous exercise.

This article presents current thoughts and findings regarding CS. Most importantly, it urges physicians to maintain a high level of suspicion when dealing with complaints of extremity pain.

Pathophysiology

CS pathophysiology follows the path of ischemic injury. Intracompartmental structures cannot withstand infinite pressure. If fluid is introduced into a fixed volume, pressure rises. Various osseofascial compartments have a relatively fixed volume; introduction of excess fluid or extraneous constriction increases pressure and decreases tissue perfusion, until no oxygen is available for cellular metabolism.

Elevated perfusion pressure is the physiologic response to rising intracompartmental pressure. As intracompartmental pressure rises, autoregulatory mechanisms are overwhelmed and a cascade of injury develops. Tissue perfusion is determined by measuring capillary perfusion pressure (CPP) minus the interstitial fluid pressure.

Normal cellular metabolism requires 5-7 mm Hg oxygen tension; this easily is maintained with the CPP averaging 25 mm Hg and interstitial pressure 4-6 mm Hg. However, rising interstitial pressure overwhelms perfusion pressure.

Matsen demonstrated that as intracompartmental pressure rises, venous pressure rises. When venous pressure is higher than CPP, capillaries collapse. The pressure at which this occurs is under debate; however, intracompartmental pressures greater than 30 mm Hg are generally agreed to require intervention.

At this point, blood flow through the capillaries stops. In the absence of flow, oxygen delivery stops. Hypoxic injury causes cells to release vasoactive substances (eg, histamine, serotonin), which increase endothelial permeability. Capillaries allow continued fluid loss, which increases tissue pressure and advances injury. Nerve conduction slows, tissue pH falls due to anaerobic metabolism, surrounding tissue suffers further damage, and muscle tissue suffers necrosis, releasing myoglobin. End result is loss of extremity and, possibly, loss of life.

Frequency

- **In the US:** Anterior distal lower extremity is the most studied CS. It is quoted as the most common, secondary to its frequency of injury. Ranges of 2-12% have been published. Another frequency cited is that 30% of limbs develop CS following vascular injury; however, this is not well documented and is most likely an estimate. Recently, McQueen retrospectively looked at 164 patients with diagnosed CS; 69% were associated with a fracture, half of those being the tibia.

Mortality/Morbidity

- CS outcome depends on both the diagnosis and the time from injury to intervention.
- Rorabeck and Macnab reported almost complete recovery of limb function if fasciotomy was performed within 6 hours.
- Matsen found necrosis after 6 hours of ischemia, which currently is the accepted upper limit of viability.

Sex

In the retrospective study by McQueen, CS was diagnosed more often in men than in women; however, this likely represents selection bias, since men are more often patients with traumatic injuries.

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Clinical

History

- Suspect CS whenever significant pain occurs in an extremity following an injury.
- As pressure rises and ischemic injury begins, the nerves malfunction.
- Often, the patient complains of severe pain out of proportion to examination and a burning sensation or tightness.
- The traditional 5 Ps (ie, pain, paraesthesia, pallor, poikilothermia, pulselessness) are not diagnostic of CS. Literature warns that, with the exception of pain and paraesthesia, these traditional signs are not reliable, and the presence or absence of them should not affect injury management.
 - Importantly, note these symptoms assume a conscious patient who did not suffer any additional injury that would hinder sensory input (eg, spinal cord injury).
 - In young children, the ability to gather a history of complaints is limited.
- High-velocity injuries particularly are worrisome.
- Determine mechanism of injury.

- Long bone fractures
- High-energy trauma
- Penetrating injuries (eg, gunshot wounds, stabbings) - Often cause arterial injury, which quickly can lead to CS
- Venous injury - May cause CS (do not be misled by palpable pulses)
- Crush injuries
- Vigorous exertion may lead to CS.
 - CS has been found in soldiers and athletes without any trauma. This can be acute or chronic with acute compartment pressures as high as those found in severe trauma.
 - If CS is suspected, check intracompartmental pressure, even without the presence of any trauma.

Physical

- Certain physical signs are associated with CS. After initial symptoms of pain or burning, decreased strength and eventually paralysis of the affected extremity occurs. Follow-up physical examinations are important to determine if any progression of symptoms exists.
- Severe pain at rest or with any movement should raise a red flag.
- Pain with certain movements, particularly passive stretching of the muscles, is the earliest clinical indicator of CS.
- Patient may report pain with active flexion.
- If patient complains of pain, determine if any neural compromise exists.
 - Sensory nerves begin to lose conductive ability, followed by motor nerves.
 - Some nerves may reveal effects of increasing pressure before others.
 - For example, in the anterior compartment of the lower leg, the superficial peroneal nerve quickly is affected, and sensation in the web space between first 2 toes may be lost.
- Compare affected limb to unaffected limb.

Causes

- Myriad of precipitating injuries leading to CS share some pathophysiology.
- Cause of CS is extremely simple; pressure is too high.
- Underlying reason for increasing pressure, as proposed by Mubarek and Hargens, is increased fluid content or decreased compartment size.
- Increased fluid content can be caused by the following:
 - Intensive muscle use (eg, tetany, vigorous exercise, seizures)
 - Burns
 - Intraarterial injection (frequently iatrogenic)
 - Envenomation
 - Decreased serum osmolarity (eg, nephrotic syndrome)
 - Infiltrated infusion
 - Hemorrhage (particularly from large vessel injury)

- Lying on a limb can cause CS.
 - In 1979, Owen et al published a landmark study in which researchers measured intracompartmental pressures in various positions common in drug overdoses.
 - Average pressures of 48 mm Hg with the head resting on forearm, 178 mm Hg when the forearm was under ribcage, and 72 mm Hg when one leg was folded under the other were reported.

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Differentials

Cellulitis
Coelenterate and Jellyfish Envenomations
Deep Venous Thrombosis and Thrombophlebitis
Gas Gangrene
Necrotizing Fasciitis
Peripheral Vascular Injuries
Rhabdomyolysis

Other Problems to be Considered

Differentials listed above often occur concurrently with CS.

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Workup

Lab Studies

- Sequential Multiple Analysis (SMA)-16
- Complete blood count (CBC) with differential
- Creatine phosphokinase (CPK) and urine myoglobin
- Serum myoglobin
- Urine toxicology screen: This subsequently may help define the etiology, but it rarely is helpful in patient treatment.
- Initial urinalysis: This may be positive for blood but negative for RBC on microscopic analysis, which may indicate myoglobin in the urine (rhabdomyolysis).

- Prothrombin time (PT) and activated partial thromboplastin time (aPTT)

Imaging Studies

- X-rays of the affected extremity
- Ultrasound
 - Ultrasound aids in evaluating arterial flow as well as visualizing any deep venous thrombosis (DVT).
 - It is not helpful in diagnosis of CS; however, it aids in the elimination of differential diagnoses.

Other Tests

- Compartment pressure measurement
 - This measure should be at the top of the list when searching for CS; perform it as soon as the diagnosis of CS is considered.
 - A number of commercial model tonometers are available (eg, Stryker, ACE). A number of "build-it-yourself" techniques, without evidence of reliability, also are available; their use is not recommended.

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Treatment

Prehospital Care

- CS can develop rapidly after an arterial injury. Therefore, speed of transport is essential. Perform only the necessary lifesaving procedures in the field if CS is suspected.

Emergency Department Care

- Many cases of CS are due to trauma. Follow advanced trauma life support (ATLS) guidelines to stabilize the patient before attempting to address CS.
- Ischemic injury is the basis for CS. Additional oxygen increases partial pressure of oxygen (PO₂).
- Keeping extremities level with the body decreases limb mean arterial pressure without changing intracompartmental pressure.
- After an elevation of 35 cm, Styf and Wiger measured a decrease in the mean arterial perfusion pressure of 23 mm Hg and no change in intracompartmental pressure.

- Early intervention is extremely important because of irreversible tissue injury approximately 6 hours after onset of CS.
 - Intravenous (IV) hydration is essential.
 - Carefully evaluate multitrauma patients with decreased mental status for CS.

Consultations

- General surgery
- Orthopedic surgery
- Vascular surgery

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Medication

Some authors have advocated the use of mannitol for CS. While its use in rhabdomyolysis is well documented, its use in acute CS is new. More recently, Daniels and Reichman treated an Israeli soldier who developed CS with mannitol. After resolution, he was discharged without a fasciotomy. Unfortunately, intracompartmental pressures were not measured, since the diagnosis was based on limb circumference and nerve conduction studies. Further investigation is warranted in this area.

HBO therapy is a logical choice for CS because it addresses the primary concern of ischemic injury. HBO has many beneficial effects. It reduces edema through oxygen-induced vasoconstriction while maintaining oxygen perfusion and supports tissue healing in a similar mechanism by allowing oxygen delivery when perfusion pressure is low. Reperfusion injury following CS often is voiced as an argument against HBO. However, HBO actually protects against reperfusion injury.

Bouachour performed a well-controlled randomized study with 31 patients following crush injury and demonstrated significant increase in complete healing ($p < 0.0005$) with HBO. Wattel et al have given an appraisal of the current literature regarding HBO therapy for CS and justifiably conclude that studies demonstrate HBO effectiveness in improving wound healing, reducing amputation rate, and lowering surgical procedure rate.

Although HBO currently is only adjunctive therapy because of its limited availability, it should not be ignored. It may extend treatment duration and it may not reverse the CS etiology, but it has been shown to be beneficial.

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Follow-up

Transfer

- Transfer if CS is suspected and any of the following are present:
 - Severe or multiple limb involvement that requires intensive care facilities
 - Inadequate surgical coverage
 - Inability to accurately diagnose CS due to lack of equipment or diagnostic imaging

Complications

- Permanent nerve damage
- Infection
- Loss of limb
- Death
- Cosmetic deformity from fasciotomy

Prognosis

- The prognosis is excellent to poor, depending on how quickly CS is treated and whether or not complications develop.

Patient Education

- Include the following discharge instructions for patients with injuries that predispose them to CS:
 - Timely follow-up examination with appropriate physician is necessary.
 - Immediately call or return to the hospital if severe pain, numbness, burning sensation, or weakness in the affected extremity develops.

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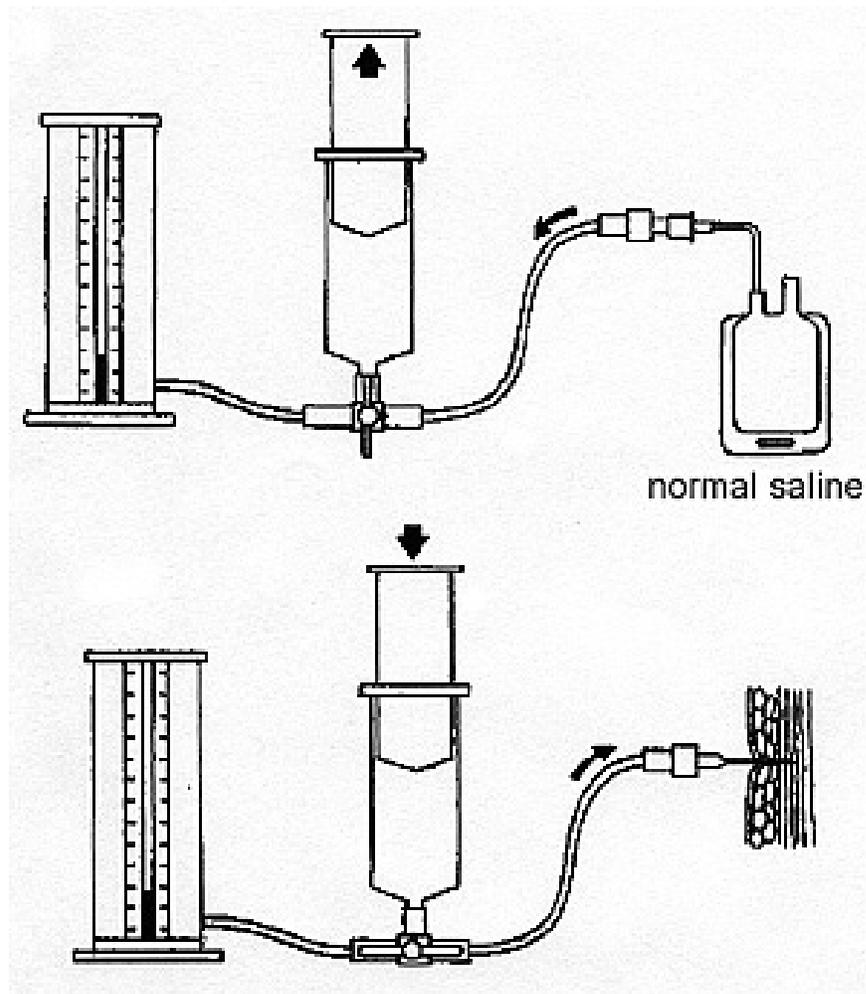
Miscellaneous

Medical/Legal Pitfalls

- Failure to measure compartment pressures

- In 1993, Templeman found an average litigation award of \$280,000 in 8 cases of missed CS.
- In all 8 cases, compartment pressures never were measured.
- Failure to consider potential errors in compartment pressure measurements
 - Equipment errors occur, and needles are misplaced into tendons, fascia, or a wrong compartment.
 - Interpret all pressure readings within the context of the clinical presentation.

Pictures



Picture 1: "

Picture type: Graph

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Diaphragmatic Injuries

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Synonyms, Key Words, and Related Terms

diaphragmatic rupture, diaphragmatic tear

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Introduction

Background

Diaphragmatic injuries are relatively rare and result from either blunt or penetrating trauma. Diagnosis and treatment are similar regardless of mechanism, although many management issues are specific to blunt trauma. Thus, this chapter focuses on blunt injuries and details their specific differences from penetrating injuries.

Diaphragmatic injuries were described first by Sennertus in 1541. Riolfi performed the first successful repair in 1886. Not until 1951, when Carter et al published the first case series, was this injury well understood and delineated.

Pathophysiology

Presently, 80-90% of blunt diaphragmatic ruptures result from motor vehicle crashes (MVCs). Falls and other traumatic events rarely are implicated. The mechanism of rupture is related to the pressure gradient between the pleural and peritoneal cavities. Lateral impact from an MVC is 3 times more likely than any other type of impact to cause a rupture, since it can distort the chest wall and shear the ipsilateral diaphragm. Frontal impact from an MVC can cause an increase in intraabdominal pressure, which results in long radial tears in the posterolateral aspect of the diaphragm, its embryologic weak point.

Review of the historical clinical literature, including the series of Carter et al, reveals that the majority (80-90%) of blunt diaphragmatic ruptures have occurred on the left side. The less common right-sided ruptures have more severe associated injuries and result in greater hemodynamic instability. They required greater force of impact, possibly because the liver provides protection or because of a weakness in the left diaphragm.

An autopsy series, however, revealed that left- and right-sided ruptures occurred almost equally. Most likely these ruptures do occur equally, but the more severe injuries associated with right-sided ruptures cause more deaths and thus a lower rate of patient survival until diagnosis in the hospital. The relative frequencies of right-sided (20-30%) and bilateral (5-10%) ruptures have increased each decade, probably because improvement in trauma care has increased survival rates of patients with significant injuries.

In MVCs, the direction of impact may determine if an injury occurs and on what side. The likelihood of injury is related directly to the direction of impact and the person's position in the car. Persons involved in an ipsilateral impact are more likely to sustain diaphragmatic injury, commonly on the ipsilateral side. In the US and Canada, this is seen as left-sided injuries in drivers and right-sided injuries in passengers.

Blunt trauma typically produces large radial tears measuring 5-15 cm, most often at the posterolateral aspect of the diaphragm. In contrast, penetrating trauma can create small linear incisions or holes, which are less than 2 cm in size and may present late after years of gradual herniation and enlargement.

Penetrating injuries to the chest or abdomen also may injure the diaphragm. This specific injury is seen commonly where penetrating trauma is prevalent. This occurs most often from gunshot wounds but can result from knife wounds. Typically, the wounds are small, although occasionally a shotgun blast or an impalement causes a large defect.

Frequency

- **In the US:** Development of diaphragmatic injury in blunt trauma is relatively rare; these injuries are seen in fewer than 5% of all patients with blunt trauma. The incidence increases each decade,

however, probably because of the increased occurrence of high-speed MVCs. Improved survival rates are probably due to advances in prehospital care, trauma center triage, and early recognition.

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Clinical

History

- Clinical presentation varies depending on the mechanism of injury (ie, blunt vs penetrating) and the presence of associated injuries. Symptoms of diaphragmatic injuries frequently are masked by associated injuries. The diaphragm is integral to normal ventilation, and injuries can result in significant ventilatory compromise. A history of respiratory difficulty and related pulmonary symptoms may indicate diaphragmatic disruption.
- Diaphragmatic tears do not occur in isolation. These patients often have associated thoracic and/or abdominal injuries or may have concomitant head or extremity trauma. The rates for associated injuries in blunt diaphragmatic rupture are as follows:
 - Pelvic fractures in 40%
 - Splenic rupture in 25%
 - Liver laceration in 25%
 - Thoracic aortic tear in 5-10%

Physical

The physical examination should focus initially on airway, ventilation, and circulation, with concomitant management of airway, ventilatory, or circulatory compromise. Examination of the neck and chest should include a particular focus on findings of tracheal deviation (ie, mediastinal shift), symmetry of chest expansion, and absence of breath sounds (ie, lung displacement). Since the incidence of associated injuries is high, physical findings typically are dictated by these other injuries.

- Early diagnosis
 - Diagnosis may not be obvious. It is made preoperatively in only 40-50% of left-sided and 0-10% of right-sided blunt diaphragmatic ruptures. In 10-50% of patients, diagnosis is not made in the first 24 hours.
 - Traumatic diaphragmatic injuries are just one of many injuries that can cause acute respiratory compromise.
 - Physical examination is limited in its utility in diagnosing this injury, but diaphragm injury may be identified by auscultation of bowel sounds in the chest or dullness on percussion of the chest. A penetrating injury to the abdomen with a suggestion of a lung or thoracic

injury indicates transgression of the diaphragm as would a chest injury with any suggestion of abdominal injury.

- The 3 clinical phases of diaphragmatic injuries were first described by Grimes.
 - The first or acute phase begins with the injury.
 - If not diagnosed early, the second or latent phase occurs. This phase is asymptomatic but may evolve into gradual herniation of abdominal contents. The diagnosis may be made later because of complications of herniation of abdominal contents into the pleural cavity.
 - The third or obstructive phase is characterized by bowel or visceral herniation, obstruction, incarceration, strangulation, and possible rupture of stomach and colon. If herniation causes significant lung compression, it can lead to tension pneumothorax. Cardiac tamponade has been described from herniation of abdominal contents into the pericardium. Diaphragmatic paralysis also may occur.

Causes

The 2 primary mechanisms of traumatic diaphragmatic injuries are blunt or penetrating trauma. Blunt traumatic injuries occur most commonly from MVCs or falls. Penetrating injuries most commonly occur from gunshot or knife injuries to the chest or abdomen.

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Differentials

Abdominal Trauma, Blunt

Abdominal Trauma, Penetrating

Pneumothorax, Tension and Traumatic

Other Problems to be Considered

Hemothorax

Pneumothorax

Pulmonary contusion

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Workup

Imaging Studies

- Chest x-ray
 - Chest x-ray is the single most important diagnostic study and may show elevation of the hemidiaphragm, a bowel pattern in the chest, or a nasogastric (NG) tube passing into the abdomen and then curling up into the chest.
 - The liver often protects a right-sided rupture from visceral herniation, and thus these ruptures may appear only as an elevated hemidiaphragm from a partially herniated liver. Left-sided ruptures are more evident when the bowel is herniated into the chest.
 - Chest x-ray in blunt left diaphragmatic injury often shows an abnormal or wide mediastinum, even when the aorta is normal. The mediastinum should be investigated because of the association with aortic injury discussed previously.
- CT scan is usually not helpful because of its poor visualization of the diaphragm. A diagnosis can be made if herniation of abdominal contents is visualized.
- MRI may aid in the diagnosis because it can accurately visualize the diaphragm's anatomy. MRI may be used in a patient in stable condition who has an equivocal diagnosis and no need for laparotomy (some penetrating injuries) or for late diagnosis.
- Thoracoscopy has been used to better visualize the diaphragm when the diagnosis is unconfirmed and laparotomy is not required.
- When considering a delayed diagnosis, chest x-ray and contrast studies (via NG or enema) often are employed. MRI typically is an ideal diagnostic test in this instance.

Other Tests

- Diagnostic peritoneal lavage
 - When diagnostic peritoneal lavage (DPL) is utilized to detect diaphragmatic injury, a false-negative result may occur. An isolated penetrating injury from the chest can cause bleeding into the lesser sac, which may not communicate with the rest of the peritoneal cavity. A DPL in this situation would show no evidence of bleeding.
 - Drainage of lavage fluid from the chest tube has been reported and is a positive result.
 - In blunt trauma, DPL result is often positive because of the associated injuries and not specifically because of the diaphragmatic tear.
 - In penetrating chest injuries, most centers use 10,000 RBC/mm³, a more sensitive criterion than normally used, to limit the number of false negatives.

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Treatment

Prehospital Care

Meticulous attention to management of the ABCs, as with all patients, is the cornerstone for prehospital management of diaphragmatic injuries. The diagnosis rarely is made in the field, and no specific prehospital treatment is required. Treat the associated injuries and ensure adequate airway control and ventilation if signs of respiratory distress are present.

Emergency Department Care

- Focus on resuscitating the patient. As in all trauma patients, the ABCs are most important. Ensure a patent airway, assist ventilation if required, and begin fluid resuscitation if necessary.
- Place an NG tube when possible. This will help in diagnosis if the NG tube appears in the chest on chest x-ray. Aspiration of gastric contents also helps to decompress any abdominal herniation and lessen the abdominoperitoneal gradient that favors herniation into the chest.
- Consider placing a chest tube to drain any associated hemothorax or pneumothorax. Perform this with caution to prevent injury to herniated abdominal contents within the pleural cavity.
- Most surgeons recommend chest tube placement prior to transfer to another facility. If this is not required immediately in the definitive care institution, it may be delayed and completed in the operating room.

Consultations

- Surgical repair is necessary, even for small tears, because the defect will not heal spontaneously. The parietoperitoneal pressure gradients favor enlargement of the defect with herniation of abdominal contents.
- Surgical management usually employs the transabdominal approach to allow a complete trauma laparotomy to search for other injuries. A thoracotomy may be necessary for repair, especially in right-sided injuries or when significant herniation has occurred. In a few situations of isolated penetrating injury where abdominal injury is thought to be unlikely, the repair can be accomplished by thoracotomy or thoracoscopy.

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Follow-up

Complications

- Early deaths usually are a result of associated injuries, not the diaphragmatic tear. Mortality rate ranges from 5-30%.
- Serious morbidity usually is related to reexpansion pulmonary edema or to the laparotomy.
- Paralysis or incoordination of the diaphragm is common, but over 50% of these conditions resolve.
- The late complications of an undiagnosed traumatic hernia include all of the following: bowel herniation, incarceration, and strangulation; tension hemothorax secondary to massive bowel herniation; pericardial tamponade from herniation into the pericardial sac; and diaphragmatic paralysis that may recover after repair.
- Death and significant morbidity rarely are related to delayed diagnosis. However, incarceration of herniated abdominal contents can lead to infarction or rupture with disastrous consequences.

Prognosis

- Generally good with immediate repair

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Miscellaneous

Medical/Legal Pitfalls

- Failure to diagnose or a delayed diagnosis: A delay in diagnosis of a small diaphragmatic tear may result in significant morbidity or death from herniation of abdominal contents.
-

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Dislocations, Ankle

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Introduction

Background

Ankle dislocations occur when significant force applied to the joint results in loss of opposition of the articular surfaces. Because of the large amount of force required and the inherent stability of the joint, dislocation rarely is seen without associated fracture.

Pathophysiology

The ankle joint is designed for a balance of stability and flexibility, particularly the former. Joint stability is provided by close articulation of the talus with the tibia and fibula. The mortise design further enhances the stability of the configuration.

The talus is trapezoidal in shape, with the greater width anteriorly. As the joint moves into plantar flexion, the talus becomes narrower, resulting in a decrease in stability. During normal walking, the ankle joint bears 3-5 times the body weight. This factor increases severalfold during running and jumping activities. As weight is applied on heel strike, the fibula descends to increase stability of the ankle joint.

Mortality/Morbidity

- Associated fractures are the rule rather than the exception.
- Ligamentous disruption varies according to the type of dislocation.
- Neurovascular injury is the principal concern, as with any dislocation. Vascular compromise may result in avascular necrosis of the talus if not promptly reduced. Tented skin may be subject to ischemic necrosis.

Sex

- Dislocations of the ankle are seen more frequently in young males than in any other group. This presumably is related to their increased risk overall for traumatic injury.
- Postmenopausal women are at higher risk for associated fractures. Increased fracture risk probably is related to osteoporotic changes in this subset of patients.

Age

Children and adolescents have the most ankle dislocations.

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Clinical

History

A detailed history regarding the mechanism of injury often helps predict the type of injuries to expect. Furthermore, an understanding of the injury mechanism aids treatment, since an opposite force is required in reduction of the joint. Four types of dislocations are seen around the ankle joint.

- Posterior
 - A posterior dislocation is the most common type of ankle dislocation. The talus moves in a posterior direction in relation to the distal tibia as force drives the foot backward. The wider anterior talus wedges back, resulting in forced widening of the joint.
 - This must be accompanied by either a disruption of the tibiofibular syndesmosis or a fracture of the lateral malleolus. This occurs most commonly when the ankle is plantar flexed.
- Lateral
 - These dislocations result from forced inversion, eversion, or rotation of the ankle.
 - They are associated uniformly with fractures of either the malleoli or distal fibula.

Physical

- Inspection of the ankle reveals significant edema with deformity ranging from trace to obvious. Tenting of the skin by the malleoli may be noted.
- Palpation of the joint reveals tenderness along the joint line, corresponding to areas of capsular or ligamentous disruption.
- In associated fractures, tenderness, deformity, or tenting proximal to the joint may be seen.

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Differentials

Ankle, Soft-tissue Injuries
Dislocations, Foot
Fractures, Ankle
Fractures, Foot

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Workup

Imaging Studies

- Routine radiographic examination of the ankle includes the following views:
 - Anteroposterior
 - Lateral
 - Mortise or oblique views: These are taken with an internal rotation of 10-20 degrees. This places both the medial and lateral malleoli in the same horizontal plane, which provides optimum viewing of the tibial plafond and talar dome.

Procedures

- Reduction of the ankle joint
 - In patients with obvious or complete neurovascular compromise, perform reduction prior to radiographic studies. Prompt reduction is important in reducing the risk of complications related to neurovascular compromise.

- Reduction is accomplished with the knee in flexion to reduce tension on the Achilles tendon. With one hand on the heel and another on the dorsum of the foot, apply traction while maintaining countertraction at the knee. Entrapment of the tibialis posterior tendon (or of a fracture fragment within the joint space) may result in an irreducible dislocation.
- Anesthesia includes Bier block, spinal block, conscious sedation with narcotics and/or benzodiazepines, or general anesthesia. Bier block is the preferred method because of its efficacy and risk profile, although time may not permit in cases of vascular compromise.

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Treatment

Prehospital Care

Prehospital personnel should immobilize the joint following standard procedure for any extremity injury. If neurovascular compromise is identified in the field by exam, revealing a cold, discolored, and pulseless or desensate foot, the joint should be realigned unless transport time is brief. This is accomplished by in-line traction with countertraction. Traction or splinting should be maintained en route to the hospital. Intravenous (IV) opioids should be used to make this procedure more tolerable for the patient. If IV opioids are unavailable, IV benzodiazepine is an alternative.

Emergency Department Care

- Early reduction is essential since delay may increase risk of neurovascular compromise or damage to articular cartilage. In patients with vascular compromise, perform reduction prior to radiologic examination.
- Postreduction radiographs should confirm proper joint alignment. Appropriate pain management is the greatest contribution an emergency physician can make to the patient's care. Postreduction splinting is discussed below.

Consultations

Dislocations of the ankle are, by definition, unstable. These require an immediate orthopedic consultation for internal fixation of any associated fractures and repair of capsular or ligamentous tears.

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Medication

Drugs used to treat the pain associated with dislocations include analgesics and anxiolytics.

Analgesics

Pain control is essential for quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained injuries.

| | |
|-------------------|---|
| Drug Name | Fentanyl citrate (Duragesic, Sublimize)- Narcotic analgesic with greater potency and much shorter half-life than morphine sulfate. DOC for conscious sedation analgesia. With short duration (30-60 min) and ease of titration, an excellent choice for pain management and sedation. Easily and quickly reversed by naloxone. After initial dose, subsequent doses should not be titrated more frequently than q3h or q6h. |
| Adult Dose | 0.5-1 mcg/kg/dose IV/IM q30-60min |
| Pediatric Dose | <2 years: 2-3 mcg/kg/dose IM/IV q30-60min 2-12 years: 1-2 mcg/kg/dose IV/IM q60min >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hypotension, respiratory depression, constipation, nausea, emesis, and urinary retention; idiosyncratic reaction, known as chest wall rigidity syndrome, may require neuromuscular blockade to increase ventilation |

| | |
|-------------------|--|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. Different strengths available. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity |

| | |
|-------------|--|
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4,000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

| | |
|-------------------|--|
| Drug Name | Oxycodone and aspirin (Percodan)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose oxycodone |
| Contraindications | Documented hypersensitivity; liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma; due to association of aspirin with Reye syndrome do not use in children (<16 y) who have the flu |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity; may potentiate anticoagulant effects of warfarin |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Duration of action may increase in elderly; caution in renal or liver impairment, peptic ulcer disease, and erosive gastritis |

| | |
|-------------------|---|
| Drug Name | Acetaminophen and codeine (Tylenol-3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg/dose (based on codeine content) PO q4-6h or 1-2 tab q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose (based on codeine content) PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

Anxiolytics

Patients with painful injuries usually experience significant anxiety. Anxiolytics allow the clinician to administer a smaller analgesic dose to achieve the same effect.

| | |
|-------------------|--|
| Drug Name | Lorazepam (Ativan)- Sedative hypnotic in benzodiazepine class that has short onset of effect and relatively long half-life. By increasing GABA, a major inhibitory neurotransmitter, may depress all levels of CNS, including limbic and reticular formation. When patient needs to be sedated for >1 day this medication is excellent. Monitor patient's blood pressure after administering dose and adjust as necessary. |
| Adult Dose | 1-10 mg/d IV divided bid/tid; not to exceed 4 mg/dose |
| Pediatric Dose | 0.05-0.1 mg/kg IV slowly over 2-5 min; may repeat a dose of 0.05 mg/kg IV slowly |
| Contraindications | Documented hypersensitivity; preexisting CNS depression; hypotension; narrow-angle glaucoma |
| Interactions | Alcohol, phenothiazines, barbiturates, or MAOIs may increase CNS toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in renal or hepatic impairment, myasthenia gravis, organic brain syndrome, or Parkinson disease |

| | |
|-------------------|--|
| Drug Name | Diazepam (Valium)- Depresses all levels of CNS, including limbic and reticular formation, possibly by increasing activity of GABA, a major inhibitory neurotransmitter. Individualize dosage and increase cautiously to avoid adverse effects. |
| Adult Dose | 5-10 mg PO/IV/IM q3-4h; repeat q2-4h prn; not to exceed 30 mg in 8-h period |
| Pediatric Dose | 0.05-.3 mg/kg/dose IV/IM over 2-3 min; repeat in 2-4 h prn; 0.12-0.8 mg/kg/d PO divided q6-8h; not to exceed 10 mg/dose |
| Contraindications | Documented hypersensitivity; narrow-angle glaucoma |
| Interactions | Phenothiazines, barbiturates, alcohols, or MAOIs may increase CNS toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution with other CNS depressants, low albumin levels, or hepatic disease (may increase toxicity) |

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Follow-up

Further Inpatient Care

- Patients with ankle dislocations should be admitted to the orthopedic surgery service for intraoperative repair.

Transfer

- In hospitals without an orthopedic surgeon, transfer to an appropriately equipped facility for operative intervention. The joint should be reduced and splinted prior to transfer.

Complications

- Nonunion or malunion
- Synostosis
- Entrapment of the tibialis posterior tendon or of a fracture fragment
- Cartilaginous injury
- Osteochondral fractures of the talar dome
- Joint stiffness and decreased range of motion
- Arterial injury (anterior and posterior tibial, peroneal)
- Compartment syndrome (rare)

Prognosis

- Dislocated ankles should not be expected to return to premorbid function. The amount of force and level of capsular disruption required to dislocate the inherently stable joint results in significant injury with lasting effects. To a limited extent, prompt intervention can reduce the risk of complications.

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Dislocations, Elbow

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Introduction

Background

The elbow joint displays an elegant balance between stability and mobility. While allowing a wide range of motion, the joint has an inherent stability that requires a considerable force to dislocate. For this reason, approximately one third of elbow dislocations are associated with fractures of bony components of the elbow.

Dislocations of the elbow fall in frequency just behind dislocations of the finger and shoulder. Most commonly, the elbow dislocates posteriorly. Immediate reduction is essential to reduce the risk of neurovascular or cartilaginous complications.

Pathophysiology

Both posterior and anterior dislocations occur.

Posterior dislocations

A fall on an extended abducted arm is the mechanism of injury seen in posterior dislocations of the elbow. An example of this is someone rollerblading who, falling backward, extends an arm behind to break the fall. Posterior dislocations account for the majority of elbow dislocations.

Anterior dislocations

A strong blow to the posterior aspect of a flexed elbow may result in anterior dislocation of the elbow. This force drives the olecranon forward in relation to the humerus.

Sex

These injuries occur more often in males than in females.

Age

Dislocations occur more commonly in adults, since the same force in children more often results in a supracondylar fracture.

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Clinical

History

Obtain history that includes the mechanism of injury, type and location of pain, amount of immediate dysfunction, treatment prior to arrival in the emergency department, timing of effusion appearance, and history of prior elbow injury.

- Mechanisms - A fall on an extended, abducted arm (posterior) or a direct blow to a flexed elbow (anterior)
- Pain - Intense, focused around the elbow joint
- Extremely limited range of motion
- Effusion

Physical

- Posterior dislocations: Elbow is flexed, with an exaggerated prominence of the olecranon. On palpation the olecranon is displaced from the plane of the epicondyles (as opposed to a

- supracondylar fracture, in which the epicondyles are palpable in the same plane as the olecranon).
- Anterior dislocations: The elbow is held in full extension. The upper arm appears shortened while the forearm is elongated and held in supination.
 - Neurovascular function should be documented in detail before and after reduction. Continued reexamination is essential.

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Differentials

Fractures, Elbow

Fractures, Forearm

Pediatrics, Nursemaid Elbow

Trauma, Peripheral Vascular Injuries

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Workup

Imaging Studies

- Radiography
 - Plain radiographs are essential prior to reduction of the suspected dislocation.
 - Postreduction films should confirm opposition of the joint surfaces and should rule out previously unidentified fractures or entrapment of bony fragments within the joint space.

Other Tests

- Arteriography for cases of suspected vascular injury

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Treatment

Prehospital Care

Prehospital personnel should splint the limb in the position found. Because of the risk of neurovascular injury, field reduction is not indicated. Successful reduction is unlikely without adequate analgesia and sedation. Patients with neurovascular compromise should be transported rapidly to the closest facility.

Emergency Department Care

- Early reduction is essential, since delay may increase risk of neurovascular compromise or damage to articular cartilage.
- The emergency physician should attempt reduction after obtaining appropriate radiologic studies if evidence of vascular compromise is noted or if orthopedic consultation is delayed significantly.
- The following 2 methods commonly are employed for posterior elbow reductions. Be certain that the patient has received adequate analgesic and sedative medications before beginning either procedure.
 - With the elbow flexed to 90 degrees and supinated, apply posterior pressure to the humerus while a second operator applies downward pressure on the proximal forearm. A coupling is felt and heard as the capitellum slides over the coronoid process and the joint realigns.
 - The second method involves placing the patient in the prone position with the humerus resting on the table and the forearm hanging perpendicular to the plane of the table. The humerus should be supported by the table, with padding, just proximal to the elbow joint. Apply 5-10 pounds of weight to the wrist. Reduction should occur over a period of minutes as the muscles relax.

Consultations

- Emergent orthopedic consultation should be sought for all patients with elbow dislocations.
- Vascular surgery consultation may be needed in patients with possible vascular injury.

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Medication

Analgesics and anxiolytics are used to treat the pain associated with dislocations.

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained injuries.

| | |
|-------------------|--|
| Drug Name | Fentanyl citrate (Duragesic, Sublimize)- Narcotic analgesic with more potency and much shorter half-life than morphine sulfate. DOC for conscious sedation analgesia. With short duration (30-60 min) and ease of titration, an excellent choice for pain management and sedation. Easily and quickly reversed by naloxone. After initial dose, subsequent doses should not be titrated more frequently than q3h or q6h. |
| Adult Dose | 0.5-1 mcg/kg/dose IV/IM q30-60min |
| Pediatric Dose | <2 years: 2-3 mcg/kg/dose IV/IM q30-60min 2-12 years: 1-2 mcg/kg/dose IV/IM q60min >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hypotension, respiratory depression, constipation, nausea, emesis, and urinary retention; idiosyncratic reaction, known as chest wall rigidity syndrome, may require neuromuscular blockade to increase ventilation |

| | |
|-------------------|--|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4,000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

| | |
|-----------|--|
| Drug Name | Oxycodone and aspirin (Percodan)- Drug combination indicated for relief of moderately severe to severe pain. |
|-----------|--|

| | |
|-------------------|--|
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity; liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma; due to association of aspirin with Reye syndrome do not use in children (<16 y) who have flu |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity; may potentiate anticoagulant effects of warfarin |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Duration of action may increase in elderly; caution in renal or liver impairment, peptic ulcer disease, and erosive gastritis |

| | |
|-------------------|---|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d acetaminophen or 5 mg of hydrocodone bitartrate/dose >12 years: 750 mg acetaminophen PO q4h; not to exceed 10 mg of hydrocodone bitartrate in a single dose; not to exceed 5 doses per day |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite, which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

Anxiolytics

Patients with painful injuries usually experience significant anxiety. Anxiolytics allow the clinician to administer a smaller analgesic dose to achieve the same effect.

| | |
|-------------------|--|
| Drug Name | Lorazepam (Ativan)- Sedative hypnotic in benzodiazepine class with short onset of effect and relatively long half-life. By increasing activity of GABA, a major inhibitory neurotransmitter, may depress all levels of CNS, including limbic and reticular formation. Excellent medication when patient needs to be sedated for >1 d. Monitor patient's BP after administering dose and adjust as necessary. |
| Adult Dose | 1-10 mg/d divided IV bid/tid; not to exceed 4 mg/dose |
| Pediatric Dose | 0.05 -0.1 mg/kg IV slowly over 2-5 min; may repeat dose of 0.05 mg/kg IV slowly; not exceed 4mg/dose |
| Contraindications | Documented hypersensitivity; preexisting CNS depression; hypotension; narrow-angle glaucoma |
| Interactions | Alcohol, phenothiazines, barbiturates, or MAOIs may increase CNS toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in renal or hepatic impairment, myasthenia gravis, organic brain syndrome, or Parkinson disease |

| | |
|-------------------|---|
| Drug Name | Diazepam (Valium)- Individualize dosage and increase cautiously to avoid adverse effects. By increasing activity of GABA, a major inhibitory neurotransmitter, depresses all levels of CNS, including limbic and reticular formation. |
| Adult Dose | 5-10 mg PO/IV/IM q3-4h; repeat q2-4h prn; not to exceed 30 mg in 8-h period |
| Pediatric Dose | 0.05-0.3 mg/kg/dose IV/IM over 2-3 min; repeat in 2-4 h prn; 0.12-0.8 mg/kg/d PO divided q6-8h; not to exceed 10 mg/dose |
| Contraindications | Documented hypersensitivity; narrow-angle glaucoma |
| Interactions | Phenothiazines, barbiturates, alcohols, or MAOIs may increase CNS toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution with other CNS depressants, low albumin levels, or hepatic disease (may increase toxicity) |

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Follow-up

Further Inpatient Care

- Indications for admission with frequent neurovascular assessment include the following:
 - Children

- Unreliable patients
- Extensive edema
- Evidence of neurovascular compromise

Further Outpatient Care

- Following reduction, splint elbow in at least 90 degrees of flexion using a posterior molded splint.
- Arrange close follow-up care with the orthopedic surgeon.

Transfer

- Patients with dislocations of the elbow should not be transferred until the elbow has been reduced.
- In hospitals without access to an orthopedic surgeon, reduction should be performed by the emergency physician prior to transfer.

Complications

- Brachial artery injury
- Medial nerve injury
- Ulnar nerve injury
- Concomitant fractures
- Avulsion of the triceps mechanism insertion (anterior dislocation only)
- Entrapment of bone fragments within the joint space
- Joint stiffness with decreased range of motion (particularly in extension)
- Myositis ossificans

Prognosis

- Up to 10 degrees limitation in full extension and some limitation in flexion are common, unless an intensive rehabilitation program is instituted.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to find vascular injury: Presence of a distal pulse does not exclude arterial injury. Severe

disruption of the joint results in brachial artery injury in 8% of patients. This complication should be suspected in cases of extreme force, massive swelling, or wide separation of the joint noted on physical or radiologic examination.

- Failure to find nerve entrapment: Loss of postreduction median nerve function should raise the concern of likely nerve entrapment. Immediate orthopedic consult is needed in these cases for operative intervention.
 - Failure to detect spontaneous reduction: Elbow dislocations can reduce spontaneously, presenting a diagnostic dilemma to the emergency physician. A high degree of suspicion is necessary to avoid overlooking the complications associated with elbow dislocations.
-

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Dislocations, Foot

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Introduction

Background

Dislocations of the foot are not common injuries but are potentially incapacitating. The mechanism of injury may vary from a simple fall to a major motor vehicle accident (MVA). A high index of suspicion for these injuries and careful examination of the appropriate radiographs are essential to avoid missing this injury.

Pathophysiology

Anatomy

The foot consists of 26 bones and numerous joints. It can be divided into 3 distinct areas: the hindfoot with the talus and calcaneus; the midfoot with the navicular, cuboid, and cuneiforms; and the forefoot with the metatarsals and phalanges.

The articulations between the hindfoot and midfoot are the midtarsal or Chopart joints. These joints are the talonavicular and the calcaneocuboid joints. The articulations between the midfoot and the forefoot

are termed the Lisfranc joints and consist of the 5 tarsometatarsal joints.

The most important joint in the foot is the subtalar joint between the talus and calcaneus. Normally, 40% of inversion and eversion occurs at this joint. The foot also contains a number of accessory bones. These occasionally are mistaken for avulsion injuries; however, the presence of a smooth cortical surface and lack of associated soft-tissue edema helps to differentiate these normal variants from fractures.

History

Chopart (1743-1795) and Desault are considered 2 of the founders of modern urologic surgery. Interestingly, they, along with Doublet, attended a dinner with the Dauphin of France. All 3 were dead within 5 days. Presumably, they were the unwitting victims of an attempted poisoning of the Dauphin! Jacques Lisfranc (1790-1847) was a French surgeon who described the joint but not the dislocation that now bears his name.

Frequency

- **In the US:** All dislocations in the foot (with the exception of simple dislocations of the toes) are uncommon injuries. The most common of these injuries are probably dislocations that involve the Lisfranc joint complex. The rarity of these injuries makes diagnosis difficult. A significant proportion of the more subtle dislocations most likely are not diagnosed on initial presentation. Dislocations through the Lisfranc joint complex are thought to have an incidence of about 1 in 50,000 persons with orthopedic trauma per year. They represent fewer than 1% of all dislocations.
- **Internationally:** No information is available on international injury rates.

Mortality/Morbidity

Dislocations of the foot may be associated with other significant injuries. For example, death after an MVA is related to the associated injuries. Conversely, morbidity may be considerable after these injuries, especially if diagnosis is delayed, because of the distracting effect of the associated injuries or because of the subtle nature of these injuries. Early reduction and immobilization may reduce morbidity. Some of these injuries are prone to avascular necrosis, especially of the talus; these patients are more likely to experience considerable long-term morbidity.

Sex

Male-to-female ratio is 6:1.

Age

Injury may occur at any age, although the more severe forms of dislocation associated with MVAs are

more common in young adult males.

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Clinical

History

Detailed medical history and history of the events surrounding the injury or appearance of symptoms are essential to identifying the type of injury and predisposition to complicating factors.

- What was the exact mechanism of injury?
- Has the patient been able to bear weight since the injury?
- Does the patient have an underlying medical condition, especially a history of diabetes?
 - Persons with diabetes may have denervation of the foot and are prone to develop Charcot joints. Charcot joints are joints that demonstrate a grossly disorganized structure, deformity, edema, extreme hypermotility, and often remarkably little pain. Function is generally good. Charcot described the arthropathies that now bear his name in a paper that reported his studies of the citizens of La Salpetriere, Paris, in 1882. Charcot joint changes were not associated with diabetes until 1936.
 - Early, accurate recognition of foot injury is particularly important in patients with diabetes, as a delayed diagnosis is associated with the development of Charcot joints.
 - Previously, the most common cause of Charcot joints was tertiary syphilis; however, especially in the Western world, diabetes (or other causes of peripheral neuritis), syringomyelia, or the rare condition of congenital indifference to pain are now the more frequent causes.
 - Current estimates indicate that 2.5% of individuals with diabetes (about 10 million people have diabetes in the US) are afflicted with neuropathic joints. The development of Charcot joint changes in diabetics may be hastened by previous injuries to the foot or ankle, especially if the injuries were complex or were not recognized and treated promptly.
- The presumed mechanism of injury responsible for each type of dislocation is discussed with that dislocation.
- In general, patients who experience dislocations of the foot have other injuries related to the mechanism of injury. A full history of the event should be obtained if possible, either from the patient or from emergency medical technicians (EMTs). As noted above, obtaining a thorough medical history is very important.
- Occasionally, these injuries may occur with minimal trauma. This is especially true with athletes. The history in these cases is usually of increasing pain and edema over a few days, resulting in a significant limitation of mobility. Often the patient gives no definitive history of a single traumatic event.

Physical

- Examination of the foot usually reveals an obvious deformity; however, some dislocations are accompanied by substantial soft-tissue edema. The exact nature of the injury may be unclear until radiographs are taken.
- Neurovascular exam
 - Assess the vascular status. If no pulse is palpable, urgent reduction of the dislocation is required. Confirm the absence of a pulse with Doppler studies in the emergency department (ED) if possible.
 - Mark the position of the pulse on the skin. This simple measure confirms that a pulse was taken and that it was palpable. It also indicates the position for reassessment. Loss of a previously palpable pulse is a sign that urgent reduction is needed.
 - Perform a neurologic examination of the foot.

Causes

- The risk factors for dislocation of the foot are the same as those for any major trauma (ie, youth, alcohol intake, drug intake). Dislocations of the foot, however, can result from an apparently simple fall (eg, twisting one's foot in a hole in the ground when jogging).
- A number of different types of dislocations of the foot exist. These dislocation types are discussed below with a review of their causes.
- Subtalar or peritalar dislocation
 - This is simultaneous dislocation of the talocalcaneal and talonavicular joints. Note that the talus remains in the ankle mortise. It typically is caused by falls from a height, MVAs, and severe twisting injuries.
 - The dislocation may be medial or lateral, although medial dislocation is more common (80%). The navicular bone and forefoot are displaced medially with a medial subtalar dislocation and laterally with a lateral dislocation.
 - These dislocations frequently are associated with fractures of the involved bones.
- Dislocations of the metatarsophalangeal joints and the toes
 - Dislocation of the first toe metatarsophalangeal joint is rare because of the strong ligaments that support this joint.
 - Dislocation of the interphalangeal joint of the great toe is not uncommon.
 - Dislocations of the other metatarsophalangeal joints are not unusual and typically are caused by trauma. The dislocation is most frequently a lateral displacement of the digit on the metatarsal head.
 - Dislocations of the interphalangeal joints occur from direct trauma (eg, stubbing the toe). Although rarely reported in the literature, these injuries are not unusual.
- Isolated fracture dislocation of the navicular on the talus has been described. It occurred following a fall from a height.

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Differentials

Fractures, Ankle

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Workup

Imaging Studies

- Routine radiography of the foot should include 4 views: anteroposterior, lateral, medial oblique, and lateral oblique. The medial oblique offers the best views of the cuneiform and cuboid bones. The lateral oblique provides the best view of the navicular and medial cuneiform.
- CT scan is being used increasingly to help evaluate fractures and dislocations in the foot, and in particular to help evaluate calcaneal and talar fractures.
- MRI often is used to diagnose stress fractures and to evaluate tendons.

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Treatment

Prehospital Care

- When the dislocated foot is seen as one of a number of injuries in a patient with major trauma, management of the other potentially life-threatening injuries takes priority.
- When the dislocation is an isolated injury, immobilize the limb to make the patient as comfortable as possible and transport the patient promptly.
- Control bleeding with direct pressure.
- Cover a compound dislocation with a sterile dressing.

Emergency Department Care

- Immediate management may be dictated by concomitant injuries. Assess neurovascular status of the foot as part of the secondary survey. Consider an urgent reduction of any dislocation causing significant neurovascular compromise.
- In cases of isolated injury, assess and record neurovascular status. Urgent radiographs should be ordered. Make arrangements for referral to an orthopedic specialist for reduction of the dislocation and further management as appropriate.
- Dislocations at the subtalar level often can be repaired with a closed reduction; however, soft tissue occasionally can become entrapped between the bones. If this occurs, an open reduction is required.
- Remember the possibility of compartment syndrome developing after severe injuries to the foot. Often the signs of compartment syndrome may be masked initially by the severe pain related to the injury. Failure to diagnose this problem can result in serious long-term sequelae for the patient, including contractures, deformities, and chronic pain. A high index of suspicion for the complication is required, and measurement of compartment pressures in the foot should be instituted if any findings suggest that this complication is present.
- Dislocations of the toes often can be reduced under local anesthesia or conscious sedation in the ED. Dislocations of the first toe may be difficult to reduce.
- Dislocations at the Lisfranc joint frequently require operative reduction. An orthopedic surgeon should be involved in the care of these injuries.

Consultations

- Management of major life-threatening injuries takes priority. If major injuries are absent, these complex and debilitating injuries need to be managed by an orthopedic specialist.

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Medication

Administer analgesia as appropriate. Ensure adequate coverage against tetanus. If dislocation is compound, broad-spectrum IV antibiotics are required. A cephalosporin generally is the DOC. Dirty wounds may need the addition of an aminoglycoside to target against gram-negative organisms. Injuries heavily contaminated with soil or farmyard waste require penicillin to protect against *Clostridium perfringens*.

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have

sustained injuries.

| | |
|-------------------|---|
| Drug Name | Fentanyl citrate (Duragesic, Sublimaze)- More potent narcotic analgesic with much shorter half-life than morphine sulfate. DOC for conscious sedation analgesia. With short duration (30-60 min) and easy titration, excellent choice for pain management and sedation. Easily and quickly reversed by naloxone. After initial dose, subsequent doses should not be titrated more frequently than q3h or q6h. |
| Adult Dose | 0.5-1 mcg/kg/dose IV/IM q30-60 min |
| Pediatric Dose | <2 years: 2-3 mcg/kg/dose IV/IM q30-60min 2-12 years: 1-2 mcg/kg/dose q60min >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hypotension, respiratory depression, constipation, nausea, emesis, and urinary retention; idiosyncratic reaction, known as chest wall rigidity syndrome, may require neuromuscular blockade in order to increase ventilation |

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| Drug Name | Meperidine (Demerol)- Narcotic analgesic with multiple actions similar to those of morphine, but may produce less constipation, smooth muscle spasm, and depression of cough reflex than similar analgesic doses of morphine. |
| Adult Dose | 50-150 mg PO/IV/IM/SC q3-4h prn |
| Pediatric Dose | 1-1.8 mg/kg PO/IV/IM/SC (0.5-0.8 mg/lb) q3-4h prn; not to exceed adult dose |
| Contraindications | Documented hypersensitivity; concurrent MAOIs; upper airway obstruction or significant respiratory depression; during labor when delivery of premature infant anticipated |
| Interactions | Monitor for increased respiratory and CNS depression with coadministration of cimetidine; hydantoins may decrease effects; avoid with protease inhibitors |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

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| Precautions | Caution in patients with head injuries since meperidine may increase respiratory depression and CSF pressure (use only if absolutely necessary); caution when using postoperatively and with history of pulmonary disease (suppresses cough reflex) Substantially increased dose levels, due to tolerance, may aggravate or cause seizures even if no prior history of convulsive disorders; monitor closely for meperidine-induced seizure activity if prior seizure history |
|-------------|---|

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|-------------------|---|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in the elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

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| Drug Name | Oxycodone and aspirin (Percodan)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity; liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma Because of association with Reye syndrome, do not use in children (<16 y) who have flu |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity; may potentiate anticoagulant effects of warfarin |
| Pregnancy | D - Unsafe in pregnancy |

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| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |

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| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d acetaminophen or 5 mg of hydrocodone bitartrate/dose >12 years: 750 mg acetaminophen q4h; not to exceed 5 doses/d acetaminophen or 10 mg of hydrocodone bitartrate/dose |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

Anxiolytics

Patients with painful injuries usually experience significant anxiety. Anxiolytics allow the clinician to administer a smaller analgesic dose to achieve the same effect.

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| Drug Name | Lorazepam (Ativan)- Sedative hypnotic in benzodiazepine class that has short onset of effect and relatively long half-life. Increasing action of GABA, a major inhibitory neurotransmitter, may depress all levels of CNS, including limbic and reticular formation. Excellent for patients who need to be sedated for longer than 24 h. Monitor patient's BP after administering dose and adjust as necessary. |
| Adult Dose | 2 mg initially or 0.044 mg/kg IV, whichever is smaller; not to exceed 4 mg/dose |
| Pediatric Dose | 0.05-0.1 mg/kg IV slowly over 2-5 min; may repeat dose of 0.05 mg/kg IV slowly; not to exceed 4 mg/dose |
| Contraindications | Documented hypersensitivity; preexisting CNS depression; hypotension; narrow-angle glaucoma |
| Interactions | Alcohol, phenothiazines, barbiturates, and MAOIs increase CNS toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in renal or hepatic impairment, myasthenia gravis, organic brain syndrome, or Parkinson disease |

Antibiotics

Prophylaxis is given to patients with compound dislocations.

| | |
|-------------------|---|
| Drug Name | Cefazolin (Ancef, Kefzol, Zolicef)- First-generation semisynthetic cephalosporin that binds to one or more penicillin-binding proteins, arrests bacterial cell wall synthesis, and inhibits bacterial replication. Primarily active against skin flora, including <i>Staphylococcus aureus</i> . Total daily dosages are same for IV and IM routes. |
| Adult Dose | 2 g IV/IM; not to exceed 12 g/d |
| Pediatric Dose | 25-100 mg/kg/d IV/IM; not to exceed 6 g/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid prolongs effect; aminoglycosides may increase renal toxicity; may yield false-positive urine dip test for glucose |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal impairment; superinfections and promotion of nonsusceptible organisms may occur with prolonged use or repeated therapy |

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| Drug Name | Gentamicin (Gentacidin, Garamycin)- Aminoglycoside antibiotic used for gram-negative bacterial coverage. Commonly used in combination with both an agent against gram-positive organisms and one that covers anaerobes. Used in conjunction with ampicillin or vancomycin for prophylaxis in patients with compound dislocations. Dosing regimens numerous and adjusted based on CrCl and changes in volume of distribution. May be given IV or IM. |
| Adult Dose | 1.5 mg/kg IV; not to exceed 80 mg |
| Pediatric Dose | <5 years with normal renal function: 2.5 mg/kg/dose IV/IM q8h >5 years: 1.5-2.5 mg/kg/dose IV/IM q8h or 6-7.5 mg/kg/d IV/IM divided q8h |
| Contraindications | Documented hypersensitivity; nondialysis-dependent renal insufficiency |
| Interactions | Other aminoglycosides, cephalosporins, penicillins, and amphotericin B may increase nephrotoxicity; enhance effects of neuromuscular blocking agents, thus prolonged respiratory depression may occur; loop diuretics may increase auditory toxicity—possible irreversible hearing loss of varying degrees may occur (monitor regularly) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Narrow therapeutic index (not intended for long-term therapy); caution in renal failure (not on dialysis), myasthenia gravis, hypocalcemia, and conditions that depress neuromuscular transmission; adjust dose in renal impairment |

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|-------------------|---|
| Drug Name | Vancomycin (Vancocin)- Potent antibiotic directed against gram-positive organisms and active against enterococcal species. Used to treat septicemia and skin-structure infections. Used in conjunction with gentamicin for prophylaxis in penicillin-allergic patients with compound dislocations. May need to adjust dose in patients with renal impairment. |
| Adult Dose | 1 g IV infusion over 1 h |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Erythema, histaminelike flushing, and anaphylactic reactions may occur when administered with anesthetic agents; with aminoglycosides, risk of nephrotoxicity may increase above that with aminoglycoside monotherapy; effects in neuromuscular blockade may be enhanced when coadministered with nondepolarizing muscle relaxants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in renal failure, neutropenia; red man syndrome caused by too rapid IV infusion (dose given over a few minutes) but rarely happens when dose given over 2 h or by PO or IP route; red man syndrome not an allergic reaction |

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|-------------------|---|
| Drug Name | Ampicillin (Omnipen, Marcillin)- Used along with gentamicin for prophylaxis in patients with compound dislocations. Interferes with bacterial cell wall synthesis during active replication, causing bactericidal activity against susceptible organisms. Given in place of amoxicillin in patients unable to take medication orally. |
| Adult Dose | 2 g IV/IM |
| Pediatric Dose | 50 mg/kg IV/IM |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid and disulfiram elevate levels; allopurinol decreases effects and has additive effects on ampicillin rash; may decrease effects of oral contraceptives |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal failure; evaluate rash and differentiate from hypersensitivity reaction |

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|----------------|--|
| Drug Name | Penicillin G (Pfizerpen)- Interferes with synthesis of cell wall mucopeptide during active replication, resulting in bactericidal activity against susceptible microorganisms. |
| Adult Dose | 2.4 million U IM as single dose in 2 injection sites |
| Pediatric Dose | 50,000 U/kg IM; maximum of 2.4 million U |

| | |
|-------------------|---|
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid can increase effects; tetracyclines can decrease effects |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Caution in impaired renal function |

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Follow-up

Further Inpatient Care

- Once the diagnosis is confirmed with x-rays, reduce the dislocation at the earliest possible opportunity. This is especially urgent if evidence suggests neurovascular compromise.
- Simple dislocations, such as those of the toes, can be reduced under a ring block. More complex dislocations, especially fracture dislocations, generally require reduction under general anesthesia in the operating room.
- Occasionally, urgent reduction of a dislocation in the ED is necessary to prevent further vascular compromise. Whenever possible, ensure adequate analgesia; conscious sedation may be required. The joint should be reduced using gentle traction and the limb then immobilized. Further therapy or operative intervention may be required after this initial reduction.
- Reduction of these dislocations, especially isolated dislocations of the talus or some of the more complex dislocations of the Lisfranc joint complex, can be very difficult. In all these cases, consulting an orthopedic specialist is always wise. Closed reduction is frequently insufficient to completely reduce these injuries, and open reduction and internal fixation are required.

Further Outpatient Care

- As noted above, except for simple dislocations of the toes, these injuries frequently require the services of an orthopedic surgeon, who is responsible for the long-term follow-up of these patients.

in/Out Patient Meds

- Analgesia is very important. Narcotics may be required. If the dislocation is compound, antibiotics are essential.

Transfer

- Most of these injuries should be managed by an orthopedic specialist. If a specialist is not available, patients should be transferred to the nearest institution able to offer this service.

Deterrence/Prevention

- Many of these injuries are due to MVAs. Strategies to reduce the number of MVAs, such as encouraging and enforcing the drinking and driving laws, will have an impact on the number of these injuries.

Complications

- One of the major complications of dislocations of the foot involves a failure to make the diagnosis. Some of these dislocations can be subtle, especially those around the Lisfranc joint complex. These dislocations often are missed, resulting in significant morbidity.
- Other complications
 - Infection as a result of compound dislocations or, occasionally, as a postoperative complication
 - Long-term stiffness of the foot
 - Foot pain not specifically localized to one area
 - Secondary osteoarthritis
 - Avascular necrosis, especially of the talus bone, after a complete dislocation of the talus
 - Damage to the medial plantar nerve with associated wasting of the intrinsic muscles of the foot (rare)

Prognosis

- Prognosis generally is good.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to diagnose dislocation of the foot. Some dislocations, at the Lisfranc joint complex particularly, can be subtle. Clues to the diagnosis include severe pain and edema of the foot. Careful examination of the appropriate radiographs should reveal the diagnosis, but in some cases, further investigation with CT scan or MRI may be required. As many as 20% of Lisfranc injuries

are thought to be missed on initial presentation.

- Failure to diagnose dislocation of the foot when other, more severe, injuries are present in a multiple-injury victim. The other injuries may be dramatic and distract attention from the foot. A full, detailed secondary survey with frequent reassessment is vital in all patients with multiple injuries.

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Dislocations, Hand

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Introduction

Background

Every emergency physician should be able to identify and manage digital dislocations. While most digital dislocations are uncomplicated, complications may ensue if the joint is reduced or splinted improperly. All complicated digital dislocations require immediate orthopedic consultation.

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Clinical

History

- Either trivial or significant trauma may contribute to digital dislocation. Eliciting the mechanism of injury may help identify injured structures within the digits.
- Gathering additional history with reference to previous injury, disability, and occupation may change how treatment is rendered.

- Forces often associated with digit dislocations include the following:
 - Axial load - May be associated with fractures
 - Direct compression - May be associated with fractures
 - Lateral force - May be associated with lateral dislocation or collateral ligament disruption
 - Hyperextension - May be associated with dorsal dislocation or volar plate injury

Physical

- With significant injury to the digits, a comprehensive examination may be hindered by pain. A thorough visual inspection, accompanied by a good history, helps gather significant information.
- Examination may reveal a gross deformity, ecchymosis, and edema.
- The finger may have reduced spontaneously. Taking a careful history and physically looking for occult signs of injury (eg, joint tenderness) are essential.
- The presence of pain should not persuade the physician to forego an assessment of the digit's range of motion (active and passive) and level of sensation.
 - Some authors advocate examination of the digit with the patient under the influence of anesthesia once the sensory examination has been completed and documented.
 - Benefits of an exam with anesthesia include improved assessment of range of motion and digit stability.

Causes

- Common mechanisms of injury include the following:
 - Industrial injuries
 - Athletic injuries
 - Falls
 - Motor vehicle accidents (MVAs)

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Differentials

Arthritis, Rheumatoid
Fractures, Hand
Gamekeeper Thumb
Hand Injuries, Soft-tissue

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Workup

Imaging Studies

- Edema, tenderness, and/or deformity at the digit should prompt radiographic evaluation. Findings can be subtle; pain out of proportion to x-ray findings should heighten the physician's suspicion for significant injury.
- The following views should be taken:
 - Anteroposterior
 - Lateral
 - Oblique
 - Stress views (occasionally)
 - Postreduction

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Treatment

Emergency Department Care

Some dislocations may not be reducible by closed means because of the interposition of the volar plate or associated ligaments or tendons in the joint. If several attempts at reduction are not successful, consultation and open reduction and internal fixation (ORIF) often is indicated. A thorough assessment of stability should be performed following a successful reduction.

- Interphalangeal joint of the thumb
 - Reductions usually are accomplished via closed means.
 - This particular dislocation may present with associated rupture of flexor pollicis longus.
 - Following evaluation and reduction, immobilize the involved joint with a thumb spica splint. The period of joint immobilization should be brief to avoid joint stiffening.
- Metacarpophalangeal joints of the fingers
 - Dislocation of an MCP joint of the fingers most often involves the index or small finger.
 - Dislocations here are relatively uncommon because of the strength of the periarticular structures.
 - Dislocations may be simple or complex. A complex dislocation nearly always needs open reduction because of an interposed volar plate.
 - Closed reduction may be accomplished by using traction along the axis of the

hyperextended phalanx and firmly pushing the base of the dislocated phalanx toward the MCP joint.

- Assess stability of the joint after reduction and follow by immobilization.
- Again, some controversy exists regarding length and position of immobilization. Some authors recommend early range of motion if no evidence of postreduction instability is observed.
- Distal interphalangeal joint of the fingers
 - The DIP joint of the finger is a very vulnerable area. Surprisingly, dislocations in this area are uncommon because of the strong support of the joint by skin and periarticular structures. With the appropriate intensity of force applied; however, the strong support network is unyielding and the skin may tear, leading to an open dislocation.
 - Reduce the dislocation with longitudinal traction and hyperextension, with firm dorsal pressure on the base of the distal phalanx. Open reduction rarely is needed in this type of dislocation.
 - After the dislocation is reduced, assess stability of the joint to rule out evidence of tendon injury.
 - Immobilize the joint with a dorsal splint in flexion if volar dislocation has occurred without tendon injury, and in extension if the dislocation is dorsal and without tendon injury.

Consultations

Complex and open dislocations should be evaluated by a hand surgeon for open reduction. In addition, those individuals with fracture-related dislocation require further evaluation by a hand surgeon.

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Medication

Drugs used to treat the pain associated with dislocations are generally NSAIDs, analgesics, and anxiolytics.

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained injuries.

| | |
|-------------------|--|
| Drug Name | Acetaminophen (Tylenol, Panadol, aspirin-free Anacin)- DOC to treat pain in patients with documented hypersensitivity to aspirin or NSAIDs and in those with upper GI disease or taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg PO q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; G-6-PD deficiency |
| Interactions | Rifampin can reduce analgesic effects; barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in patients with chronic alcoholism following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; APAP is contained in many OTC products, and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

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| Drug Name | Acetaminophen and codeine (Tylenol-3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tab q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates, since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
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| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen or 5 mg hydrocodone bitartrate/dose >12 years: 750 mg acetaminophen PO q4h; not to exceed 5 doses/d acetaminophen or 10 mg hydrocodone bitartrate/dose |

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|-------------------|---|
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite, which may cause hypersensitivity; caution in patients dependent on opiates, since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. Different strengths available. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

| | |
|-------------------|---|
| Drug Name | Oxycodone and aspirin (Percodan)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity; liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma Due to association of aspirin with Reye syndrome, do not use in children (<16 y) who have the flu |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity; may potentiate anticoagulant effects of warfarin |
| Pregnancy | D - Unsafe in pregnancy |

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|-------------|---|
| Precautions | Duration of action may increase in elderly; caution in renal or liver impairment, peptic ulcer disease, and erosive gastritis |
|-------------|---|

Anxiolytics

Patients with painful injuries usually experience significant anxiety. Anxiolytics allow the clinician to administer a smaller analgesic dose to achieve the same effect.

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|-------------------|--|
| Drug Name | Lorazepam (Ativan)- Sedative hypnotic in benzodiazepine class that has short onset of effect and relatively long half-life. By increasing action of GABA, a major inhibitory neurotransmitter, may depress all levels of CNS, including limbic and reticular formation. Excellent for patients who need to be sedated for >24 h. |
| Adult Dose | 1-10 mg/d PO divided bid/qid; not to exceed 4 mg/dose |
| Pediatric Dose | 0.05-0.1 mg/kg IV slowly over 2-5 min; may repeat a dose of 0.05 mg/kg IV slowly |
| Contraindications | Documented hypersensitivity; preexisting CNS depression; hypotension; narrow-angle glaucoma |
| Interactions | Alcohol, phenothiazines, barbiturates, or MAOIs increase CNS toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in renal or hepatic impairment, myasthenia gravis, organic brain syndrome, or Parkinson disease |

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

These agents are most commonly used for the relief of mild to moderately severe pain. Although the effects of NSAIDs in the treatment of pain tend to be patient specific, ibuprofen is usually the DOC for initial therapy. Other options include ketoprofen and naproxen.

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|-------------------|---|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- DOC for treatment of mild to moderately severe pain, if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, inhibiting prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |

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| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants—monitor PT regularly and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in CHF, hypertension, and decreased renal or hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for relief of mild to moderately severe pain and inflammation. Administer small dosages initially to patients with small body size, the elderly, and those with renal or liver disease. Doses higher than 75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe patient for response. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 14 years: 0.1â€1 mg/kg PO q6-8h > 12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants—monitor PT regularly and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in CHF, hypertension, and decreased renal or hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|------------|---|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, which decreases prostaglandin synthesis. |
| Adult Dose | 500 mg PO initial dose, followed by 250 mg PO q6-8h; not to exceed 1.25 g/d |

| | |
|-------------------|--|
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants—monitor PT regularly and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

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Follow-up

Complications

- Instability, joint stiffness, hyperextension, and flexion deformity may develop as a result of the dislocation or damaged periarticular structures. Additionally, overly aggressive attempts at reduction of a dislocation can lead to fracture of the digit.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to take special precautions and considerations. As with other dislocations of the

extremities, however, the majority of hand dislocations are fairly obvious as a result of the associated deformity.

- Failure to consider an occult fracture. If x-rays are obtained and no identifiable fracture is visible, yet the patient remains in a significant amount of discomfort, an occult fracture may be present. Proper splinting and urgent referral may be indicated.
- Failure to consider a growth plate injury. A child or adolescent with open growth plates who remains in pain even though x-rays reveal no fracture may have a growth plate injury. Proper splinting and urgent referral may be indicated.

Special Concerns

- Every emergency physician should have a firm understanding of the acute management of simple dislocations of the digits. Historical, physical, and radiographic findings often guide the management of the dislocation. When the dislocation is complicated, consult with and/or refer to a hand surgeon. Generally, reduced dislocations without evidence of instability and near-normal range of motion can be treated by brief immobilization and subsequent referral.

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Dislocations, Hip

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Introduction

Background

Trauma is the most common etiology of hip dislocation seen in the ED. Although children may sustain a hip dislocation from relatively minor trauma, such as athletic activities, adult hip dislocations generally are associated with more severe mechanisms of injury. Since the energy required to dislocate an adult's hip is significant, the most common etiologies are motor vehicle accidents (MVAs) and falls from a significant height.

Consequently, many patients with hip dislocation have multiple injuries that may take precedence in the resuscitation sequence. Conversely, the physical findings of a hip dislocation may be overlooked on initial resuscitation of a trauma patient, especially an unconscious one. The secondary trauma survey should include an assessment of the hips and other large joints.

Three main types of hip dislocation

- Traumatic dislocation of a previously normal hip

- Dislocation of a prosthetic hip
- Developmental dysplasia of the hip resulting in spontaneous and often chronic dislocation

Pathophysiology

The hip is a ball and socket joint with the inherent stability associated with such joints. In addition, the hip has tremendous reinforcement by ligaments, the joint capsule, and large muscle insertions that provide additional stability. Consequently, a large amount of force is required to dislocate the hip.

A hip dislocation is a true orthopedic emergency. The incidence of subsequent avascular necrosis (AVN) of the femoral head is a time-dependent phenomenon, becoming more likely to occur if relocation is delayed beyond 6 hours. The hip may dislocate posteriorly (the most common), anteriorly, or centrally through the acetabulum into the pelvis. It may be a simple dislocation or a fracture/dislocation involving the acetabulum or the head, surgical neck, or shaft of the femur.

Posterior hip dislocations occur typically during MVAs, especially head-on collisions, when the knees of the front seat occupant strike the dashboard. Energy is transmitted along the femoral shaft to the hip joint. If the leg is struck while in an adducted position, a posterior dislocation may result. If the leg is in neutral or an abducted position when struck, an anterior dislocation or fracture/dislocation may occur. In the latter case, the posterior wall of the acetabulum is fractured, making subsequent reduction less stable.

Anterior dislocation of the hip occurs either from a direct blow to the posterior aspect of the hip or, more commonly, to a force applied to an abducted leg that levers the hip anteriorly out of the acetabulum.

The third type of hip dislocation is a central dislocation in which a direct impact to the lateral aspect of the hip forces the hip centrally through the acetabulum into the pelvis. This is a fracture/dislocation.

Frequency

- **In the US:** Posterior hip dislocations are more common than anterior ones, accounting for almost 90% of hip dislocations. The frequency has decreased in the US with the increased use of automobile safety belts and air bags. Anterior dislocations and central fracture/dislocations account for fewer than 10% of hip dislocations.

Mortality/Morbidity

Mortality associated with hip dislocation is primarily due to associated injuries of the pelvis, head, or thorax. Approximately 50% of patients with hip dislocation have other fractures as well.

- The local venous injury and prolonged immobilization associated with hip dislocations lead to a significant incidence of deep venous thrombosis (DVT) and potentially lethal pulmonary embolus in affected patients.
- Osteoarthritis is a common and potentially disabling complication, occurring in 23-50% of cases.
- AVN is common, occurring in 8-13% of patients.
- Injury to the sciatic nerve occurs in 10-14% of posterior dislocations during the initial trauma or during relocation.
- Anterior dislocations occasionally are associated with injury to the femoral artery or nerve.

Race

Hip dislocations occur more commonly in whites than in other races in the US.

Sex

Hip dislocations are seen more commonly in young males, often associated with MVAs and sports injuries.

Age

Hip dislocations resulting from traumatic injuries (especially MVAs) are more common in those younger than 35 years. Hip dislocations resulting from falls are more common in those older than 65 years.

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Clinical

History

- An anterior or posterior hip dislocation presents with a unique history of applied force.
- Posterior hip dislocations
 - Patient typically relates a history of great force applied to a flexed knee and hip. The conscious patient reports pain in the hip and buttock area.
 - Associated injury of the sciatic nerve may be caused by compression or laceration by bony fragments. Resultant neurologic deficit ranges from pain in the sciatic nerve distribution to loss of sensation in the posterior leg and foot and loss of dorsiflexion (peroneal branch) or plantar flexion (tibial branch) of the foot.
 - Vascular injury is relatively rare with posterior dislocations compared to anterior, but it may result in local hematoma formation. Soft tissues tend to tamponade bleeding before

hemorrhagic shock ensues. The presence of shock should lead to a search for other injuries.

Physical

A neurovascular examination is imperative prior to any attempt at reduction.

- Posterior hip dislocations
 - The affected limb is shortened, adducted, and internally rotated, with the hip and knee held in slight flexion.
 - Patient may be unable to walk or adduct the leg.
 - Signs of vascular or sciatic nerve injury may be present.
 - Pain in hip, buttock, and posterior leg
 - Loss of sensation in posterior leg and foot
 - Loss of dorsiflexion (peroneal branch) or plantar flexion (tibial branch)
 - Loss of deep tendon reflexes (DTRs) at the ankle
 - Local hematoma
- Central dislocation
 - The leg is shortened, abducted or adducted, and internally or externally rotated depending on the type and extent of penetration into the pelvis.
 - The typical posture of the leg with anterior or posterior hip dislocation may not be seen if an associated femoral shaft fracture is present. The leg distal to the fracture assumes a neutral position, masking the usual rotation seen with a dislocation. The incidence of missed hip dislocation is much higher in the presence of a femoral shaft fracture.

Causes

- Posterior dislocations: These usually occur during MVAs, especially head-on collisions, when the knees of the front seat occupant with adducted hips strike the dashboard. This injury also may result from a fall from a significant height.
- Anterior dislocations: Anterior hip dislocations occur when force is applied to an abducted leg that levers the hip anteriorly out of its articulation.
- Central dislocations: Central dislocation occurs when force is transmitted axially along the shaft of the femur, fracturing the hip through the acetabulum. This occurs mainly in falls from a significant height or from lateral impact on the hip.
- General causes
 - The most common cause of a hip dislocation is a MVA, in which a front seat occupant strikes a flexed knee against the dashboard during a head-on impact. Transmitted forces displace the hip posteriorly out of the acetabulum.
 - Patients with hip prostheses may undergo hip dislocation with relatively little trauma, as the ligaments supporting the joint are no longer functioning.
 - Patients with Down syndrome are prone to hip dislocations.

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Differentials

Abdominal Trauma, Blunt
Dislocations, Hip
Fractures, Femur
Fractures, Hip
Fractures, Pelvic
Legg-Calve-Perthes Disease
Pediatrics, Limp

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Workup

Lab Studies

- No laboratory tests specifically assist in the diagnosis of hip dislocation. Depending on the mechanism of injury and the patient's clinical presentation, a variety of tests may be indicated in the overall trauma workup, including the following:
 - Complete blood count (CBC) including serial spun hematocrits
 - Urinalysis
 - Type and cross-match for blood
 - Additional tests appropriate in certain circumstances - Pregnancy test, toxicology screen, coagulation profile, and serum chemistry panel

Imaging Studies

- Plain radiographs
 - Plain radiographs of the pelvis should be obtained routinely in patients with a severe mechanism of injury, such as a MVA or fall from a significant height. Pelvic fractures may occur in as many as 10% of these patients. Alert patients with no distracting injuries and no pelvic pain may not need pelvic films.
 - The appearance of a hip dislocation may be very subtle on a single anteroposterior (AP) pelvis view, because the femoral head may lie in an apparently normal position on this

view even though it is dislocated. Central dislocations usually are seen easily on this view. Associated acetabular fractures may be seen as well.

- If a hip dislocation is suspected, AP and lateral x-rays of the involved hip should be taken. In a posterior dislocation, the femur is adducted and internally rotated, while the head of the femur is situated lateral and superior to the acetabulum. In an anterior dislocation, the femur is abducted and externally rotated while the head of the femur is medial and inferior to the acetabulum.
- X-rays of the hip also may reveal concomitant fractures around the head and neck of the femur.
- A Johnson lateral x-ray of the hip (a lateral x-ray of the hip from the opposite side with the contralateral thigh flexed) can be done without moving the involved hip. This view also may reveal the dislocated position (anterior or poster) of the femoral head.
- Although not a routine part of the trauma workup, oblique views are useful if a hip dislocation is suspected. Oblique (Judet) views of the pelvis show the anterior and posterior walls of the pelvis as well as each acetabular column separately and may reveal a hip dislocation that was not apparent on the AP view.
- Magnetic resonance imaging
 - MRI of the hip is usually impractical in the initial evaluation of a trauma patient. It is, however, the best imaging modality in detecting and assessing AVN of the hip and in detecting undisplaced stress fractures of the femoral neck.
 - MRI is also useful in the diagnosis of bone tumors, osteomyelitis, osteoarthritis, and congenital abnormalities of the hip joint.

Other Tests

- Radionucleotide scanning: This sensitive method detects early AVN. It is currently the criterion standard for diagnosis for AVN, although it is being replaced by MRI, which reveals greater anatomic detail and appears to be equally sensitive.

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Treatment

Prehospital Care

- Patients with hip dislocation often have associated injuries that may take precedence for stabilization both in the field and in the ED. Attempts to reduce the dislocation in the field are ill-advised.
- Establish ABCs with appropriate spinal immobilization.

- If hip dislocation is detected in the field, the patient should be placed on a backboard and allowed to assume the leg position that is most comfortable (ie, hip slightly flexed, leg adducted).
- The patient should be transported to a level of trauma center appropriate for his or her overall clinical status.

Emergency Department Care

- Patients with hip dislocations often have other injuries that require immediate stabilization or emergent investigation in the ED. Reduction of the dislocation may have to wait until more severe injuries are treated. Intravenous (IV) access is indicated to provide fluid resuscitation and administration of analgesia, as well as other medications as needed.
- Once life-threatening injuries have been stabilized or ruled out, evaluate the hip dislocation. Obtain orthopedic consultation whenever practical.
- Reduction of a hip dislocation should be deferred to the orthopedic specialist, if possible. Emergency physicians should be familiar with reduction techniques and should perform the reduction if a specialist is unavailable within a reasonable time period, within a 6-hour window from the time of injury, or if a neurovascular deficit is present.
- Even if an orthopedist attempts a closed reduction, the emergency physician can often provide assistance, such as countertraction, during the procedure and often is responsible for the conscious sedation required to perform a closed reduction. Attempts at closed reduction can be made even with central dislocations. Urgent indications are the presence of neurovascular deficits or delays in care approaching 6 hours.
- Additional radiographs or a CT scan may be useful in delineating the extent and exact nature of the dislocation and the presence of associated fractures, which may complicate the reduction or stability of the joint following reduction.
- If the patient's vital signs and overall clinical condition permit, adequate analgesia is indicated. Reduction is greatly facilitated by the use of conscious sedation, and a variety of medications may be used for this purpose. A combination of agents with muscle relaxant and analgesic effects is optimal. Monitor the patient appropriately during conscious sedation.
- Once sufficient relaxation has been achieved, initiate attempts at closed reduction using one of the following techniques. Make no more than 3 attempts at closed reduction, as the incidence of AVN increases with multiple attempts.
- The particular approach to reduction should be based on the exact nature of the dislocation and the position of the femoral head in relation to the acetabulum, not on the physician's favorite method. For example, a pure superior dislocation may best be treated by simple longitudinal traction.
- Either of 2 techniques is used most often for reducing a simple posterior hip dislocation.
 - Allis maneuver
 - Place patient in supine position under deep conscious sedation (eg, methohexital infusion).
 - Monitor vital signs, cardiac rhythm, and pulse oximetry.
 - While an assistant stabilizes the pelvis with direct pressure, the operator stands on

the bed over the patient.

- Flex the hip and knee to 90 degrees and apply axial traction with gradually increasing force and a rocking motion until the hip relocates.
- Additional lateral traction to the proximal femur may help disengage the femoral head and facilitate reduction.
- Other techniques
 - Reverse Bigelow maneuver: This technique seldom is used in the ED. It involves the application of a firm jerk to a partially flexed thigh while holding the proximal tibia/knee area.
 - Whistler technique: This recently described technique for the ED is relatively simple, and physicians have had success with it. The dislocated hip is relocated using the physician's arm to raise and maneuver the affected leg as the physician's shoulder is raised. The physician's hand rests on the opposite thigh. An assistant provides countertraction on the tibia/fibula.
 - Leg crossing maneuver: Occasionally, a dislocation can be reduced gently by coaxing the patient to gradually cross the affected leg over the other (adduction) and then applying gentle traction to the leg while an assistant guides the femoral head back into position by direct pressure in an anterior direction.
 - Longitudinal traction: This may be adequate to reduce a purely superior dislocation.
- The duration of traction and nonweight-bearing immobilization is controversial. Some evidence suggests that early weight bearing (eg, 2 weeks after relocation) may increase the severity of aseptic necrosis when it occurs. Early weight bearing decreases the incidence of other complications (eg, venous thromboembolism, decubiti), and some studies have found equivalent outcomes with early and delayed weight bearing.
- Indications for open reduction
 - Irreducible dislocation (approximately 10% of all dislocations)
 - Persistent instability of the joint following reduction (eg, fracture/dislocation of the posterior acetabulum)
 - Fracture of the femoral head or shaft
 - Neurovascular deficits that occur after closed reduction

Consultations

Consult an orthopedic surgeon for all hip dislocations.

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Medication

Administer adequate analgesia if the patient's clinical status allows. Conscious sedation with agents that

provide muscle relaxation, amnesia, and analgesia are indicated for ED reductions. General anesthesia may be required for patients with dislocations that are irreducible by closed means as well as those with significant associated fractures, central dislocations, or associated neurovascular injury.

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained injuries.

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|-------------------|---|
| Drug Name | Fentanyl citrate (Duragesic, Sublimaze)- More potent narcotic analgesic with much shorter half-life than morphine sulfate. DOC for conscious sedation analgesia. Excellent choice for pain management and sedation; has short duration (30-60 min) and easy to titrate. Easily and quickly reversed by naloxone. After initial dose, subsequent doses should not be titrated more frequently than q3h or q6h. |
| Adult Dose | 0.5-2 mcg/kg/dose IV/IM |
| Pediatric Dose | <2 years: 2-3 mcg/kg/dose IV/IM q30-60min 2-12 years: 1-2 mcg/kg/dose q60min >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hypotension, respiratory depression, constipation, nausea, emesis, and urinary retention; idiosyncratic reaction, known as chest wall rigidity syndrome, may require neuromuscular blockade in order to increase ventilation |

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| Drug Name | Meperidine (Demerol)- Narcotic analgesic with multiple actions similar to those of morphine. May produce less constipation, smooth muscle spasm, and depression of cough reflex than similar analgesic doses of morphine. |
| Adult Dose | 50-150 mg PO/IV/IM/SC q3-4h prn |
| Pediatric Dose | 1-1.8 mg/kg (0.5-0.8 mg/lb) PO/IV/IM/SC q3-4h prn; not to exceed adult dose |
| Contraindications | Documented hypersensitivity; concurrent MAOIs; upper airway obstruction or significant respiratory depression; during labor when delivery of premature infant anticipated |

| | |
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| Interactions | Monitor for increased respiratory and CNS depression with coadministration of cimetidine; hydantoin may decrease effects; avoid with protease inhibitors |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients with head injuries, since may increase respiratory depression and CSF pressure (use only if absolutely necessary); caution when using postoperatively and with history of pulmonary disease (suppresses cough reflex); substantially increased dose levels, because of tolerance, may aggravate or cause seizures even if no history of convulsive disorders; monitor closely for meperidine-induced seizure activity if prior seizure history |

Anxiolytics

Patients with painful injuries usually experience significant anxiety. Anxiolytics allow the clinician to administer a smaller analgesic dose to achieve the same effect.

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|-------------------|---|
| Drug Name | Diazepam (Valium)- By increasing activity of GABA, major inhibitory neurotransmitter, depresses all levels of CNS including limbic and reticular formation. Individualize dose and increase it cautiously to avoid adverse effects. |
| Adult Dose | 5 mg PO/IV/IM q2-4h prn; not to exceed 30 mg/8h |
| Pediatric Dose | 0.05-.3 mg/kg/dose IV/IM over 2-3 min; repeat in 2-4h prn; 0.12-0.8 mg/kg/d PO divided q6-8h; not to exceed 10 mg/dose |
| Contraindications | Documented hypersensitivity; narrow-angle glaucoma |
| Interactions | Phenothiazines, barbiturates, alcohols, and MAOIs increase CNS toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution with other CNS depressants, low albumin levels, or hepatic disease (may increase toxicity) |

| | |
|-------------------|--|
| Drug Name | Lorazepam (Ativan)- Sedative hypnotic in benzodiazepine class that has short onset of effect and relatively long half-life. By increasing activity of GABA, major inhibitory neurotransmitter, may depress all levels of CNS, including limbic and reticular formation. Excellent medication when patient needs to be sedated for >24 h. |
| Adult Dose | 1-10 mg/d divided bid/tid; not to exceed 4 mg/dose |
| Pediatric Dose | 0.05-.1 mg/kg IV slowly over 2-5 min; may repeat a dose of 0.5 mg/kg IV slowly; not to exceed 4 mg/dose |
| Contraindications | Documented hypersensitivity; preexisting CNS depression; hypotension; narrow-angle glaucoma |

| | |
|--------------|---|
| Interactions | Alcohol, phenothiazines, barbiturates, and MAOIs increase CNS toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in renal or hepatic impairment, myasthenia gravis, organic brain syndrome, or Parkinson disease |

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Follow-up

Further Inpatient Care

- A variety of techniques that accomplish open reduction, acetabular repair, and fixation of associated fractures are beyond the scope of this text.
- Following reduction, obtain repeat AP and lateral x-rays of the hip as well as repeat CT scan or MRI of the hip to verify proper reduction.
- After either open or closed reduction of a hip dislocation, the patient is placed on bed rest with the legs abducted and with skeletal traction designed to keep the hip from displacing posteriorly.
- The duration of traction is approximately 2 weeks, but the recommended nonweight-bearing period is controversial and varies from 9 days to 3 months.

Further Outpatient Care

- Obtain repeat CT or MRI scans 2-3 months after reduction to verify proper location and to screen for complications such as AVN, osteoarthritis, and heterotopic calcification at an early stage.
- Development of AVN or severe osteoarthritis following a hip dislocation may require replacement of the hip with a prosthetic joint.

in/Out Patient Meds

- Appropriate analgesia and sedation are required during hospitalization.
- Nonsteroidal anti-inflammatory medications (NSAIDs) may be required on an outpatient basis.

Transfer

- Once stabilized, patients with multiple trauma may be transferred.
- A patient with an isolated hip dislocation may be transferred providing no neurovascular deficit is suspected and the transfer does not extend the dislocation time longer than 6 hours. Generally, hip dislocations are reduced at the receiving facility and, if necessary, the patient is transferred for

ongoing inpatient care with appropriate immobilization en route.

Complications

- Avascular necrosis of the hip
 - AVN is common, occurring in 8-13% of patients.
 - Early diagnosis and treatment of dislocations decreases the incidence of AVN.
 - The effect of early weight bearing on the occurrence of AVN is controversial; most studies have found that early weight bearing following reduction is associated with more severe AVN but does not appear to increase the incidence.
 - Incidence of AVN is increased with delayed reduction, repeated attempts at reduction, and open reduction (40% versus 15.5% with closed reduction). This may be due to operative trauma or because those dislocations requiring surgery are inherently more severe.
 - Adults develop AVN more commonly than children.
 - AVN may not become apparent on plain radiographs for several months. Earlier diagnosis can be made with MRI or nuclear scans, and these modalities should be considered in a patient who develops late and persistent pain following a dislocation.
- Heterotopic calcification
- Recurrent dislocation
- Ligamentous injury of the knee, other fractures
- Complications of immobilization (DVT, pulmonary embolus, decubiti, pneumonia)
- Sciatic nerve injury (posterior dislocation)
 - Injury to the sciatic nerve occurs in 10-14% of posterior dislocations during the initial trauma or during relocation.
 - Sciatic nerve function should be verified before and after relocation to detect this complication. The finding of sciatic nerve dysfunction mandates surgical exploration to release or repair the nerve.
- Femoral artery injury (anterior dislocation)

Prognosis

- Prognosis of hip dislocation varies with the type of dislocation, associated fractures of the femoral head or acetabulum, and the presence of other injuries. Overall, good to excellent results are obtained in 76-93% of patients. The principal determinants of a poor prognosis are as follows:
 - AVN occurs in 4-21.8% of patients in some reviews and 8-13% in others. The incidence is increased with delays in reduction beyond 6 hours and open reduction. The severity of AVN increases in patients who undergo early weight bearing. AVN is a severe complication that usually requires replacement with a prosthetic hip.
 - Severe osteoarthritis occurs in at least 10% of patients and is more common in older patients. This seems to be an increased incidence compared to populations without hip dislocations of a similar age, and some authors have found the incidence to range from 30-71% after open reduction.

- Injury to either the femoral or sciatic nerve usually consists of a neuropraxia, and eventual recovery of function can be expected in these cases. Permanent injury to these nerves can occur, resulting in disabling deficits.
- Recurrent dislocation is a common complication, because supporting ligaments have been disrupted.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to diagnose hip dislocation can be the basis for medical liability, because delayed diagnosis (beyond 6 hours after dislocation) is associated with a poor prognosis.
- Failure to transfer patient promptly to an orthopedic specialist at an early stage may be the basis for liability. (Since transfer inevitably occurs at an early stage, delayed complications are not usually a source of liability for emergency physicians but may be for the orthopedist.)
- Clinical pitfalls in the management of hip dislocation include the following:
 - Failure to diagnose hip dislocation in the presence of associated femoral shaft fracture
 - Reliance on a single anteroposterior pelvis film, which may result in a missed posterior hip dislocation as the femoral head appears to be in the proper place
 - Ascribing hemorrhagic shock to blood loss associated with a hip fracture (eg, missing associated intrathoracic or intraabdominal injuries)
 - Failure to test femoral and sciatic nerve function and distal perfusion before and after attempts at closed reduction

Special Concerns

- Dislocation of a prosthetic hip
 - Hip prostheses frequently deteriorate over time and may dislocate with minimal trauma, such as crossing the legs. Reducing such dislocations is much less urgent than reducing acute dislocation, as the concern regarding AVN and osteoarthritis is nonexistent.
 - Reduction is accomplished in identical fashion and treatment is the same as for nonprosthetic hips, but these patients can be mobilized to bear weight sooner than those with nonprosthetic hip dislocation.
- Children
 - If an increased acetabular angle is noted, that is, an increased slope in a line drawn from the upper outermost acetabulum to the center of the acetabulum, this is a sign of possible acetabular dysplasia or subluxation. This condition warrants further investigation.

- Children may dislocate a hip more easily and with a lesser mechanism of injury than adults. Interpretation of radiographs is complicated by the presence of open epiphyses. Salter fractures may occur.
 - Reduction should be accomplished in very gentle fashion, under general anesthesia or deep conscious sedation, to avoid producing iatrogenic fracture, slipped capital femoral epiphysis, or other epiphyseal injury.
-

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Dislocations, Interphalangeal

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Introduction

Background

Interphalangeal (IP) joint dislocations of the fingers and toes are common. Typically associated with forced hyperextension or hyperflexion of the digit, they require immediate reduction. The IP joint is a hinge joint that allows only flexion and extension and consists of several ligamentous complexes. The volar plate provides stability against hyperextension injury and dorsal dislocation of the phalanx. It often ruptures during a dorsal dislocation and may be associated with an avulsion fracture at the base of the phalanx. The strong collateral ligament complex resists hyperextension and lateral dislocation injury. The extensor hood complex stabilizes against hyperflexion injury and volar displacement of the phalanx.

Pathophysiology

Forced hyperextension with axial compression causes a dorsal dislocation of the proximal IP (PIP) or distal IP (DIP) joint, in which the middle (or distal) phalanx is dislocated dorsal to the proximal (middle) phalanx. Forced hyperflexion results in a volar IP joint dislocation (eg, where the distal phalanx is dislocated volar to the middle phalanx).

Patients whose digits have neurovascular compromise, an open joint dislocation, ligamentous or volar

plate rupture, joint instability, or an associated fracture should have immediate orthopedic consultation. All finger dislocations should be reevaluated subsequently by an orthopedic or hand specialist to manage potential subtle ligamentous, cartilaginous, or bony injury. A lateral or volar PIP joint dislocation, although rare, requires an orthopedist for possible open reduction with internal fixation. A dislocation of the metacarpophalangeal (MCP) joint, although rare in adults, may be more common in children. MCP dislocation usually requires open reduction by a pediatric orthopedist.

Frequency

- **In the US:** Dorsal PIP dislocation is the most common IP dislocation. Volar IP joint dislocations are relatively uncommon. PIP joint dislocations occur more frequently than DIP joint dislocations.

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Clinical

History

- History usually reveals a traumatic athletic injury or entrapment of the finger between objects. Typically, the finger was jammed or bent backwards during basketball, football, or other sports activity. The patient often experiences diffuse pain, swelling, and tingling.
- Determine the following aspects of the patient's history:
 - Which is the dominant hand of the patient and which hand is injured?
 - What is the patient's occupation?
 - Where did injury occur (eg, job, assault)?
 - How much time has passed since the initial injury?

Physical

- An accurate and detailed examination often requires digital block anesthesia. The clinician should test and document each of the following:
 - Gross deformity, diffuse edema, ecchymosis, and tenderness of the involved digit
 - Possible anesthesia or paresthesia in the distal aspect of the involved digit
 - Range of motion, function, and stability of involved joint
 - Detailed neurovascular examination of entire involved hand
- Skin laceration after a blunt hyperextension injury suggests volar plate rupture.

Causes

- Axial compression or lateral forces directed to the digit
- Forced hyperextension or hyperflexion of digit from traumatic athletic injury, entrapment of finger between objects, or a fall
- Predisposition to ligamentous injury possible in those with lax ligaments (eg, Down syndrome)

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Differentials

Dislocations, Hand
Fractures, Hand
Gamekeeper Thumb
Hand Injuries, Soft-tissue

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Workup

Imaging Studies

- Radiographs
 - Take anteroposterior, true lateral, and oblique radiographs of the affected digit. Obtain 3 views prior to and after reduction (see [Picture 1](#), [Picture 2](#), [Picture 3](#), [Picture 4](#)).
 - Physeal, avulsion, or distal tuft fractures as well as osteochondral fragments are often subtle and seen only on 1 or 2 views.
 - Obtain stress views to assess joint stability.

Procedures

- Administer digital block anesthesia 10-15 minutes before any reduction maneuver.
- Be sure to remove all rings.

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Treatment

Prehospital Care

- Splint, ice, and elevate the affected digit.
- Evaluate neurovascular status before and after transport to the ED.

Emergency Department Care

- Reduction and postreduction procedures
 - With the patient's hand or foot securely braced, grasp dislocated phalanx with dry gauze loosely wrapped around the phalanx (gauze improves grip). Hyperextend joint slightly with gentle longitudinal traction for a dorsal dislocation or hyperflex for a volar dislocation. Gradually push dislocated phalanx into its normal anatomical position.
 - Do not apply vigorous traction in a child, because that may interpose soft tissue or an osteochondral fragment into the distracted joint space and prevent reduction.
 - After reduction, examine the affected joint for flexor-extensor tendon function, active range of motion, localized tenderness, and instability in the medial-lateral and dorsal-volar directions.
 - Immobilize the joint with a foam-padded splint immediately after reduction to prevent redislocation or instability. Immobilize for 14-21 days for a PIP joint dislocation and 10-14 days for a DIP joint dislocation. Buddy taping for 3-6 weeks thereafter allows active range of motion and prevents hyperextension.
 - For a dorsal PIP dislocation, apply the splint dorsally with the joint in 20-30 degrees of flexion.
 - For a volar DIP dislocation, apply the splint only to the DIP joint on the volar aspect; the DIP should be in full extension. Allow the PIP joint full range of motion.
 - In children, the cause of dislocation is more likely ligamentous laxity rather than rupture. Immobilization by buddy taping to an adjacent digit for 10-14 days is an acceptable alternative treatment.
 - Obtain postreduction radiographs. Assess functional stability with stress views. This confirms correct joint alignment and congruity and identifies subtle fractures, especially chip or avulsion fractures.
 - Assess neurovascular status following reduction.

Consultations

- Joint instability or neurovascular compromise after reduction requires immediate orthopedic or hand consultation.
- Because joint instability or dysfunction and subtle ligamentous, cartilaginous, or bony injury often are obscured by extensive edema and pain immediately after the injury, all finger joint

dislocations should be referred for orthopedic or hand specialist evaluation within 2-3 weeks following reduction.

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Medication

NSAIDs, analgesics, and anxiolytics are used to treat the pain associated with dislocations.

Nonsteroidal Anti-Inflammatory Drugs (NsAIDs)

These agents are used most commonly for the relief of mild to moderately severe pain. Although the effects of NSAIDs in the treatment of pain tend to be patient specific, ibuprofen is the DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Ibuprin, Advil, and Motrin)- DOC for treatment of mild to moderately severe pain, if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, inhibiting prostaglandin synthesis. |
| Adult Dose | 400-800 mg/dose PO q6-8h |
| Pediatric Dose | 10 mg/kg/dose PO q8h; not to exceed adult dose |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants (monitor PT carefully and instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in CHF, hypertension, and decreased renal or hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy (instruct patients to monitor for signs of bleeding) |

| | |
|-------------------|--|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for relief of mild to moderately severe pain and inflammation. Administer small dosages initially to patients with small body size, the elderly, and those with renal or liver disease. Doses higher than 75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe patient. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 14 years: 0.1-1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants (monitor PT carefully and instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in CHF, hypertension, and decreased renal or hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy (instruct patients to monitor for signs of bleeding) |

| | |
|-------------------|--|
| Drug Name | Flurbiprofen (Ansaid)- Has analgesic, antipyretic, and anti-inflammatory effects. May inhibit cyclooxygenase enzymes, inhibiting prostaglandin biosynthesis. |
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients on anticoagulants (monitor PT carefully and instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|--|
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |
|-------------|--|

| | |
|-------------------|--|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, decreasing prostaglandin synthesis. |
| Adult Dose | 500 mg PO initial dose, followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants (monitor PT carefully and instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients with injuries.

| | |
|-----------|--|
| Drug Name | Acetaminophen (Tylenol, Panadol, Aspirin-free Anacin)- DOC for treatment of pain in patients with documented hypersensitivity to aspirin and NSAIDs, those with upper GI disease, or those taking oral anticoagulants. |
|-----------|--|

| | |
|-------------------|---|
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg PO q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Rifampin can reduce analgesic effects; barbiturates, carbamazepine, hydantoin, or isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; acetaminophen is contained in many OTC products and combined use with these products may result in cumulative acetaminophen doses exceeding recommended maximum dose |

| | |
|-------------------|--|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4,000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

| | |
|-------------------|--|
| Drug Name | Oxycodone and aspirin (Percodan)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity; liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma; due to association of aspirin with Reye syndrome do not use in children (<16 y) who have flu |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity; may potentiate anticoagulant effects of warfarin |

| | |
|-------------|---|
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Duration of action may increase in elderly; caution in renal or liver impairment, peptic ulcer disease, and erosive gastritis |

| | |
|-------------------|--|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tab q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates, since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|--|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen or 5 mg of hydrocodone bitartrate >12 years: 750 mg acetaminophen PO q4h; not to exceed 5 doses/d acetaminophen or 10 mg of hydrocodone bitartrate |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates, since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

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Follow-up

Further Inpatient Care

- Admission may be warranted as dictated by a hand consultant or concurrent injuries.

Further Outpatient Care

- Apply ice and elevate the digit. Splint at all times.
- The patient should not participate in sports activities involving the hand.
- The patient should have a follow-up evaluation with an orthopedist or hand specialist.

in/Out Patient Meds

- NSAIDs may be taken as needed.

Transfer

- If an orthopedic or hand specialist is not immediately available for consultation, transfer patients whose reductions are unsuccessful or those who have an unstable joint, open joint injury, or associated epiphyseal or avulsion fracture.

Deterrence/Prevention

- Patients may use supportive taping during future sports activities.

Complications

- Complications are rare with early reduction, although persistent pain or swelling is common. Despite appropriate management with rest, ice, and elevation, pain and swelling may persist for 6-12 months.
- Inadequate immobilization after reduction may result in redislocation.
- Prolonged immobilization may result in muscle contracture.
- Volar plate injury may lead to recurrent dislocation with chronic laxity, hyperextensibility (swan-neck deformity on active extension), or flexion contracture (pseudoboutonnière deformity without DIP hyperextension).
- Late or delayed reduction commonly results in loss of joint motion, joint instability, and limitation of hand function.

Prognosis

- The prognosis is excellent with proper reduction and follow-up evaluation by orthopedic or hand specialist.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to perform successful reduction. Typical causes include intraarticular entrapment of the volar plate, extensor hood ligaments, or osteochondral fragment from an associated avulsion fracture. Buttonhole dislocation of the phalangeal neck through the joint capsule also may complicate reduction.
- Failure to diagnose an unstable joint, open joint injury, or associated epiphyseal or avulsion fracture

Special Concerns

- In a patient whose occupation requires manual dexterity, especially if the dominant hand is injured, carefully document range of joint motion and neurovascular status.
-

Pictures





Picture 1: Anteroposterior view of distal interphalangeal (DIP) joint dislocation

Picture type: X-RAY



Picture 2: Lateral view of distal interphalangeal (DIP) joint dislocation

Picture type: X-RAY



Picture 3: Oblique view of distal interphalangeal (DIP) joint dislocation

Picture type: X-RAY





Picture 4: Oblique view of proximal interphalangeal (PIP) joint dislocation

Picture type: X-RAY

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Dislocations, Knee

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Introduction

Background

Knee dislocation is a relatively rare injury but an important one to recognize, since coexistent vascular injury, if missed, often leads to limb loss. In addition, knee dislocation often presents in the context of multisystem trauma or spontaneous relocation, which makes detection more difficult.

[Top](#)

Clinical

History

Knee dislocation is classified according to the position of the tibia relative to the femur. The 5 major types of dislocation, which are illustrated in [Picture 1](#), are as follows:

- Anterior: Anterior dislocation often is caused by severe knee hyperextension. Cadaver research has shown that approximately 30 degrees of hyperextension is required before dislocation will occur.
- Posterior: Posterior dislocation occurs with anterior-to-posterior force to the proximal tibia, such as a dashboard type of injury or a high-energy fall on a flexed knee. [Picture 2](#) shows an x-ray of a posterior dislocation.
- Medial, lateral, or rotatory: Medial, lateral, and rotatory dislocations require varus, valgus, or rotatory components of applied force. A lateral dislocation is illustrated in [Picture 3](#).
- More than half of all dislocations are anterior or posterior, and both of these have a high incidence of popliteal artery injury. Twenty to thirty percent of all knee dislocations are complicated further by open joint injury (see [Picture 4](#)).

Physical

- Most often the affected limb has a gross deformity around the knee with swelling and immobility. Occasionally, the knee will have relocated spontaneously prior to the patient's arrival at the ED. This makes a careful physical examination very important. The finding of varus or valgus instability in full extension of the knee is suggestive of a grossly unstable knee and of a spontaneously reduced dislocation.
- A careful vascular examination is essential, as popliteal artery injury occurs in 35-45% of all knee dislocations. The popliteal artery may be damaged severely in both closed and open dislocations, and such injury must be ruled out in knees that have relocated spontaneously. Palpation of the dorsalis pedis and posterior tibial arteries along with capillary refill evaluation is necessary. The presence of normal pulses does not rule out the presence of significant vascular injury. Coexistent peroneal nerve injury occurs in 25-35% of patients and manifests with decreased sensation at the first webspace with impaired dorsiflexion of the foot.

Causes

- The knee is a very stable joint requiring high-energy trauma to produce dislocation. At least 3 major ligaments must rupture for dislocation to occur. Common mechanisms of injury include the following:
 - Motor vehicle collisions
 - Auto-pedestrian impact
 - Industrial injuries
 - Falls
 - Athletic injuries

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Differentials

Fractures, Femur

Fractures, Knee

Fractures, Tibia and Fibula

[Top](#)

Workup

Imaging Studies

- Plain radiographs
- Arteriogram

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Treatment

Prehospital Care

- Prehospital personnel should splint the extremity and provide rapid transport to a medical facility.
- Perform field reduction for patients with evidence of vascular compromise.

Emergency Department Care

- Reduction procedures
 - Do not delay reduction in limbs with obvious vascular impairment. Only patients with good peripheral pulses should undergo prerelation radiographs.
 - Reduction is straightforward and often easily accomplished in the ED. After adequate sedation, longitudinal traction will relocate the majority of knee dislocations. A postreduction lateral knee dislocation is shown in [Picture 5](#). Posterolateral dislocations are particularly difficult and often require operative reduction.
 - After reduction, splint the lower extremity, apply ice, and keep the knee elevated.

Consultations

Always consult an orthopedic surgeon. In addition, consult a vascular surgeon, even in patients without clinical findings or arteriography suggestive of popliteal arterial injury.

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Medication

NSAIDs, analgesics, and anxiolytics are used to treat the pain associated with dislocations.

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients with injuries.

| | |
|-------------------|--|
| Drug Name | Fentanyl citrate (Duragesic, Sublimaze)- Narcotic analgesic with greater potency and much shorter half-life than morphine sulfate. Excellent choice for pain management and sedation with its short duration time (30-60 min) and ease of titration. Easily and quickly reversed by naloxone. After initial dose, subsequent doses should not be titrated more frequently than q3h or q6h. |
| Adult Dose | 0.5-1 mcg/kg/dose IV/IM q30-60min |
| Pediatric Dose | <2 years: 2-3 mcg/kg/dose IV/IM q30-60min 2-12 years: 1-2 mcg/kg/dose IV/IM q60min >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hypotension, respiratory depression, constipation, nausea, emesis, and urinary retention; idiosyncratic reaction, known as chest wall rigidity syndrome, may require neuromuscular blockade to increase ventilation |

| | |
|-------------------|---|
| Drug Name | Meperidine (Demerol)- Narcotic analgesic with multiple actions similar to those of morphine. May produce less constipation, smooth muscle spasm, and depression of cough reflex than similar analgesic doses of morphine. |
| Adult Dose | 50-150 mg PO/IV/IM/SC q3-4h prn |
| Pediatric Dose | 1-1.8 mg/kg (0.5-0.8 mg/lb) PO/IV/IM/SC q3-4h prn; not to exceed adult dose |
| Contraindications | Documented hypersensitivity; concurrent MAOIs; upper airway obstruction or significant respiratory depression; during labor when delivery of premature infant is anticipated |
| Interactions | Cimetidine may increase respiratory and CNS depression; hydantoins may decrease effects; avoid with protease inhibitors |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients with head injuries, since meperidine may increase respiratory depression and CSF pressure (use only if absolutely necessary); caution when using postoperatively and with history of pulmonary disease (suppresses cough reflex); substantially increased dose levels, due to tolerance, may aggravate or cause seizures even if no prior history of convulsive disorders; monitor closely for meperidine-induced seizure activity if prior seizure history |

| | |
|-------------------|--|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4,000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

| | |
|------------|--|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe -to- pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tab q4h; not to exceed 12 tabs/d |

| | |
|-------------------|--|
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates, since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn pain |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d acetaminophen or 5 mg of hydrocodone bitartrate/dose >12 years: 750 mg acetaminophen PO q4h; not to exceed 5 doses/d acetaminophen or 10 mg of hydrocodone bitartrate/dose |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates, since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Oxycodone and aspirin (Percodan)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity; liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma; due to association of aspirin with Reye syndrome, do not use in children (<16 y) who have flu |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity; may potentiate anticoagulant effects of warfarin |
| Pregnancy | D - Unsafe in pregnancy |

| | |
|-------------|--|
| Precautions | Duration of action may increase in elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4,000 mg/24h of acetaminophen; higher doses may cause liver toxicity |
|-------------|--|

Anxiolytics

Patients with painful injuries usually experience significant anxiety. Anxiolytics allow the clinician to administer a smaller analgesic dose to achieve the same effect.

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|-------------------|---|
| Drug Name | Lorazepam (Ativan)- Sedative hypnotic in benzodiazepine class that has short onset of effect and relatively long half-life. By increasing action of GABA, a major inhibitory neurotransmitter, may depress all levels of CNS, including limbic and reticular formation. Excellent for patients who require sedation for longer than 24 h. Monitor BP after administering and adjust as necessary. |
| Adult Dose | 1-10 mg/d PO/IV/IM divided bid/tid; not to exceed 4 mg/dose |
| Pediatric Dose | 0.05-0.1 mg/kg IV slowly over 2-5 min; may repeat a dose of 0.05 mg/kg IV slowly; not to exceed 4 mg/dose |
| Contraindications | Documented hypersensitivity, preexisting CNS depression; hypotension; narrow-angle glaucoma |
| Interactions | alcohol, phenothiazines, barbiturates, or MAOIs may increase CNS toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in renal or hepatic impairment, myasthenia gravis, organic brain syndrome, or Parkinson disease |

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

These agents are used most commonly for the relief of mild to moderately severe pain. Although the effects of NSAIDs in the treatment of pain tend to be patient specific, ibuprofen is usually the DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

| | |
|----------------|---|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- DOC for treatment of mild to moderately severe pain, if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, inhibiting prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid 12 years: Administer as in adults |

| | |
|-------------------|--|
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants (monitor PT carefully and instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in CHF, hypertension, and decreased renal or hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy (monitor PT carefully and instruct patients to watch for signs of bleeding) |

| | |
|-------------------|--|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for relief of mild to moderately severe pain and inflammation. Administer small dosages initially to patients with a small body size, the elderly, and those with renal or liver disease. Doses higher than 75 mg do not increase its therapeutic effects. Administer high doses with caution and closely observe the patient for response. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 14 years: 0.1-1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants (monitor PT carefully and instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in CHF, hypertension, and decreased renal or hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy (monitor PT carefully and instruct patient to monitor for signs of bleeding) |

| | |
|-----------|---|
| Drug Name | Flurbiprofen (Ansaid, Ocuferen)- Has analgesic, antipyretic, and anti-inflammatory effects. May inhibit cyclooxygenase enzyme, inhibiting prostaglandin biosynthesis. |
|-----------|---|

| | |
|-------------------|--|
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants (monitor PT carefully and instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

| | |
|-------------------|--|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, decreasing prostaglandin synthesis. |
| Adult Dose | 500 mg PO initial dose, followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants (monitor PT closely and instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

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Follow-up

Further Inpatient Care

- Vascular examination findings may be normal in the presence of significant popliteal artery injury. Most authors recommend mandatory arteriograms or operative exploration for all knee dislocations.
- Time is of utmost concern, as vascular repair more than 8 hours after injury carries an amputation rate of greater than 80%. In contrast, operative vascular repair within 8 hours of injury yields a limb-salvage rate of 80%.
- The repair of coexistent popliteal vein injury is controversial. Fasciotomy is recommended after vascular repair, as severe swelling and development of compartment syndrome are common in the postoperative phase.
- Operative repair of nerve injury remains controversial, as a poor prognosis is common with both operative and nonoperative care.
- Operative ligamentous repair is recommended by most authors, as functional results are better than those of nonoperative care.

Transfer

- Patients considered for transfer should have undergone emergency reduction of the knee dislocation. Since time is crucial in salvaging the limb after a vascular injury, transfer should be initiated only if vascular consultation and/or evaluation are not available at the transferring institution or if an arteriogram has been performed and results are normal.

Complications

- Popliteal artery injury
- Popliteal vein injury
- Peroneal nerve injury
- Ligamentous injury
- Compartment syndrome

Prognosis

- When treated expeditiously and appropriately, 60-70% of patients will have a painless, stable

knee. Of the remaining patients, one half will eventually have reasonable function, while the other half will have a chronically unstable and painful knee.

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Miscellaneous

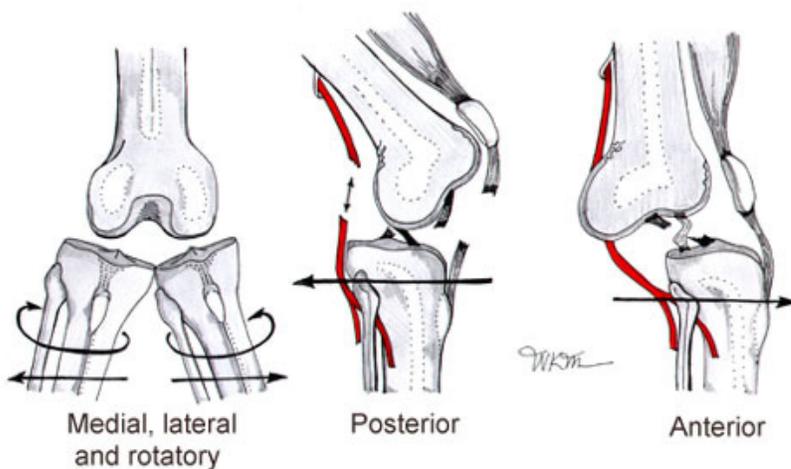
Medical/Legal Pitfalls

- Failure to perform reduction expeditiously
- Failure to consider an associated vascular injury
- Failure to recognize a spontaneously reduced dislocation

Special Concerns

- Knee dislocation is a serious limb-threatening injury that needs to be recognized and treated expeditiously. The possibility of vascular injury must be pursued actively, as it contributes significantly to the morbidity associated with this disorder.
-

Pictures



Picture 1: Types of knee dislocation

Picture type: Graph



Picture 2: Posterior knee dislocation

Picture type: X-RAY



Picture 3: Lateral knee dislocation

Picture type: Photo



Picture 4: Open knee dislocation

Picture type: Photo



Picture 5: Lateral knee dislocation from Picture 2 after reduction

Picture type: Photo

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Dislocations, Mandible

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Synonyms, Key Words, and Related Terms

jaw dislocation

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Introduction

Background

Dislocation of the mandible is an infrequent presentation in the ED. The condition causes the patient discomfort, although most are not in severe pain. In a majority of cases, the mandible can be reduced by using simple techniques. Rarely, a mandibular dislocation may require open reduction under general anesthesia.

Pathophysiology

Certain patients are predisposed to mandibular dislocation by virtue of a shallow mandibular fossa or an underdeveloped condyle. Most cases of dislocation occur spontaneously when the jaw is opened wide (eg, while yawning, yelling, eating, singing, during prolonged dental work) or during a seizure. Traumatic dislocations occur when downward force is applied to a partially opened mandible.

Once the condyle is pried out of its fossa, it lies anterior to the articular eminence and is blocked mechanically from spontaneously reducing. Spasm of the masseter and pterygoid muscles results in trismus and further traps the condyle in its dislocated position. The resulting dislocation may be unilateral or bilateral. In either case, patients are unable to close their mouths completely and often have difficulty speaking. The dislocation is surprisingly not very painful unless an associated mandibular fracture is present.

Mortality/Morbidity

Mortality is not associated with mandibular dislocation. Morbidity is due to an increased tendency for recurrent dislocation and for eventual development of painful osteoarthritis of the traumatized joint or temporomandibular joint (TMJ) syndrome.

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Clinical

History

- The patient often is unable to enunciate clearly; this may make the history difficult to ascertain. Most patients relate an uncomfortable sensation of jaw movement following maximal mouth opening, such as that associated with yawning. This is followed by an inability to close the mouth and variable amounts of pain and discomfort in the area of the mandibular fossa.
- Most patients with traumatic dislocations relate a history of being struck on a partially opened jaw with similar resulting symptoms. Rarely, a patient presents with mandible dislocation as part of a multiple trauma scenario, in which case head injuries, intoxication, or other causes of altered mental status may preclude the patient's complaint. In these rare cases, the dislocation must be discovered on physical examination.
- Patients may have experienced previous dislocations and complain of a recurrent dislocation.

Physical

- The physical findings depend on the duration of dislocation, presence of associated fracture, and whether the dislocation is bilateral or unilateral.

- Unilateral dislocation: The mandible is tilted and lies lower on the affected side. Associated edema, tenderness, and palpable deformity may be present in the TMJ area. The teeth cannot be closed actively or passively.
- Bilateral dislocation: When both mandibular condyles are dislocated, the patient appears to have prognathia (underbite) and has bilateral edema and tenderness in the TMJ areas. The teeth do not close actively or passively as a result of mechanical obstruction. Bilateral masseter spasm often is palpable.
- Associated fractures
 - A fracture at the base of the condyle allows the mandible to slide forward and mimics a dislocation. The pain associated with a fracture is greater than with a simple dislocation.
 - Since reduction of a dislocation may result in an iatrogenic fracture of the condyle, perform this reduction gradually and gently.

Causes

- Patients with a congenitally shallow mandibular fossa or underdeveloped condyle are at risk for dislocation.
- Previous dislocations, whether from preexisting anatomic abnormalities or destruction of stabilizing ligaments, predispose patients to repeated dislocations.
- Rare causes of dislocation include malignancy, osteomyelitis, and rheumatoid arthritis, although the latter condition usually results in ankylosis of the TMJ.
- The immediate cause of dislocation is usually an exaggerated opening of the mouth that pries the condyle out of the fossa.

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Differentials

Fractures, Mandible

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Workup

Imaging Studies

- Plain radiography

- Plain radiographs of the mandible, including bilateral oblique views, virtually always show the affected condyle lying anterior to the articular eminence.
- Obtain radiographs prior to attempts at reduction because of the risk of associated fracture of the mandible.
- A Panorex view of the mandible is most accurate in detecting and characterizing mandibular fractures and reliably detects dislocations; however, the availability of Panorex is variable.

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Treatment

Prehospital Care

No specific treatment is indicated in the field. The decision regarding self-transport versus paramedic transport is based upon factors other than the mandibular dislocation (eg, presence of multiple trauma, patient's level of pain and distress).

Emergency Department Care

- ED treatment is confined to establishing airway patency and adequate ventilation. In a majority of cases, diagnosis and treatment can be rendered in the ED.
- Associated injuries, such as cervical spine injury, take precedence over treatment of the mandible dislocation.
- The diagnosis and reduction of the mandibular dislocation should take place after more life-threatening injuries have been addressed.
- More complex cases, such as irreducible dislocations, chronic dislocations, or those with associated fractures should be treated with analgesics and referred urgently to appropriate specialists.
- Reduction
 - Once adequate radiographs have been obtained that confirm a dislocation and exclude a mandibular fracture, proceed with reduction attempts. Refer patients with mandibular fractures to appropriate specialists.
 - Uncomplicated dislocations can be managed in the ED, generally using conscious sedation. With very recent dislocations, one attempt at reduction can be made without conscious sedation.
 - Conscious sedation is appropriate providing adequate monitoring can be instituted and depending on the experience and training of the physician. Most clinicians prefer to have the patient seated in front of them during the relocation, which makes deep conscious

sedation impractical. Combining single doses of a benzodiazepine and opiate usually sedates the patient and relaxes masseter spasm sufficiently to allow manipulation of the mandible. Local anesthesia into the TMJ also may facilitate reduction. Alternatively, the patient may be placed in a supine position with the clinician standing behind the patient's head and pushing caudally.

- Reduction technique 1: Clinician faces the patient.
 - Place gloved thumbs on the retromolar pad (ie, behind the last molar) on either side of the mandible and grasp the inferior surface of the mandible with the fingers on each side.
 - Exert downward pressure on the lower molars to free the condyle from its entrapped position anterior to the articular eminence.
 - Ease the mandible posteriorly to return it to its anatomic position.
 - Successful reduction is usually evident, as the teeth close rapidly due to masseter spasm, and a palpable (and sometimes audible) clunk occurs on reduction. The physician must beware of having her or his thumbs trapped in an inadvertent human bite as the mandible relocates. Because of this risk, the common practice of wrapping both thumbs with gauze and pressing down on both molars is discouraged.
- Confirmation of relocation: Repeat radiographs to confirm reduction and exclude the possibility of fracture during reduction. Observe the patient for airway patency and monitor vital signs until the effects of the sedatives have worn off. Caution the patient to avoid opening the mouth widely to prevent recurrent dislocation. Barton bandages rarely are used and are indicated primarily in patients who are unable to understand or follow discharge instructions (eg, developmentally delayed). For most patients, the Barton bandage is unnecessary, and instructions to avoid wide mouth opening for several days and to begin range of motion exercises are sufficient.

Consultations

All patients should be referred for follow-up by an otolaryngologist or oral-maxillofacial surgeon. More urgent consultation is indicated for irreducible dislocations or fracture/dislocations of the mandible that may require operative intervention.

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Medication

Sedation and analgesia are indicated if reduction is attempted. The medications traditionally employed for this purpose are diazepam and meperidine. Other conscious sedation protocols can be employed providing the patient maintains an adequate gag reflex. Deep conscious sedation is not desirable, as the

patient should remain seated during relocation. Certain medications that can cause masseter spasm (eg, methohexital, chlorthalidone, phenothiazines) should be avoided, as this complication would prevent relocation of the mandible.

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients with injuries.

| | |
|-------------------|---|
| Drug Name | Meperidine (Demerol)- Narcotic analgesic with multiple actions similar to those of morphine. May produce less constipation, smooth muscle spasm, and depression of cough reflex than similar analgesic doses of morphine. |
| Adult Dose | 1-2 mg/kg IV/IM/SC q3-4h prn |
| Pediatric Dose | 1-1.8 mg/kg (0.5-0.8 mg/lb) IV/IM/SC q3-4h prn; not to exceed adult dose |
| Contraindications | Documented hypersensitivity; concurrent MAOIs; upper airway obstruction or significant respiratory depression; during labor when delivery of premature infant is anticipated |
| Interactions | Cimetidine may increase respiratory and CNS depression; hydantoins may decrease effects; avoid with protease inhibitors |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients with head injuries, since meperidine may increase respiratory depression and CSF pressure (use only if absolutely necessary); caution when using postoperatively and with history of pulmonary disease (suppresses cough reflex); substantially increased dose levels, due to tolerance, may aggravate or cause seizures even if no prior history of convulsive disorders; monitor closely for meperidine-induced seizure activity if prior seizure history |

| | |
|-------------------|--|
| Drug Name | Fentanyl citrate (Duragesic, Sublimaze)- Potent narcotic analgesic with much shorter half-life than morphine sulfate. With short duration (30-60 min) and easy titration, an excellent choice for pain management and sedation. Easily and quickly reversed by naloxone. |
| Adult Dose | 0.5-1 mcg/kg/dose IV/IM q30-60min |
| Pediatric Dose | <2 years: 2-3 mcg/kg/dose IV/IM q30-60min 2-12 years: 1-2 mcg/kg/dose q60min >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |

| | |
|--------------|--|
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hypotension, respiratory depression, constipation, nausea, emesis, and urinary retention; idiosyncratic reaction, known as chest wall rigidity syndrome, may require neuromuscular blockade to increase ventilation |

Anxiolytics

Patients with painful injuries usually experience significant anxiety. Anxiolytics allow the clinician to administer a smaller analgesic dose to achieve the same effect.

| | |
|-------------------|--|
| Drug Name | Diazepam (Valium)- Individualize dosage and increase cautiously to avoid adverse effects. |
| Adult Dose | 5 mg IV/IM q2-4h prn |
| Pediatric Dose | 0.1-0.3 mg/kg IV q4-8h |
| Contraindications | Documented hypersensitivity; narrow-angle glaucoma |
| Interactions | Phenothiazines, barbiturates, alcohols, and MAOIs may increase CNS toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution with other CNS depressants, low albumin levels, or hepatic disease (may increase toxicity) |

| | |
|-------------------|---|
| Drug Name | Lorazepam (Ativan)- Sedative hypnotic in benzodiazepine class that has short onset of effect and relatively long half-life. By increasing action of GABA, a major inhibitory neurotransmitter, may depress all levels of CNS, including limbic and reticular formation. Excellent medication for patients requiring sedation for >24h. Monitor BP after administering dose and adjust as necessary. |
| Adult Dose | 1-10 mg/d IV/IM divided bid/tid; not to exceed 4 mg/dose |
| Pediatric Dose | 0.05-0.1 mg/kg IV slowly over 2-5 min; may repeat a dose of 0.05 mg/kg IV slowly |
| Contraindications | Documented hypersensitivity; preexisting CNS depression; hypotension; narrow-angle glaucoma |
| Interactions | Alcohol, phenothiazines, barbiturates, and MAOIs may increase CNS toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in renal or hepatic impairment, myasthenia gravis, organic brain syndrome, or Parkinson disease |

Follow-up

Further Inpatient Care

- In the rare cases of mandible dislocation that cannot be reduced by the methods described above, closed reduction under general anesthesia or open reduction may be required. Similarly, chronic dislocations or fractures/dislocations of the mandible are best reduced by oral-maxillofacial (OMF) or ear, nose, and throat (ENT) specialists.

Further Outpatient Care

- Successfully relocated mandible dislocations do not require any specific ongoing treatment, although the patient should be cautioned against opening the mouth wide, which could easily cause a recurrence.
- All patients with reduced mandible dislocations should be followed up by an appropriate specialist because of the possibility of jaw instability, ligamentous damage, and chronic TMJ pain.

Transfer

- Patients with dislocation of the mandible can be transferred providing no severe associated injuries are present, vital signs are stable, and the airway is patent.
- In many cases, relocation is simple to perform at the initial ED, and the patient can be referred for ongoing care at another facility, precluding the need for transfer.

Complications

- Serious complications from mandibular dislocation are rare. Several complications are associated with the dislocation and reduction, however.
- Dislocation complications
 - Fracture of the mandibular condyle can occur during dislocation. Open fractures are at risk of infection and osteomyelitis. Interposition of soft tissues may make the dislocation irreducible.
 - Theoretically, massive edema or bleeding into the pharynx may compromise the airway, although this complication has not been reported. Injury to the external carotid artery and facial nerve have been reported.

Prognosis

- As the dislocation occurs in anatomically predisposed individuals and disrupts the joint capsule and ligaments that stabilize the TMJ, recurrent dislocation is very common. Recurrent dislocation often results in osteoarthritis of the TMJ with chronic pain and inflammation.
- Many surgical interventions are available to correct chronic dislocation and painful TMJ syndrome described in the OMF and ENT literature. As many patients with mandible dislocation experience recurrent dislocation, refer all of these patients to an appropriate specialist for follow-up.

Patient Education

- Patients should be instructed to avoid opening their mouths widely to prevent recurrent dislocation.
-

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Dislocations, Shoulder

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Introduction

Background

Shoulder dislocation is documented in Egyptian tomb murals as early as 3000 BC, with depiction of a manipulation for glenohumeral dislocation resembling the Kocher technique. Hippocrates detailed the oldest known reduction method still in use today.

Pathophysiology

The shoulder dislocates more than any other joint. It moves almost without restriction, but pays the price of vulnerability. The shoulder's integrity is maintained by the glenohumeral joint capsule, the cartilaginous glenoid labrum (which extends the shallow glenoid fossa), and muscles of the rotator cuff.

Anterior dislocations account for over 95% of dislocations, with posterior dislocations making up 4% and inferior dislocations about 0.5%. Superior and intrathoracic dislocations are extremely rare.

Frequency

- **In the US:** The shoulder accounts for over half of the major joint dislocations seen in the ED.
- **Internationally:** A Dutch study estimated the incidence of shoulder dislocation at 17 per 100,000.

Sex

Distribution is bimodal, with peak incidence in men aged 20-30 years and women aged 61-80 years.

Age

Shoulder dislocation occurs more frequently in adolescents than children, because the weaker epiphyseal growth plates in children tend to fracture before dislocation occurs. In older adults, collagen fibers have fewer cross-links, making the joint capsule and supporting tendons and ligaments weaker and dislocation more likely. Older adults also fall more frequently.

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Clinical

History

Patients generally complain of severe shoulder pain and decreased range of motion with a history of trauma.

Physical

- Anterior shoulder dislocation
 - Arm is held in slight abduction and external rotation.
 - Shoulder is "squared off" (ie, boxlike) with loss of deltoid contour compared to contralateral side.
 - Humeral head is palpable anteriorly (in the subcoracoid region, beneath the clavicle).
 - Patient resists abduction and internal rotation and is unable to touch the opposite shoulder.
 - Compare bilateral radial pulses to help rule out vascular injury.
 - In all cases, evaluate the axillary nerve before and after reduction by testing both pinprick sensation in the "regimental badge" area of the deltoid and palpable contraction of the deltoid during attempted abduction. Evaluate sensory and motor function of the musculocutaneous and radial nerves.

- Inferior shoulder dislocation (luxatio erecta)
 - Arm is fully abducted with elbow commonly flexed on or behind head (see [Picture 1](#)).
 - Humeral head may be palpable on the lateral chest wall.

Causes

- Anterior shoulder dislocations usually result from abduction, extension, and external rotation, such as when preparing for a volleyball spike. Falls on an outstretched hand are a common cause in older adults. The humeral head is forced out of the glenohumeral joint, rupturing or detaching the anterior capsule from its attachment to the head of the humerus or from its insertion to the edge of the glenoid fossa. This occurs with or without lateral detachment.
- Posterior dislocations are caused by severe internal rotation and adduction. This usually occurs during a seizure, a fall on an outstretched arm, or electrocution. Occasionally, a severe direct blow may cause a posterior dislocation. Bilateral posterior dislocation is rare and almost always results from seizure activity.
- Rare, but serious, inferior dislocations (luxatio erecta) may be due to axial force applied to an arm raised overhead, such as when a motorcycle collision victim tumbles to the ground. More commonly, the shoulder is dislocated inferiorly by indirect forces hyperabducting the arm. The neck of the humerus is levered against the acromion and the inferior capsule tears as the humeral head is forced out inferiorly. This injury always is accompanied by fracture and/or serious soft-tissue injury.

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Differentials

Acromioclavicular Injury

Fractures, Humerus

Other Problems to be Considered

Humeral fractures most commonly involve the greater tuberosity, head, and neck.

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Workup

Lab Studies

- None are needed specifically for shoulder dislocation.

Imaging Studies

- Shoulder trauma series - Anteroposterior (AP) and axillary or scapular "Y" views
 - Anterior dislocation is characterized by subcoracoid position of the humeral head in the AP view. The dislocation is often more obvious in a scapular "Y" view where the humeral head lies anterior to the "Y." In an axillary view, the "golf ball" (ie, humeral head) is said to have fallen anterior to the "tee" (ie, glenoid).
 - In posterior dislocation, the AP view may show a normal walking stick contour of the humeral head, or it may resemble a light bulb or ice cream cone depending upon the degree of rotation. The scapular "Y" view reveals the humeral head behind the glenoid (the center of the "Y"). In an axillary view, the "golf ball" falls posteriorly off the "tee."
 - In lateral dislocation, the AP view may show the arm raised over the head with the radial head inferior to the glenoid (see [Picture 2](#)).
- Postreduction films confirm relocation of the humerus and may reveal new or previously obscured pathology. Immobilization prior to roentgenography is imperative.

Other Tests

- Arteriography may be used to evaluate suspected arterial injury.
- Electromyography (EMG) may be used later to characterize nerve injuries.

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Treatment

Prehospital Care

- Stabilize and treat associated trauma as indicated.
- Allow the patient to assume a position of comfort while maintaining cervical spine immobilization if necessary.
- Place a pillow between the patient's arm and torso to increase comfort.

Emergency Department Care

- Offer analgesia to decrease pain.
- Prereduction and postreduction radiographs are recommended. Patients with frequent recurrent dislocations can safely avoid radiographs.
- Conscious sedation helps relax surrounding musculature, making reduction easier.
- The key to successful reduction is slow and steady application of a maneuver with adequate analgesia and relaxation.
- Successful reduction is evinced by a palpable or audible relocation, marked reduction in pain, and increased range of motion. The patient may be asked to touch the uninjured shoulder to safely demonstrate a successful reduction.
- Orthopedic consultation is recommended by some prior to reduction of posterior and inferior dislocations.
- After all reductions, apply a shoulder immobilizer and perform a careful neurovascular examination. Many recommend postreduction radiography, especially if the procedure was difficult.
- Reduction of anterior dislocation
 - Leverage methods such as the Kocher and Hippocratic techniques are discouraged because of increased incidence of humeral shaft fractures and capsule or axillary nerve injuries.
 - Stimson technique: Patient lies prone on the bed with the dislocated arm hanging over the side. Traction is provided by up to 10 kg of weight attached to the wrist or above the elbow. Apply gentle internal/external humeral rotation. Reduction may take 20-30 minutes.
 - External rotation method: While the patient lies supine, adduct the arm and flex it to 90 degrees at the elbow. Slowly rotate the arm externally, pausing for pain. Reduce the shoulder before reaching the coronal plane. Often successful, this procedure requires only one physician and little force (see [Special Concerns](#)).
 - Traction-countertraction: While the patient lies supine, apply axial traction to the arm with a sheet wrapped around the forearm and the elbow bent 90 degrees. An assistant should apply countertraction using a sheet wrapped under the arm and across the chest.
 - Scapular rotation: This less traumatic technique has success rates over 90% in experienced hands, often without sedation. With the patient lying prone, apply manual traction or 5-15 lb of hanging weight to the wrist. After relaxation, rotate the inferior tip of the scapula medially and the superior aspect laterally. Alternatively, the patient can be seated while an assistant provides traction-countertraction by pulling on the wrist with one hand and bracing the upper chest with the other. The same scapular rotation is then performed.
 - Many other reduction techniques have been described. Roberts and Hedges provide a more complete list.
- Reduction of inferior dislocation
 - Maintain gentle axial traction on the humerus while gentle abduction is applied.
 - Apply countertraction across the ipsilateral shoulder (see [Picture 3](#)).
 - Following reduction, slowly adduct the arm.
 - Buttonholing of the humeral head through the capsule usually requires open reduction.

Consultations

Orthopedic consultation may be helpful for dislocations with concomitant fractures, posterior or inferior dislocations, and cases in which the patient's shoulder cannot be reduced in a timely fashion.

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Medication

Opiate analgesia should be given as needed for pain. Conscious sedation generally is required to relax muscles adequately for reduction. Some orthopedic surgeons advocate relaxation and analgesia with intraarticular anesthetics.

Analgesics

These agents may be used for the relief of pain and relaxation of shoulder muscles. Pain control is essential to quality patient care. It ensures patient comfort, improves likelihood of successful reduction, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients with injuries.

| | |
|-------------------|---|
| Drug Name | Fentanyl citrate (Duragesic, Sublimaze)- DOC because of its rapid, almost immediate onset and short duration of 30-60 min. Can be reversed easily by naloxone 2 mg IV as needed for respiratory depression. Often used as part of conscious sedation with midazolam (see Sedation article). Useful for emergency department visits only. Not intended to be given on an outpatient basis. |
| Adult Dose | 0.5-1 mcg/kg/dose IV/IM q30-60min; titrate to pain relief in 50 mcg IV increments |
| Pediatric Dose | 2-3 mcg/kg IV; titrate to pain relief |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hypotension, respiratory depression, constipation, nausea, emesis, and urinary retention; idiosyncratic reaction, known as chest wall rigidity syndrome, may require neuromuscular blockade to increase ventilation |

| | |
|-------------------|--|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4,000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

| | |
|-------------------|---|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen q4-6h prn; not to exceed 2.6 g/d acetaminophen or 5 mg of hydrocodone bitartrate/dose >12 years: 750 mg acetaminophen q4h; not to exceed 5 doses/d acetaminophen or 10 mg of hydrocodone bitartrate/dose |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates, since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

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Follow-up

Further Inpatient Care

- Following conscious sedation, the patient should be observed for at least 1 hour before being discharged in the care of family or friends.
- Patients requiring operative reduction and repair must be admitted by the orthopedic surgery service.

Further Outpatient Care

- Arrange for orthopedic follow-up in 5-7 days.
- The patient's shoulder should remain in the immobilizer until the orthopedic clinic appointment.

in/Out Patient Meds

- NSAIDs, such as ibuprofen, may be taken as needed for pain and inflammation.
- A few days of narcotic analgesia, with an agent such as hydrocodone or oxycodone, is often helpful.

Deterrence/Prevention

- The patient should remain in the immobilizer until under the care of an orthopedic surgeon.
- To prevent recurrent dislocation, patients should avoid activities that involve abduction and external rotation of the arm, such as combing their hair.

Complications

- Recurrence
- Fractures and soft-tissue injuries
 - Hill-Sachs lesions occur when the edge of the glenoid causes an impaction fracture in the posterolateral aspect of the humeral head during anterior dislocation and in the anterolateral aspect in posterior dislocation. This lesion is reported in 11-50% of anterior dislocations.
 - Fracture of anterior or posterior glenoid rim also may occur, and significant displacement necessitates operative management. The greater tuberosity, acromion, coracoid, clavicle, and humeral neck and shaft are also common sites of fractures.
 - Bankart lesion is a detachment of the anterior part of the glenoid labrum and capsule.
 - Rotator cuff traction injury is more common in adults aged 40 years and older and with inferior dislocation. In an ongoing prospective study of patients older than 40 years sustaining an initial dislocation, all treated with arthroscopic evaluation, 86% had rotator cuff tears. This is a commonly missed injury. The average time from injury to diagnosis of rotator cuff rupture in patients older than 40 years is over 7 months.
- Vascular injury

- Axillary artery injury, though unlikely, is possible; it is associated with decreased radial pulse.
- Axillary mass or hematoma with possible bruit may be observed. Lateral chest bruising is common.

Prognosis

- Approximately 90% of patients younger than 20 years at the time of the initial dislocation have a recurrence; however, dislocation recurs in only 14% of patients older than 40 years. Many orthopedic surgeons believe that more than 1 complete anterior dislocation justifies considering surgical repair.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to find posterior dislocations. These are missed commonly, so careful examination and radiographs are imperative. Hawkins found an average interval of 1 year between injury and diagnosis of posterior dislocation in a series of 40 patients.
- Failure to find and document associated fractures and neurovascular injuries.

Special Concerns

- Pregnant patients
 - Patients in the third trimester should be placed in the left lateral decubitus position to avoid compression of the inferior vena cava by the uterus.
 - The abdomen should be shielded during roentgenography.
 - Relocation techniques requiring placement of the patient in a prone position may be problematic.
- Geriatric patients: Since fractures can occur easily with vigorous manipulation, choose a gentle relocation technique. Avoid the Hippocratic and Kocher techniques. The external rotation method also uses leverage primarily, and although reported complications are extremely rare, choosing another technique is advised. Towels or sheets used for traction or countertraction can cause friction injury to the fragile skin of older adults.

Pictures



Picture 1: Presentation of luxatio erecta (inferior shoulder) dislocation in a person involved in a motorcycle accident

Picture type: Photo

**IMAGE
TEMPORARILY
UNAVAILABLE**



Picture 2: Anteroposterior shoulder radiograph showing humeral head inferior to glenoid and arm raised overhead in a patient who sustained luxatio erecta (inferior shoulder) dislocation in a motorcycle accident
Picture type: Photo

**IMAGE
TEMPORARILY
UNAVAILABLE**



Picture 3: Relocation of luxatio erecta (inferior shoulder) dislocation. An emergency resident is applying axial traction, an orthopedic resident is providing countertraction, and the ED attending physician is pressing the radial head back into the glenoid fossa.

Picture type: Photo

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Dislocations, Wrist

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Introduction

Background

Carpal dislocations represent a continuum of wrist injury that can lead to lunate or perilunate dislocation. The lunate cup commonly is directed in a volar direction in dislocation because of the mechanism of the injury. Perilunate dislocations result from dislocation of the distal carpal row. The capitate normally rests within the lunate cup, as seen on a lateral view. With perilunate dislocations, the capitate is seen most commonly as dorsal, but it also may be volar to the lunate on lateral x-ray evaluation. As a result of the stresses involved, scaphoid fractures often accompany perilunate dislocation. Carpal instability may take many forms and represents a spectrum of injury including scapholunate dissociation, lunate and perilunate dislocations, scaphoid fracture, and other intercarpal instabilities.

Pathophysiology

The mechanism of injury is usually a fall onto an outstretched hand with hand rotation, which may lead to a variety of injuries. These injuries range from scapholunate strain to carpal dislocation, with scaphoid fracture at the end of the spectrum. Unfortunately, most of these injuries are not diagnosed in the ED. The injury may lead to chronic pain and instability of the wrist.

Frequency

- **In the US:** Incidence of wrist injuries is estimated as 2.5% of ED visits. Wrist dislocations represent a very small portion of these visits.
- **Internationally:** Same incidence as in the US.

Mortality/Morbidity

- The morbidity of wrist dislocations is tied to the frequently missed diagnosis of lunate or perilunate dislocation in the ED. Often, patients are not diagnosed with these injuries until weeks following the initial injury.
- Many patients with undiagnosed wrist dislocation have chronic pain.
- Carpal instability, including radiocarpal instability, is a frequent complication.

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Clinical

History

- Patients usually present to the ED fairly soon after a fall onto an outstretched hand.
- The mechanism of injury is ulnar deviation of the wrist coupled with dorsiflexion.
- The resulting intercarpal supination places great stress on the carpals. The result can be a lunate or perilunate dislocation.
- Often, the only symptom is wrist pain.

Physical

The patient may have diffuse pain on palpation that is difficult to distinguish from other causes of wrist pain, including scapholunate strain, scaphoid fracture, triangular fibrocartilage complex tears, and other disorders.

Causes

- Carpal stability is based on the lunate as the central anchor for the proximal and distal carpal rows.
- The lunate is apposed to the radius, and the capitate rests within the lunate cup.

- The proximal row of carpals is connected by interosseous ligaments.
- Carpal stress is characterized as radial or ulnar, with some degree of axial loading. This stress is translated to all bones.
- Ligamentous injury results in a spectrum of injuries, including lunate and perilunate dislocations.
- The lunate-scaphoid ligaments may not be disrupted; in this is the case, scaphoid fracture may occur.

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Differentials

Arthritis, Rheumatoid
Carpal Tunnel Syndrome
Dislocations, Hand
Fractures, Hand
Fractures, Wrist
Hand Injuries, Soft-tissue

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Workup

Imaging Studies

- Plain x-rays of the wrist, both anteroposterior (AP) and lateral views, are essential to diagnose wrist dislocations (as well as other carpal instabilities).
 - On an AP view, 2 arcs should be identified. The first arc consists of the radiocarpal row, which should be smooth and continuous. Disruption is suggestive of a lunate dislocation.
 - The second arc consists of the midcarpal row, which also should be smooth and continuous. Disruption of this arc is suggestive of a perilunate dislocation.
 - The appearance of the lunate is important on the AP view. Normally, the lunate is quadrangular. With lunate dislocations, it becomes triangular. This may be an additional clue to dislocation.
 - On the lateral view, visualize the column, which consists of the radius, lunate, and capitate. The lunate should lie within the radius cup and the capitate should rest within the lunate cup. Loss of this normal column implies lunate or perilunate dislocation.
- Stress x-rays obtained with radial and ulnar deviation of the hand may demonstrate scapholunate dissociation.

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Treatment

Prehospital Care

- Prehospital care includes assessment for other injuries that may accompany the wrist injury.
- If no other injuries are identified, splint the wrist.
- Patients may be transported in their private vehicles, but the prehospital provider must emphasize the potential seriousness of the injury.
- Under no circumstances should a prehospital provider attempt a reduction of a suspected wrist dislocation. It may be a distal radius fracture, which requires significant care to reduce.

Emergency Department Care

- Patients with wrist injuries have an entire spectrum of possible injuries that represent potential disability.
- Although no specific fracture or dislocation may be seen on x-ray, carpal instability still may be present.
- Therefore, splint with plaster even if no injury is found on x-ray.
- Carefully splint with AP splints to the fingers until a hand specialist can evaluate the injury.

Consultations

- Patients in whom a wrist dislocation has been identified require referral to a hand specialist who is either an orthopedic or plastic surgeon, depending on local custom.
- Wrist dislocations may be reduced by emergency physicians, but only after consulting with the hand specialist.
- The patient's own primary care physician may follow up, but it is important to stress to the primary care physician the need for hand specialist referral.

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Medication

The goals of pharmacotherapy are to reduce morbidity and prevent complications.

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit these patients.

| | |
|-------------------|--|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tab q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates, since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|--|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may decrease toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4,000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

| | |
|------------|--|
| Drug Name | Oxycodone and aspirin (Percodan)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |

| | |
|-------------------|--|
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity; liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma; due to association of aspirin with Reye syndrome do not use in children (<16 y) who have flu |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity; may potentiate anticoagulant effects of warfarin |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Duration of action may increase in elderly; caution in renal or liver impairment, peptic ulcer disease, and erosive gastritis |

| | |
|-------------------|--|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d acetaminophen >12 years: 750 mg acetaminophen PO q4h; not to exceed 10 mg hydrocodone bitartrate per dose or 5 doses/24 h |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates, since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|--|
| Drug Name | Acetaminophen (Tylenol, Panadol, Aspirin-free Anacin)- DOC for pain in patients with documented hypersensitivity to aspirin or NSAIDs, those with upper GI disease, or those taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg PO q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; known G-6-PD deficiency |

| | |
|--------------|---|
| Interactions | Rifampin can reduce analgesic effects; barbiturates, carbamazepine, hydantoins, or isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; acetaminophen is contained in many OTC products and combined use with these products may result in cumulative acetaminophen doses exceeding recommended maximum dose |

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

These agents are used most commonly for the relief of mild to moderately severe pain. Although the effects of NSAIDs tend to be patient specific, ibuprofen is usually the DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- DOC for treatment of mild to moderately severe pain if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, which inhibits prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants (monitor PT closely and instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in CHF, hypertension, and decreased renal or hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy (monitor PT carefully and instruct patients to watch for signs of bleeding) |

| | |
|-------------------|--|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for relief of mild to moderately severe pain and inflammation. Administer small dosages initially to patients with small body size, the elderly, and those with renal or liver disease. Doses higher than 75 mg do not increase its therapeutic effects. Administer high doses with caution and closely observe patient. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 14 years: 0.1-1 mg/kg PO q6-8h >14 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants (monitor PT closely and instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in CHF, hypertension, and decreased renal or hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy (monitor PT carefully and instruct patients to watch for signs of bleeding) |

| | |
|-------------------|--|
| Drug Name | Flurbiprofen (Ansaid)- Has analgesic, antipyretic, and anti-inflammatory effects. May inhibit cyclooxygenase enzyme, inhibiting prostaglandin biosynthesis. |
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants (monitor PT closely and instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|--|
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |
|-------------|--|

| | |
|-------------------|--|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, decreasing prostaglandin synthesis. |
| Adult Dose | 500 mg initial dose followed by 250 mg PO q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants (monitor PT closely and instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

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Follow-up

Further Inpatient Care

- Admission is not indicated for isolated wrist dislocation.

Further Outpatient Care

- Patients with lunate or perilunate dislocations, if reduced in the ED, may safely be discharged home with careful warnings of the potential for compartment syndrome, pain, and other postinjury conditions.
- Close follow-up must be arranged with a hand specialist.

in/Out Patient Meds

- Because of the severity of pain, narcotic pain medication often is required for the first 3 days.

Transfer

- Transfer is required if the emergency physician is unable to achieve reduction and a hand specialist is not available to evaluate the injury.

Complications

- Vascular complications are unusual but may occur if an associated fracture is present, particularly of the distal radius.
- Soft-tissue complications include carpal ligamentous disruption, which results in carpal instability.

Prognosis

- Many patients who sustain lunate or perilunate dislocation develop chronic wrist pain or wrist instability.
- Remember that lunate and perilunate dislocations are part of a continuum of injury that arises from significant carpal ligamentous injury. This often results in chronic carpal instability.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to diagnose wrist injury

- Failure to evaluate for the possibility of wrist dislocation in every patient who has a wrist injury. Systematically evaluating the AP and lateral wrist x-rays should eliminate this mistake.
- Failure to properly follow up. Patients with a wrist dislocation suffer a significant, potentially disabling injury. Follow-up should include referral to a hand specialist.
- Failure to adequately immobilize. Patients who have sustained a wrist dislocation should have AP splints applied, boxed to the fingertips. The hand specialist decides whether a long arm cast extension should be applied to avoid pronation/supination.

Special Concerns

- A concomitant scaphoid fracture may occur as part of the injury pattern.
-

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Epidural Hematoma

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Synonyms, Key Words, and Related Terms

EDH

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Introduction

Background

Epidural hematoma (EDH) is a traumatic accumulation of blood between the inner table of the skull and the stripped-off dural membrane. The inciting event often is a focused blow to the head, such as that produced by a hammer or baseball bat. In 85-95% of patients, this trauma results in an overlying fracture. Blood vessels in close proximity to the fracture are the sources of the hemorrhage. Because the underlying brain usually has been minimally injured, prognosis is excellent if treated aggressively. Outcome from surgical decompression and repair is related directly to patient's preoperative neurologic condition.

Pathophysiology

Approximately 70-80% of EDHs are located in the temporoparietal region where skull fractures cross the path of the middle meningeal artery or its dural branches. Frontal and occipital EDHs each constitute about 10%, with the latter occasionally extending above and below the tentorium. Association of hematoma and skull fracture is less common in young children because of calvarial plasticity.

EDHs usually are arterial in origin but result from venous bleeding in one third of patients. Occasionally, torn venous sinuses cause EDH, particularly in the parietal-occipital region or posterior fossa. These injuries tend to be smaller and associated with a more benign course. Usually, venous EDHs only form with a depressed skull fracture, which strips the dura from the bone and, thus, creates a space for blood to accumulate. In certain patients, especially those with delayed presentations, venous EDHs are treated nonsurgically.

Expanding high-volume EDHs can produce a midline shift and subfalcine herniation. Compressed cerebral tissue can impinge on the third cranial nerve, resulting in ipsilateral pupillary dilation and contralateral hemiparesis or extensor motor response.

EDHs usually are stable, attaining maximum size within minutes of injury; however, Borovich demonstrated progression of EDH in 9% of patients during the first 24 hours. Rebleeding or continuous oozing presumably causes this progression. EDH occasionally runs a more chronic course and is detected only days after injury.

Frequency

- **In the US:** EDH occurs in 1-2% of all head trauma cases and in about 10% of patients who present with traumatic coma.

Mortality/Morbidity

- Reported mortality rates range from 5-43%.
- Higher rates are associated with the following:
 - Advanced age
 - Intradural lesions
 - Temporal location
 - Increased hematoma volume
 - Rapid clinical progression
 - Pupillary abnormalities
 - Increased intracranial pressure (ICP)
 - Lower Glasgow coma scale (GCS)

Age

- Patients younger than 5 years and older than 55 years have an increased mortality rate.
- Patients younger than 20 years account for 60% of EDH incidences.
- EDH is uncommon in patients who are elderly because the dura is strongly adhered to the inner table of the skull. In case series of EDH, fewer than 10% of patients are older than 50 years.

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Clinical

History

- Fewer than 20% of patients demonstrate the classic presentation of a lucid interval.
- Following injury, the patient may be continually comatose, briefly comatose and recovered, or continually conscious.
- Severe headache
- Vomiting
- Seizure
- Patients with posterior fossa EDH may have a dramatic delayed deterioration. The patient can be conscious and talking and a minute later apneic, comatose, and minutes from death.

Physical

- Cushing response consisting of the following can indicate increased ICP:
 - Hypertension
 - Bradycardia
 - Bradypnea
- Contusion, laceration, or bony step-off may be observed in the area of injury.
- Dilated, sluggish, or fixed pupil(s), bilateral or ipsilateral to injury, suggest increased ICP or herniation.
- Classic triad indicating transtentorial herniation consists of the following:
 - Coma
 - Fixed and dilated pupil(s)
 - Decerebration

Causes

- EDH results from traumatic head injury, usually with an associated skull fracture and arterial laceration.

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Differentials

Subarachnoid Hemorrhage
Subdural Hematoma

Other Problems to be Considered

Cerebral contusion

Diffuse axonal injury

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Workup

Lab Studies

- Perform appropriate lab work for associated trauma.
- No specific tests are required.
- Coagulation abnormalities are a marker of severe head injury. Breakdown of the blood-brain barrier with exposed brain tissue is a potent cause of disseminated intravascular coagulation (DIC).

Imaging Studies

- Head CT scan
 - Immediate unenhanced CT scan is the procedure of choice for diagnosis.
 - Head CT scan shows location, volume, effect, and other potential intracranial injuries.
 - EDH forms an extraaxial, smoothly marginated, lenticular, or biconvex homogenous density.
 - EDH rarely crosses the suture line because the dura is attached more firmly to the skull at

sutures.

- Focal isodense or hypodense zones within EDH indicate active bleeding.
- Irregular hypodense swirling indicates active bleeding in the majority of patients.
- Air in acute EDH suggests fracture of sinuses or mastoid air cells.
- At surgery or autopsy, 20% of patients have blood in both epidural and subdural spaces.

Other Tests

- Cervical spine evaluation usually is necessary because of the risk of neck injury associated with EDH.

Procedures

- Perform burr hole(s) if the following occur:
 - Patient is herniating
 - All other treatments prove insufficient
 - Neurosurgery is unavailable
 - Air or ground medical transport is prolonged

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Treatment

Prehospital Care

- Stabilize acute life-threatening conditions and initiate supportive therapy. Airway control and blood pressure support are the most important issues.
- Establish IV access, administer oxygen, and monitor.
- Administer IV crystalloids to maintain systolic blood pressure (SBP) greater than 90 mm Hg.
- Intubation, sedation, and neuromuscular blockade per protocol

Emergency Department Care

- Establish IV access, administer oxygen, monitor, and administer IV crystalloids as necessary to keep SBP greater than 90 mm Hg.
- Intubate using rapid sequence induction (RSI), which generally includes premedication with lidocaine, a cerebroprotective sedating agent (eg, etomidate), and neuromuscular blockade. Lidocaine may have limited effect in this situation, yet it carries virtually no risk. Intubate after a

basic neurologic examination to facilitate oxygenation, protect airway, and allow for hyperventilation as needed.

- Elevate head 30° after the spine is cleared, or use reverse Trendelenburg position to reduce ICP and increase venous drainage.
- Administer mannitol 0.25-1 g/kg IV after consulting a neurosurgeon if SBP is greater than 90 mm Hg with continued clinical signs of increased ICP. This reduces both ICP (by osmotically reducing brain edema) and blood viscosity, which increases cerebral blood flow and oxygen delivery.
- Hyperventilation to partial pressure of carbon dioxide (PCO₂) of about 30 mm Hg treats incipient herniation or signs of increasing ICP; however, this is controversial. Be careful not to lower PCO₂ too far (<25 mm Hg). Perform hyperventilation if clinical signs of increased ICP progress; this procedure reduces ICP by hypocarbic vasoconstriction and reduces risks of hypoperfusion and death of injured cells.
- Phenytoin reduces the incidence of early posttraumatic seizures, although it does not affect late-onset seizures or the development of a persistent seizure disorder.

Consultations

- Consult neurosurgery immediately for EDH evacuation and repair.
- Consult trauma surgery for other life-threatening injuries.

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Medication

Use RSI prn. Etomidate, when used as RSI sedating agent, maintains SBP, lowers ICP and brain metabolism, and has rapid onset and brief duration. Thiopental is not recommended because of its propensity to drop SBP, which is the leading cause of secondary brain injury. Mannitol osmotically reduces ICP and improves blood flow. Phenytoin provides prophylaxis against early posttraumatic seizure. Once the patient has received adequate fluids (central venous pressure [CVP] >6 cm H₂O), pressors such as norepinephrine can be used to maintain SPB >90 mm Hg.

Osmotic Diuretic

Osmotically reduces brain edema and ICP and reduces blood viscosity, improving cerebral blood flow and oxygen delivery.

| | |
|-------------------|---|
| Drug Name | Mannitol (Osmitrol)- Keeps serum osmolality <320 mOsm to prevent renal failure. Maintain euvolemia with adequate IV fluid replacement. Foley catheter is essential. |
| Adult Dose | 0.25-1 g/kg IV q30-60min |
| Pediatric Dose | Administer by weight as in adults |
| Contraindications | Documented hypersensitivity; anuria; severe pulmonary congestion; severe dehydration; active intracranial bleeding; progressive renal damage; progressive heart failure; SBP <90 mm Hg |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Large crystals may form in a cold solution; carefully evaluate cardiovascular status before rapid administration, as sudden increase in extracellular fluid may lead to fulminating CHF; if blood is coadministered, add at least 20 mEq of sodium chloride to each L of mannitol solution to avoid pseudoagglutination; do not administer electrolyte-free mannitol solutions with blood |

Antiepileptic

Prevents early posttraumatic seizure, which can increase ICP and neurotransmitter release as well as alter blood pressure and oxygen delivery.

| | |
|-------------------|--|
| Drug Name | Phenytoin (Dilantin)- DOC for seizure prophylaxis. May be supplanted by fosphenytoin. If actively seizing, coadminister benzodiazepine. |
| Adult Dose | 17 mg/kg IV; mix in NS (precipitates in D5W); infuse no faster than 50 mg/min |
| Pediatric Dose | Administer by weight as in adults |
| Contraindications | Documented hypersensitivity; because of effects on ventricular automaticity, do not use in sino-atrial block, sinus bradycardia, second- and third-degree AV block, or in patients with Adams-Stokes syndrome |
| Interactions | Increased toxicity with amiodarone, benzodiazepines, chloramphenicol, cimetidine, fluconazole, isoniazid, metronidazole, miconazole, phenylbutazone, succinimides, sulfonamides, omeprazole, phenacetamide, disulfiram, ethanol (acute ingestion), trimethoprim, valproic acid; effects may decrease when taken concurrently with barbiturates, diazoxide, ethanol (chronic ingestion), rifampin, antacids, charcoal, carbamazepine, theophylline, sucralfate; may decrease effects of acetaminophen, corticosteroids, dicumarol, disopyramide, doxycycline, estrogens, haloperidol, amiodarone, carbamazepine, cardiac glycosides, quinidine, theophylline, methadone, metyrapone, mexiletine, oral contraceptives, valproic acid |
| Pregnancy | D - Unsafe in pregnancy |

| | |
|-------------|--|
| Precautions | Administer slowly (50 mg/min) to avoid hypotension; avoid extravasation; perform blood counts and urinalyses when initiating therapy and at monthly intervals for several mo to monitor for blood dyscrasias; discontinue use if skin rash appears and do not resume use if rash is exfoliative, bullous, or purpuric; after too-rapid IV administrations death from cardiac arrest may occur, which is sometimes preceded by marked QRS widening; administer cautiously to patients with acute intermittent porphyria; exercise caution with diabetes, as it may raise blood sugar levels; discontinue drug if hepatic dysfunction occurs |
|-------------|--|

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Follow-up

Further Inpatient Care

- Transfer to operating room (OR) for EDH evacuation and repair.
- Admit to neurosurgical ICU after surgery or directly for monitoring. This will likely include ICP, partial pressure oxygen (PO₂), or other intracranial monitoring devices.
- Repeat CT scan in the event of clinical deterioration.

Transfer

- Transfer to hospital with a CT scanner and neurosurgeon.
- Consider air transport if a trauma center is distant; timely decompression is critical for a good outcome.

Deterrence/Prevention

- Encourage use of seat belts and car seats.
- Advocate helmets for bicycling, skateboarding, snowboarding, rollerblading, and horse and motorcycle riding.

Complications

- Neurobehavioral changes: Postconcussive syndrome can last hours to months (see [Postconcussive Syndrome](#)).
- Vegetative state
- Death

Prognosis

- Mortality rates approximate 0% for patients not in coma preoperatively, 9% for obtunded patients, and 20% for patients in a deep coma before surgery.
- If treated early, prognosis usually is excellent, because the underlying brain injury generally is limited.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to consider diagnosis, especially in a conscious patient with normal pupils
- Failure to transfer expeditiously to a trauma center with a neurosurgeon (air medical transport may be warranted)
- Failure to ascribe altered mental status to EDH (instead naming alcohol or another intoxicant as the cause)
- Failure to perform frequent routine neurologic checks in patients who are being observed rather than sent for CT scan

Special Concerns

- Pediatric patients
 - May not fracture the skull
 - Lower mortality rates, except in infants
-

Pictures

**IMAGE
TEMPORARILY
UNAVAILABLE**



Picture 1: Movie of a young, professional skateboarder incurring an epidural hematoma, among other injuries, during a fall.

Picture type: Image



Picture 2: Right temporal epidural hematoma with midline shift. Patient should be taken immediately to the operating room for neurosurgery. This may require emergent transport to a trauma center or other facility with a neurosurgeon available.

Picture type: CT

**IMAGE
TEMPORARILY
UNAVAILABLE**



Picture 3: Aeromedical transport to a neurosurgical facility may make the difference between a neurologically intact patient, a severely disabled patient, and a dead patient.

Picture type: Image



Picture 4: Brain CT scan of 90-year-old man who slipped on a waxed floor. Witnesses reported loss of consciousness followed by a "lucid interval." Patient arrived in ED unconscious. CT scan shows epidural hematoma. (Image courtesy of Dr Dana Stearns, Massachusetts General Hospital)

Picture type: CT

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Fingertip Injuries

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Introduction

Background

The fingertip is the part of the terminal phalanx that is distal to the insertion of extensor and flexor tendons. Fingertip injuries are extremely common. A functioning fingertip has sensation without pain, stable padding, and an acceptable appearance.

Pathophysiology

Fingertip injuries occur frequently because hands are used to explore surroundings. Blunt or crush injuries create subungual hematomas, nail root avulsions, and fractures of the terminal phalanx. Sharp or shearing injuries from knives and glass result in lacerations. Occasionally, the end of the fingertip is completely avulsed. Burns and frostbite commonly involve fingertips.

Frequency

- **In the US:** About 10% of all accidents encountered in the ED involve the hand. Hand injuries represent 11-14% of on-the-job injuries and 6% of compensation paid injuries. They account for approximately two thirds of hand injuries in children. Damage to the nail bed is reported to occur in 15-24% of fingertip injuries.

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Clinical

History

Ascertain the following information when gathering patient history:

- Mechanism of injury
- Hand dominance
- Occupation and hobbies
- Length of time since injury
- Tetanus immunization status

Physical

Evaluate the fingertip injury to determine the following:

- Crush versus sharp injuries
- Nail or nail bed involvement
- Bone involvement
- Viability of tip
- Presence of foreign body

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Workup

Lab Studies

- No laboratory studies are indicated, other than standard preoperative labs if surgery is required.

Imaging Studies

- Radiographs may be necessary either to assess alignment of distal phalanx fractures or to detect

presence of foreign bodies.

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Treatment

Prehospital Care

- Care for amputated part
 - Cleanse gently.
 - Cover in a saline-soaked gauze wrap.
 - Place amputated part in a watertight bag.
 - Place watertight bag with amputated part on water and ice for preservation. Do not place part directly on ice.

Emergency Department Care

- Preserve length, padding, and sensation of finger.
- Preserve proper nail growth capacity and function.
- Prevent infection.
- Minimize joint stiffness.
- Limit employment disability.
- Use of a digital block provides excellent local anesthesia and does not cause further swelling of fingertip.
- Lacerations
 - Suture simple lacerations with 5-0 or 6-0 nylon. Subcutaneous or deep dermal sutures are not indicated.
 - Remove nail and inspect matrix when fingertip lacerations involve nail and injuries that avulse, split, or disrupt it. Replace all retrievable fragments of nail matrix as free grafts.
 - Repair nail matrix according to the following steps:
 - Administer anesthesia with a digital block and establish a bloodless field with a Penrose drain.
 - Remove nail.
 - Debride gently.
 - Clean and remove all foreign bodies.
 - Repair nail matrix meticulously with fine absorbable suture (6-0 Monocryl). Histoacryl blue works well to secure nail plate in place of sutures.
 - Reinsert nail plate or substitute.
 - Apply sterile nonadherent dressing and splint.

- Fingertip amputations
 - Various methods are used for amputation injuries including simple revision amputation, full- or partial-thickness skin grafts, local flaps, distal flaps, and neurovascular island pedicle flaps.
 - Distal fingertip amputations may be treated conservatively in the ED. Various treatments may be provided in ED depending on the emergency physician's skills, training, and time availability. However, for distal amputations that involve significant tissue loss, the physician should discuss a treatment plan with the follow-up hand surgeon.
- Removal of splinter under nail: Unroof the splinter by trimming the nail with iris scissors.

Consultations

- Hand surgeon

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Medication

The goal of pharmacotherapy is to reduce pain. Tetanus immunization also may be indicated.

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

Commonly used for relief of mild to moderate pain. Effects of NSAIDs in treating pain tend to be patient specific, yet ibuprofen is usually the DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and ketoprofen.

| | |
|-------------------|---|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- DOC for treatment of mild to moderate pain, if no contraindications are present. Inhibits inflammatory reactions and pain probably by decreasing activity of the enzyme cyclooxygenase, which inhibits prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 10 mg/kg q6-8h; not to exceed 2.4 g/d >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |

| | |
|--------------|--|
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for relief of mild to moderate pain and inflammation. Small dosages initially are indicated in small and elderly patients and in those with renal or liver disease. Doses >75 mg do not increase its therapeutic effects. Administer high doses with caution and closely observe patient for response. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 14 years: 0.1-1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

| | |
|----------------|--|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderate pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, which decreases prostaglandin synthesis. |
| Adult Dose | 500 mg PO, followed by 250 mg PO q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg PO bid; not to exceed 10 mg/kg/d |

| | |
|-------------------|--|
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

| | |
|-------------------|---|
| Drug Name | Flurbiprofen (Ansaid)- Has analgesic, antipyretic, and anti-inflammatory effects. May inhibit cyclooxygenase enzyme, inhibiting prostaglandin biosynthesis that may result in analgesic and anti-inflammatory activities. |
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion, risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures.

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|-------------------|---|
| Drug Name | Acetaminophen (Tylenol, Panadol, Aspirin-free Anacin)- DOC for pain in patients with documented hypersensitivity to aspirin or NSAIDs, with upper GI disease, or who are taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg PO tid/qid; not to exceed 4 g/d PO is fine |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; known G-6-P deficiency |
| Interactions | Rifampin can reduce analgesic effects of acetaminophen; coadministration with barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; APAP is contained in many OTC products and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

| | |
|-------------------|---|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for the treatment of mild to moderate pain. |
| Adult Dose | 30-60 mg/dose PO based on codeine content q4-6h or 1-2 tabs q4h; not to exceed 12 tabs/d |
| Pediatric Dose | 0.5-1 mg/kg/dose PO based on codeine q4-6h; 10-15 mg/kg/dose PO based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | Toxicity increases with CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|------------|--|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for the relief of moderate to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |

| | |
|-------------------|---|
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d acetaminophen >12 years: 750 mg acetaminophen PO q4h; not to exceed 10 mg hydrocodone bitartrate per dose or 5 doses/24 h |
| Contraindications | Documented hypersensitivity; high altitude cerebral edema (HACE) or elevated intracranial pressure (ICP) |
| Interactions | Coadministration with phenothiazines may decrease analgesic effects; toxicity increases with CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for the relief of moderate to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose PO oxycodone; not to exceed 5 mg/dose of oxycodone q4-6h prn |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects of this medication; toxicity increases with coadministration of either CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in elderly patients; be aware of total daily dose of acetaminophen that patient is receiving; do not exceed 4000 mg/24 h of acetaminophen (higher doses may cause liver toxicity) |

Toxoid

Used for tetanus immunization. Administer booster injection in previously immunized individuals to prevent this potentially lethal syndrome.

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|-------------------|--|
| Drug Name | Tetanus toxoid- Used to induce active immunity against tetanus in selected patients. The immunizing agent of choice for most adults and children aged >7 years are tetanus and diphtheria toxoids. Necessary to administer booster doses to maintain tetanus immunity throughout life. Pregnant patients should receive only tetanus toxoid, not a diphtheria antigen-containing product. May administer into deltoid or midlateral thigh muscles in children and adults. In infants, preferred site of administration is the mid-thigh laterally. |
| Adult Dose | Primary immunization: 0.5 mL IM, give 2 injections 4-8 wk apart and a third dose 6-12 mo after second injection Booster dose: 0.5 mL q 10 years |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; a history of any type of neurological symptoms or signs following administration of this product; FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are an important cause of provocative poliomyelitis |
| Interactions | Patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization due to poor immune response; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent use of medication with systemic chloramphenicol because it may impair amnestic response to tetanus toxoid; concurrent use of tetanus immune globulin may delay development of active immunity by several days (interaction is, nevertheless, clinically insignificant and does not preclude its concurrent use) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Do not use to treat actual tetanus infections, or for immediate prophylaxis of unimmunized individuals (use instead tetanus antitoxin, preferably human tetanus immune globulin) diminished antibody response to active immunization may be observed in patients receiving immunosuppressive therapy; better to defer primary diphtheria immunization until immunosuppressive therapy discontinued; routine immunization of symptomatic and asymptomatic HIV-infected persons is recommended |

Immunoglobulins

Patients who may not have been immunized against *Clostridium tetani* products should receive tetanus immune globulin (Hyper-Tet).

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|-----------|--|
| Drug Name | Tetanus immune globulins (Hyper-Tet)- Used for the passive immunization of persons with wounds that may be contaminated with tetanus spores. |
|-----------|--|

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|-------------------|--|
| Adult Dose | Prophylaxis: 250-500 U IM in opposite extremity to tetanus toxoid lesion Clinical tetanus: 3000-10,000 U IM |
| Pediatric Dose | Prophylaxis: 250 U IM in opposite extremity to tetanus toxoid Clinical tetanus: 3000-10,000 U IM |
| Contraindications | Since antibodies in globulin preparation may interfere with immune response to vaccination, do not administer within 3 mo of live virus immune globulin administration; may be necessary to revaccinate persons who received immune globulin shortly after live virus vaccination |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Persons with isolated IgA deficiency have potential for developing antibodies to IgA and could have anaphylactic reactions to subsequent administration of blood products that contain IgA; do not perform skin testing since intradermal injection of concentrated gamma globulin may cause localized area of inflammation and can be misinterpreted, causing the medication to be withheld from a patient not allergic to this material; true allergic responses to human gamma globulin given in prescribed IM manner are extremely rare; do not admix with other medications because they are usually incompatible |

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Follow-up

Further Inpatient Care

- Keep hand elevated.

Further Outpatient Care

- Keep hand elevated.
- Check wound 2 days after ED treatment.
- Analgesics may be necessary for first few days.
- Splint fractures in extension for 2 weeks.

Complications

- Untreated nailbed lacerations may lead to subsequent nail deformities.

- When amputation with loss of two thirds of the nail occurs, half of the fingers develop beaking or a curved nail.

Prognosis

- Oldest recorded patient to show fingertip regeneration was aged 11 years.

Patient Education

- Full growth of nail takes an average of 100 days, but fingertip trauma may delay growth by 20 days.
- Average healing time for fingertip amputation is 21-27 days.
- Remove sutures after 7-10 days.

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Miscellaneous

Special Concerns

- In 60% of patients with subungual hematomas that involve more than 50% of the nail surface, laceration is repairable. This figure increases to over 95% when an associated fracture of distal phalanx is present.
 - Conservative treatment without nail removal is advocated for patients with closed hematomas (regardless of the size of hematoma) and intact nails with no skin-fold laceration or nail disruption.
 - Nail complex requires distal bone and soft tissue support to prevent a hook-nail deformity.
 - Because of its flammability, do not use ethyl chloride for anesthesia before trephinating a subungual hematoma with cautery. Be careful with artificial nails as well.
-

Pictures



Picture 1: Significant nail bed injuries can occur from nail root avulsions.

Picture type: Photo



Picture 2: Removal of nail plate with iris scissors

Picture type: Photo



Picture 3: Suturing of nail bed laceration

Picture type: Photo



Picture 4: Sutured nail bed injury

Picture type: Photo



Picture 5: U-stitch method of securing nail plate

Picture type: Photo

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Fractures, Ankle

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Introduction

Background

Ankle joints are highly susceptible to injury because they are relatively mobile and bear much of the stress associated with weight bearing. Four bones provide the joint framework: the distal tibia, distal fibula, talus, and calcaneus. On occasion, such as during vigorous exercise, the ankle must endure forces greater than 5 times the body weight. Ankles must support more weight per unit area than any other joint in the body. Helping to stabilize and secure the ankle are the medial (deltoid) and lateral malleolar ligaments, along with the interosseous membrane that bridges the tibia and fibula. Conceptually, it helps to view the ankle as a closed ring structure encircling the talus.

Pathophysiology

The primary motion of the ankle at the tibiotalar junction is plantarflexion and dorsiflexion with inversion and eversion occurring at the subtalar joint located between the talus and calcaneus.

Excessive inversion stress is the most common cause of ankle injuries for 2 anatomic reasons. First, the medial malleolus is shorter than the lateral malleolus, thus allowing the talus to invert more than evert. Second, the deltoid ligament stabilizing the medial aspect of the ankle joint is a stronger support than the thinner lateral ligaments. The ankle is much more stable and resistant to eversion injury than inversion injury as a result; however, when eversion injury does occur, the individual often suffers substantial

damage to bony and ligamentous supporting structures and loss of joint stability.

Transverse malleolar fractures usually represent an avulsion-type injury, while vertical malleolar fractures result from talar impaction.

Frequency

- **In the US:** Estimates of the frequency of ankle injuries in the US range from 1 million to 10 million per year. Ankle fractures account for 15% of all ankle injuries. Malleolar fractures account for 30% of all ankle fractures, with lateral malleoli fractures predominating.

Mortality/Morbidity

- Patients with unrecognized or undertreated open ankle fractures are at high risk for both local infection (including osteomyelitis) and sepsis. Gas gangrene is the most serious infectious complication and can be life threatening as well as limb threatening.
- Vascular supply to the ankle and foot may become compromised by development of a compartment syndrome or direct injury to blood vessels from bone fragments.
- Inadequate fracture reduction and/or fixation may lead to mechanical instability, chronic pain, and stiffness.

Race

No race predilection is noted.

Sex

Male-to-female ratio is 2:1, while women older than 40 years have a higher proportion of fracture-dislocations.

Age

Ankle fractures can occur in any age group, yet they are more common in elderly and young individuals.

- Osteoporosis increases the risk of fractures in older persons.
- Younger patients (ie, pre-adolescents) prove more vulnerable to ankle fractures, as the growth plate rather than supporting ligaments represent the weak link. Growth plate fractures are classified using the Salter-Harris system.

Clinical

History

- Knowledge of the direction of torque force applied to the ankle and the foot's position at the time of injury helps predict the nature and severity of an ankle injury. While patients tend to recall the event, they often cannot depict the exact manner in which their injury occurred. Perhaps more important than the precise mechanism of ankle injury are the circumstances surrounding the injury, which may predict more serious trauma to other bones or organ systems.
- Document chronic medical conditions, especially the presence of diabetes, peripheral vascular disease, and metabolic bone disease, because this may affect exam findings and treatment plans.
- Establish history of prior trauma to the affected ankle because antecedent laxity, instability, or former radiographic abnormalities may be misinterpreted as an acute event.
- Inquire about medication usage that may provoke premature osteoporosis, such as corticosteroids, or drugs (eg, nonsteroidal anti-inflammatory drugs [NSAIDs]) that may mitigate the degree of swelling normally expected with fractures.

Physical

As an ankle fracture often presents with symptoms similar to those of an ankle sprain, complete a thorough examination of the involved extremity to avoid misdiagnosis and prevent unnecessary radiographs.

- Indicators suggesting fracture include gross deformity, swelling (especially perimalleolar), bony tenderness, discoloration, and ecchymosis. Inability to bear weight on the injured foot also indicates fracture.
- Inspect carefully, noting the presence of open wounds close to the injured ankle.
- Palpate for focal bony tenderness, especially along the medial and lateral malleoli and posterior aspect of the joint.
- Corroborate any visible deformity by gently manipulating the affected area.
- Assess passive and active range of motion of the ankle joint, noting limitations. During the immediate acute phase, most patients' ankles are too tender to cooperate with stress testing of the joint.
- Assess the neurovascular status of the foot and ankle. Compare findings to unaffected extremity.
 - Check presence and quality of pulse of the posterior tibial artery.
 - Check presence and quality of pulse of dorsalis pedis artery. Note that the dorsalis pedis is congenitally absent in as many as 10-15% of the population.
 - Confirm adequate capillary refill.

Causes

Two classification schemes, the Lauge-Hansen and Danis-Weber, currently are employed to help categorize ankle fractures. Derived from cadaver studies, the Lauge-Hansen system categorizes ankle fractures based on the position of the foot and the forces acting on it at the time of injury, while Danis-Weber relies on the level of fibular fracture.

- Lauge-Hansen classification: Position of the foot (eg, supination, pronation) is described first. Second, the deforming force is described as external rotation, abduction, or adduction. A definitive number of combinations exist, and the severity of the injury conforms to the amount of force applied in each direction. This classification does not include axial compression injuries.
 - Supination-adduction class
 - Stage I - Transverse fracture of lateral malleolus
 - Stage II - Steep oblique fracture of medial malleolus
 - Pronation-abduction class
 - Stage I - Transverse fracture of medial malleolus or torn deltoid ligament
 - Stage II - Disruption of posterior and anterior tibiofibular ligaments with or without avulsion of posterior malleolus
 - Stage III - Oblique fracture of distal fibula
- Fracture eponyms
 - Pilon fracture
 - A pilon fracture designates a fracture of the distal tibial metaphysis combined with disruption of the talar dome. This fracture originates from an axial loading mechanism in which the talus drives into the tibial plafond, such as when a patient involved in an auto accident compresses his foot on the floorboard to brace against injury. Skiers coming to an unexpected sudden stop and victims of free fall from heights also may sustain pilon fractures. Incidence of pilon fractures ranges from 1-10% of all tibial fractures.
 - Establish vascular and integument integrity. Perform a meticulous exam of the skin, because marked swelling and breaching of the integument frequently accompany these fractures. Skin sloughing is not uncommon. Subsequent edema, fracture blisters, and skin necrosis from the original injury may convert closed fractures to open injuries.
 - Associated injuries include spinal compression fractures, especially of L1, and ipsilateral or contralateral fractures of the os calcis, tibial plateau, pelvis, or acetabulum.
 - Tillaux fracture
 - A Tillaux fracture describes a Salter-Harris (SH) type III injury of the lateral tibial epiphysis caused by extreme eversion and lateral rotation of the ankle. Incidence is highest in adolescents because the fracture occurs after the medial aspect of the epiphyseal plate of the tibia closes but before the lateral aspect arrests.
 - Distinguish a Tillaux fracture from a triplane fracture. Triplane fracture is a

combination of a SH II and III fracture and is more likely than a Tillaux fracture to require open reduction and internal fixation.

- Bimalleolar fractures, termed Pott fractures, involve at least 2 elements of the ankle ring. These fractures should be considered unstable and require urgent orthopedic attention.
- A trimalleolar, or Cotton, fracture involves the medial, lateral, and posterior malleoli. These fractures are considered unstable and require urgent orthopedic attention.

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Differentials

Ankle, Soft-tissue Injuries
Arthritis, Rheumatoid
Compartment Syndrome, Extremity
Deep Venous Thrombosis and Thrombophlebitis
Dislocations, Ankle
Fractures, Foot
Fractures, Tibia and Fibula
Gout and Pseudogout

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Workup

Lab Studies

- No lab studies are necessary in patients with isolated ankle fracture.

Imaging Studies

- Routinely ordering radiographs following an ankle injury is not cost-effective, because fewer than 15% of affected patients have fractures. Patients without fractures are identified reliably from the physical examination. Ottawa ankle rules provide practical guidelines to select patients for radiographic studies.
- Indications for ankle radiographs in patients with acute ankle pain include pain in the ankle region

plus 1 of the following:

- Bony tenderness at the posterior edge or tip of the medial malleolus
- Bony tenderness at the posterior edge or tip of the lateral malleolus
- Inability to bear weight both immediately and in the ED
- Confounding variables to the Ottawa rules are (1) younger than 18 years, (2) underlying neurologic deficit affecting lower limb(s), (3) altered mental status, and (4) multisystem trauma.
- Perform a standard 3-view radiographic examination (anteroposterior [AP], lateral, and mortise views). In the mortise view, the foot is rotated approximately 15° internally, allowing better visualization of the ankle mortise. Check x-ray for headset sign (ie, tibia sits atop the talus resembling a headpiece on a receiver). Normally, the space between the cradle and handle should be equal. Lack of symmetry suggests injury. Stress views help assess ankle joint stability but usually are deferred during the initial ED evaluation.
- Ankle joint usually adheres to the ring axiom (eg, a fracture in one part of the ring often is associated with a second injury). Always look for an associated medial malleolar fracture when a spiral fracture of the fibula proximal to the ankle mortise is seen. A vertical fracture of the medial malleolus also is associated with either a lateral malleolar fracture or rupture of the lateral ligaments.
- Accessory ossicles appear frequently adjacent to the medial and lateral malleoli and may mimic fractures. Clinical correlation is important. Accessory ossicles demonstrate well-corticated margins, while fracture fragments exhibit less-defined borders.
- CT scan and MRI
 - Occasionally useful, these expensive and time-consuming studies typically are not recommended for routine diagnosis of ankle fractures.
 - Advanced imaging is most useful to diagnose talar dome and triplane fractures, distinguish pilon from trimalleolar fractures, and differentiate an accessory ossicle from an avulsion fracture. Occasionally, these tests are used to assess the complexity of the fracture and any associated ligamentous and intraarticular injuries.

Other Tests

- Stress radiographs assess the ankle during stress testing; however, results of this test generally do not affect immediate ED management.

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Treatment

Prehospital Care

- For an isolated ankle injury, confirm neurovascular status of the concerned limb, decrease pain, and prevent further damage.
 - Stabilize the suspected fracture site with a pillow splint, air splint, or bulky Jones dressing before transporting patient. Try to immobilize ankle in a neutral position if possible but avoid excessive handling.
 - Immobilization helps decrease pain, bleeding, and damage to surrounding soft tissue.
 - Prehospital reduction of a fracture is not advised unless neurovascular compromise is evident (eg, presence of a cool, dusky foot) and a significantly prolonged transport time is anticipated.

Emergency Department Care

- Definitive treatment of ankle fractures is complex and multifactorial (eg, level and stability of fracture, patient's age, activity goals). Once associated injuries and neurovascular compromise are excluded, determine integrity of the ankle mortise. Simple, uncomplicated lateral malleolar fractures usually can be splinted in the ED, followed by arrangement of timely orthopedic follow-up care; bimalleolar, trimalleolar, and pilon fractures necessitate urgent orthopedic attention for possible open reduction and internal fixation (ORIF).
- Standards guiding treatment of displaced fractures and dislocations of the ankle include reduction of injuries as soon as possible and maintaining reduction during healing period (eg, cast, external fixator, ORIF).
- Safeguard open fractures from further contamination by covering wounds with a wet, sterile dressing secured by loosely wrapped dry sterile gauze. Affirm current tetanus immunization, administering tetanus immunoglobulin when patients lack immunity and harbor a grossly contaminated wound.
 - Consider antibiotic prophylaxis, administering cephalexin for mildly to moderately contaminated wounds and adding an aminoglycoside for highly contaminated wounds. Administer vancomycin and gentamicin when the patient is allergic to penicillin.
 - Leave fracture blisters intact. Once ruptured, blisters are more likely to become contaminated by skin flora.
- Closed reduction is accomplished as follows (refer to [Dislocations, Ankle](#) for specific techniques):
 - The orthopedic consultant typically reduces ankle fractures. Ankle dislocations are reduced easily, and emergency physicians should be skilled in their initial management; however, immediate reduction of a dislocation is not required unless blood flow to the foot is compromised.
 - Provide either local anesthesia with a hematoma block or employ conscious sedation. As hematoma blocks prevent respiratory depression, they are useful in high-risk patients such as intoxicated patients, older persons, and children.
 - Closed reduction is best achieved by manipulating the limb to reverse the direction of the original deforming forces. For example, a fracture-dislocation resulting from abductive stress requires pushing the affected site in an adduct direction to restore. Applying a concurrent distracting force often assists reduction attempts.

- Splinting and casting
- Ankle splints are commercially available or may be constructed by sandwiching 10-12 layers of plaster between 4 sheets of cotton padding.
- Posterior splint: Stable injuries can be treated initially with a posterior splint. Ask the patient to lie prone with the knee bent to a 90-degree angle when applying a posterior splint. Extend the splint from the metatarsal heads along the posterior surface of the leg to the level of the fibular head. Maintain the ankle at a 90-degree angle, and mold the splint in the malleolar region. Discharge instructions include informing the patient to elevate the affected leg, apply ice, and refrain from bearing weight on the injured joint. As swelling diminishes, apply a walking cast.
- Sugar tong/short leg stirrup splint: An alternative to the posterior splint is a sugar tong or short leg stirrup splint. Using 4- or 6-inch plaster, pass the splint under the plantar aspect of the foot, between the calcaneus and metatarsal heads. Secure in place with an elastic wrap.
- Splinting of a fracture with bulky padding (eg, Jones dressing) is indicated when immobilization and compression are needed but swelling is expected to progress. In very unstable ankle fractures, apply a bivalve cast. A normal cast is bivalved by cutting completely through the casting material on the medial and lateral aspects longitudinally to avoid extremity compression. Next, the bivalved cast is overwrapped with an elastic bandage to stabilize the fracture site, while still allowing for swelling and expansion.

Consultations

- Request orthopedic consultation for the following conditions:
 - Displaced medial, lateral, or posterior malleolar fracture
 - Medial malleolar fracture with lateral ligament damage
 - Lateral malleolar fracture with deltoid ligament damage
 - Fibula fracture at or proximal to tibiotalar joint line
 - All bimalleolar fractures
 - All trimalleolar fractures
 - All intraarticular fractures
 - All open fractures
 - All pilon fractures

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Medication

Provide sufficient analgesia to patients sustaining an ankle fracture. A variety of medications can be used, ranging from oral acetaminophen to parenteral narcotics. For conscious sedation, agents include short-acting sedative-hypnotics and opiate analgesics, usually in combination. In addition, administer tetanus prophylaxis for open fractures.

Narcotic/Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Sedating properties of narcotics benefit patients who have sustained fractures.

| | |
|-------------------|---|
| Drug Name | Morphine sulfate (Duramorph, Astramorph, MS Contin)- Used to achieve a desired anxiolytic and analgesic effect because easily titrated to desired level of pain control or sedation. Reversed by naloxone. |
| Adult Dose | 2.5-5 mg IV q10-15min prn |
| Pediatric Dose | Neonates: 0.05-0.2 mg/kg/dose prn Children: 0.1-0.2 mg/kg q2-4h prn |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

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| Drug Name | Fentanyl citrate (Duragesic, Sublimaze)- Good choice for immediate pain relief and conscious sedation because of its rapid onset and short duration (30-60 min). Easily titrated to desired level of pain control or sedation. Easily reversed by naloxone. |
| Adult Dose | 0.5-2.0 mcg/kg IV/IM; titrate to desired level of pain control and/or sedation in increments of 25-50 mcg IV |
| Pediatric Dose | <2 years: 2-3 mcg/kg/dose IV/IM q30-60min 2-12 years: 1-2 mcg/kg q60min >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

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| Precautions | Caution in hypotension, respiratory depression, constipation, nausea, emesis, and urinary retention; idiosyncratic reaction, known as chest wall rigidity syndrome, may require neuromuscular blockade to increase ventilation |
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Anxiolytic/Hypnotics

Patients with painful injuries usually experience significant anxiety. Anxiolytics allow administration of a smaller analgesic dose to achieve the same effect.

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| Drug Name | Midazolam hydrochloride (Versed)- Short-acting benzodiazepine/sedative hypnotic used for its anxiolytic, amnestic, and sedating properties. Easily titrated and easily reversed with flumazenil. |
| Adult Dose | Loading dose: 0.05-0.2 mg IV q2 min Maintenance dose: Infuse 1-2 mcg/kg/min and titrate to desired effect; 0.5-1 mg IV q3min prn; titrate to desired level of sedation |
| Pediatric Dose | Infants <6 months: Not recommended 6 months to 5 years: 0.05-0.1 mg/kg IV; not to exceed total dose of 0.6 mg/kg 6-12 years: 0.025-0.05 mg/kg IV; not to exceed total dose of 0.4 mg/kg >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; preexisting hypotension; narrow-angle glaucoma; sensitivity to propylene glycol (diluent) |
| Interactions | Sedative effects may be antagonized by theophyllines; narcotics and erythromycin may accentuate sedative effects due to decreased clearance |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in congestive heart failure, pulmonary disease, renal impairment, and hepatic failure |

Antidotes

In conscious sedation, a benzodiazepine antagonist may be needed to reverse the sedation and respiratory depression resulting from benzodiazepines and narcotics.

An opioid antagonist also can be used to reverse oversedation in a patient manifesting significant respiratory depression.

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| Drug Name | Flumazenil (Romazicon)- Selective antagonist of benzodiazepine receptor. |
| Adult Dose | 0.2 mg IV q1min; total dose 1 mg at 1 time or 3 mg q1h |

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| Pediatric Dose | Not established Recommended dose: Initially, 0.01 mg/kg over 15 sec, then 0.005-0.01 mg/kg q1min intervals; not to exceed 0.2 mg |
| Contraindications | Documented hypersensitivity; serious cyclic-antidepressant overdose; patients given a benzodiazepine for control of potentially life-threatening condition (eg, increased intracranial pressure or status epilepticus) |
| Interactions | Caution in cases of mixed drug overdose; toxic effects due to other drugs taken in overdose (eg, cyclic antidepressants) may occur with reversal of benzodiazepine effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Patients on benzodiazepines for prolonged periods may experience seizures |

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| Drug Name | Naloxone (Narcan)- Prevents or reverses opioid effects including hypotension, respiratory depression, and sedation, possibly by displacing opiates from their receptor. Rapid onset of 1-2 min. Oversedation or respiratory depression should reverse rapidly. |
| Adult Dose | 0.4-2 mg IV |
| Pediatric Dose | 0.01 mg/kg IV |
| Contraindications | Documented hypersensitivity |
| Interactions | Decreases analgesic effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in cardiovascular disease; may precipitate withdrawal symptoms in patients addicted to opiates |

Antibiotics

Therapy must cover all likely pathogens in the clinical setting.

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| Drug Name | Cefazolin (Ancef, Kefzol, Zolicef)- Cephalosporin that binds to 1 or more penicillin-binding proteins, arrests bacterial cell wall synthesis, and inhibits bacterial replication. Primarily active against skin flora, including <i>Staphylococcus aureus</i> . Total daily dosages are the same for IV and IM routes. |
| Adult Dose | 2 g IV/IM q6-12h; not to exceed 12 g/d |
| Pediatric Dose | 25-100 mg/kg/d IV/IM; not to exceed 6 g/d |
| Contraindications | Documented hypersensitivity |

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| Interactions | Probenecid prolongs effect; aminoglycosides may increase renal toxicity; may yield false positive urine-dip test for glucose |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal impairment; superinfections and promotion of nonsusceptible organisms may occur with prolonged use or repeated therapy |

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| Drug Name | Gentamicin (Gentacidin, Garamycin)- Aminoglycoside antibiotic used for gram-negative bacterial coverage. Commonly used in combination with both an agent against gram-positive organisms and one that covers anaerobes. Used in conjunction with ampicillin or vancomycin for prophylaxis in patients with open fractures. |
| Adult Dose | 1.5 mg/kg IV; not to exceed 80 mg |
| Pediatric Dose | 2 mg/kg |
| Contraindications | Documented hypersensitivity; non-dialysis-dependent renal insufficiency |
| Interactions | Other aminoglycosides, cephalosporins, penicillins, or amphotericin B may increase nephrotoxicity; enhances effects of neuromuscular blocking agents, thus prolonged respiratory depression may occur; loop diuretics may increase auditory toxicity €possible irreversible hearing loss of varying degrees may occur (monitor regularly) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Narrow therapeutic index (not intended for long-term therapy); caution in renal failure (not on dialysis), myasthenia gravis, hypocalcemia, and conditions that depress neuromuscular transmission; adjust dose in renal impairment |

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| Drug Name | Vancomycin (Vancocin)- Potent antibiotic directed against gram-positive organisms and active against enterococcal species. Also useful in treatment of septicemia and skin structure infections. Used in conjunction with gentamicin for prophylaxis in patients with open fractures. May need to adjust dose in patients with renal impairment. |
| Adult Dose | 1 g IV over 1 h |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Erythema, histamine-like flushing, and anaphylactic reactions may occur when administered with anesthetic agents; taken concurrently with aminoglycosides, risk of nephrotoxicity may increase above that with aminoglycoside monotherapy; effects in neuromuscular blockade may be enhanced when coadministered with nondepolarizing muscle relaxants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

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| Precautions | Caution in renal failure, neutropenia; red man syndrome is caused by too rapid IV infusion (dose given over a few minutes) but rarely happens when dose given over 2 h or by PO or IP route; red man syndrome not an allergic reaction |
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| Drug Name | Ampicillin (Omnipen, Marcillin)- Used for prophylaxis in patients with open fractures; interferes with bacterial cell wall synthesis during active replication, causing bactericidal activity against susceptible organisms. |
| Adult Dose | 2 g IV/IM |
| Pediatric Dose | 50 mg/kg IV/IM |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid and disulfiram elevate levels; allopurinol decreases effects and has additive effects on ampicillin rash; may decrease effects of oral contraceptives |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal failure; evaluate rash and differentiate from hypersensitivity reaction |

Toxoids

Used for tetanus immunization. A booster injection in previously immunized individuals is recommended to prevent this potentially lethal syndrome.

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| Drug Name | Tetanus toxoid- Used to induce active immunity against tetanus in selected patients; tetanus and diphtheria toxoids are immunizing agents of choice for most adults and children >7 y; administer booster doses throughout life to maintain tetanus immunity; pregnant patients should receive only tetanus toxoid, not a diphtheria antigen-containing product. In children and adults, may administer into deltoid or midlateral thigh muscles. In infants, preferred site is midhigh laterally. |
| Adult Dose | Primary immunization: 0.5 mL IM; 2 injections 4-8 wk apart; third dose 6-12 mo after second injection Booster dose: 0.5 mL q10y |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; history of any type of neurological symptoms or signs following administration of this product FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are an important cause of provocative poliomyelitis |

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| Interactions | Patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization due to poor immune response; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent use of medication with systemic chloramphenicol since it may impair amnestic response to tetanus toxoid; concurrent use of tetanus immune globulin may delay development of active immunity by several days (interaction is nevertheless clinically insignificant and does not preclude its concurrent use) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Do not use to treat actual tetanus infections, or for immediate prophylaxis of unimmunized individuals (use instead tetanus antitoxin, preferably human tetanus immune globulin); diminished antibody response to active immunization may be seen in patients receiving immunosuppressive therapy; better to defer primary diphtheria immunization until immunosuppressive therapy discontinued; routine immunization of symptomatic and asymptomatic HIV-infected persons recommended |

Immunoglobulins

Administer tetanus immune globulin to patients who may not have been immunized against *Clostridium tetani* products.

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| Drug Name | Tetanus immune globulins (Hyper-Tet)- For passive immunization of persons with wounds that may be contaminated with tetanus spores. |
| Adult Dose | For prophylaxis: 250-500 U IM in opposite extremity to tetanus toxoid lesion For clinical tetanus: 3,000-10,000 U IM |
| Pediatric Dose | For prophylaxis: 250 U IM in opposite extremity to tetanus toxoid For clinical tetanus: 3,000-10,000 U IM |
| Contraindications | Since antibodies in globulin preparation may interfere with immune response to vaccination, do not administer within 3 mo of live virus immune globulin administration; may be necessary to revaccinate persons who received immune globulin shortly after live virus vaccination |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

Precautions

Persons with isolated IgA deficiency have potential for developing antibodies to IgA and could have anaphylactic reactions to subsequent administration of blood products that contain IgA; do not perform skin testing, since intradermal injection of concentrated gamma globulin may cause localized area of inflammation and can be misinterpreted, causing medication to be withheld from a patient not allergic to this material; true allergic responses to human gamma globulin given in prescribed IM manner are extremely rare; do not admix with other medications since usually incompatible

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Follow-up

Further Inpatient Care

- Admission criteria
 - Open fracture
 - Unstable fracture requiring urgent operative stabilization
 - Presence of or potential for neurovascular compromise (eg, severely comminuted pilon fracture)

Further Outpatient Care

- Stable fractures treated in ED should receive follow-up care by an orthopedist.
- Advise patients to refrain from bearing weight on ankle until seen by orthopedist.
- Provide crutches and instructions on their proper use.
- Ensure the patient can use the crutches properly.
- Instruct patients to elevate injured ankle to reduce swelling and pain.
- Apply ice packs to areas of swelling for 10-15 minutes every 3-4 hours while awake for first 24-48 hours (may be helpful).
- For unstable fractures that require operative care at a later date, consult an orthopedist and arrange timely follow-up care.
- Especially for older persons, assess the patient's ability to modify activities of daily living (ADL) due to imposed restrictions resulting from the injury. Occasionally, patients lacking family support may require admission for social reasons.
- Provide written and oral information on cast and/or splint care, and ensure patient understands which symptoms warrant immediate physician notification and/or return to ED.

in/Out Patient Meds

- Prescribe oral analgesics (eg, acetaminophen with or without codeine, hydrocodone) for pain relief. Some controversy exists whether NSAIDs impair fracture and ligament healing.

Transfer

- When appropriate orthopedic care is not available at current facility, transfer patient to a facility that provides a higher level of care (comply with Emergency Medical Treatment and Active Labor Act [EMTALA] regulations).
- Stabilize fracture prior to transfer.

Deterrence/Prevention

- Encourage patient to undergo rehabilitation to regain strength of ankle joint.
- Orthotics and proper shoe gear may help prevent future injury.

Complications

- Nonunion of fracture site requires orthopedic referral for operative repair.
- Malunion of the fracture site occurs more frequently than nonunion and potentially proceeds to degenerative changes of the joint. Chronic persistent symptoms such as pain, weakness, and instability of the ankle may develop. Refer such patients to a qualified orthopedist for evaluation and possible surgical revision.
- Traumatic arthritis complicates 20-40% of ankle fractures. Generally, the more severe the fracture, the greater the likelihood of posttraumatic arthritis; comminuted pilon fractures are most at risk. Older patients have an increased risk of arthritic complications.
- Sudeck atrophy, a type of reflex sympathetic dystrophy (RSD), may precede ankle fractures. Clinical features include complex pain, muscle atrophy, cyanosis, and edema. The term Sudeck atrophy is reserved for RSD-like conditions accompanied by a characteristic radiographic appearance (ie, spotty rarefaction), as opposed to the ground-glass appearance seen with disuse atrophy of bone.
- Osteochondral fractures of the talar surface can easily go unrecognized and if left untreated may result in chronic pain, locking, and swelling. If suspected, arrange appropriate orthopedic follow-up care.
- In children, ankle fractures involving the growth plate may cause chronic deformity with disturbance of growth of the limb.

Prognosis

- Prognosis can be improved with prompt, accurate diagnosis and appropriate treatment and referral.

- Complex open fractures with substantial soft-tissue damage have a worse prognosis than isolated closed ankle fractures.
- Isolated, nondisplaced lateral malleolus fracture, the most common ankle fracture, has a favorable prognosis and heals unremarkably.
- Aggressive rehabilitation helps reduce the majority of morbidity associated with ankle fractures.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to find fractures. This accounts for 20% of malpractice claims against emergency physicians. Causes include inadequate exams, acceptance of inadequate films, lack of real-time radiology consultation, failure to promptly treat or consult when evidence of vascular compromise exists, failure to explain limits of initial x-ray interpretation, failure to immobilize and prevent further injury, and failure to arrange follow-up care.
 - Failure to appreciate subtle fractures (eg, osteochondral lesions). These may not be conspicuous or go unrecognized on initial radiographs. When suspicion for a fracture remains high despite seemingly normal radiographs, splint the extremity, have the patient refrain from weight bearing, and arrange timely orthopedic referral.
 - Failure to provide clear and concise aftercare and follow-up instructions to all patients discharged from the ED. Give details of splint or cast care and list symptoms that warrant immediate physician notification and/or return to the ED.
-

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Fractures, Cervical Spine

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Introduction

Background

Approximately 5-10% of unconscious patients who present to the ED as the result of a motor vehicle accident or fall have a major injury to the cervical spine. Most cervical spine fractures occur predominantly at 2 levels. One third occur at the level of C2, and one half occur at the level of C6 or C7. Most fatal cervical spine injuries occur in upper cervical levels, either at craniocervical junction C1 or C2.

The normal anatomy of the cervical spine consists of 7 cervical vertebrae separated by intervertebral disks and joined by a complex network of ligaments. These ligaments keep individual bony elements behaving as a single unit.

View the cervical spine as 3 distinct columns: anterior, middle, and posterior. The anterior column is composed of two thirds of the vertebral bodies, the annulus fibrosus, intervertebral disks, and anterior longitudinal ligament. The middle column is composed of one third of the vertebral bodies, the annulus, intervertebral disk, and posterior longitudinal ligament. The posterior column contains all of the remaining posterior elements formed by the pedicles, transverse processes, articulating facets, laminae, and spinous processes.

The anterior and posterior longitudinal ligaments maintain the structural integrity of the anterior and middle columns. The posterior column is held in alignment by a complex ligamentous system, including the nuchal ligament complex, capsular ligaments, and ligamenta flava.

If 1 column is disrupted, other columns may provide sufficient stability to prevent spinal cord injury. If 2 columns are disrupted, the spine may move as 2 separate units, increasing the likelihood of spinal cord injury.

The atlas, C1, and the axis, C2, differ markedly from other cervical vertebrae. The atlas has no vertebral body; however, it is composed of a thick anterior arch with 2 prominent lateral masses and a thin posterior arch. The axis contains the odontoid process that represents fused remnants of the atlas body. The odontoid process is held in tight approximation to the posterior aspect of the anterior arch of C1 by the transverse ligament, which stabilizes the atlantoaxial joint.

Apical, alar, and transverse ligaments provide further stabilization by allowing spinal column rotation, preventing posterior displacement of the dens in relation to the atlas.

Pathophysiology

Cervical spine injuries are best classified according to several mechanisms of injury. These include flexion, flexion-rotation, extension, extension-rotation, vertical compression, lateral flexion, and imprecisely understood mechanisms that may result in odontoid fractures and atlanto-occipital dislocation.

Flexion injury

Common injuries associated with a flexion mechanism include the following:

- Simple wedge compression fracture without posterior disruption
- Flexion teardrop fracture
- Anterior subluxation
- Bilateral facet dislocation
- Clay shoveler fracture
- Anterior atlantoaxial dislocation

Simple wedge fracture

With a pure flexion injury, a longitudinal pull is exerted on the nuchal ligament complex that, because of its strength, usually remains intact. The anterior vertebral body bears most of the force, sustaining simple wedge compression anteriorly without any posterior disruption.

Radiographically, the anterior border of the vertebral body has diminished height and increased concavity along with increased density due to bony impaction (see [Picture 2A](#)). The prevertebral soft tissues are swollen.

The posterior column remains intact, making this a stable fracture that requires only use of a cervical orthosis for treatment.

Flexion teardrop fracture

A flexion teardrop fracture occurs when flexion of the spine, along with vertical axial compression, causes a fracture of the anteroinferior aspect of the vertebral body. This fragment is displaced anteriorly and resembles a teardrop (see [Picture 2B](#)). For this fragment to be produced, significant posterior ligamentous disruption must occur. Since the fragment displaces anteriorly, a significant degree of anterior ligamentous disruption exists.

This injury involves disruption of all 3 columns, making this an extremely unstable fracture that frequently is associated with spinal cord injury. Initial management is application of traction with cervical tongs.

Anterior subluxation

Anterior subluxation in the cervical spine occurs when posterior ligamentous complexes (nuchal ligament, capsular ligaments, ligamenta flava, posterior longitudinal ligament) rupture. The anterior longitudinal ligament remains intact. No associated bony injury is seen.

Radiographically, the lateral view shows widening of interspinous processes, and anterior and posterior contour lines are disrupted in flexion views (see [Picture 3](#)). Since the anterior columns remain intact, this fracture is considered mechanically stable by definition.

Anterior subluxation is rarely associated with neurologic sequelae. Nevertheless, most authorities approach this injury as if it were potentially unstable because of the significant displacement that can occur with flexion, and very rare cases have associated neurologic deficit.

Bilateral facet dislocation

Bilateral facet dislocation is an extreme form of anterior subluxation that occurs when a significant degree of flexion and anterior subluxation causes ligamentous disruption to extend anteriorly, which causes significant anterior displacement of the spine at the level of injury. This injury involves the annulus fibrosus, anterior longitudinal ligament, and posterior ligamentous complex. At the level of injury, ie, the upper vertebrae, inferior articulating facets pass superior and anterior to the superior articulating facets of the lower involved vertebrae because of extreme flexion of the spine.

Radiographically, this is seen as a displacement of more than half of the anteroposterior diameter of the vertebral body in the lateral view (see [Picture 4](#)).

This is an extremely unstable condition and is associated with a high prevalence of spinal cord injuries. Initial management is closed reduction and traction with cervical tongs. A significant number of bilateral facet dislocations are accompanied by disk herniation. In patients with these injuries, further neurologic damage may occur if the injured disk retropulses into the canal during the application of cervical traction. Therefore, a careful neurologic examination should accompany closed reduction in these patients.

Clay shoveler fracture

Abrupt flexion of the neck, combined with a heavy upper body and lower neck muscular contraction, results in an oblique fracture of the base of the spinous process, which is avulsed by the intact and powerful supraspinous ligament. Fracture also occurs with direct blows to the spinous process or with trauma to the occiput that causes forced flexion of the neck.

Injury commonly is observed in a lateral view, since the avulsed fragment is readily evident (see [Picture 5A](#)). Injury commonly occurs in lower cervical vertebrae; therefore, visualization of the C7-T1 junction in the lateral view is imperative. Injury also may be seen in the anteroposterior view as a vertically split appearance of the spinous process in the lower vertebrae (see [Picture 5B](#)).

Since injury involves only the spinous process, this fracture is considered stable, and it is not associated with neurologic impairment. Management involves only cervical immobilization with an orthotic device for comfort.

Flexion-rotation injury

Common injuries associated with a flexion-rotation mechanism include unilateral facet dislocation and rotary atlantoaxial dislocation.

Unilateral facet dislocation

Unilateral facet dislocation occurs when flexion, along with rotation, forces one inferior articular facet of an upper vertebra to pass superior and anterior to the superior articular facet of a lower vertebra, coming to rest in the intervertebral foramen (see [Picture 6A](#)). Although the posterior ligament is disrupted, vertebrae are locked in place, making this injury stable.

Radiographically, the lateral view shows an anterior displacement of the spine at the involved level of less than one half the diameter of the vertebral body. This is in contrast to the greater displacement seen with a bilateral facet dislocation, as discussed above. The anteroposterior view is useful in diagnosis of

unilateral dislocation because it shows a disruption in the line connecting the spinous processes at the level of the dislocation (see [Picture 6B](#)). The oblique view is also useful because it shows a disruption of the typical shingles appearance at the level of the involved vertebra (see [Picture 6C](#)). The dislocated superior articulating facet of the lower vertebra is seen projecting within the neural foramina.

The injury seldom is associated with neurologic deficits. The orthopedic consultant performs initial management, applying cervical traction to attempt closed reduction.

Rotary atlantoaxial dislocation

This injury is a specific type of unilateral facet dislocation.

Radiographically, the odontoid view shows asymmetry of the lateral masses of C1 with respect to the dens along with unilateral magnification of a lateral mass of C1 (wink sign). However, since the atlantoaxial joint permits flexion, extension, rotation, and lateral bending, radiographic asymmetry is produced when the head is tilted laterally or rotated or if a slightly oblique odontoid view is obtained despite perfect head positioning. To confirm true dislocation, basilar skull structures (jugular foramina) should appear symmetric in the presence of the findings described above.

This injury is considered unstable because of its location.

Extension injury

Common injuries associated with an extension mechanism include hangman fracture, extension teardrop fracture, fracture of the posterior arch of C1 (posterior neural arch fracture of C1), and posterior atlantoaxial dislocation.

Hangman fracture (traumatic spondylolisthesis of C2)

The name of this injury is derived from the typical fracture that occurs after hangings. Presently, it commonly is caused by motor vehicle accidents and entails bilateral fractures through the pedicles of C2 due to hyperextension.

Radiographically, a fracture line should be evident, extending through the pedicles of C2, along with obvious disruption of the spinolaminar contour line (see [Picture 7](#)).

Although considered an unstable fracture, it seldom is associated with spinal injury, since the anteroposterior diameter of the spinal canal is greatest at this level, and the fractured pedicles allow decompression. When associated with unilateral or bilateral facet dislocation at the level of C2, this particular type of hangman fracture is unstable and has a high rate of neurologic complications that require immediate referral for cervical traction to reduce the facet dislocation. All other types of

hangman fracture can be managed initially with a cervical orthotic device.

Extension teardrop fracture

As with flexion teardrop fracture, extension teardrop fracture also manifests with a displaced anteroinferior bony fragment. This fracture occurs when the anterior longitudinal ligament pulls fragment away from the inferior aspect of the vertebra because of sudden hyperextension. The fragment is a true avulsion, in contrast to the flexion teardrop fracture in which the fragment is produced by compression of the anterior vertebral aspect due to hyperflexion.

The fracture is common after diving accidents and tends to occur in lower cervical levels. It also may be associated with the central cord syndrome due to buckling of the ligamenta flava into spinal canal during the hyperextension phase of the injury.

This injury is stable in flexion but highly unstable in extension. Initial management is avoidance of iatrogenic extension and cervical traction with tongs.

Fracture of the posterior arch of C1 fracture (posterior neural arch fracture)

This fracture occurs when the head is hyperextended and the posterior neural arch of C1 is compressed between the occiput and the strong and prominent spinous process of C2, causing the weak posterior arch of C1 to fracture (see [Picture 8A](#)).

Radiographically, the lateral projection shows a fracture line through the posterior neural arch. The odontoid view fails to show any displacement of the lateral masses of C1 with respect to the articular pillars of C2, a finding that distinguishes this fracture from a Jefferson fracture.

The transverse ligament and the anterior arch of C1 are not involved, making this fracture stable. Initial management involves the differentiation of this benign fracture from a Jefferson fracture. Once this is accomplished, only use of a cervical orthosis is required.

Vertical (axial) compression injury

Common injuries associated with a vertical compression mechanism include Jefferson fracture (burst fracture of the ring of C1), burst fracture (dispersion, axial loading), atlas fracture, and isolated fracture of the lateral mass of C1 (pillar fracture).

Jefferson fracture (burst fracture of the ring of C1)

This fracture is caused by a compressive downward force that is transmitted evenly through the occipital condyles to the superior articular surfaces of the lateral masses of C1. The process displaces the masses

laterally and causes fractures of the anterior and posterior arches, along with possible disruption of the transverse ligament. Quadruple fracture of all 4 aspects of the C1 ring occurs.

Radiographically, it is characterized by bilateral lateral displacement of the articular masses of C1. The odontoid view shows unilateral or bilateral displacement of the lateral masses of C1 with respect to the articular pillars of C2; this finding differentiates it from a simple fracture of the posterior neural arch of C1 (see [Picture 8B](#)). The lateral projection usually reveals a striking amount of prevertebral soft tissue edema.

When displacement of the lateral masses is more than 6.9 mm, complete disruption of the transverse ligament has occurred, and immediate referral for cervical traction is warranted. If displacement is less than 6.9 mm, the transverse ligament is still competent, and neurologic injury is unlikely.

Burst fracture of the vertebral body

When downward compressive force is transmitted to lower levels in the cervical spine, the body of the cervical vertebra can shatter outward, causing a burst fracture. This fracture involves disruption of the anterior and middle columns, with a variable degree of posterior protrusion of the latter.

Radiographically, this fracture is evidenced by a vertical fracture line in the frontal projection and by comminution and protrusion of the vertebral body anteriorly and posteriorly with respect to the contiguous vertebrae in the lateral view (see [Picture 9](#)). Middle column posterior protrusion may extend into the spinal canal and can be associated with anterior cord syndrome. Burst fractures always require an axial CT scan or MRI to document amount of middle column retropulsion.

Initially manage burst fractures with a loss in height of more than 25%, retropulsion, or neurologic deficit by applying traction with cervical tongs. When none of those problems exist, the fracture is considered stable.

Multiple or complex injuries

Common injuries associated with multiple or complex mechanisms include odontoid fracture, fracture of the transverse process of C2 (lateral flexion), atlanto-occipital dislocation (flexion or extension with a shearing component), and occipital condyle fracture (vertical compression with lateral bending).

Mechanism of injury, location, and clinical relevance

Upper cervical spine (occiput to C2) injuries

Injuries at the upper cervical level are considered unstable because of their location. Nevertheless, since the diameter of the spinal canal is greatest at the level of C2, spinal cord injury from compression is the

exception rather than the rule. Incompletely understood mechanisms or a combination of them usually produces injuries encountered at this level.

Common injuries include fracture of the atlas, atlantoaxial subluxation, odontoid fracture, and hangman fracture (see [Extension injury](#)). Less common injuries include occipital condyle fracture, atlanto-occipital dislocation, atlantoaxial rotary subluxation (see [Flexion-rotation injury](#)), and C2 lateral mass fracture.

Atlas (C1) fractures

Four types of atlas fractures (I, II, III, IV) result from impaction of the occipital condyles on the atlas, causing single or multiple fractures around the ring. The first 2 types of atlas fracture are stable and include isolated fractures of the anterior and posterior arch of C1, respectively (posterior arch fracture is described under [Extension injury](#)). Anterior arch fractures usually are avulsion fractures from the anterior portion of the ring and have a low morbidity rate and little clinical significance. The third type of atlas fracture is a fracture through the lateral mass of C1. Radiographically, asymmetric displacement of the mass from the rest of the vertebra is seen in odontoid view. This fracture also has a low morbidity rate and little clinical significance. The fourth type of atlas fracture is the burst fracture of the ring of C1 and also is known as a Jefferson fracture (discussed under [Vertical \(axial\) compression injury](#)). This is

the most significant type of atlas fracture from a clinical standpoint because it is associated with neurologic impairment.

Initial management of types I, II, and III atlas fractures consists of placement of a cervical orthosis. Type IV fracture, or Jefferson fracture, is managed with cervical traction.

Atlantoaxial subluxation

When flexion occurs without a lateral or rotatory component at the upper cervical level, it can cause an anterior dislocation at the atlantoaxial joint if the transverse ligament is disrupted. Because this joint is near the skull, shearing forces also play a part in the mechanism causing this injury, as the skull grinds the C1-C2 complex in flexion. Since the transverse ligament is the main stabilizing force of the atlantoaxial joint, this injury is unstable. Neurologic injury may occur from cord compression between the odontoid and posterior arch of C1.

Radiographically, this injury is suspected if the predental space is more than 3.5 mm (5 mm in children). Use axial CT to confirm the diagnosis. These injuries may require fusion of C1 and C2 for definitive management.

Atlanto-occipital dislocation

When severe flexion or extension exists at the upper cervical level, atlanto-occipital dislocation may

occur. Atlanto-occipital dislocation involves complete disruption of all ligamentous relationships between the occiput and the atlas. Death usually occurs immediately from stretching of the brainstem, which causes respiratory arrest.

Radiographically, disassociation between the base of the occiput and the arch of C1 is seen. Cervical traction is absolutely contraindicated, since further stretching of the brainstem can occur.

Odontoid process fractures

The 3 types of odontoid process fractures are classified based on the anatomic level at which the fracture occurs (see [Picture 1](#)).

- Type I odontoid fracture is an avulsion of the tip of the dens at the insertion site of the alar ligament. Although a type I fracture is mechanically stable, it often is seen in association with atlanto-occipital dislocation and must be ruled out because it is life threatening.
- Type II fractures occur at the base of the dens and are the most common odontoid fractures. This type is associated with a high prevalence of nonunion because of the limited vascular supply and a small area of cancellous bone.
- Type III odontoid fracture occurs when the fracture line extends into the body of the axis. Nonunion is not a major problem with these injuries because of a good blood supply and the greater amount of cancellous bone.

With types II and III fractures, the fractured segment may be displaced anteriorly, laterally, or posteriorly. Since posterior displacement of segment is more common, the prevalence of spinal cord injury is as high as 10% with these fractures.

Initial management of a type I dens fracture is use of a cervical orthosis. Manage types II and III fractures by applying traction with cervical tongs.

Occipital condyle fracture

Occipital condyle fractures are caused by a combination of vertical compression and lateral bending. Avulsion of the condylar process or a comminuted compression fracture may occur secondary to this mechanism. These fractures are associated with significant head trauma and usually are accompanied by cranial nerve deficits.

Radiographically, they are difficult to delineate, and axial CT may be required to identify them.

These mechanically stable injuries require only orthotic immobilization for management, and most heal uneventfully. These fractures are significant because of the injuries that usually accompany them.

Mechanical instability

Column disruption may lead to mechanical instability of the cervical spine. The degree of instability depends on several factors that may translate into neurologic disability, secondary to spinal cord compression. A full spectrum of cervical injuries with varying degrees of clinical importance, from the clinically insignificant to the potentially disastrous, exists. As many as 39% of cervical fractures have some degree of associated neurologic deficit.

The risk of neurologic injury, secondary to spinal injury, increases with degenerative changes related to aging, arthritic conditions (rheumatoid arthritis, ankylosing spondylitis), spinal stenosis, spina bifida, and os odontoideum, as well as the specific mechanism and location of the injury.

Trafton has ranked specific cervical injuries based on their degree of mechanical instability. The list below ranks cervical spine injuries in order of instability (most to least unstable):

- Rupture of the transverse ligament of the atlas
- Fracture of the dens (odontoid fracture)
- Burst fracture with posterior ligamentous disruption (flexion teardrop fracture)
- Bilateral facet dislocation
- Burst fracture without posterior ligamentous disruption
- Hyperextension fracture dislocation
- Hangman fracture
- Extension teardrop (stable in flexion)
- Jefferson fracture (burst fracture of the ring of C1)
- Unilateral facet dislocation
- Anterior subluxation
- Simple wedge compression fracture without posterior disruption
- Pillar fracture
- Fracture of the posterior arch of C1
- Spinous process fracture (clay shoveler fracture)

Frequency

- **In the US:** Cervical spine injuries cause an estimated 6000 deaths and 5000 new cases of quadriplegia each year.

Sex

Male-to-female ratio is 4:1.

Age

- Most patients with a cervical spine injury are in their prime and leading an active lifestyle prior to injury.
- About 80% of patients are aged 18-25 years.

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Clinical

History

Common presentations include the following:

- Posterior neck pain on palpation of spinous processes
- Limited range of motion associated with pain
- Weakness, numbness, or paresthesias along affected nerve roots

Physical

Clinical evaluation of the cervical spine in a patient with blunt trauma is unreliable. In a study of surgical residents' ability to predict cervical injuries on the basis of clinical examination alone, sensitivity and specificity were 46% and 94%, respectively. Because of these limitations and potential for catastrophic morbidity if injury is missed, most patients with complex blunt trauma seen in the ED undergo radiographic evaluation before clearance, with some exceptions.

Common findings on physical examination in cervical spine injury include the following:

- Spinal shock
 - Flaccidity
 - Areflexia
 - Loss of anal sphincter tone
 - Fecal incontinence
 - Priapism
 - Loss of bulbocavernosus reflex
- Autonomic dysfunction
 - Ileus
 - Urinary retention
 - Poikilothermia

Causes

Motor vehicle accidents and falls account for 50% and 20% of these injuries, respectively. Sports-related activities account for 15%. The remaining injuries are attributed to interpersonal violence.

The following athletic activities have the highest incidence of associated cervical spine injuries. Participants in these events should be considered at high risk.

- Diving
- Equestrian activities
- Football
- Gymnastics
- Skiing
- Hang gliding

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Differentials

Cauda Equina Syndrome
Cervical Strain
Dissection, Vertebral Artery
Hanging Injuries and Strangulation
Neck Trauma
Neoplasms, Spinal Cord
Shock, Septic
Spinal Cord Infections
Spinal Cord Injuries
Thoracic Outlet Syndrome
Torticollis

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Workup

Imaging Studies

- Radiographic evaluation
 - Patients who exhibit neurologic deficits consistent with a cord lesion
 - Patients with an altered sensorium from head injury or intoxication
 - Patients who complain about neck pain or tenderness
 - Patients who do not complain about neck pain or tenderness but have significant distracting injuries
 - Controversy surrounds trauma patients who are alert, not intoxicated, neurologically intact, and without neck pain or tenderness. One study did not find a single cervical spine injury in 549 of such patients. Similarly, another study did not identify any injury in 155 alert trauma patients without neck complaints. These and other studies seem to indicate that this subgroup of patients has virtually no risk of having a cervical spine injury; thus, radiographic evaluation is not warranted.
- Cross-table lateral view
 - Approximately 85-90% of cervical spine injuries are evident in lateral view, making it the most useful view from a clinical standpoint.
 - A technically acceptable lateral view shows all 7 vertebral bodies and the cervicothoracic junction. Approach analysis of this view methodically to avoid missing significant pathology.
 - Check alignment of cervical spine by following 3 imaginary contour lines.
 1. The first line connects the anterior margins of all the vertebrae and is referred to as the anterior contour line (see [Picture 10A](#)).
 2. The second line should connect the posterior aspect of all vertebrae in a similar way and is referred to as the posterior contour line.
 3. The third line should connect the bases of the spinous processes and is referred to as the spinolaminar contour line.
 - An exception occurs in young children who, because of immature muscular development, may have a benign pseudosubluxation in the upper cervical spine. An imaginary straight line should connect the points bisecting the base of the spinous processes of C1, C2, and C3. In pseudosubluxation, these imaginary points should not be displaced more than 2 mm in front of or behind the straight line.
 - Check individual vertebrae thoroughly for obvious fracture or changes in bone density. Areas of decreased bone density are seen in patients with osteoporosis, osteomalacia, or osteolytic lesions and may represent weak areas predisposed to injury. Areas of increased bony density may be seen with osteoblastic lesions or may represent compression fractures of an acute nature.
 - Look for soft tissue changes in predental and prevertebral spaces. The predental space, also known as the atlantodental interval, is the distance between the anterior aspect of the odontoid and the posterior aspect of the anterior arch of C1 (see [Picture 10A](#)). This space should be no more than 3 mm in an adult and 5 mm in a child. Suspect transverse ligament disruption if these limits are exceeded.
 - Prevertebral space extends between the anterior border of the vertebra to the posterior wall of the pharynx in the upper vertebral level (C2-C4) or to the trachea in the lower vertebral level (C6).

- At the level of C2, prevertebral space should not exceed 7 mm.
- At the level of C3 and C4, it should not exceed 5 mm, or it should be less than half the width of the involved vertebrae.
- At the level of C6, prevertebral space is widened by the presence of the esophagus and cricopharyngeal muscle. At this level, the space should be no more than 22 mm in adults or 14 mm in children younger than 15 years.
- Children younger than 24 months may exhibit a physiologic widening of the prevertebral space during expiration; therefore, obtain images in small children during inspiration to assess prevertebral space adequately.
- If the prevertebral space is widened at any level, a hematoma secondary to a fracture is the most likely diagnosis.
- Check for fanning of the spinous processes. This is evident as an exaggerated widening of the space between 2 spinous process tips and suggests posterior ligamentous disruption.
- Check for an abrupt change in angulation of greater than 11 degrees at a single interspace. This also suggests bony injury with possible ligamentous involvement.
- Oblique view
 - This view also is considered a laminar view because most pathologic conditions assessed on it manifest with some disruption in the normal overlapping appearance of the vertebral laminae.
 - The normal structural appearance of the laminae is described as shingles on a roof, forming a regular elliptical curve with equal interlaminar spaces (see [Picture 10B](#)).
 - If interlaminar space between 2 continuous laminae is increased, suspect subluxation of the involved vertebrae.
 - Similarly, if the expected tiling of shingles is disrupted, suspect a unilateral facet dislocation.
 - A posterior laminar fracture should be evident as disruption of the body of a single shingle.
- Anteroposterior view
 - This is the least useful view from a clinical standpoint.
 - A straight line should connect the spinous processes bisecting the cervical spine. If this is not seen, consider a rotation injury (ie, unilateral facet dislocation). Also consider a clay shoveler fracture if a spinous process appears vertically split.

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Treatment

Prehospital Care

When a cervical spine injury is suspected, minimize neck movement during transport to the treating facility. Ideally, transport the patient on a backboard with a semirigid collar, with the neck stabilized on

the sides of the head with sand bags or foam blocks taped from side to side (of the board), across the forehead.

Emergency Department Care

- If spinal malalignment is identified, place the patient in skeletal traction with tongs as soon as possible (with very few exceptions), even if no evidence of neurologic deficit exists.
- The specific injury involved and capabilities of the consulting staff guide further management.
- Place tongs 1 finger width above the ear lobes in alignment with the external auditory canal.
- The consultant applies the tongs for traction under close neurologic and radiograph surveillance.

Consultations

- An orthopedic surgeon or neurosurgeon, depending on local availability, custom, or referral system, should be available for immediate referral.
- If the treating physician notes spinal cord injury, consult a neurosurgeon.

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Medication

Administer steroids to any patient with blunt cervical spine injury and associated neurologic symptoms of less than 8 hours in onset.

Corticosteroids

Agents have anti-inflammatory properties and cause profound, varied metabolic effects. In addition, these agents modify the body's immune response to diverse stimuli.

| | |
|-------------------|---|
| Drug Name | Methylprednisolone (Solu-Medrol, Depo-Medrol)- Decrease inflammation by suppressing the migration of polymorphonuclear leukocytes and reversing increased capillary permeability. |
| Adult Dose | 30 mg/kg IV q30min; followed by continuous IV drip 5.4 mg/kg q1h for 1 d |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; viral, fungal, or tubercular skin infections |

| | |
|--------------|--|
| Interactions | Coadministration with digoxin, may increase digitalis toxicity secondary to hypokalemia; estrogens may increase levels of methylprednisolone; phenobarbital, phenytoin, and rifampin may decrease levels of methylprednisolone (adjust dose); monitor patients for hypokalemia when they are taking medication concurrently with diuretics |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Hyperglycemia, edema, osteonecrosis, peptic ulcer disease, hypokalemia, osteoporosis, euphoria, psychosis, growth suppression, myopathy, and infections are possible complications of glucocorticoid use |

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Follow-up

Complications

- Spinal shock
 - Severe spinal cord injury may cause a concussive injury of the spinal cord termed spinal shock syndrome.
 - Spinal shock manifests as distal areflexia of a transient nature that may last from a few hours to weeks. Initially, the patient experiences a flaccid quadriplegia along with areflexia. Segmental reflexes start to return usually within 24 hours as spinal shock starts to resolve. At that point, flaccid quadriplegia changes to spastic paralysis.
 - Eventually, total resolution can be expected.
- Complete and incomplete cord syndromes
 - Besides spinal shock, complete and incomplete spinal cord syndromes may occur.
 - Spinal shock mimics a complete spinal cord lesion. Emergency physicians should wait until spinal shock resolves to make an accurate estimate of the patient's prognosis.
 - Incomplete cord syndromes are described and include anterior spinal cord syndrome, central spinal cord syndrome, Brown-Séquard syndrome, and less frequent, cord syndromes at high cervical levels (ie, Horner syndrome, posteroinferior cerebellar artery syndrome).
 - The prognosis of a patient with a complete lesion, after spinal shock subsides, is permanent paraplegia.
 - Patients with an incomplete lesion (partial motor or sensory function) can expect to regain some degree of function.
- Central spinal cord syndrome
 - This syndrome is caused by damage to the corticospinal tract.
 - It is characterized by weakness, greater in the upper extremities than the lower extremities and more pronounced in the distal aspect of extremity.

- The syndrome usually is associated with a hyperextension injury in patients with spondylosis or congenital stenosis of the cervical canal.
- Extension of the cervical spine, causing buckling of the ligamentum flavum into the spinal cord, is believed to cause central spinal cord syndrome.
- High cervical spinal cord syndromes
 - These syndromes are associated with damage to the spinal tract of the trigeminal nerve in the high cervical region.
 - A characteristic onion-skin pattern of anesthesia in the face may occur.
- Posteroinferior cerebellar artery syndrome
 - A diverse constellation of symptoms, including dysphagia, dysphonia, hiccups, vertigo, vomiting, or cerebellar ataxia, may occur.
 - Any of the high cervical cord syndromes may result from direct injury to the upper cervical level and/or cervicomedullary junction.
 - Vertebral artery occlusion from dislocation or hyperextension of the cervical column also can produce them.

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Miscellaneous

Medical/Legal Pitfalls

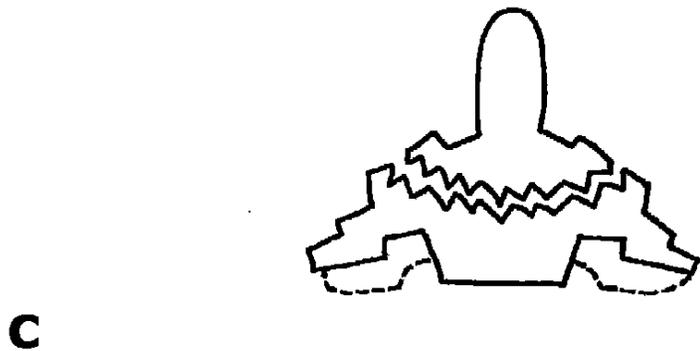
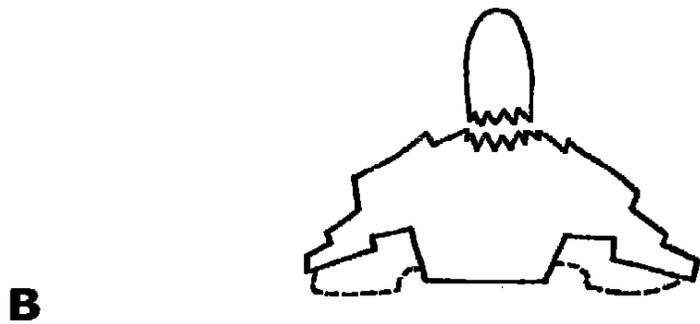
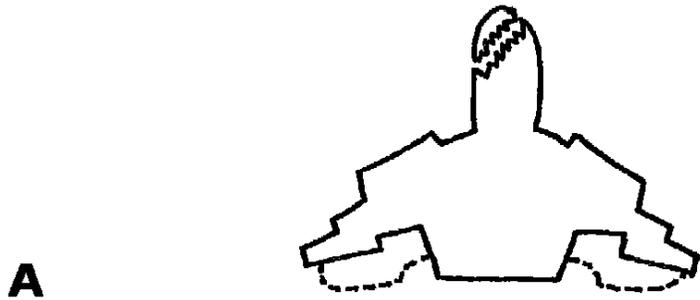
- Failure to fully visualize these areas has resulted in patient morbidity and successful malpractice litigation against emergency physicians.

Special Concerns

- Pediatric considerations
 - Structural differences between pediatric and adult cervical spines alter injury patterns and cause distinct pathology in young children.
 - The more elastic intervertebral ligaments and more horizontally aligned facet joints in young children predispose them to subluxation of the cervical spine without bony injury.
 - Immature neck muscles and a proportionally large head further compound this effect, making pediatric cervical spines act like a fulcrum and increasing the chance of injury.
 - This fulcrum starts in the upper cervical levels and changes progressively to lower levels as the pediatric cervical spine matures, until it reaches adult levels at C5 and C6. Most injuries occur at the C1-C3 levels in children younger than 8 years.
-

Pictures

FIGURE 1

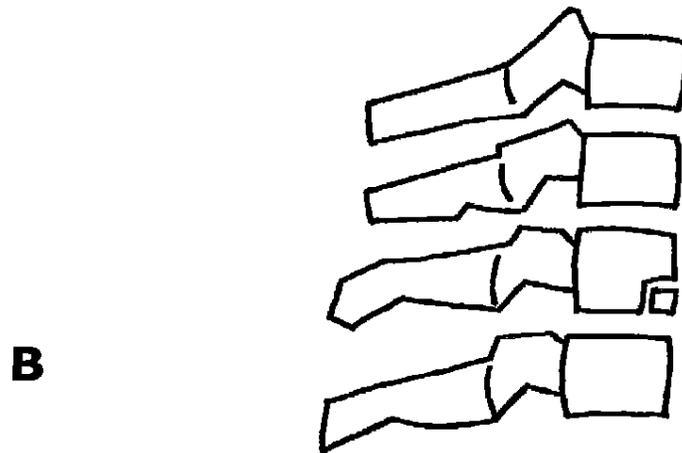
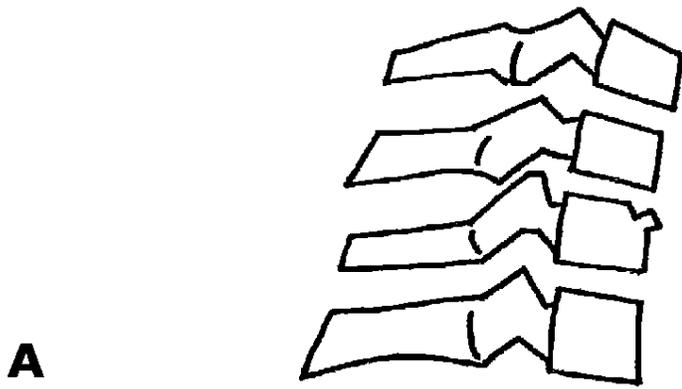


Picture 1: Odontoid fractures. A. Type I odontoid fracture represents an avulsion of the tip of the dens at the insertion site of the alar ligament. Although mechanically stable, it is associated with life-threatening

atlanto-occipital dislocation. B. Type II odontoid fracture is a fracture at the base of the dens. This is the most common type of odontoid fracture. C. With type III odontoid fracture, the fracture line extends into the body of the axis.

Picture type: Photo

FIGURE 2

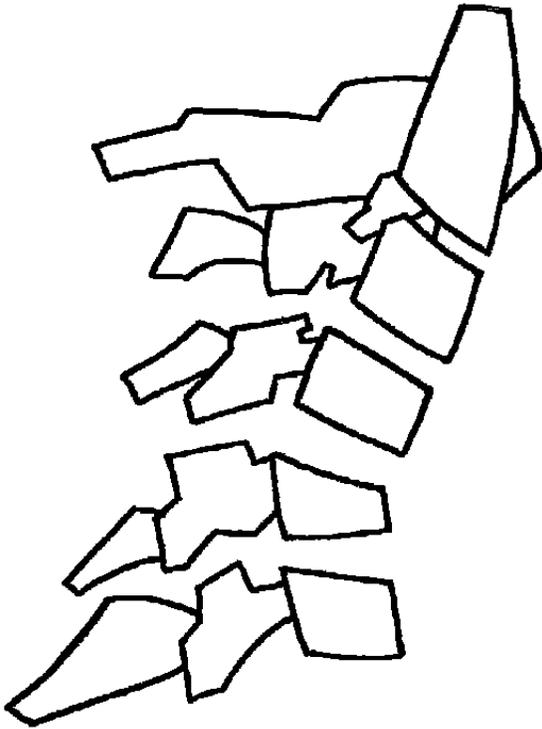


Picture 2: A. Simple wedge fracture with a flexion mechanism of injury is stable. B. Flexion teardrop

fracture with a flexion mechanism is unstable.

Picture type: Photo

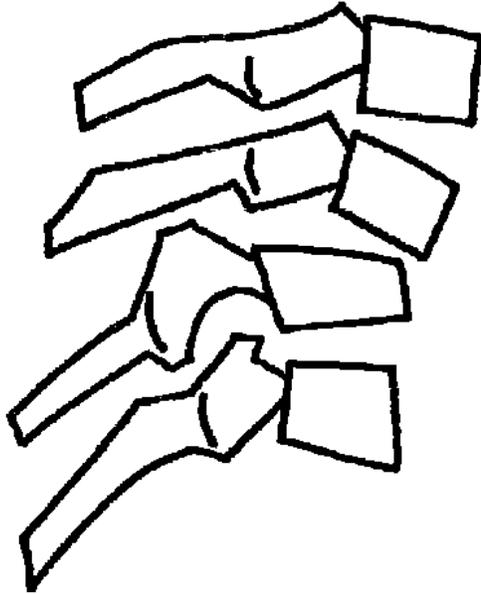
FIGURE 3



Picture 3: Anterior subluxation with a flexion mechanism is stable in extension but potentially unstable in flexion.

Picture type: Photo

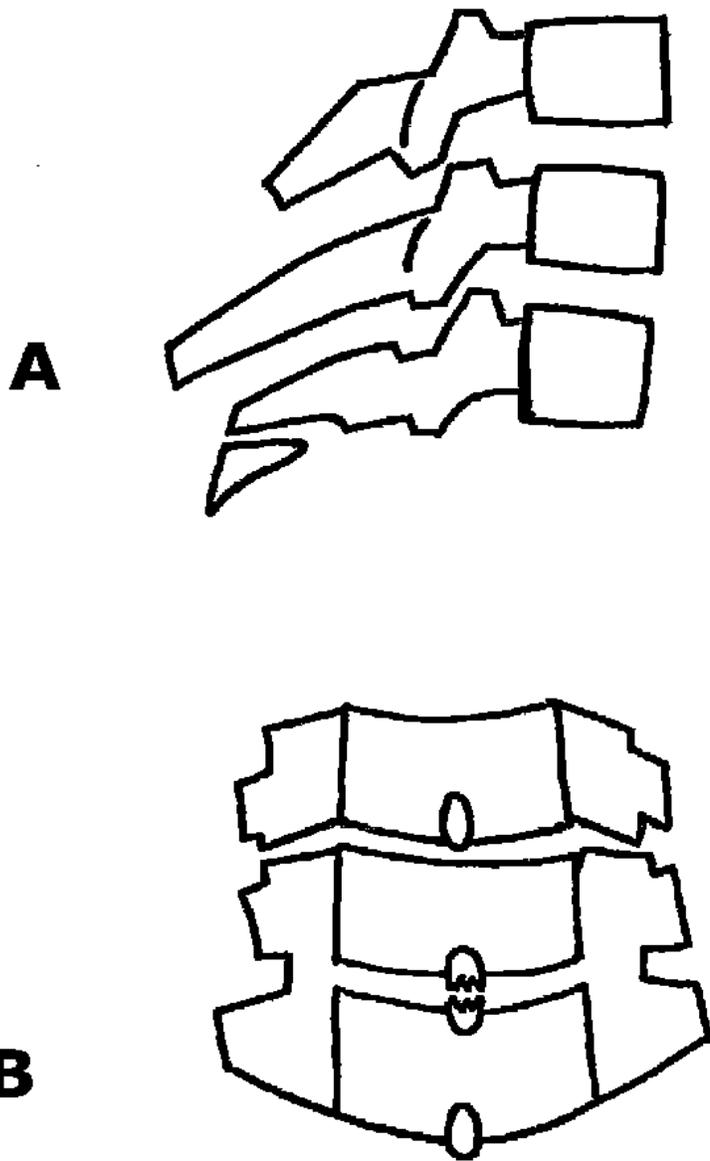
FIGURE 4



Picture 4: Bilateral facet dislocation with a flexion mechanism is extremely unstable and can have an associated disk herniation that impinges on the spinal cord during reduction.

Picture type: Photo

FIGURE 5



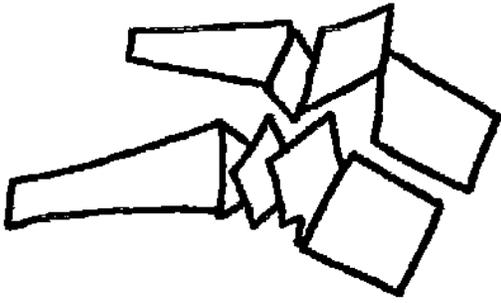
Picture 5: Clay shoveler fracture. A. Lateral view of this fracture caused by a flexion mechanism shows that it is stable and represents an avulsion fracture of the base of the spinous process near the supraspinous ligament. B. Anteroposterior view shows the vertically split appearance of the spinous process.

Picture type: Photo

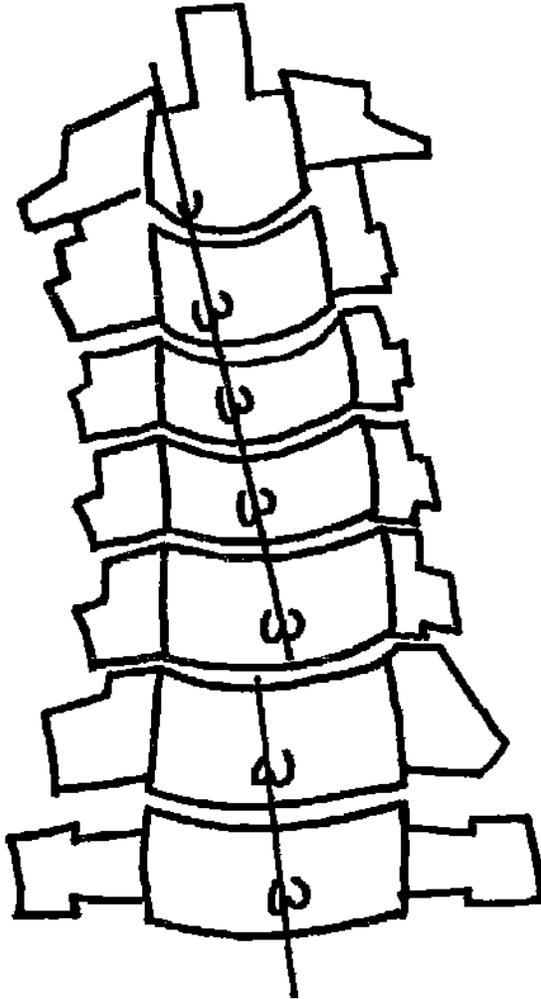
FIGURE 6



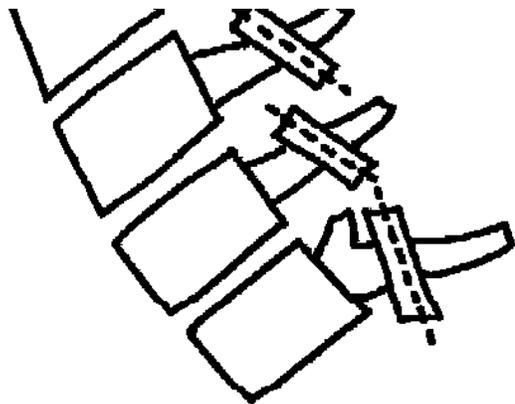
A



B



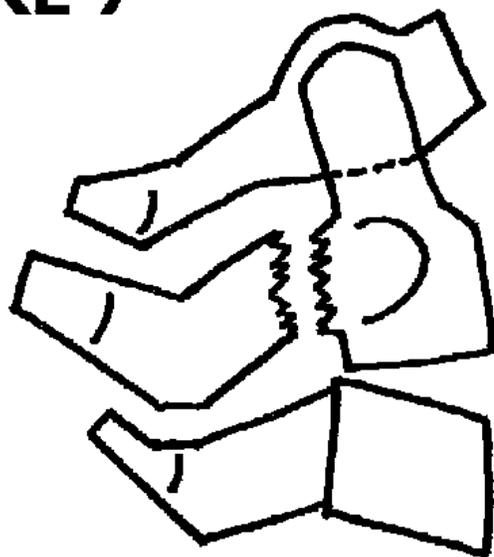
C



Picture 6: Unilateral facet dislocation. A. Lateral view of this fracture caused by a flexion-rotation mechanism shows that it is stable. Anterior displacement of spine is less than one half of the diameter of a vertebral body. B. Anteroposterior view shows disruption of a line connecting spinous processes at the level of the dislocation. C. Oblique view shows that the expected tiling of the laminae is disrupted, and the dislocated superior articulating facet of the lower vertebra is seen projecting within the neural foramina.

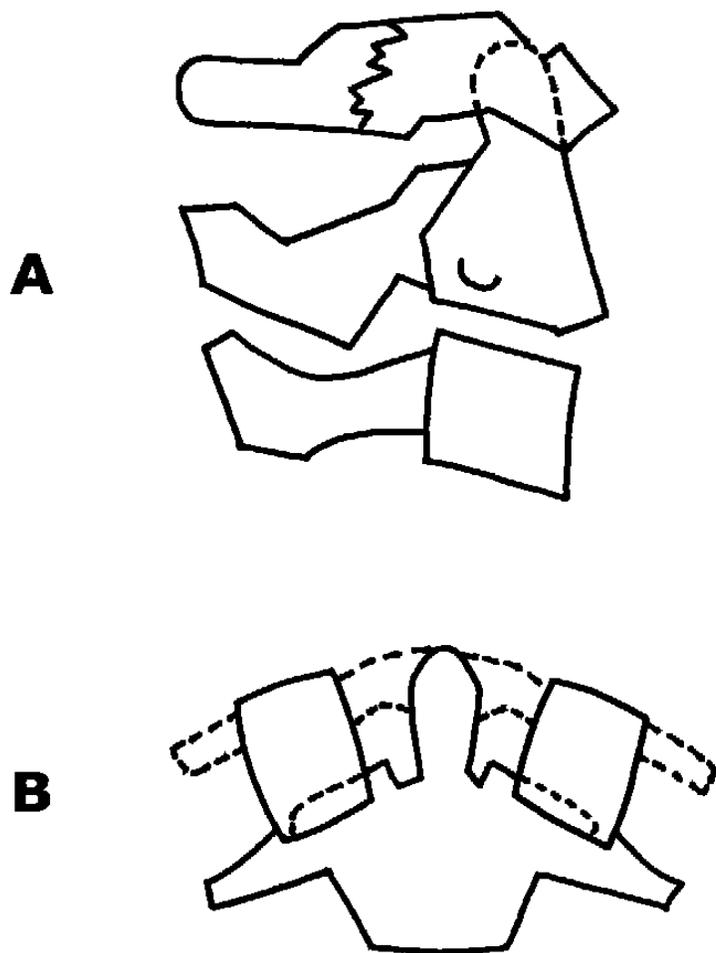
Picture type: Photo

FIGURE 7



Picture 7: Hangman fracture caused by an extension mechanism is unstable. Fracture line is evident in the lateral projection extending through pedicles of C2, along with disruption of the spinolaminar line. Sometimes, this fracture is associated with unilateral or bilateral facet dislocation, which makes it highly unstable.

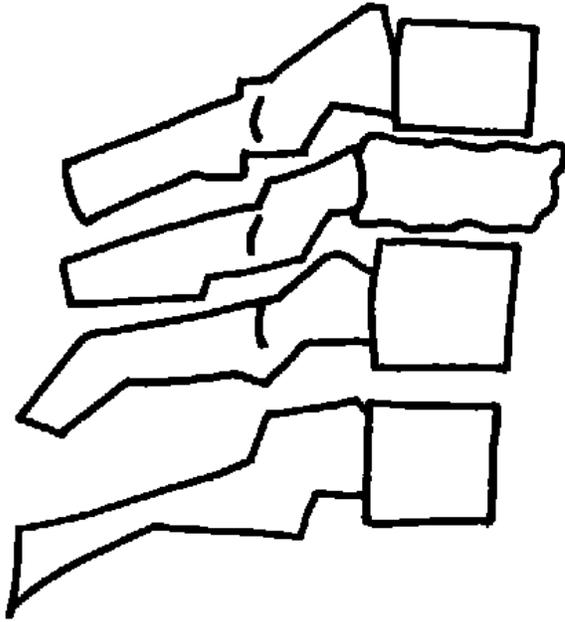
Picture type: Photo

FIGURE 8

Picture 8: A. Fracture of the posterior arch of C1 fracture caused by an extension mechanism is stable. Lateral projection shows a fracture line through the posterior neural arch without widening predental space. An odontoid view must be obtained to differentiate this benign fracture from a Jefferson fracture. B. Jefferson fracture caused by a vertical (axial) compression mechanism is unstable. This fracture of all aspects of the C1 ring is associated with possible disruption of the transverse ligament of the atlas. Lateral projection may show a widened predental space and a fracture through the posterior arch of C1. Odontoid view shows displacement of the lateral masses of C1, allowing distinction of this fracture from a simple fracture of the posterior neural arch of C1.

Picture type: Photo

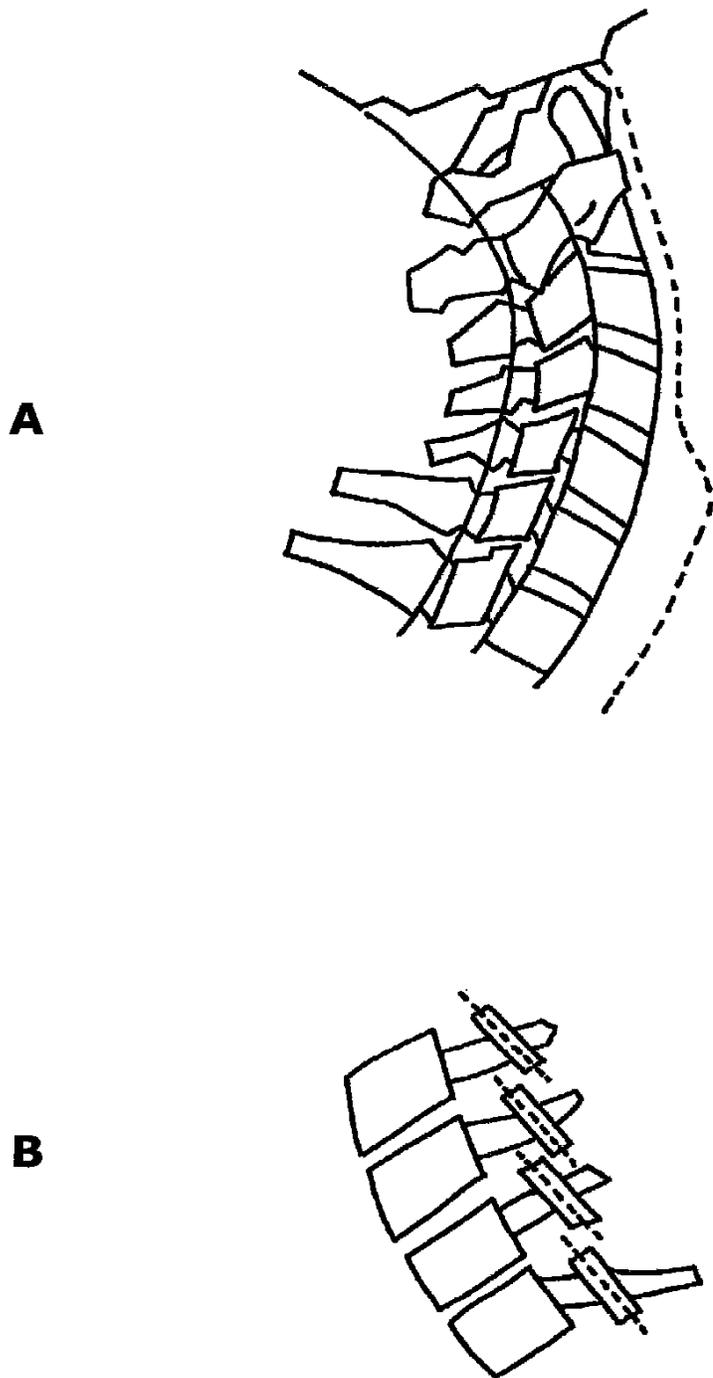
FIGURE 9



Picture 9: Burst fracture of vertebral body caused by a vertical (axial) compression mechanism is stable mechanically and involves disruption of the anterior and middle columns, with variable degree of protrusion of the latter. This middle column posterior protrusion may extend into the spinal canal and be associated with an anterior cord syndrome.

Picture type: Photo

FIGURE 10



Picture 10: A. Normal lateral projection shows the relationships of anterior, posterior, and spinolaminar lines and prevertebral spaces. B. Normal oblique projection shows the normal appearance of the laminae as shingles on a roof forming a regular elliptical curve with equal interlaminar spaces.

Picture type: Photo

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Fractures, Clavicle

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Introduction

Background

Clavicular fractures are common injuries that account for approximately 5% of all fractures seen in the ED. In neonates and children, these fractures are very common and generally heal well. In adults, the force required to fracture the clavicle is greater, healing occurs at a slower rate, and risk of potential complications is higher.

Pathophysiology

The clavicle is the sole articulation of the shoulder girdle to the trunk. It protects major underlying vessels, lung, and brachial plexus. Displaced clavicle fractures can injure these structures because of their proximity and sharp edges.

Approximately 80% of clavicle fractures occur in the middle third (class A), 15% involve the distal or lateral third (class B), and 5% the proximal or medial third (class C). The anatomy of the clavicle with potential fracture sites marked is shown in [Picture 1](#).

Class B fractures are classified further as type I or nondisplaced, in which supporting ligaments remain

intact with no significant displacement of fracture fragments (see [Picture 2](#)), type II or displaced, in which the coracoclavicular ligament ruptures with resultant upward displacement of the proximal segment because of sternocleidomastoid muscle (see [Picture 3](#), [Picture 4](#)), and type III or articular surface (involving acromioclavicular joint).

Frequency

- **In the US:** Clavicular fractures account for approximately 5% of ED visits for fractures.

Mortality/Morbidity

While the overwhelming majority of clavicle fractures are benign, associated life-threatening intrathoracic injuries are possible. Complications vary based on location of fracture (see [Complications](#)).

Age

Clavicle fractures are the most common of all pediatric fractures. They can present in the newborn period, especially following a difficult delivery, and nearly half of all clavicle fractures occur in children younger than 7 years. In young children, the fracture is often incomplete (ie, greenstick fracture) or a bowing deformity without definite fracture.

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Clinical

History

- Patient typically reports a fall onto an outstretched upper extremity, a fall onto a shoulder, or direct clavicular trauma.
- Pain, especially with upper extremity movement
- Swelling

Physical

- Tenderness
- Crepitus
- Edema
- Deformity

- Ecchymosis, especially when severe displacement causes tenting of skin
- Bleeding from open fracture (rare)
- Decreased breath sounds on auscultation, indicating possible pneumothorax
- Decreased pulses or evidence of decreased perfusion on vascular exam, suggesting vascular compromise
- Diminished sensation or weakness on distal neurovascular exam, suggesting neurologic compromise
- Nonuse of arm on affected side in neonates

Causes

- Fall onto shoulder or outstretched upper extremity
- Direct blow to clavicle

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Differentials

Dislocations, Shoulder

Fractures, Rib

Pneumothorax, Tension and Traumatic

Rotator Cuff Injuries

Sternoclavicular Joint Injury

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Workup

Imaging Studies

- Routine clavicle radiographs
 - Fracture usually is demonstrated on an anteroposterior (AP) view.
 - Apical lordotic views may be required to define degree of displacement.

Other Tests

- Other tests may be required when clinically indicated to assess possibility of life-threatening

associated injuries.

- Chest radiograph, if pneumothorax suspected
- Angiography, if vascular injury suspected

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Treatment

Prehospital Care

- Identify and treat associated life-threatening injuries.
 - Employ ABC approach to evaluation, and stabilize patient.
 - Perform a careful secondary survey.
 - Apply a cold pack to injury.
 - Immobilize upper extremity with a sling.

Emergency Department Care

- Identify and treat associated life- and limb-threatening injuries. If fracture is open, treat patient with prophylactic antibiotics, tetanus immunization (if needed), irrigation, and placement of a sterile dressing while awaiting urgent orthopedic consultation.
- Class A (middle third fractures): Treat with sling immobilization. Some prefer using a figure-of-eight clavicular splint, especially for displaced fractures.
- Class B (distal third fractures): Treat type I (nondisplaced) and type III (articular surface) fractures with sling immobilization. Immobilize type II (displaced) fractures in a sling and swathe. These may require orthopedic surgical fixation.
- Class C (proximal third): Treat nondisplaced fractures with sling immobilization. Displaced injuries may require orthopedic referral for surgical reduction. Neonatal fractures generally heal spontaneously in several weeks without special treatment.

Consultations

- Consult a trauma surgeon immediately when the patient has evidence of multisystem involvement.
- Orthopedic surgery
 - Open fractures necessitate urgent consultation.
 - Displaced fractures may need surgical repair, necessitating referral.

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Medication

Prophylactic intravenous antibiotics that cover typical skin flora (such as cefazolin sodium) are necessary with open fractures. Control discomfort with nonsteroidal anti-inflammatory drugs (NSAIDs), and if pain continues, add a narcotic analgesic. Tetanus immunization also may be indicated.

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

These agents are used most commonly for relief of mild to moderately severe pain. Effects of NSAIDs in treatment of pain tend to be patient specific, yet ibuprofen is usually DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually DOC for treatment of mild to moderately severe pain, if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, which inhibits prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ €monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for relief of mild to moderately severe pain and inflammation. Administer small dosages initially to patients with small bodies, older patients, and those with renal or liver disease. Doses higher than 75 mg do not increase therapeutic effects. Administer high doses with caution and observe. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 14 years: 0.1-1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€”monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Flurbiprofen (Ansaid)- Has analgesic, antipyretic, and anti-inflammatory effects. May inhibit cyclooxygenase enzyme, inhibiting prostaglandin biosynthesis. |
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€”monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|--|
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |
|-------------|--|

| | |
|-------------------|--|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of cyclooxygenase, which decreases prostaglandin synthesis. |
| Adult Dose | 500 mg PO followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures.

| | |
|-----------|---|
| Drug Name | Acetaminophen (Tylenol, Panadol, aspirin-free Anacin)- DOC for treatment of pain in patients with documented hypersensitivity to aspirin or NSAIDs or in those with upper GI disease or taking oral anticoagulants. |
|-----------|---|

| | |
|-------------------|---|
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; known G-6-P deficiency |
| Interactions | Rifampin can reduce analgesic effects; barbiturates, carbamazepine, hydantoin, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; APAP is contained in many OTC products and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

| | |
|-------------------|---|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tabs or caps PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|--|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tab q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose PO based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants increase toxicity |

| | |
|-------------|---|
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in the elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

Anxiolytics

Patients with painful injuries usually experience significant anxiety. Anxiolytics allow smaller analgesic dose to achieve the same effect.

| | |
|-------------------|--|
| Drug Name | Lorazepam (Ativan)- Sedative hypnotic in benzodiazepine class that has short onset of effect and relatively long half-life. By increasing action of GABA, a major inhibitory neurotransmitter, may depress all levels of CNS, including limbic and reticular formation. Excellent for patients who need to be sedated for >24 h. Monitor patient's BP after administering dose. Adjust as necessary. |
| Adult Dose | Initial dose: 2 mg total or 0.044 mg/kg IV, whichever is smaller; not to exceed 4 mg/dose |
| Pediatric Dose | 0.05-0.1 mg/kg IV slowly q2-5min; may repeat dose of 0.05 mg/kg IV slowly |
| Contraindications | Documented hypersensitivity; preexisting CNS depression; hypotension; narrow-angle glaucoma |
| Interactions | Alcohol, phenothiazines, barbiturates, and MAOIs increase toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in renal or hepatic impairment, myasthenia gravis, organic brain syndrome, or Parkinson disease |

Antibiotics

Therapy must cover all likely pathogens in the clinical setting.

| | |
|-------------------|---|
| Drug Name | Cefazolin (Ancef, Kefzol, Zolicef)- First-generation semisynthetic cephalosporin that, by binding to 1 or more penicillin-binding proteins, arrests bacterial cell wall synthesis and inhibits bacterial replication. Primarily active against skin flora, including <i>Staphylococcus aureus</i> . |
| Adult Dose | 2 g IV/IM q6-12h; not to exceed 12 g/d |
| Pediatric Dose | 25-100 mg/kg/d IV/IM divided q6-8h, depending on severity of infection; not to exceed 6 g/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid prolongs effects; aminoglycosides may increase renal toxicity; may yield false-positive urine-dip test for glucose |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal impairment; superinfections and promotion of nonsusceptible organisms may occur with prolonged use or repeated therapy |

| | |
|-------------------|--|
| Drug Name | Gentamicin (Gentacidin, Garamycin)- Aminoglycoside antibiotic used for gram-negative bacterial coverage. Commonly used in combination with both an agent against gram-positive organisms and one that covers anaerobes. Used in conjunction with ampicillin or vancomycin for prophylaxis in patients with open fractures. Dosing regimens are numerous and are adjusted based on CrCl and changes in volume of distribution. Gentamicin may be given IV/IM. |
| Adult Dose | 1.5 mg/kg IV; not to exceed 80 mg |
| Pediatric Dose | 2 mg/kg IV |
| Contraindications | Documented hypersensitivity; nondialysis-dependent renal insufficiency |
| Interactions | Other aminoglycosides, cephalosporins, penicillins, and amphotericin B may increase nephrotoxicity; aminoglycosides enhance effects of neuromuscular blocking agents, thus prolonged respiratory depression may occur; loop diuretics may increase auditory toxicity of aminoglycosides—possible irreversible hearing loss of varying degrees may occur (monitor regularly) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Narrow therapeutic index (not intended for long-term therapy); caution in renal failure (not on dialysis), myasthenia gravis, hypocalcemia, and conditions that depress neuromuscular transmission; adjust dose in renal impairment |

| | |
|-------------------|--|
| Drug Name | Ampicillin (Omnipen, Marcillin)- Used along with gentamicin for prophylaxis in patients with open fractures. Interferes with bacterial cell wall synthesis during active replication, causing bactericidal activity against susceptible organisms. |
| Adult Dose | 2 g IV/IM |
| Pediatric Dose | 50 mg/kg IV/IM |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid and disulfiram elevate levels; allopurinol decreases effects and has additive effects on ampicillin rash; may decrease effects of oral contraceptives |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal failure; evaluate rash and differentiate from hypersensitivity reaction |

| | |
|-------------------|---|
| Drug Name | Vancomycin (Vancocin)- Potent antibiotic directed against gram-positive organisms and active against enterococcal species. Useful in septicemia and skin structure infections. Used in conjunction with gentamicin for prophylaxis in penicillin-allergic patients with open fractures. May need to adjust dose in patients diagnosed with renal impairment. |
| Adult Dose | 1 g IV infused over 1 h |
| Pediatric Dose | 1 g IV over 1 h |
| Contraindications | Documented hypersensitivity |
| Interactions | Erythema, histaminelike flushing, and anaphylactic reactions may occur when administered with anesthetic agents; taken concurrently with aminoglycosides, risk of nephrotoxicity may increase above that with aminoglycoside monotherapy; effects in neuromuscular blockade may be enhanced, when coadministered with nondepolarizing muscle relaxants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in renal failure, neutropenia; red man syndrome caused by too rapid IV infusion (dose given over a few minutes) but rarely happens when dose given over 2 h or by PO or IP route; red man syndrome not an allergic reaction |

Toxoid

This agent is used for tetanus immunization. A booster injection in previously immunized individuals is recommended to prevent this potentially lethal syndrome.

| | |
|-------------------|--|
| Drug Name | <p>Tetanus toxoid- Induces active immunity against tetanus in selected patients. Immunizing agents of choice for most adults and children older than 7 years are tetanus and diphtheria toxoids. Administer booster doses to maintain tetanus immunity throughout life.</p> <p>Pregnant patients should receive only tetanus toxoid, not a diphtheria antigen-containing product.</p> <p>In children and adults, may administer into deltoid or midlateral thigh muscles. In infants, preferred site of administration is midhigh laterally.</p> |
| Adult Dose | <p>Primary immunization: 0.5 mL IM; give 2 injections 4-8 wk apart and a third dose 6-12 mo after second injection</p> <p>Booster dose: 0.5 mL q10y</p> |
| Pediatric Dose | Administer as in adults |
| Contraindications | <p>Documented hypersensitivity; history of any type of neurological symptoms or signs following administration of this product</p> <p>FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are an important cause of provocative poliomyelitis</p> |
| Interactions | <p>Patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization due to poor immune response; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent use of chloramphenicol since it may impair amnestic response to tetanus toxoid; concurrent use of tetanus immune globulin may delay development of active immunity by several days (interaction is nevertheless clinically insignificant and does not preclude its concurrent use)</p> |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | <p>Do not use to treat actual tetanus infections, or for immediate prophylaxis of unimmunized individuals (use instead tetanus antitoxin, preferably human tetanus immune globulin); diminished antibody response to active immunization may be seen in patients receiving immunosuppressive therapy; better to defer primary diphtheria immunization until immunosuppressive therapy discontinued; routine immunization of symptomatic and asymptomatic HIV-infected persons recommended</p> |

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Follow-up

Further Outpatient Care

- Orthopedic follow-up care
- Patient education
- Early physical therapy (eg, range of motion exercises) if indicated

Deterrence/Prevention

- Injury avoidance education
 - Adequate protective gear for participation in certain sports
 - Seat belt utilization
 - Drug and alcohol counseling as needed

Complications

- Brachial plexus compression resulting from hypertrophic callus formation (may cause peripheral neuropathy)
- Delayed union or nonunion (especially with distal third fractures)
- Poor cosmetic appearance
- Posttraumatic arthritis
- Intrathoracic injury
 - As with first rib fractures, great force is necessary to cause proximal third clavicle fractures; excluding underlying injuries is imperative (see [Picture 5](#)).
 - Pneumothorax
 - Subclavian artery and vein injury
 - Internal jugular vein injury
 - Axillary artery injury

Prognosis

- Excellent in children
- Excellent in adults with proper follow-up care, early detection, and treatment of complications

Patient Education

- Use of sling and/or shoulder immobilizer
- Use of figure-of-eight bandage (clavicle strap)
 - Educate patients as to proper placement and adjustment techniques.
 - Paresthesias or edema in hands or fingers indicate strap is too tight and should be removed.
 - Purpose of this bandage is to reduce pain by decreasing fracture fragment movement, not necessarily to maintain perfect alignment.
 - May be combined with a sling for added comfort.

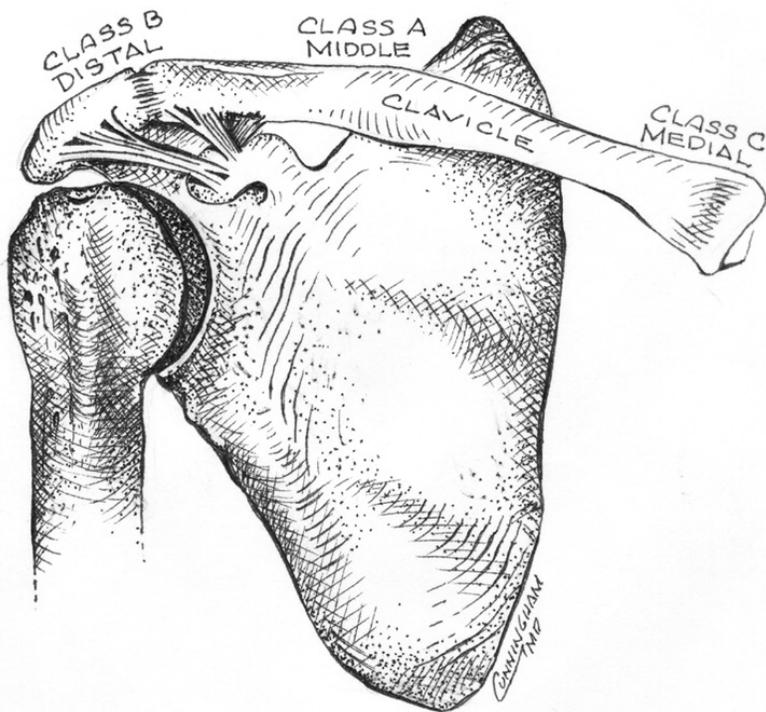
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Miscellaneous

Medical/Legal Pitfalls

- Failure to recognize and treat associated severe injuries
 - Failure to refer patients at risk of complications to orthopedist
-

Pictures

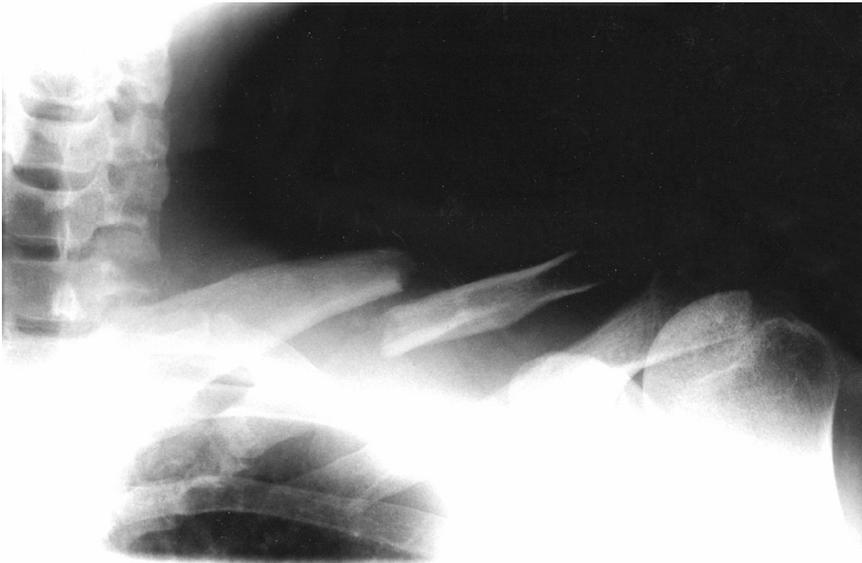


Picture 1: Anatomy of the clavicle indicating potential fracture sites (Courtesy of Michael Cunningham, MD)

Picture type: Photo



Picture 2: Nondisplaced middle clavicle fracture
Picture type: X-RAY



Picture 3: Displaced fracture of middle clavicle
Picture type: X-RAY



Picture 4: Displaced middle clavicle fracture

Picture type: X-RAY



Picture 5: Clavicle fracture with rib fractures. Remember to look for associated injuries.
Picture type: X-RAY

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Fractures, Elbow

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Introduction

Background

Elbow fractures are encountered commonly in the ED. Injury patterns for children and adults are quite different. Emergency physicians must recognize whether fractures require admission, immediate orthopedic evaluation, or less urgent referral.

Pathophysiology

Direct trauma or a fall onto an outstretched hand is responsible for most elbow fractures. Triceps muscle insertion on the olecranon often causes its displacement following fracture. Elbow joints are composed of 3 distinct articulations: radiocapitellar, ulnotrochlear, and proximal radioulnar, all contained in 1 synovial-lined capsule. This capsule typically encases hemarthrosis following injury.

The brachial artery is the most commonly injured artery. This is especially common in supracondylar fractures.

The median nerve is the most commonly injured nerve. This injury often is due to displaced supracondylar humerus fracture.

Age

- Fracture patterns vary markedly among different age groups.
- Supracondylar humerus fractures are most common in children aged 4-10 years because of this age group's relative strength of surrounding ligaments in comparison to bone.
- Injuries to proximal radius often manifest as radial neck fractures in children and radial head fractures in adults.
- Intraarticular condyle fractures are seen in children and adults.

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Clinical

History

- Mechanism of injury for most elbow fractures is direct elbow trauma or a fall onto an outstretched hand. Patients may experience the following:
 - Pain
 - Swelling
 - Decreased range of motion

Physical

- Patient is unable to fully extend elbow, and pain is present with pronation/supination of the forearm.
- Edema and ecchymosis near the elbow may be evident.
- Perform careful shoulder and wrist examinations with all elbow injuries.
- Radial head fracture is characterized by point tenderness at the radial head (located along lateral aspect of elbow) and pain with pronation/supination.
- Supracondylar fracture
 - Perform and document a careful vascular examination, as the brachial artery may be disrupted.
 - Perform and document a careful neurologic exam, as nerves (most commonly the median nerve or one of its branches, the anterior interosseus nerve) may be injured.

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Differentials

Dislocations, Elbow
Fractures, Forearm
Fractures, Humerus
Pediatrics, Nursemaid Elbow

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Workup

Imaging Studies

- Anteroposterior (AP), lateral, and oblique x-rays of the elbow adequately visualize most elbow fractures.
 - On all views, the center of the radial head should align with the center of the capitellum.
 - Misalignment of the radial head and capitellum indicates a subluxed or dislocated radial head.
- Olecranon fracture ([Picture 2](#))
 - Lateral radiograph demonstrates a displaced intraarticular fracture.
 - Use AP view to identify associated fracture or dislocation.

Procedures

- Supracondylar fracture
 - In general, an orthopedic consultant best handles decisions regarding reduction of significantly angulated and displaced fractures.
 - If neurovascular structures are compromised, the emergency physician may need to apply forearm traction to reestablish distal pulses.
 - If pulse is not restored with traction, emergent operative intervention for brachial artery exploration or fasciotomy is indicated.

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Treatment

Emergency Department Care

- Provide adequate analgesia to achieve patient comfort in ED.
- Radial head fracture
 - For nondisplaced fractures, a sling is all that is necessary.
 - For displaced fractures, closed reduction rarely is required or possible.
 - Patients can be placed in a posterior long arm splint with the elbow in 90° of flexion and forearm in full supination, then given a sling for comfort.
- Supracondylar fracture
 - Adult patients usually require surgical intervention.
 - In children, nondisplaced, nonangulated fractures can be splinted (elbow in 90° of flexion); angulated fractures require reduction and splinting; and displaced fractures require reduction and percutaneous pinning on an urgent basis within 12-24 hours.

Consultations

- Bleeding around the elbow raises suspicion of an open fracture or open joint and requires urgent orthopedic consultation.
- Supracondylar fracture: A displaced supracondylar fracture requires urgent orthopedic consultation because of significant risk of neurovascular injury and compromise. Admit patient for serial neurovascular checks.
- Olecranon fracture: Loss of active extension or intraarticular displacement of greater than 1 mm are indications for surgical treatment.
- Radial head fracture
 - Occult or small radial head fractures are treated symptomatically with early range of motion exercises.
 - For displaced or comminuted fractures mechanically blocking joint motion, surgical intervention may be necessary. Refer these patients to an orthopedic surgeon.
- Condylar fracture: Displaced fractures of the trochlea or capitellum require surgical intervention.

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Medication

Drugs used to treat fractures are generally NSAIDs, analgesics, and anxiolytics. In addition, administer proper antibiotics and tetanus prophylaxis for open fractures.

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

These drugs are used commonly for relief of mild to moderately severe pain. Effects of NSAIDs in treatment of pain tend to be patient specific, yet ibuprofen is usually DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually DOC for treatment of mild to moderately severe pain, if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, which inhibits prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients on anticoagulants—monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

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|-------------------|--|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Relieves mild to moderately severe pain and inflammation. Administer small dosages initially to patients with small bodies, older persons, and those with renal or liver disease. Doses higher than 75 mg do not increase therapeutic effects. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 14 years: 0.1-1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |

| | |
|--------------|--|
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants—monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, which decreases prostaglandin synthesis. |
| Adult Dose | 500 mg PO followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 5-7 mg/kg/dose PO q8-12h prn |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants—monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

| | |
|----------------|---|
| Drug Name | Flurbiprofen (Ansaid)- Has analgesic, antipyretic, and anti-inflammatory effects. May inhibit cyclooxygenase enzyme, inhibiting prostaglandin biosynthesis. |
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |

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|-------------------|--|
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants—monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients with fractures.

| | |
|-------------------|---|
| Drug Name | Acetaminophen (Tylenol, Panadol, aspirin-free Anacin)- DOC for treatment of pain in patients with documented hypersensitivity to aspirin or NSAIDs, those with upper GI disease, or those taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; known G-6-P deficiency |
| Interactions | Rifampin can reduce analgesic effects; barbiturates, carbamazepine, hydantoin, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; APAP is contained in many OTC products and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

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| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
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| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | <12 years: 750 mg acetaminophen PO q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d >12 years: 10-15 mg/kg/dose acetaminophen q4-6h prn; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates, since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in the elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

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| Drug Name | Oxycodone and aspirin (Percodan)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity; liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma Due to association of aspirin with Reye syndrome do not use in children (<16 y) who have flu |

| | |
|--------------|---|
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity; may potentiate anticoagulant effects of warfarin |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Duration of action may increase in the elderly; caution in renal or liver impairment, peptic ulcer disease, and erosive gastritis |

Anxiolytics

Patients with painful injuries usually experience significant anxiety. Anxiolytics allow a smaller analgesic dose to achieve the same effect.

| | |
|-------------------|--|
| Drug Name | Lorazepam (Ativan)- Sedative hypnotic in benzodiazepine class that has a short onset of effect and relatively long half-life. By increasing action of GABA, a major inhibitory neurotransmitter, may depress all levels of CNS, including limbic and reticular formation. Excellent for sedating patient for longer than 24-h period. Monitor patient's BP after administering dose and adjust as necessary. |
| Adult Dose | Initial dose: 2 mg total or 0.044 mg/kg IV, whichever is smaller |
| Pediatric Dose | 0.05 - 0.1 mg/kg IV slowly q2-5min; may repeat dose of 0.05 mg/kg IV slowly |
| Contraindications | Documented hypersensitivity; preexisting CNS depression; hypotension; and narrow-angle glaucoma |
| Interactions | Alcohol, phenothiazines, barbiturates, and MAOIs increase CNS toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in renal or hepatic impairment, myasthenia gravis, organic brain syndrome, or Parkinson disease |

Antibiotics

These agents are given as prophylaxis to patients with open fractures.

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| Drug Name | Gentamicin (Gentacidin, Garamycin)- Aminoglycoside antibiotic used for gram-negative bacterial coverage. Commonly used in combination with both an agent against gram-positive organisms and one that covers anaerobes. Dosing regimens are numerous and adjusted based on renal function (CrCl) and changes in volume of distribution. Dose may be given IV/IM. |
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| Adult Dose | 1.5 mg/kg/dose IV q8-24h; not to exceed 80 mg; may need dosage adjustment in patients diagnosed with renal impairment |
| Pediatric Dose | <5 years with normal renal function: 2.5 mg/kg/dose q8h IV/IM >5 years: 1.5-2.5 mg/kg/dose IV/IM q8h or 6-7.5 mg/kg/d q8h; not to exceed 300 mg/d, with adjustments for renal function prn; monitor levels as in adults |
| Contraindications | Documented hypersensitivity; non-dialysis-dependent renal insufficiency |
| Interactions | Other aminoglycosides, cephalosporins, penicillins, and amphotericin B may increase nephrotoxicity; enhances effects of neuromuscular blocking agents and thus may cause prolonged respiratory depression; loop diuretics may increase auditory toxicity—possible irreversible hearing loss of varying degrees may occur (monitor regularly) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Narrow therapeutic index (not intended for long-term therapy); caution in renal failure (not on dialysis), myasthenia gravis, hypocalcemia, and conditions that depress neuromuscular transmission; adjust dose in renal impairment |

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| Drug Name | Ampicillin (Omnipen, Marcillin)- Used along with gentamicin for prophylaxis in patients with open fractures. |
| Adult Dose | 1-2 g IV/IM q6h |
| Pediatric Dose | 100-200 mg/kg/day IV/IM divided q6h |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid and disulfiram elevate levels; allopurinol decreases effects and has additive effects on ampicillin rash; may decrease effects of oral contraceptives |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal failure; evaluate rash and differentiate from hypersensitivity reaction |

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| Drug Name | Vancomycin (Vancocin)- Potent antibiotic directed against gram-positive organisms and active against enterococcal species. Also useful in treatment of septicemia and skin structure infections. Used in conjunction with gentamicin for prophylaxis in penicillin-allergic patients with open fractures. Dosing interval based on renal function. |
| Adult Dose | 10 mg/kg/dose IV q8-24h; adjust interval on basis of renal function Usual dose: 1000 mg IV q12h; infuse over 1 h |
| Pediatric Dose | 10 mg/kg/dose IV q8h; infuse over 1 h |
| Contraindications | Documented hypersensitivity |

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| Interactions | Anesthetic agents may cause erythema, histamine-like flushing, and anaphylactic reactions; taken concurrently with aminoglycosides, risk of nephrotoxicity may increase above that with aminoglycoside monotherapy; nondepolarizing muscle relaxants may enhance effects in neuromuscular blockade |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in renal failure, neutropenia; red man syndrome is caused by too rapid IV infusion (dose given over a few minutes) but rarely happens when dose given over 2 h or by PO or IP route; red man syndrome is not an allergic reaction |

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| Drug Name | Ceftriaxone (Rocephin)- Third-generation cephalosporin that has broad-spectrum efficacy against gram-negative organisms, lower efficacy against gram-positive organisms, and higher efficacy against resistant organisms. By binding to 1 or more penicillin-binding proteins, arrests bacterial cell wall synthesis and inhibits bacterial replication. |
| Adult Dose | 1-2 g IV qd; not to exceed 4 g/d |
| Pediatric Dose | Neonates >7 days: 25-50 mg/kg/d IV; not to exceed 125 mg/d Infants and children: 50-75 mg/kg/d IV qd; not to exceed 2 g/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid may increase levels; ethacrynic acid, furosemide, and aminoglycosides may increase nephrotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal impairment; caution in breastfeeding women and allergy to penicillin |

Toxoid

This agent is used for tetanus immunization. Booster injection in previously immunized individuals is recommended to prevent this potentially lethal syndrome.

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|-----------|--|
| Drug Name | Tetanus toxoid- Induces active immunity against tetanus in selected patients. Immunizing DOC for most adults and children >7 y are tetanus and diphtheria toxoids. Necessary to administer booster doses to maintain tetanus immunity throughout life. Pregnant patients should receive only tetanus toxoid, not a diphtheria antigen-containing product. In children and adults, may administer into deltoid or midlateral thigh muscles. In infants, preferred site of administration is midthigh, lateral. |
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| Adult Dose | Primary immunization: 0.5 mL IM, give 2 injections q4-8 wk apart and a third dose q6-12 mo after second injection Booster dose: 0.5 mL q10y |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; history of any type of neurological symptoms or signs following administration of this product FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are an important cause of provocative poliomyelitis |
| Interactions | Patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization due to poor immune response; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent use of chloramphenicol since it may impair anesthetic response to tetanus toxoid; concurrent use of tetanus immune globulin may delay development of active immunity by several days (interaction is nevertheless clinically insignificant and does not preclude its concurrent use) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Do not use to treat actual tetanus infections, or for immediate prophylaxis of unimmunized individuals (use instead tetanus antitoxin, preferably human tetanus immune globulin); diminished antibody response to active immunization may be seen in patients receiving immunosuppressive therapy; better to defer primary diphtheria immunization until immunosuppressive therapy discontinued; routine immunization of symptomatic and asymptomatic HIV-infected persons is recommended |

Immunoglobulins

Patients who may not have been immunized against *Clostridium tetani* products should receive tetanus immune globulin.

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|----------------|---|
| Drug Name | Tetanus immune globulins (Hyper-Tet)- For passive immunization of any person with a wound that may be contaminated with tetanus spores. |
| Adult Dose | For prophylaxis: 250-500 U IM in opposite extremity to tetanus toxoid For clinical tetanus: 3,000-10,000 U IM |
| Pediatric Dose | For prophylaxis: 250 U IM in opposite extremity to tetanus toxoid For clinical tetanus: 3,000-10,000 U IM |

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| Contraindications | Since antibodies in globulin preparation may interfere with immune response to vaccination, do not administer within 3 mo of live virus immune globulin administration; may be necessary to revaccinate persons who received immune globulin shortly after live virus vaccination |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Persons with isolated IgA deficiency have potential for developing antibodies to IgA and could have anaphylactic reactions to subsequent administration of blood products that contain IgA; do not perform skin testing since intradermal injection of concentrated gamma globulin may cause localized area of inflammation and can be misinterpreted, causing the medication to be withheld from a patient not allergic to this material; true allergic responses to human gamma globulin given in prescribed IM manner are extremely rare; do not admix with other medications since usually incompatible |

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Follow-up

Further Inpatient Care

- Open fractures require extensive irrigation. Administer appropriate prophylactic antibiotics, such as cefazolin sodium 1 g IV and gentamicin.
- Supracondylar fractures: In children, displaced fractures require surgical intervention and continual monitoring of neurovascular status.

Further Outpatient Care

- Radial head fracture
 - For uncomplicated fractures, begin range of motion exercises within 3-7 days to reduce risk of permanent loss of motion from elbow joint contracture.
 - Intraarticular fractures, which may require radial head excision or fixation, should be seen by an orthopedist within 1 week for definitive management.
- Supracondylar fracture
 - Refer patients with nondisplaced fractures to an orthopedist within 24 hours to evaluate and recheck neurovascular status.
 - Upon dissipation of edema, apply a long arm cast that holds elbow in 90° of flexion for approximately 6 weeks.

in/Out Patient Meds

- As with all fractures, address adequate outpatient analgesia, especially during first few days. Acetaminophen, with codeine or hydrocodone, may be appropriate treatment.

Complications

- Radial head fracture
 - Nondisplaced fractures that are immobilized for prolonged periods of time may have permanently decreased range of motion.
 - Comminuted radial head fractures associated with undiagnosed distal radial-ulnar joint injuries can lead to permanent wrist injuries and loss of pronation/supination motion.
- Olecranon fracture: An ulnar nerve injury, although rare, may be associated with a displaced olecranon fracture.

Prognosis

- Radial head fracture - Usually no functional loss with nonoperative treatment
- Supracondylar humerus fracture
 - Children: Undisplaced fractures and properly managed displaced/angulated fractures result in no long-term functional deficits.
 - Adults: Usually range of motion decreases somewhat but without functional deficit.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to document a neurovascular exam in a child with a supracondylar fracture
- Failure to reexamine and document neurovascular exam following application of a splint. Elbow flexion greater than 90° may obliterate pulse or compromise neurologic function.
- Failure to pad olecranon when splinting an olecranon fracture may lead to skin breakdown and iatrogenic open fracture.
- Failure to maintain prolonged immobilization of radial head fractures leads to permanently decreased range of motion.
- Failure to recognize a radial head dislocation associated with a fracture of the proximal ulna (Monteggia fracture or dislocation)

Pictures



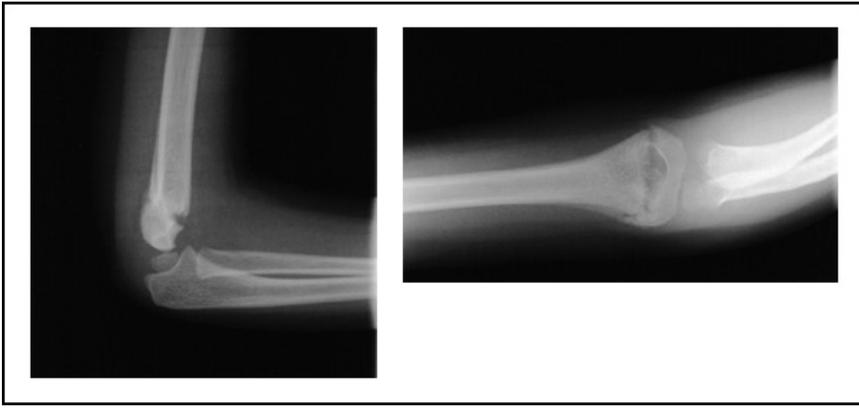
Picture 1: Fracture of radial head best seen on lateral projection

Picture type: X-RAY



Picture 2: Lateral radiograph of a displaced olecranon fracture

Picture type: X-RAY



Picture 3: Displaced supracondylar humerus fracture

Picture type: X-RAY

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Fractures, Face

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Synonyms, Key Words, and Related Terms

alveolar fracture, Le Fort fracture, maxillofacial fracture, nasal fracture, zygoma fracture

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Introduction

Background

In approximately 400 BC, Hippocrates provided the first description of a variety of facial injuries. Rene Le Fort used cadaver studies in 1900 to provide detailed descriptions of 3 basic types of facial fracture.

Endotracheal anesthesia and radiography developed during the First World War led to better understanding and treatment of facial fractures. During the Second World War, a multidisciplinary approach to treatment of facial fractures continued to improve the outcomes of severely injured soldiers. The more recent introduction of CT reconstruction, along with new surgical techniques, has improved cosmetic results immensely.

Pathophysiology

Maxillofacial fractures result from blunt or penetrating trauma. Blunt injuries are far more common, including vehicular accidents, altercations, sports-related trauma, occupational injuries, and falls. Penetrating injuries include gunshot wounds, stabbings, and explosions.

Mass, density, and shape of the striking object, as well as speed of impact, directly affect type and severity of facial injury. The amount of force required to fracture various facial bones may be classified as high impact (greater than 50 times force of gravity [g]) or low impact (less than 50 g).

- High impact
 - Supraorbital rim: 200 g
 - Symphysis mandible: 100 g
 - Frontal-glabellar: 100 g
 - Angle of mandible: 70 g

Simple nasal fractures are the most common of all facial fractures. They must be distinguished from the more serious nasoethmoidal (NOE) fractures. NOE fractures extend into the nose through the ethmoid bones. Fractures through the ethmoid are prone to cerebrospinal fluid (CSF) leaks from dural tears.

Zygomatic arch fractures tend to occur in 2-3 places along the arch. Often 3 breaks occur, 1 at each end of the arch and a third in the middle, forming a V-shaped fracture; this often impinges on the temporalis muscle below, causing trismus.

Zygomaxillary (tripod) fractures result from a direct blow to the cheek. Fracture occurs at articulations of the zygoma with the frontal bone maxillae and zygomatic arch and often extends through the orbital floor. Because the infraorbital nerve passes through the orbital floor, hypesthesia often occurs in its sensory distribution.

Alveolar fractures occur just above the level of the teeth through the alveolar portion of the maxilla. Usually a group of teeth is loose, and blood is noted at the gingival line.

Le Fort fractures

Le Fort or midface fractures are classified into 3 types and occasionally are mixed from one side of the face to the other.

- Le Fort I: Horizontal maxillary fracture separates the maxillary process (hard palate) from the rest of the maxilla. Fracture extends through the lower third of the septum and involves the maxillary sinus. This is below the level of the infraorbital nerve and thus does not cause hypesthesia.
- Le Fort II: Pyramidal fracture starts at the nasal bone, extends through the lacrimal bone, and courses downward through the zygomaxillary suture. It courses posteriorly through the maxilla and below the zygoma into the upper pterygoid plates. The inner canthus of the nasal

bridge is widened. Because the fracture extends through the zygoma, near the exit of the infraorbital nerve, hypesthesia often is present. Bilateral subcutaneous hematomas often are present.

- **Le Fort III:** Craniofacial dysjunction also starts at the nasal bridge. It extends posteriorly through the ethmoid bones and laterally through the orbits below the optic foramen, through the pterygomaxillary suture into the sphenopalatine fossa. This fracture separates facial bones from cranium, causing the face to appear long and flat (ie, dish face).

Frequency

- **In the US:** Approximately 3 million facial injuries occur annually, but most do not involve maxillofacial fractures. One study placed the incidence of severe maxillofacial injury (fractures and lacerations) at 0.04-0.09% for motor vehicle collisions. Motor vehicle-related injuries are more common in rural areas, and altercation-related injuries are more frequent in inner cities.

Mortality/Morbidity

Incidence of other major injuries is as high as 50% in high-impact facial fractures, compared with 21% for low-impact fractures. Motor vehicle collision-related fractures are more likely to have associated injuries than violence-related fractures. The mortality rate is as high as 12% in high-impact fractures but is rarely due to maxillofacial injury. The incidence of associated cervical spine injuries has been reported in the 0.2-6% range.

Sex

Adult male-to-female ratio is 3:1.

Age

Male predominance is reduced to 3:2 in children.

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Clinical

History

- First priority is to perform a primary survey and attend to ABCs, as maxillofacial fractures are

caused by significant trauma. Initially, focus on assessment of airway patency, breathing, circulation, and gross neurologic function, as well as control of the cervical spine.

- Once life-threatening issues are addressed, obtain a thorough history.
 - Allergies
 - Medications
 - Past medical history
 - Last meal
 - Events leading to injury

Physical

- Complete examination of the face is necessary, since multiple injuries easily occur. Portions of the examination specific for facial bones are marked with an asterisk (*).
 - Inspect face for asymmetry, which is often easiest to do looking down from the head of the bed.*
 - Inspect open wounds for foreign bodies and palpate for bony injury.*
 - Palpate the bony structures of the supraorbital ridge and frontal bone for step-off fractures.
 - Thoroughly examine eyes for injury, abnormality of ocular movements, and visual acuity.*
 - Inspect nares for telecanthus and widening of the nasal bridge, and palpate for tenderness and crepitus.*
 - Inspect nasal septum for septal hematoma and clear rhinorrhea, which may suggest a CSF leak.*
 - Palpate zygoma along its arch as well as its articulations with the frontal bone, temporal bone, and maxillae.*
 - Check facial stability by grasping teeth and hard palate and gently pushing back and forth, then up and down, feeling for movement or instability of midface.*
 - Inspect teeth for fracture and bleeding at the gum line (a sign of fracture through the alveolar bone), and test for stability.*
 - Check teeth for malocclusion and step-off.*
 - Palpate mandible for tenderness, swelling, and step-off along its symphysis, body, angle, and condyle anterior to the ear canal.
 - Evaluate supraorbital, infraorbital*, inferior alveolar, and mental nerve distributions for hypesthesia or anesthesia.
- NOE fracture: Suspect NOE if the patient has evidence of a nasal fracture with telecanthus, widening of the nasal bridge with detached medial canthus, and epistaxis or CSF rhinorrhea.
- Zygoma fracture: Physical findings of a depressed malar eminence with tenderness suggest a zygoma or zygomatic arch fracture. Often edema is marked, which can obscure the depression. The patient may complain of pain in the cheek on movement of the jaw. The patient may have trismus or difficulty opening the mouth from impingement of the temporalis muscle as it passes under the zygoma.
- Tripod fracture
 - Suspect tripod fracture after blunt force to the cheek with physical findings of marked periorbital edema and ecchymosis. Malar flattening may be seen early, but marked

swelling of overlying tissues often obscures this finding. Lateral canthus may be depressed if the zygoma is displaced inferiorly. Hypesthesia of the infraorbital nerve often is present, because the fracture extends through the orbit into the zygomaticomaxillary area where the nerve exits.

- Palpating the zygomaticomaxillary arch from inside the mouth may reveal a step-off fracture. A step-off may be noted at the zygomaticofrontal suture or on the zygomatic arch as well. Eye injuries may be associated with these fractures, thus a thorough eye examination is important to document and act upon.

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Differentials

Corneal Abrasion
Corneal Laceration
Dislocations, Mandible
Domestic Violence
Elder Abuse
Epidural Hematoma
Fractures, Frontal
Fractures, Mandible
Fractures, Orbital
Globe Rupture
Neck Trauma
Pediatrics, Child Abuse
Retinal Detachment
Sexual Assault
Subdural Hematoma

Other Problems to be Considered

Dentate, avulsed

Dentate, displaced

Dentate, fractures

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Workup

Lab Studies

- Base need for laboratory studies upon extent of concomitant nonfacial trauma.
- If injuries are isolated to face and surgery is planned, order preoperative laboratory tests.

Imaging Studies

- Nasal bone fractures
 - Nasal bone fractures can be diagnosed clinically by history and physical examination. Plain nasal films consisting of a lateral view coning down on the nose and a Waters view can confirm the diagnosis but are of little practical use. If edema has resolved and no deformity is noted, x-rays are unnecessary.
 - If deformity persists after resolution of edema, films may be obtained at follow-up to help plan the repair. Omission of ED films is cost-effective, since most nasal fractures do not need to be reduced.
- Zygoma fracture
 - Best film for evaluating zygomatic arch is an underexposed submental view, also known as bucket handle view, because arches appear as bucket handles.
 - Fracture also can be seen on a Waters view, and in some cases on a Towne view, of a facial series.
- Le Fort fractures
 - Coronal CT scan of facial bones has replaced plain films in evaluation of Le Fort fractures, especially with use of 3-D reconstruction. Since Le Fort fractures often are mixed from one side to the other, CT scan is superior to plain films and makes visualization of the fracture for repair much easier. If CT is not available, a facial series with lateral, Waters, and Caldwell views can be used to evaluate the fracture. Almost all Le Fort fractures cause blood to collect in the maxillary sinus.
 - Le Fort I fractures: Imaging demonstrates a fracture extending horizontally across the inferior maxilla, sometimes including a fracture of the lateral sinus wall, extending into the palatine bones and pterygoid plates.
 - Le Fort II fractures: Imaging demonstrates disruption of the inferior orbital rim lateral to the infraorbital canal and a fracture of the medial orbital wall and nasal bone. The fracture extends posteriorly into the pterygoid plates.
 - Le Fort III fractures: Imaging demonstrates fractures at the zygomaticofrontal suture, zygoma, medial orbital wall, and nasal bone extending posteriorly through the orbit at the pterygomaxillary suture into the sphenopalatine fossa.

Other Tests

- Perform chest films if teeth are missing to rule out tooth aspiration.
- Test clear rhinorrhea for glucose. Nasal secretions, unlike CSF, are normally low in glucose. If blood is present, this test is unreliable. Blood-tinged fluid can be placed on filter paper to look for a double ring sign of CSF around blood, but this is not a reliable test.

Procedures

- When CSF rhinorrhea is suspected, fluorescein may be injected into the lumbar subarachnoid space. Observe with a Wood lamp 30 minutes later for fluorescence of nasal discharge; if present, this confirms CSF rhinorrhea. This procedure is not usually performed by emergency physicians.

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Treatment

Prehospital Care

- ABCs are first priority. If necessary, hold airway open by chin lift or jaw thrust. Avoid nasotracheal route of intubation because of the risk of intracranial tube placement.
- Place patient on a backboard and collar if cervical spine injury is suspected.
- Treat hypoventilation with intubation and bag ventilation.
- Control actively bleeding wounds with direct pressure.

Emergency Department Care

- ABCs take priority. Reassess airway frequently. Early intubation, before edema occurs, can make airway control much easier than waiting until a problem arises from obstruction. When intubation by oral route is impossible, perform cricothyroidotomy to secure airway.
- Avoid the temptation to focus on the obvious facial deformity, thereby failing to perform a complete primary survey. Other life-threatening conditions need to be diagnosed rapidly and appropriate resuscitation undertaken. Follow this with a complete secondary survey.
- Evaluation of facial fractures is part of the secondary survey.
- Epistaxis may require anterior nasal packing to control bleeding. Posterior packing occasionally may be needed.
- Drain septal hematomas to avoid necrosis of septal cartilage.

Consultations

- Refer patients with facial fractures to an oral and maxillofacial surgeon, ear, nose, and throat (ENT) surgeon, or plastic surgeon who is experienced in care of these injuries.
- Consult a neurosurgeon if a CSF leak is diagnosed or suspected.
- Refer care of patients with multiple injuries to a surgeon with experience in trauma care. If a surgeon with trauma experience is not available, transfer patient to a higher level trauma center.

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Medication

When airway control is needed, rapid sequence induction often is the preferred method. Perform rapid sequence induction, utilizing medications to induce unconsciousness and muscle paralysis to facilitate intubation. A cricothyroidotomy kit should be at bedside if oral intubation cannot be accomplished.

Provide adequate analgesia, including opioids, NSAIDs, or local anesthetics.

Prophylactic antibiotics are controversial when a CSF leak is identified or when the fracture involves the sinuses. It is usually left to the discretion of the specialist assuming care of the patient.

If the patient has an open wound, update tetanus immunization.

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

These drugs are used most commonly for relief of mild to moderately severe pain. Effects of NSAIDs in treatment of pain tend to be patient specific, yet ibuprofen is usually DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

| | |
|-------------------|---|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually DOC for treatment of mild to moderately severe pain, if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, which inhibits prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |

| | |
|--------------|--|
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- For relief of mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, which decreases prostaglandin synthesis. |
| Adult Dose | 500 mg PO followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

| | |
|----------------|---|
| Drug Name | Flurbiprofen (Ansaid)- Has analgesic, antipyretic, and anti-inflammatory effects. May inhibit cyclooxygenase enzyme, inhibiting prostaglandin biosynthesis. |
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |

| | |
|-------------------|--|
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€”monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

| | |
|-------------------|--|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for relief of mild to moderately severe pain and inflammation. Administer small dosages initially to patients with small bodies, older persons, and those with renal or liver disease. Doses higher than 75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe patients for response. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 14 years: 0.1â€”1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€”monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and

aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures.

| | |
|-------------------|--|
| Drug Name | Acetaminophen (Tylenol, Panadol, aspirin-free Anacin)- DOC for treatment of pain in patients with documented hypersensitivity to aspirin and NSAIDs, those with upper GI disease, or those taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; known G-6-P deficiency. |
| Interactions | Rifampin can reduce analgesic effects; barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; APAP is contained in many OTC products, and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

| | |
|-------------------|---|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg based on codeine content PO q4-6h or 1-2 tabs q4h; not to exceed 12 tabs/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|------------|---|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |

| | |
|-------------------|---|
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen PO q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in the elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

| | |
|-------------------|--|
| Drug Name | Oxycodone and aspirin (Percodan)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity; liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma Due to association of aspirin with Reye syndrome do not use in children (<16 y) who have flu |

| | |
|--------------|---|
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity; may potentiate anticoagulant effects of warfarin |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Duration of action may increase in the elderly; caution in renal or liver impairment, peptic ulcer disease, and erosive gastritis |

| | |
|-------------------|---|
| Drug Name | Morphine Sulfate (Duramorph, Astramorph, MS Contin)- DOC for narcotic analgesia due to its reliable and predictable effects, safety, and ease of reversibility with naloxone. Morphine sulfate administered IV may be dosed in a number of ways and commonly is titrated until desired effect obtained. |
| Adult Dose | Starting dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h Relatively hypovolemic patients: Start with 2 mg IV/IM/SC and reassess hemodynamic effects of dose |
| Pediatric Dose | Neonates: 0.05-0.2 mg/kg IV prn Children: 0.1-0.2 mg/kg IV q2-4h prn |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

Anxiolytics

Patients with painful injuries usually experience significant anxiety. Anxiolytics allow a smaller analgesic dose to achieve the same effect.

| | |
|----------------|--|
| Drug Name | Lorazepam (Ativan)- Sedative hypnotic in benzodiazepine class that has short onset of effect and relatively long half-life. By increasing action of GABA, a major inhibitory neurotransmitter, may depress all levels of CNS, including limbic and reticular formation. Excellent for sedating patients for longer than 24-h period. Monitor patient's BP after administering dose. Adjust as necessary. |
| Adult Dose | Initial dose: 2 mg total or 0.044 mg/kg IV, whichever is smaller |
| Pediatric Dose | 0.05-0.1 mg/kg IV slowly q2-5min; may repeat dose of 0.05 mg/kg IV slowly |

| | |
|-------------------|---|
| Contraindications | Documented hypersensitivity; preexisting CNS depression; hypotension; and narrow-angle glaucoma |
| Interactions | Alcohol, phenothiazines, barbiturates, and MAOIs increase toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in renal or hepatic impairment, myasthenia gravis, organic brain syndrome, or Parkinson disease |

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Follow-up

Further Inpatient Care

- Patients with NOE fractures generally require admission to monitor for a CSF leak and observe for signs of meningitis or brain abscess, which are known complications.
- Patients with zygomatic arch fractures who have significant trismus or inability to open the mouth may require admission for observation because of potential problems with aspiration or airway obstruction from vomiting.
- Patients with tripod fractures with eye involvement generally require admission to ophthalmology.
- Patients with Le Fort fractures may require admission for further workup prior to open reduction and internal fixation. Patients also may need a short admission if arch wires are used, because of the risk of obstruction or aspiration should they vomit. During the hospital stay, teach patients how to remove the crossband so the mouth can be opened if they need to vomit.
- Patients with multiple traumas should be admitted to a surgeon with trauma experience to coordinate care of all injuries.

Further Outpatient Care

- Patients with simple nasal fractures can be discharged home with follow-up in 5-7 days when edema has decreased. Avoid delaying follow-up care, because fracture healing may begin prior to a necessary reduction. Give patients epistaxis instructions and instruct to return if clear fluid from nose is noted.
- Patients with simple zygomatic arch fractures, without trismus or mouth opening problems, can be discharged home with proper follow-up care.
- Patients with tripod fractures without eye involvement can be discharged home with appropriate follow-up care.

in/Out Patient Meds

- Facial fractures tend to be very painful. Provide adequate analgesia, including oral opioids and NSAIDs.

Transfer

- If appropriate specialists are not available, transfer the patient to a higher level hospital. This is particularly important in patients with multiple injuries.

Deterrence/Prevention

- Use of seatbelts and airbags can reduce incidence of facial injuries in motor vehicle accidents. Use of helmets with facial guards can reduce injury in motorcycle accidents and in accidents in such sports as skiing, snowboarding, hockey, and football.

Complications

- Continued CSF leaks can occur, although most stop by 2-3 weeks after the injury.
- Meningitis and abscesses are serious infections that can occur when a CSF leak is present. Observe patients closely for signs and symptoms.
- Sepsis
- Scars and facial deformity
- Injury to infraorbital nerve in tripod and Le Fort II fractures that extends through the infraorbital foramen where the nerve exits

Patient Education

- If band arch wires are placed, teach patients how to release the crossband in an emergency.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to diagnose fractures correctly or fully

- Failure to diagnose associated intracranial, intrathoracic, intraabdominal, or cervical spine injuries. This occurs when the physician focuses on an obvious injury, missing a life-threatening injury because a complete workup in the secondary survey was never completed.
- Failure to evaluate associated ocular injuries adequately

Special Concerns

- Always consider loss of airway and cervical spine, intracranial, and intraabdominal injuries.
- Evaluate possibility of eye injury.
- Carefully evaluate eye and extraocular muscle function.

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Fractures, Femur

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Introduction

Background

This article discusses fractures of the femoral diaphysis. For proximal femur fractures (subtrochanteric to femoral head), see the article [Fractures, Hip](#). For fractures of the distal femur (supracondylar to condylar), see the article [Fractures, Knee](#).

The femur is the largest and strongest bone and has a good blood supply. Because of this and its protective surrounding muscle, the shaft requires a large amount of force to fracture. Once a fracture does occur, this same protective musculature usually is the cause of displacement, which commonly occurs with femoral shaft fractures.

As with many orthopedic injuries, neurovascular complications and pain management are the most significant issues in patients who come to the ED. The rich blood supply, when disrupted, can result in significant bleeding. Open fractures have added potential for infection.

The 3 types of femoral shaft fractures are as follows:

- Type I - Spiral or transverse (most common)

- Type II - Comminuted
- Type III - Open

Associated injuries are common.

Pathophysiology

Diaphyseal fractures result from significant force transmitted by a direct blow or from indirect force transmitted at the knee.

Pathologic fractures may occur with relatively little force. These may be the result of bone weakness from osteoporosis or lytic lesions.

Mortality/Morbidity

Morbidity and mortality rates have been reduced in femoral shaft fractures, mainly as the result of changes in methods of fracture immobilization. Current therapies allow for early mobilization, thus reducing the risk of complications associated with prolonged bed rest.

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Clinical

History

History usually is obvious in cases of femoral diaphyseal fractures. Typically, patients describe a significant force applied to the extremity. Significant pain and deformity are reported as well.

Physical

- Conduct a thorough exam to rule out associated injury. Hip fractures and ligamentous knee injuries commonly are observed in association.
- At the site of fracture, tenderness on exam and visible deformity typically are noted.
- Extremity may appear shortened, and crepitus may be noted with movement.
- Thigh is often swollen secondary to hematoma formation.
- Perform a thorough vascular exam on the extremity. Signs of vascular compromise should prompt arteriography and a vascular surgery consult. Physical signs of arterial injury include the following.
 - Expanding hematoma

- Absent or diminished pulses
- Progressive neurologic deficits in a closed fracture
- Test distal neurologic function, though exam is frequently unreliable because of the amount of pain associated with these fractures. Nerve injury is rare because of protective surrounding musculature.

Causes

- Trauma
- Lytic lesions
 - Cancerous metastasis
 - Paget disease
 - Bone cysts

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Differentials

Fractures, Hip
Fractures, Knee
Fractures, Pelvic
Trauma, Peripheral Vascular Injuries

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Workup

Lab Studies

- No specific lab studies are indicated. In a patient with large expanding hematoma, measure serial hematocrits and order type and cross-match in case blood transfusion becomes necessary.
- For patients requiring open reduction and internal fixation (ORIF), obtain preoperative tests, including chest x-ray (CXR) and ECG.

Imaging Studies

- Anteroposterior (AP) and lateral views of the femur normally are sufficient. As with all long-bone

fractures, obtain images of the joint above and below fracture.

- If a vascular injury is suspected, perform arteriography.

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Treatment

Prehospital Care

- Prehospital personnel should splint the extremity in the position it was found. If signs of neurovascular compromise are observed, the limb may be reduced after administering adequate analgesia. Well-aligned fractures, with or without neurovascular injury, can be immobilized using a traction device. Hare or Thomas traction splints are used most commonly.
- Apply wet sterile dressings over an open fracture. If the wound is grossly contaminated, sterile saline irrigation may be used to remove large contaminants.

Emergency Department Care

- Fracture reduction and immobilization
 - Reduce fractures to near-anatomic alignment using in-line traction. This reduces pain and helps prevent hematoma formation. Hold reduction by a traction device (eg, Hare, Buck) or long-leg posterior splint.
 - Pneumatic splint may have additional benefits of reducing blood loss by direct pressure and tamponade of hematoma formation. Traction is often required to hold femur out to length because of contraction of large muscle mass in thigh.
- Infection prophylaxis: With open fractures, administer tetanus toxoid (unless given within 5 y) and use antibiotics with excellent staphylococcal coverage and good tissue penetration. Often, a first-generation cephalosporin (ie, cefazolin sodium) is administered in combination with gentamicin.
- Other: In addition to maintenance IV fluids, patients suspected of significant blood loss should be resuscitated with crystalloids. Place Foley catheter and restrict all patients to taking nothing by mouth (NPO) until seen by an orthopedic surgeon.

Consultations

- Emergently consult an orthopedic surgeon.
- Evidence of vascular or progressing neurologic compromise should prompt emergent consultation with a vascular surgeon. In some hospitals, the general surgeon may have privileges for vascular intervention.

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Medication

Drugs used to treat fractures are generally NSAIDs, analgesics, and anxiolytics.

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

These drugs are used most commonly for relief of mild to moderately severe pain. Effects of NSAIDs in treatment of pain tend to be patient specific, yet ibuprofen is usually DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually DOC for treatment of mild to moderately severe pain, if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, which inhibits prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients on anticoagulants—monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

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|-------------------|--|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for relief of mild to moderately severe pain and inflammation. Administer small dosages initially to patients with small bodies, older persons, and those with renal or liver disease. Doses higher than 75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe patient for response. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 12 years: 0.10-1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ €monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

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| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, which decreases prostaglandin synthesis. |
| Adult Dose | 500 mg PO followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/d PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ €monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |

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| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |
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|-------------------|--|
| Drug Name | Flurbiprofen (Ansaid)- Has analgesic, antipyretic, and anti-inflammatory effects. May inhibit cyclooxygenase enzyme, inhibiting prostaglandin biosynthesis. |
| Adult Dose | 200-300 mg/d PO bid/qid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€”monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures.

| | |
|----------------|---|
| Drug Name | Acetaminophen (Tylenol, Panadol, aspirin-free Anacin)- DOC for treatment of pain in patients with documented hypersensitivity to aspirin or NSAIDs, with upper GI disease, or taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/d PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg q4h; not to exceed 5 doses/d |

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|-------------------|---|
| Contraindications | Documented hypersensitivity |
| Interactions | Rifampin can reduce analgesic effects; barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; APAP is contained in many OTC products and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

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| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/d acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

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| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg based on codeine content PO q4-6h or 1-2 tabs q4h; not to exceed 12 tabs/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg oxycodone PO q4-6h prn; not to exceed 5 mg of oxycodone per dose |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in the elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

Anxiolytics

Patients with painful injuries usually experience significant anxiety. Anxiolytics allow administration of smaller analgesic dose to achieve same effect.

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| Drug Name | Lorazepam (Ativan)- Sedative hypnotic in benzodiazepine class that has short onset of effect and relatively long half-life. By increasing action of GABA, a major inhibitory neurotransmitter, may depress all levels of CNS, including limbic and reticular formation. Excellent for sedating patients for >24 h. Monitor patient's BP after administering dose and adjust as necessary. |
| Adult Dose | Initial dose: 2 mg total or 0.044 mg/kg IV, whichever is smaller |
| Pediatric Dose | 0.05 - 0.1 mg/kg IV slowly q2-5min; may repeat dose of 0.05 mg/kg IV slowly |
| Contraindications | Documented hypersensitivity; preexisting CNS depression; hypotension; narrow-angle glaucoma |
| Interactions | Alcohol, phenothiazines, barbiturates, and MAOIs increase CNS toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in renal or hepatic impairment, myasthenia gravis, organic brain syndrome, or Parkinson disease |

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Follow-up

Further Inpatient Care

- Adults are best treated with immediate operative fixation, typically intramedullary nailing.
- Young children typically are treated with skeletal or skin traction for approximately 4 weeks, followed by a body spica cast.
- Depending on stage of skeletal maturity, some adolescents may be treated with initial external fixation, intramedullary nailing, or compression screw plate fixation.
- In presence of contraindications to surgery, this repair may be delayed for days without significant complications if leg length is maintained with traction.
- Open fractures require immediate operative debridement followed by delayed intramedullary nailing.

Transfer

- Transfer patients with femur fractures when fracture is immobilized adequately. This is best accomplished with a traction device. As an alternative, use a pneumatic or posterior molded splint.
- Reasons for transfer include the following:
 - Lack of appropriate orthopedic staff or operative facilities at presenting center necessitates transfer.
 - Associated serious injuries, which are common, may require trauma center for ideal evaluation and management.

Complications

- Hemorrhagic shock
 - Closed fractures of the femur can result in significant blood loss (eg, 1 L) within the thigh. Open fractures have potential for even greater blood loss.
 - Because of the high rate of associated injuries, actively seek out other sources of blood loss in patients with femur fractures and hypovolemic shock.
- Infection: While open fractures are at high risk of soft-tissue and bony infection, postoperative infection is rare following repair of closed fractures.
- Respiratory demise: Fat embolism and adult respiratory distress syndrome (ARDS) can occur.
- More delayed complications include permanent stiffness of hip or knee, shortening of extremity, or malrotation, resulting in permanent deformity and decreased performance.
- Complications directly related to repair include (in order of increasing frequency) breakage of fixator hardware, nonunion, malunion, or delayed union.
- Finally, refracture has occurred at the initial injury site.

Prognosis

- Those who survive initial trauma associated with injury typically heal well. Early mobilization following intramedullary nailing greatly reduces complications associated with prolonged immobilization.
- Age affects speed and quality of recovery. Fractures may be caused by underlying medical conditions such as osteoporosis or cancer metastasis; these conditions may complicate recovery further.
- Patients older than 60 years with closed fractures of femur have a mortality rate of 17% and complication rate of 54%.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to conduct a careful assessment to exclude other, potentially life-threatening, injuries in the presence of femur fracture, which generally denotes high-energy trauma
 - Failure to consider the possibility of child abuse in young children
 - Failure to reduce and stabilize angulated femur fractures as soon as possible to minimize neurovascular injury and hematoma formation
-

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Fractures, Foot

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Introduction

Background

Approximately 10% of all fractures occur in the 26 bones of the foot. These bones comprise 2 bones in the hindfoot (calcaneus, talus), 5 bones in the midfoot (navicular, cuboid, 3 cuneiforms), and 19 bones in the forefoot (5 metatarsals, 14 phalanges). In addition, the foot contains sesamoid bones, most commonly the os trigonum, os tibiale externum, os peroneum, and os vesalianum pedis. Their smooth sclerotic bony margins and relatively consistent locations help distinguish them from fractures. Hindfoot connects to the midfoot at the Chopart joint; forefoot connects to the midfoot at the Lisfranc joint.

Age

In contrast to adults, children have relatively stronger ligaments than bone or cartilage. As a result, fractures are more common than sprains in children. However, a child's forefoot is flexible and very resilient to injury. When metatarsal or phalangeal fractures do occur, they may be difficult to recognize because of multiple growth centers. In such cases, comparison views of the uninjured foot often are helpful.

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Clinical

History

- Mechanism of injury
- Time between injury and presentation
- Prior injuries

Physical

- Inspect injured foot for swelling, bruises, deformity, and open wounds.
- Uncover uninjured foot for side-by-side comparison.
- Palpate for pulses, capillary refill, tenderness, instability, and crepitus.
- Test range of motion and joint function. Normal ranges of motion of the foot relative to the ankle are 45° plantarflexion, 20° dorsiflexion, 30° inversion, 20° eversion, 20° internal rotation, and 10° external rotation. Comparisons with the uninjured foot are helpful.
- Explore all open wounds.
- Conduct and document a careful neurologic exam of foot, including both motor and sensory functions.

Causes

Trauma - Direct, indirect, or overuse

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Differentials

Abdominal Pain in Elderly Persons
Ankle, Soft-tissue Injures
Compartment Syndrome, Extremity
Dislocations, Foot

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Workup

Imaging Studies

- Plain film radiography
 - Ottawa foot rules are a tool that predicts significant midfoot fractures. They are guidelines used to determine whether radiographs are necessary.
 - If any of the following are present, a radiograph is required.
 - Point tenderness over the base of fifth metatarsal
 - Point tenderness over the navicular bone
 - Inability to take 4 steps, both immediately after injury and in the ED

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Treatment

Prehospital Care

Stabilize and elevate foot.

Emergency Department Care

- Ice, immobilize, and elevate foot and provide analgesia to all patients with significant foot fractures.
- Options for initial immobilization
 - Posterior or stirrup splints
 - Reinforced bulky dressing, also termed Jones splint, which consists of a web roll and an elastic compression bandage
 - Rigid, flat-bottom orthopedic shoe also termed postop or Reece shoe
 - Definitive immobilization often requires application of a cylindrical cast, applied during the acute phase (and often bivalved to accommodate further swelling) or after a few days when edema has begun to decrease.
- First metatarsal fracture: This is the least commonly fractured metatarsal. The first metatarsal head bears twice the weight of other metatarsal heads. Treat minimally displaced or nondisplaced fractures with immobilization without weight bearing. Displaced fractures usually require open reduction and internal fixation (see [Picture 7](#)).
- Internal metatarsal fracture
 - Fractures of internal (second, third, fourth) metatarsals are very common. Nondisplaced and displaced fractures usually heal well, with weight bearing as tolerated, in a cast or rigid flat-bottom orthopedic shoe. Exclude disruptions of the Lisfranc (tarsometatarsal)

joint by maintaining a high level of suspicion.

- March fracture is a stress fracture of the second and/or third metatarsal that commonly occurs in joggers. Radiographs are often negative, and sometimes a bone scan helps determine this diagnosis. Treatment is cessation of aggravating activity for 4-6 weeks.

- Fifth metatarsal fracture: The proximal fifth metatarsal is the most common site of midfoot fractures. Fractures are of 2 general types, the Jones fracture and the pseudo-Jones or tennis fracture. Midshaft (see [Picture 8](#)) and distal fifth metatarsal fractures (see [Picture 9](#)) are less common.
 - Proximal avulsion fracture: Fractures at the proximal tuberosity are very common and termed pseudo-Jones or tennis fractures (see [Picture 1](#)). This avulsion injury usually is associated with a lateral ankle strain and occurs at the attachment of the peroneus brevis tendon. It heals well with a compression dressing and weight bearing as tolerated.
 - Jones fracture: This less common but more problematic fracture occurs transversely at the base of the fifth metatarsal, 1.5-3 cm distal to the proximal tuberosity (see [Picture 2](#)). Displacement of this fracture tends to increase with continued weight bearing. Patients with this fracture often (35-50%) develop persistent nonunions requiring bone grafting and internal fixation. Initial therapy must include immobilization without weight bearing.
- The Lisfranc joint is found at the base of second metatarsal and is formed by a 6-bone arch that includes the first, second, and third cuneiforms and first, second, and third metatarsals. Fracture-dislocations at this joint are rare, yet are still the most commonly misdiagnosed foot injuries (see [Picture 3](#), [Picture 6](#)). They can result in posttraumatic arthritis and reflex sympathetic dystrophy. Displaced fractures are clinically and radiographically obvious, yet nondisplaced or minimally displaced fractures may be subtle.
- To facilitate diagnosis, grasp first and second metatarsals and move them alternately through plantarflexion and dorsiflexion.
- Radiographic diagnosis is made by detecting widening (diastasis) of 2-5 mm between the bases of the first and second metatarsals or between the middle and medial cuneiforms. Fracture at the base of the second metatarsal strongly suggests the diagnosis. If standard radiographs appear normal despite clinical suspicion, radiographs of the injured foot bearing weight may reveal the fracture. These fractures require immediate orthopedic consultation for reduction and fixation.
- Neck and body fracture: These are the most common talar fractures and may be associated with subtalar dislocation. Displaced fractures usually require surgical fixation. Nondisplaced fractures are treated with non-weight-bearing short leg cast for 6-10 weeks.
- Lateral process fracture: This type was previously rare, yet now is more common because of snowboarding injuries. Treatment should include immobilization with strict avoidance of weight bearing.

- Posterior process (Shepherd) fracture: Caused by damage to the posterior process of the talus, this fracture's usual mechanism is sudden plantarflexion or repetitive motion, especially in athletes who dance or kick. Diagnosis usually is not confirmed in the ED, because clinical examination is typically nonspecific and plain film radiography normal. Suspicion warrants referral to an orthopedist. Treatment includes immobilization with either partial or full weight bearing. Note that this fracture often is confused with an accessory bone that occurs at this location, the os trigonum.
- Transchondral/osteochondral talar dome fracture: This rare injury often presents as a nonhealing ankle sprain and is caused by small cartilaginous avulsions or body chips in tibial articulation. Tenderness of the talar dome can be appreciated with the foot in dorsiflexion. Radiographs may be normal, and injuries cannot be distinguished clinically from ankle sprains. Delayed presentation may show crepitus, joint locking, and laxity of lateral and anterior ankle ligaments. Suspicion warrants referral to an orthopedist for bone scan or other definitive imaging. Initial therapy for this injury is immobilization without weight bearing.
 - Calcaneal fractures usually occur in patients aged 30-50 years, with a peak incidence at 45 years. They occur in males 5 times more often than in females.
- Intraarticular joint depression fracture: This is the most common form of calcaneal fracture. Lateral foot radiograph reveals a reduction in the Böhler angle, the posterior angle formed by intersection of a line from the posterior to the middle facet and a line from the anterior to the middle facet (see [Picture 7](#), [Picture 8](#)). Böhler angle is normally between 20-40°. Angles less than 20°, or more than 5° smaller than that of uninjured side, indicate a fracture. Obtain an urgent orthopedic consultation, since open reduction and internal fixation is usually necessary.
- Extraarticular fracture: Treat these calcaneal fractures with a bulky compression dressing, rest, ice, and elevation. Arrange orthopedic follow-up care.

Consultations

Nonemergent referral or urgent consultation with an orthopedic surgeon (or podiatrist if appropriate) is often necessary; which is appropriate depends on the type of fracture.

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Medication

Analgesics (narcotics, NSAIDs) are generally the only medications needed to treat foot fractures. Administer antibiotics and tetanus prophylaxis to patients with open fractures.

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

These agents are used most commonly for relief of mild to moderately severe pain. Effects of NSAIDs in treatment of pain tend to be patient specific, yet ibuprofen is usually DOC for initial therapy. Other NSAIDs also may be used.

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| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually DOC for treatment of mild to moderately severe pain, if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, which inhibits prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

Narcotic Combination Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures. Hydrocodone and oxycodone preparations are generally more effective and better tolerated than other narcotic-acetaminophen combinations such as those containing codeine.

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| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |

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| Pediatric Dose | <12 years: 10-15 mg/kg acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

Antibiotics

Prophylaxis is given to patients with open fractures.

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| Drug Name | Penicillin G (Pfizerpen)- Interferes with synthesis of cell wall mucopeptide during active replication, resulting in bactericidal activity against susceptible microorganisms. |
| Adult Dose | 2.4 million U IM single dose in 2 injection sites |
| Pediatric Dose | 50,000 U/kg IM to maximum of 2.4 million U |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid can increase effects; tetracyclines can decrease effects |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Caution in impaired renal function |

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| Drug Name | Clindamycin (Cleocin)- Lincosamide is useful as treatment against serious skin and soft-tissue infections caused by most staphylococcal strains. Also effective against aerobic and anaerobic streptococci, except enterococci. Clindamycin is used for prophylaxis in penicillin-allergic patients. Useful as treatment against streptococci and most staphylococcal strains. |
| Adult Dose | 600 mg PO/IV q6-8h for 5-7d |
| Pediatric Dose | 20-40 mg/kg IM/IV tid/qid for 5-7 d |
| Contraindications | Documented hypersensitivity; regional enteritis; ulcerative colitis; hepatic impairment; antibiotic-associated colitis |

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| Interactions | Increases duration of neuromuscular blockade induced by tubocurarine and pancuronium; erythromycin may antagonize effects; antidiarrheals may delay absorption |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in severe hepatic dysfunction; no adjustment necessary in renal insufficiency; associated with severe and possibly fatal colitis |

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| Drug Name | Gentamicin (Gentacidin, Garamycin)- Aminoglycoside antibiotic used for gram-negative bacterial coverage. Commonly used in combination with both an agent against gram-positive organisms and one that covers anaerobes. Used in conjunction with ampicillin or vancomycin for prophylaxis in patients with open fractures. |
| Adult Dose | 1.5 mg/kg/dose IV q8-24h; not to exceed 80 mg; dosing interval based on renal function |
| Pediatric Dose | 2 mg/kg/dose IV q8h; dosing interval based on renal function |
| Contraindications | Documented hypersensitivity; nondialysis-dependent renal insufficiency |
| Interactions | Other aminoglycosides, cephalosporins, penicillins, and amphotericin B may increase nephrotoxicity; enhances effects of neuromuscular blocking agents, thus prolonged respiratory depression may occur; loop diuretics may increase auditory toxicity of aminoglycosides—irreversible hearing loss of varying degrees may occur (monitor regularly) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Narrow therapeutic index (not intended for long-term therapy); caution in renal failure (not on dialysis), myasthenia gravis, hypocalcemia, and conditions that depress neuromuscular transmission; adjust dose in renal impairment |

Tetanus Toxoid

This agent is used for tetanus immunization. Booster injection is recommended in previously immunized individuals to prevent this potentially lethal syndrome.

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| Drug Name | Tetanus toxoid- Induces active immunity against tetanus in selected patients. Tetanus and diphtheria toxoids are the immunizing DOC for most adults and children older than 7 y. Necessary to administer booster doses to maintain tetanus immunity throughout life. Pregnant patients should receive only tetanus toxoid, not a diphtheria antigen-containing product. In children and adults, may administer into deltoid or midlateral thigh muscles. In infants, preferred site of administration is midthigh laterally. |
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|-------------------|---|
| Adult Dose | Primary immunization: 0.5 mL IM; give 2 injections 4-8 wk apart and a third dose 6-12 mo after second injection Booster dose: 0.5 mL q10y |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; history of any type of neurological symptoms or signs following administration of this product FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are an important cause of provocative poliomyelitis |
| Interactions | Patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization due to poor immune response; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent use of chloramphenicol since it may impair amnestic response to tetanus toxoid; concurrent use of tetanus immune globulin may delay development of active immunity by several days (interaction is nevertheless clinically insignificant and does not preclude its concurrent use) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Do not use to treat actual tetanus infections, or for immediate prophylaxis of unimmunized individuals (use instead tetanus antitoxin, preferably human tetanus immune globulin); diminished antibody response to active immunization may be seen in patients receiving immunosuppressive therapy; better to defer primary diphtheria immunization until immunosuppressive therapy discontinued; routine immunization of symptomatic and asymptomatic HIV-infected persons is recommended |

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Follow-up

Complications

- Compartment syndrome is the most dangerous acute complication of foot fractures. This syndrome is associated primarily with midfoot fractures sustained as the result of a crush mechanism. Clinical signs include marked swelling (early) and neurovascular compromise (late). Suspicion warrants emergent orthopedic consultation; treatment is fasciotomy when diagnosis is confirmed.
- Long-term complications
 - Arthritis
 - Infection

- Nonunion or instability
- Gait disturbances

Prognosis

- Generally excellent with appropriate treatment

Patient Education

- Proper instruction in crutch-walking is required for those unable to bear weight.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to note fractures because radiographs not obtained or misread
 - Failure to consult or refer to an orthopedic surgeon when necessary
-

Pictures



Picture 1: Proximal fifth metatarsal avulsion fracture (also termed pseudo-Jones, tennis, or dancer fracture)

Picture type: X-RAY



Picture 2: Jones fracture of the fifth metatarsal

Picture type: X-RAY



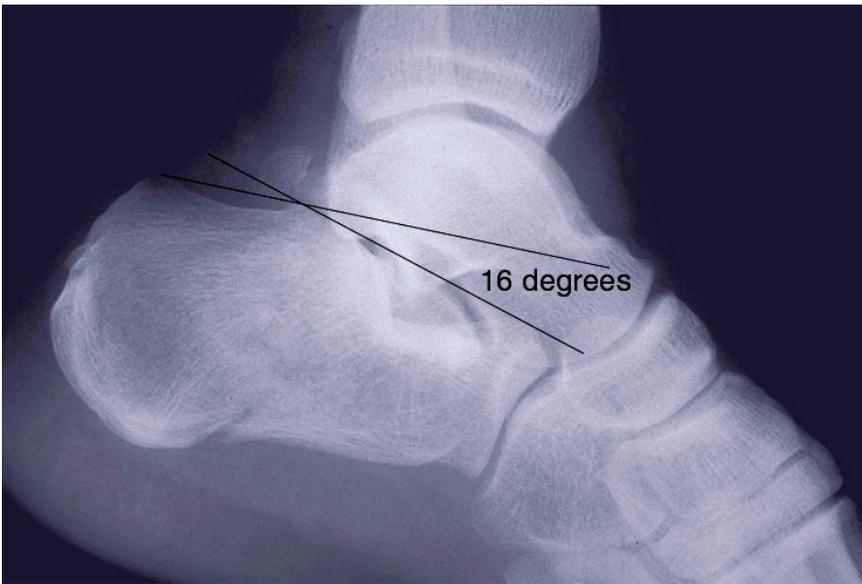
Picture 3: Lisfranc fracture-dislocation

Picture type: X-RAY



Picture 4: Calcaneal fracture with intraarticular involvement and joint depression

Picture type: X-RAY



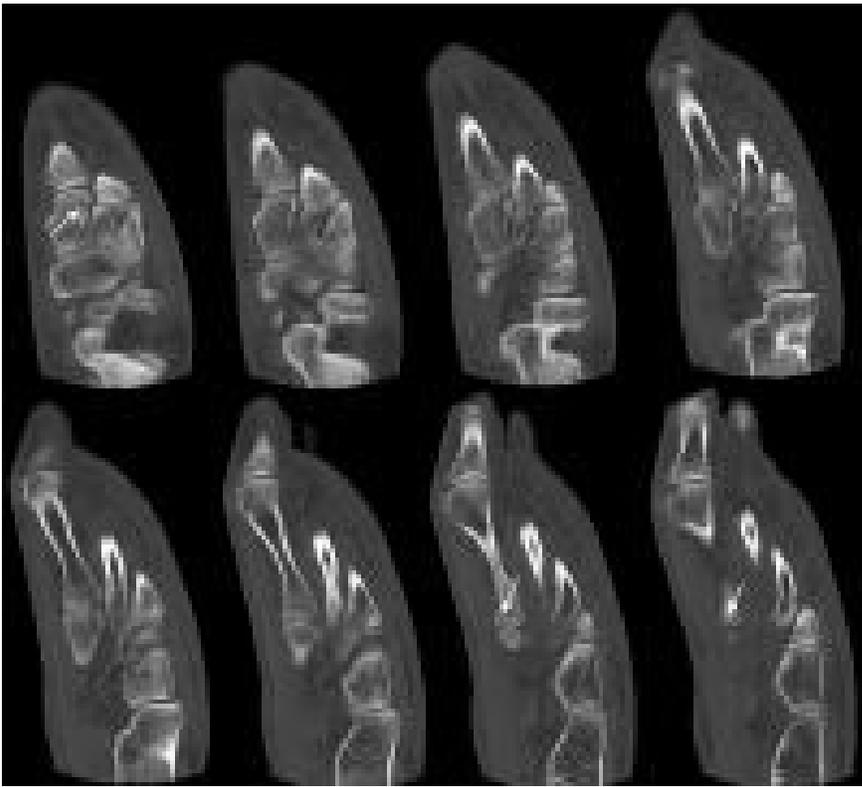
Picture 5: Calcaneal fracture with intraarticular involvement and joint depression with Böhler angle imposed. Reduced angle of 16° is pathologic.

Picture type: X-RAY



Picture 6: Subtle fracture of the first cuneiform at the Lisfranc joint. Another fracture at the base of the first metatarsal is not seen here but was found on subsequent computed tomography.

Picture type: X-RAY

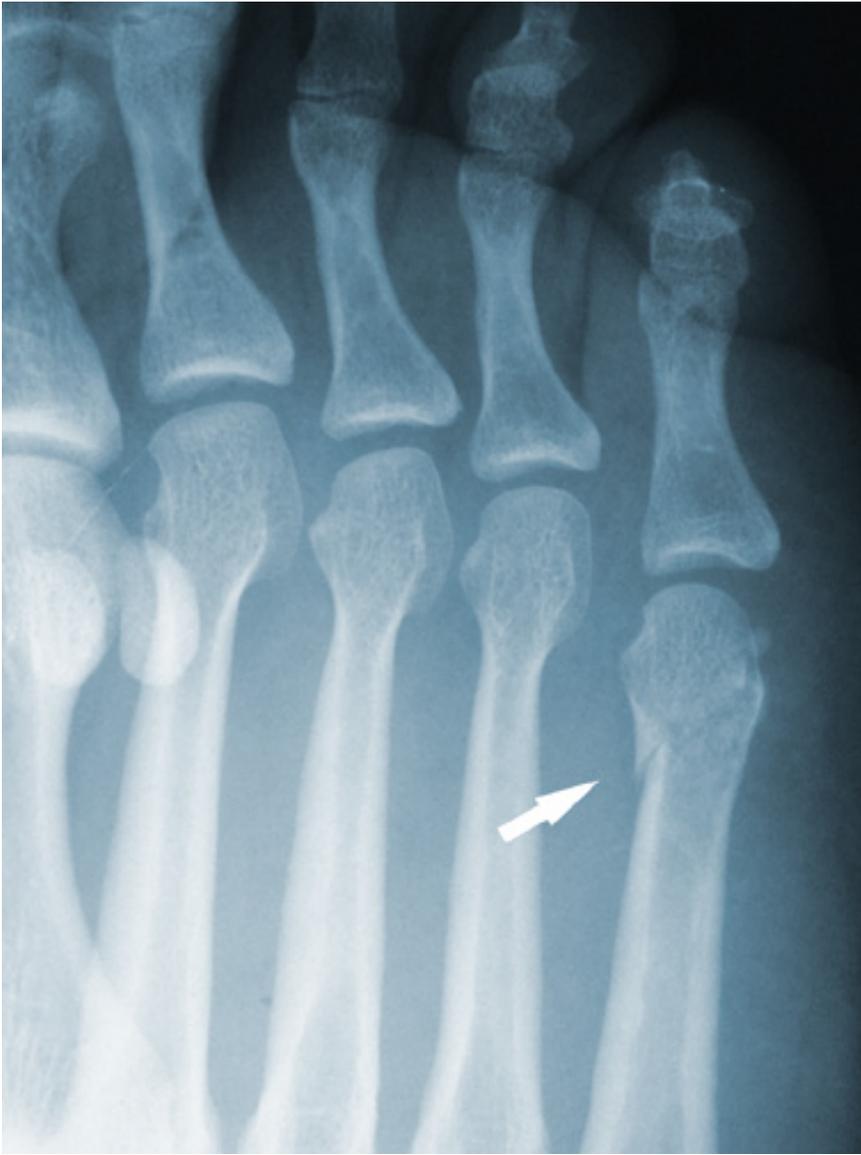


Picture 7: CT scan showing fracture of first cuneiform and proximal first metatarsal
Picture type: CT



Picture 8: Spiral fracture of the shaft of the fifth metatarsal. This fracture was treated conservatively with immobilization.

Picture type: X-RAY



Picture 9: Minimally displaced fracture of the distal fifth metatarsal. This fracture was treated conservatively with immobilization in a rigid flat bottom shoe.

Picture type: X-RAY

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Fractures, Forearm

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Introduction

Background

Because forearm fractures account for most limb fractures, emergency physicians should be familiar with evaluation and management of each type. Forearm fractures are classified as involving the proximal, middle, or distal shaft. Fractures of the radius and ulna, intimately associated with the elbow and wrist, are discussed in those articles (see [Fractures, Elbow](#) and [Fractures, Wrist](#)).

Pathophysiology

Fractures of the wrist and elbow usually involve a fall onto the outstretched arm, while forearm shaft fractures more commonly are the result of a direct blow.

Frequency

- **In the US:** The upper extremity is involved in nearly half of all fractures seen, and wrist fractures account for about one third of these.

Mortality/Morbidity

Postmenopausal women, because of osteoporosis, have a higher rate of forearm fractures than other adults. When the mechanism of injury seems trivial, suspect a pathologic fracture associated with a cyst or tumor. Forearm fractures in older persons are associated with increased risk of future vertebral and hip fractures.

Race

Forearm fractures are less common in blacks because of a lower incidence of osteoporosis.

Sex

- In infants and toddlers, forearm fractures have no sex predilection.
- In children older than 2 years, fractures are more common in boys than girls.
- In older persons, fractures are more common in women than men.

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Clinical

History

History is usually consistent with a direct blow to the forearm or a fall directly onto the forearm. Understanding the mechanism of injury helps direct the physical exam to detect injuries.

Physical

- Patients usually have localized pain, tenderness, and swelling at the fracture site.
- Fractures are classified as open or closed.
 - Consider any puncture or break in the skin over a fracture site evidence of an open fracture unless proven otherwise.
 - Incidence of open forearm fractures is second only to those of the tibia.
 - Open fracture classification system (Smith)
 - Type I - Clean wound less than 1 cm
 - Type II - 1-cm wound without extensive soft-tissue damage
 - Type III - Segmental fracture or extensive soft-tissue damage
 - Subtype IIIA - Fracture from gunshot wound

- Subtype IIIB - Farm injuries with extensive soft-tissue damage and wound contamination
- Perform a vascular exam. Check capillary refill, radial pulse, and Allen test.
- Examine wrist and elbow for tenderness and range of motion.
 - Palpate wrist to evaluate for ulnar styloid fracture, dorsal prominence of the ulna, or wrist pain with rotation.
 - Tenderness or prominence of radial head may be the only physical finding in patients with reduced Monteggia lesion or radial head fracture.

Causes

- Sports, particularly in-line skating, skateboarding, mountain biking, and contact sports
- Trauma, commonly from automobile collisions, blows with a blunt object, or child abuse

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Differentials

Dislocations, Elbow

Dislocations, Hand

Dislocations, Wrist

Fractures, Elbow

Fractures, Hand

Fractures, Wrist

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Workup

Imaging Studies

- General radiography principles
 - Anteroposterior and lateral views of wrist, forearm, and elbow are required when forearm fracture is suspected from clinical findings.
 - Forearm radiographs, which include distal joints, are inadequate for absolutely excluding associated wrist and elbow injuries, as diagnosis of radioulnar dislocation requires the x-ray beam to be centered at the joint.

- Monteggia fracture
 - Defined as a fracture of the ulna (usually proximal one third) with dislocation of the radial head. Anterior radial head dislocation is most common (60%), yet medial, lateral, and posterior dislocations also occur.
 - Isolated proximal ulnar fractures are rare. Always suspect a Monteggia fracture/dislocation and closely examine radial head for dislocation or other evidence of injury.
 - Radial head dislocation can be missed when radiographs are misinterpreted, falsely negative, or inadequate. It also may go unrecognized when the dislocation reduces spontaneously prior to imaging. A line drawn through the radial shaft and head must align with the capitellum in all views to exclude dislocation.
 - Immobilize with a long-arm splint (with elbow flexed 90° and forearm neutral). Children may be treated by reduction and casting, while adults require admission for ORIF.
- Concomitant radius and ulna fractures: Concomitant fractures usually result from a significant force applied directly to the forearm or major multisystem trauma. Swelling and deformity indicate the diagnosis, and radiographic confirmation is usually straightforward (see [Picture 1](#)). Compartment syndrome is a potential complication because of the degree of tissue injury and swelling involved. Treatment usually requires admission for an urgent ORIF, though in children younger than 10 years, if reduced to less than 10° of angulation, these fractures may be treated by casting alone.
- Essex-Lopresti fracture: This is defined as a fracture of the radial head and dislocation of DRUJ, with partial or complete disruption of radioulnar interosseous membrane.
- Torus (greenstick) fracture: This occurs in children with only a moderate degree of trauma and can be managed with a long-arm cast when angulation is less than 10° (see [Picture 2](#), [Picture 3](#)). All require orthopedic referral.

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Treatment

Prehospital Care

Stabilize arm to prevent or limit neurovascular injury from sharp bone fragments.

Emergency Department Care

- Immobilize forearm and upper arm and provide effective analgesia unless patient has other injuries with the potential for hemodynamic or respiratory instability.
- Identify other injuries. Because forearm fractures require considerable force, perform a complete physical exam to exclude other injuries.

- Assess injured forearm.
 - Perform a careful exam of the upper extremity to identify neurovascular deficits, tense muscle compartments, and disruptions of the skin.
 - Obtain appropriate radiographs to define fracture(s) and evaluate for associated dislocation.
- Provide adequate analgesia/anesthesia.
- Perform emergent reduction, if necessary.
- Immobilize injury.
- Administer antibiotics and tetanus immunization, as indicated.
- Immediate fracture reduction is indicated when any of the following exists:
 - Neurovascular compromise
 - Severe displacement
 - Tenting of the skin

Consultations

- Consult orthopedist for open fractures, operative fractures, or dislocations, and arrange close follow-up care.
- Fracture reductions typically are deferred to an orthopedist unless evidence of neurovascular compromise is noted.

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Medication

Drugs used to treat fractures are generally NSAIDs and analgesics. In addition, administer proper antibiotics and tetanus prophylaxis for open fractures.

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

These drugs are used most commonly for relief of mild to moderately severe pain. Although effects of NSAIDs in treatment of pain tend to be patient specific, ibuprofen is usually DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

| | |
|-----------|---|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually DOC for treatment of mild to moderately severe pain, if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, inhibiting prostaglandin synthesis. |
|-----------|---|

| | |
|-------------------|--|
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for relief of mild to moderately severe pain and inflammation. Administer small dosages initially to patients with small bodies, older persons, and those with renal or liver disease. Doses higher than 75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 12 years: 0.1â€1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, decreasing prostaglandin synthesis. |
| Adult Dose | 500 mg PO followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ €monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

| | |
|-------------------|--|
| Drug Name | Flurbiprofen (Ansaid, Ocufer)- Has analgesic, antipyretic, and anti-inflammatory effects. May inhibit cyclooxygenase enzyme, inhibiting prostaglandin biosynthesis. |
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ €monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|--|
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |
|-------------|--|

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and enables physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures.

| | |
|-------------------|---|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tab q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|--|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen PO q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |

| | |
|-------------|---|
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |
|-------------|---|

| | |
|-------------------|---|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone q4-6h |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in the elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

Antibiotics

Empiric antimicrobial therapy must be comprehensive and should cover all likely pathogens in the clinical setting.

| | |
|-------------------|---|
| Drug Name | Gentamicin (Gentacidin, Garamycin)- Aminoglycoside antibiotics used for gram-negative bacterial coverage. Commonly used in combination with both an agent against gram-positive organisms and one that covers anaerobes. Used in conjunction with ampicillin or vancomycin for prophylaxis in patients with open fractures. |
| Adult Dose | 1.5 mg/kg IV; not to exceed 80 mg |
| Pediatric Dose | 2 mg/kg IV |
| Contraindications | Documented hypersensitivity; nondialysis-dependent renal insufficiency |
| Interactions | Other aminoglycosides, cephalosporins, penicillins, and amphotericin B may increase nephrotoxicity; enhances effects of neuromuscular blocking agents, thus prolonged respiratory depression may occur; loop diuretics may increase auditory toxicity—possible irreversible hearing loss of varying degrees may occur (monitor regularly) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|---|
| Precautions | Narrow therapeutic index (not intended for long-term therapy); caution in renal failure (not on dialysis), myasthenia gravis, hypocalcemia, and conditions that depress neuromuscular transmission; adjust dose in renal impairment |
|-------------|---|

| | |
|-------------------|---|
| Drug Name | Ampicillin (Omnipen, Marcillin)- Used for prophylaxis in patients undergoing dental, oral, or respiratory tract procedures. Interferes with bacterial cell wall synthesis during active replication, causing bactericidal activity against susceptible organisms. This drug is given in place of amoxicillin in patients unable to take medication orally. It is also used along with gentamicin for prophylaxis in patients with open fractures. |
| Adult Dose | 2 g IV/IM |
| Pediatric Dose | 50 mg/kg IV/IM |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid and disulfiram elevate levels; allopurinol decreases effects and has additive effects on ampicillin rash; may decrease effects of oral contraceptives |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal failure; evaluate rash and differentiate from hypersensitivity reaction |

| | |
|-------------------|---|
| Drug Name | Vancomycin (Vancocin)- Potent antibiotic directed against gram-positive organisms and active against enterococcal species. Also useful in treatment of septicemia and skin structure infections. Used in conjunction with gentamicin for prophylaxis in penicillin-allergic patients undergoing GI or GU procedures. May need to adjust the dose in patients with renal impairment. |
| Adult Dose | 1 g IV infused over 1 h |
| Pediatric Dose | 1.5 mg/kg IV infused over 1 h |
| Contraindications | Documented hypersensitivity |
| Interactions | Erythema, histaminelike flushing, and anaphylactic reactions may occur when administered with anesthetic agents; taken concurrently with aminoglycosides, risk of nephrotoxicity may increase above that with aminoglycoside monotherapy; effects in neuromuscular blockade may be enhanced when coadministered with nondepolarizing muscle relaxants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in renal failure, neutropenia; red man syndrome is caused by too rapid IV infusion (dose given over a few minutes) but rarely happens when dose given over 2 h or by PO or IP route; red man syndrome not an allergic reaction |

Toxoid

This agent is used for tetanus immunization. A booster injection in previously immunized individuals is recommended to prevent this potentially lethal syndrome.

| | |
|-------------------|---|
| Drug Name | Tetanus toxoid- Used to induce active immunity against tetanus in selected patients. Tetanus and diphtheria toxoids are immunizing AOC for most adults and children >7 y. Necessary to administer booster doses to maintain tetanus immunity throughout life. Pregnant patients should receive only tetanus toxoid, not a diphtheria antigen-containing product. In children and adults, may administer into deltoid or midlateral thigh muscles. In infants, preferred site of administration is midhigh laterally. |
| Adult Dose | Primary immunization: 0.5 mL IM, give 2 injections 4-8 wk apart and a third dose 6-12 mo after a second injection Booster dose: 0.5 mL q10y |
| Pediatric Dose | Primary immunization: 0.5 mL IM, give 2 injections 4-8 wk apart and a third dose 6-12 mo after the second injection. Booster dose: 0.5 mL q10y |
| Contraindications | Documented hypersensitivity; history of any type of neurological symptoms or signs following administration of this product FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are an important cause of provocative poliomyelitis |
| Interactions | Patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization due to poor immune response; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent use of chloramphenicol since it may impair amnestic response to tetanus toxoid; concurrent use of tetanus immune globulin may delay development of active immunity by several days (interaction is nevertheless clinically insignificant and does not preclude its concurrent use) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Do not use to treat actual tetanus infections, or for immediate prophylaxis of unimmunized individuals (use instead tetanus antitoxin, preferably human tetanus immune globulin) diminished antibody response to active immunization may be seen in patients receiving immunosuppressive therapy; better to defer primary diphtheria immunization until immunosuppressive therapy discontinued; routine immunization of symptomatic and asymptomatic HIV-infected persons is recommended |

Immunoglobulins

Patients who may not have been immunized against *Clostridium tetani* products should receive tetanus immune globulin.

| | |
|-------------------|---|
| Drug Name | Tetanus immune globulins (Hyper-Tet)- Used for passive immunization of any person with a wound that may be contaminated with tetanus spores. |
| Adult Dose | For prophylaxis: 250-500 U IM in opposite extremity to tetanus toxoid lesion For clinical tetanus: 3,000-10,000 U IM |
| Pediatric Dose | For prophylaxis: 250 U IM in the opposite extremity to tetanus toxoid For clinical tetanus: 3,000-10,000 U IM |
| Contraindications | Since antibodies in globulin preparation may interfere with immune response to vaccination, do not administer within 3 mo of live virus immune globulin administration; may be necessary to revaccinate persons who received immune globulin shortly after live virus vaccination |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Persons with isolated IgA deficiency have potential for developing antibodies to IgA and could have anaphylactic reactions to subsequent administration of blood products that contain IgA; do not perform skin testing since intradermal injection of concentrated gamma globulin may cause localized area of inflammation and can be misinterpreted, causing the medication to be withheld from a patient not allergic to this material; true allergic responses to human gamma globulin given in prescribed IM manner are extremely rare; do not admix with other medications since usually incompatible |

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Follow-up

Further Inpatient Care

- Admit patient whenever the following conditions are present:
 - Open fracture
 - Presence of or potential for neurovascular compromise
 - Fracture requiring ORIF and orthopedist plans to operate expeditiously

Further Outpatient Care

- Most cases can be treated safely by splinting and referral to an orthopedist who will then schedule surgical repair (if necessary).
- Elevate injured extremity and limit physical activities to prevent further injury.
- Provide instructional material on cast/splint care and symptoms requiring a return to ED.

in/Out Patient Meds

- Prescribe oral analgesics (eg, NSAIDs, acetaminophen with codeine/hydrocodone).

Transfer

- Transfer to a facility with a higher level of care when no orthopedist is available and admission or urgent surgery is necessary.

Deterrence/Prevention

- Recommend wearing wrist guards while in-line skating, roller skating, or skateboarding.
- Prevent osteoporosis in postmenopausal women.

Complications

- Direct neurovascular injury
- Physeal arrest if fracture involves growth plate
- Radioulnar synostosis after delayed treatment
- Compartment syndrome - Associated with closed shaft fractures of radius or ulna and with tight casts. It is less common in upper than in lower extremities.
- Loss of supination-pronation after a forearm fracture

Prognosis

- Prognosis for recovery of forearm fractures (ie, good bony union, maintenance of function), is related to severity and type of fracture and is optimized by treating fractures early and appropriately.
- Morbidity is related to missed or delayed diagnosis of an open fracture or dislocation associated with fracture.
- Improvements in internal and external fixation materials and techniques have allowed more aggressive treatment of forearm fractures, with fewer complications and improved recovery of function.

- Midshaft fractures tend to have worse outcomes than fractures in the distal or proximal third of forearm.

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Miscellaneous

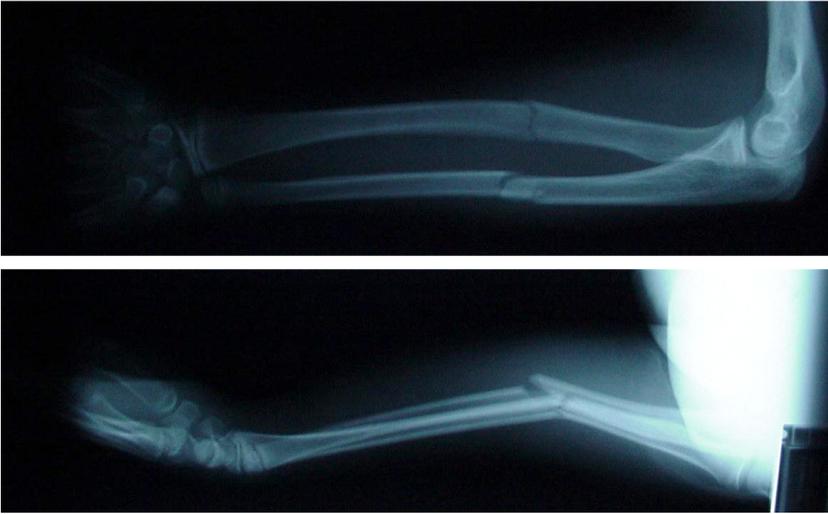
Medical/Legal Pitfalls

- Failure to suspect DRUJ pathology in face of isolated radial fracture (Galeazzi type)
- Failure to suspect radial head dislocation in face of isolated ulnar fracture (Monteggia type)
 - Radial head dislocations usually can be reduced or closed early in presentation, but delayed diagnosis commonly requires open reduction.
 - Lesions undiagnosed by the emergency physician are likely to be missed on outpatient follow-up visit.
 - Spontaneous reduction during splinting and loss of physical findings of pain at the radial head by the time of follow-up contribute to delayed or missed diagnosis
- Failure to recognize neurovascular injury

Special Concerns

- Suspect child abuse when mechanism of injury is inconsistent with fracture type, especially in newborns and infants.
 - Realize that lesser amounts of mechanical force may result in fracture, especially in postmenopausal women.
-

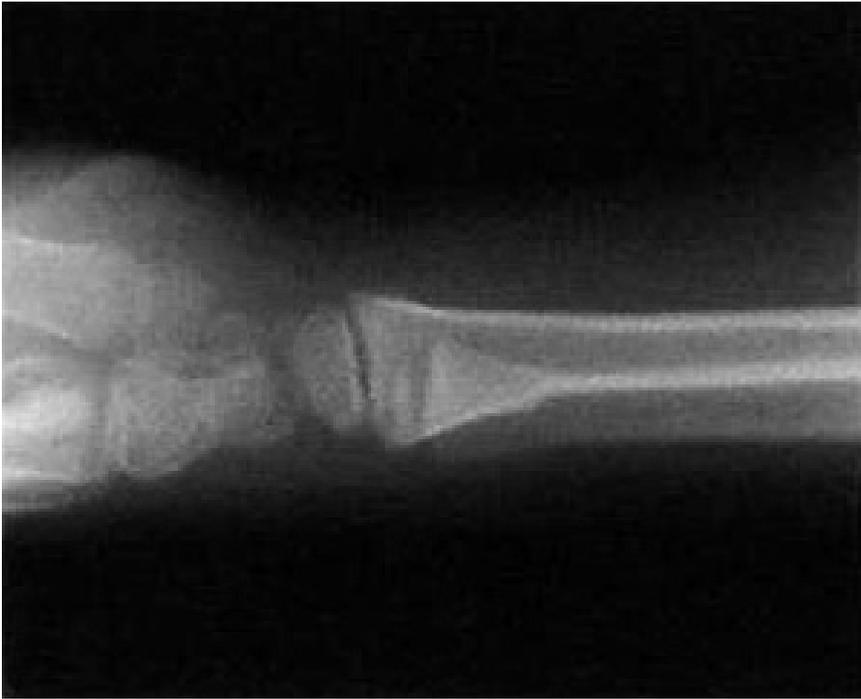
Pictures



Picture 1: Fractures of the radius and ulna with dorsal angulation of distal fragments
Picture type: X-RAY



Picture 2: Torus fracture of the radius
Picture type: X-RAY



Picture 3: Torus fracture of the radius

Picture type: X-RAY

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Fractures, Frontal

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Introduction

Background

Hippocrates described an array of facial injuries as long ago as 400 BC. In 1823, von Graeffe described an elastic tube placed in the nose to maintain an open airway. During the early 20th century, Sir Harold Gilles, father of plastic surgery, taught army personnel about breathing problems in patients with facial injuries and to place them supine to maintain an airway.

René Le Fort, a Frenchman, studied cadavers in 1901. He described 3 basic types of fractures. Endotracheal anesthesia and radiography developed during the First World War led to a better understanding and treatment of facial fractures. During the Second World War, a multidisciplinary approach to treatment of facial fractures continued to improve the outcomes of severely injured soldiers. Advent of CT reconstruction of facial bones, along with new surgical techniques, has dramatically improved the final appearance patients who have sustained bony injuries.

Pathophysiology

Maxillofacial fractures result from blunt or penetrating injury. Blunt injuries are far more common, resulting from vehicular accidents, altercations, sporting-related trauma, occupational injuries, and falls. Penetrating injuries mainly are the result of gunshot wounds, stabbings, and explosions.

Type of object striking the face and force behind the object are the main determinants of whether a person sustains soft-tissue or bony injury. In automobile accidents, striking a hard dashboard is more likely to cause bony injury than striking a padded dashboard or an airbag. Striking the steering wheel concentrates the force more than striking the flat surface of the dashboard. This also holds true for altercations with a bat, as compared to a bare fist or boxing glove. Penetrating injury from a shot gun at a distance is not likely to cause fractures. Bullets from low-velocity guns are likely to cause fractures; high-velocity bullets cause fractures and extensive soft-tissue damage.

The amount of force needed to fracture different bones of the face has been studied; injuries have been divided into those that require high impact to fracture (greater than 50 times the force of gravity [g]) and those that require a low impact to fracture (50 g or less).

- High impact
 - Supraorbital rim - 200 g
 - Symphysis of the mandible - 100 g
 - Frontal-glabellar bone - 100 g
 - Angle of mandible - 70 g

Frequency

- **In the US:** Approximately 3 million facial injuries occur annually; however, most do not involve maxillofacial fractures. One study placed the incidence of severe maxillofacial injury (fractures and lacerations) at 0.04-0.09% for persons in motor vehicle accidents. Incidence of fractures due to motor vehicle injuries is higher in rural areas, and altercation-related fractures are more frequent in urban areas.

Mortality/Morbidity

Incidence of other major injuries is as high as 50% in high-impact fractures, while it is 21% for low-impact fractures. Motor vehicle accidents are more likely than violent altercations to cause other injuries. Mortality rate in high-impact fractures is as high as 12%, yet deaths rarely occur from maxillofacial injury. The incidence of cervical spine injuries associated with frontal fractures has been reported in the 0.2-6.0% range.

Sex

Adult male-to-female ratio is 3:1.

Age

Male predominance is reduced to 2:1 in children.

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Clinical

History

- Since maxillofacial fractures are the result of trauma, primary survey and attention to ABCs take priority. Focus initially on patency of airway, control of cervical spine, and whether the patient is having difficulty breathing, and determine if the patient is experiencing symptoms of shock or neurologic impairment.
- Once life threats have been addressed, obtain a thorough history.
 - Allergies
 - Medications
 - Medical history
 - Last meal
 - Events leading to injury

Physical

- Complete exam of the face is necessary since multiple injuries can occur easily. Portions of the exam specific for the frontal bone are marked with an asterisk (*).
 - Inspect face for asymmetry, which is often easiest to do looking down from the head of the bed.
 - *Inspect open wounds for foreign bodies and palpate for bony injury.
 - *Palpate bony structures of the supraorbital ridge and frontal bone for step-off fractures.
 - *Examine eyes thoroughly for injury, abnormality of ocular movements, and visual acuity.
 - Inspect nares for telecanthus and widening of the nasal bridge. Palpate for tenderness and crepitus.
 - Inspect nasal septum for septal hematoma and clear rhinorrhea, which suggests cerebrospinal fluid (CSF) leak.
 - Palpate zygoma along its arch as well as its articulations with frontal bone, temporal bone, and maxillae.
 - Check facial stability by grasping teeth and hard palate, then gently pushing back and forth, then up and down, feeling for movement or instability of midface.
 - Inspect teeth for fracture and bleeding at gum line, a sign of fracture through alveolar bone. Test for stability.
 - Check teeth for malocclusion and step-off.
 - Palpate mandible for tenderness, edema, and step-off along its symphysis, body, angle, and condyle anterior to ear canal.

- *Evaluate supraorbital, infraorbital, inferior alveolar, and mental nerve distributions for hypoesthesia and anesthesia.
- Frontal fracture is suspected in patients who experience high-impact, blunt trauma and have a physical exam demonstrating step-off of the frontal bone or supraorbital ridge. Epistaxis or CSF leak merits further evaluation if the patient has a forehead injury.

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Differentials

Blast Injuries

Corneal Abrasion

Corneal Laceration

Dislocations, Mandible

Domestic Violence

Epidural Hematoma

Epistaxis

Fractures, Cervical Spine

Fractures, Face

Fractures, Frontal

Fractures, Mandible

Fractures, Orbital

Globe Rupture

Neck Trauma

Pediatrics, Child Abuse

Retinal Detachment

Sexual Assault

Shock, Hemorrhagic

Spinal Cord Injuries

Subarachnoid Hemorrhage

Subdural Hematoma

Other Problems to be Considered

Airway obstruction

Dental fractures

Foreign body aspiration, teeth

Nerve injury - Supraorbital, infraorbital, inferior alveolar, facial

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Workup

Lab Studies

- Direct lab studies toward workup of trauma patient.
- If fracture is an isolated injury, obtain preoperative labs if surgery is planned.

Imaging Studies

- Radiographs
 - Obtain routine facial views, including Waters, Caldwell, and lateral projections.
 - Caldwell projection provides the best view of the anterior table; however, the posterior table is difficult to assess in any of the standard plain film views.

Other Tests

- Test clear rhinorrhea for glucose to help determine if it is CSF, as nasal secretions are normally low in glucose.
- If blood is present, this test is unreliable.
- Blood-tinged fluid can be placed on filter paper to look for a double ring sign of CSF around blood; however, this is not reliable.

Procedures

- When dural leak causing CSF rhinorrhea is suspected yet cannot be proven, the following procedure, which is generally not performed in the Emergency Department, may be done. Inject fluorescein dye into the lumbar subarachnoid space. Examine the discharged nasal fluid 30 minutes later with a Wood lamp for fluorescence; fluorescence confirms CSF rhinorrhea.

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Treatment

Prehospital Care

- ABCs are first priority. Hold airway open by chin lift, jaw thrust, or airway adjuncts, including endotracheal intubation.
- Because of concerns over intracranial placement of endotracheal tubes, avoid using the nasotracheal route for intubation if the patient has extensive facial damage or midface fracture is suspected.
- Place the patient on a backboard with a collar if cervical spine injury is a possibility.
- Treat hypoventilation with intubation and bag ventilation.
- Control actively bleeding wounds by applying a bandage with direct pressure.

Emergency Department Care

- ABCs take priority; reassess airway frequently.
- Do not focus solely on the obvious deformity, thereby failing to perform a complete primary survey.
- Rapidly diagnose other life threats and undertake appropriate resuscitation. Follow with a complete secondary survey.
- Diagnosis of frontal bone fracture in the ED is part of secondary survey.

Consultations

- If a frontal fracture is diagnosed, refer patient to a neurosurgeon, as these injuries often are associated with intracranial injury.
- Provide care for a patient with multiple injuries in conjunction with a surgeon experienced in trauma care.

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Medication

When airway control is needed, rapid-sequence induction is often the preferred method. Rapid-sequence induction utilizes medications to induce unconsciousness and muscle paralysis to facilitate intubation. A cricothyroidotomy kit should be at bedside in case problems arise.

Medication for pain control is appropriate, including NSAIDs, narcotics, or local anesthetics.

Use of prophylactic antibiotics is controversial when a CSF leak is identified. It is usually left to the discretion of the specialist assuming care of the patient.

In cases of open wounds, administer tetanus toxoid if the patient is not up to date.

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

These drugs are used most commonly for relief of mild to moderately severe pain. Effects of NSAIDs in the treatment of pain tend to be patient specific, yet ibuprofen is usually DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually DOC for treatment of mild to moderately severe pain, if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, which inhibits prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|----------------|---|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Relieves mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, which decreases prostaglandin synthesis. |
| Adult Dose | 500 mg PO followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose; not to exceed 10 mg/kg/d |

| | |
|-------------------|--|
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

| | |
|-------------------|--|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- For relief of mild to moderately severe pain and inflammation. Administer small dosages initially to patients with small bodies, older persons, and those with renal or liver disease. Doses higher than 75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe patient for response. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 12 years: 0.1â€1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Flurbiprofen (Ansaid, Ocufer)- Has analgesic, antipyretic, and anti-inflammatory effects. May inhibit cyclooxygenase enzyme, inhibiting prostaglandin biosynthesis. |
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

Analgesics

Pain control is essential to quality care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures.

| | |
|-------------------|---|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tab q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in the elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

| | |
|------------|--|
| Drug Name | Morphine sulfate (Duramorph, Astramorph, MS Contin)- DOC for narcotic analgesia because of its reliable and predictable effects, safety, and ease of reversibility with naloxone. Morphine sulfate administered IV may be dosed in a number of ways and commonly is titrated until desired effect obtained. |
| Adult Dose | Starting dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h Relatively hypovolemic patients: Start with 2 mg IV/IM/SC and reassess hemodynamic effects of dose |

| | |
|-------------------|---|
| Pediatric Dose | Neonates: 0.05-0.2 mg/kg IV prn Children: 0.1-0.2 mg/kg IV q2-4h prn |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

Immunoglobulins

Patients who may not have been immunized against *Clostridium tetani* products should receive tetanus immune globulin.

| | |
|-------------------|---|
| Drug Name | Tetanus immune globulins (Hyper-Tet)- For passive immunization of any person with a wound that may be contaminated with tetanus spores. |
| Adult Dose | For prophylaxis: 250-500 U IM in opposite extremity to tetanus toxoid For clinical tetanus: 3,000-10,000 U IM |
| Pediatric Dose | For prophylaxis: 250 U IM in opposite extremity to tetanus toxoid For clinical tetanus: 3,000-10,000 U IM |
| Contraindications | Since antibodies in globulin preparation may interfere with immune response to vaccination, do not administer within 3 mo of live virus immune globulin administration; may be necessary to revaccinate persons who received immune globulin shortly after live virus vaccination |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Persons with isolated IgA deficiency have potential for developing antibodies to IgA and could have anaphylactic reactions to subsequent administration of blood products that contain IgA; do not perform skin testing since intradermal injection of concentrated gamma globulin may cause localized area of inflammation and can be misinterpreted, causing the medication to be withheld from a patient not allergic to this material; true allergic responses to human gamma globulin given in prescribed IM manner are extremely rare; do not admix with other medications since usually incompatible |

Toxoid

This agent is used for tetanus immunization. Booster injection in previously immunized individuals is recommended to prevent this potentially lethal syndrome.

| | |
|-------------------|---|
| Drug Name | <p>Tetanus toxoid- Used to induce active immunity against tetanus in selected patients. Tetanus and diphtheria toxoids are the immunizing DOC for most adults and children >7 y. Necessary to administer booster doses to maintain tetanus immunity throughout life.</p> <p>Pregnant patients should receive only tetanus toxoid, not a diphtheria antigen-containing product.</p> <p>In children and adults, may administer into deltoid or midlateral thigh muscles. In infants, preferred site of administration is midhigh laterally.</p> |
| Adult Dose | <p>Primary immunization: 0.5 mL IM, give 2 injections 4-8 wk apart and a third dose 6-12 mo after second injection</p> <p>Booster dose: 0.5 mL q10y</p> |
| Pediatric Dose | Administer as in adults |
| Contraindications | <p>Documented hypersensitivity; history of any type of neurological symptoms or signs following administration of this product</p> <p>FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are important cause of provocative poliomyelitis</p> |
| Interactions | <p>Patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization due to poor immune response; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent chloramphenicol since it may impair anamnestic response to tetanus toxoid; concurrent use of tetanus immune globulin may delay development of active immunity by several days (interaction is nevertheless clinically insignificant and does not preclude its concurrent use)</p> |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | <p>Do not use to treat actual tetanus infections, or for immediate prophylaxis of unimmunized individuals (use instead tetanus antitoxin, preferably human tetanus immune globulin) diminished antibody response to active immunization may be seen in patients receiving immunosuppressive therapy; better to defer primary diphtheria immunization until immunosuppressive therapy discontinued; routine immunization of symptomatic and asymptomatic HIV-infected persons recommended</p> |

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Follow-up

Further Inpatient Care

- Since these fractures require extreme force, admitting all except those few patients with isolated, nondisplaced anterior table fractures is appropriate.
- Patients with depression of the inner table often require neurosurgical intervention to elevate the fragment.
- Those with continued CSF leak may require a frontal sinus procedure involving ablation of the sinus and removal of the inner table to allow the frontal sinus to become part of cranium.

Transfer

- If appropriate specialists are not available, arrange transfer to a higher level hospital. Regulations of the Emergency Medical Treatment and Active Labor Act (EMTALA) must be followed.

Deterrence/Prevention

- Use of seat belts and airbags can reduce incidence of facial injuries in motor vehicle accidents. Use of helmets with facial guards can reduce injury in motorcycle accidents and in such sports as skiing, snowboarding, hockey, and football.

Complications

- CSF leaks may continue, though most cease by 2-3 weeks after the injury.
- Observe patient closely for signs and symptoms of meningitis or abscess formation.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to diagnose frontal fracture
- Failure to diagnose associated intracranial or cervical spine injuries because focus was on obvious injury

Special Concerns

- Always consider loss of airway and intracranial and intraabdominal injury.
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Fractures, Hand

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Introduction

Background

Hand fractures, a frequent emergency department complaint, are the most common fractures of the body. Much morbidity and disability can be prevented by instituting proper management at initial evaluation. Emergency physicians, often first to assess these fractures, must possess skills to properly evaluate and manage these injuries.

This chapter focuses entirely on fractures of the hand. Please see other chapters for information on [wrist injuries](#), [soft-tissue hand injuries](#), and [dislocations](#).

Pathophysiology

Basic concepts about bony structures of the hand help to understand injury patterns and manage hand fractures. The hand is a group of gliding bones surrounded by soft tissue. A relatively immobile center consisting of the second and third metacarpal bones provides fixed support around which intrinsic movements of the hand depend. More mobile bones of the hand, such as the first, fourth, and fifth metacarpals, may tolerate a greater degree of angulation without disability, while the less mobile second and third metacarpal bones must have more precise reduction to ensure proper function.

Frequency

- **In the US:** More than 16 million people each year receive emergency care for hand injuries. Common emergencies include fractures, ligamentous injuries, and infections.

Mortality/Morbidity

- Disability from hand injuries may result in loss of sensation, strength, and flexibility, the chief functions of the hands. Preserving function relies on maintaining the structural relationships of the intrinsic hand structures as well as musculotendinous connections from the forearm.
- Prevention of disability from hand injuries is the primary goal of treatment. Maintenance of function, rather than cosmesis, is of paramount concern in the management of hand injuries.

Age

Hand fractures occur in all age groups, although fractures in young children should prompt suspicion of child abuse.

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Clinical

History

Hand fractures usually are not difficult to diagnose. Most patients provide a history of preceding trauma. Physicians must elicit details of the trauma, as this may benefit the hand surgeon. If an industrial injury is the cause, details may help prevent injury to others. Document the following important details in ED records:

- Hand dominance of patient
- Hand that is injured
- Occupation and hobbies requiring dexterity
- Mechanism of injury
 - Did injury occur in a clean or dirty environment?
 - Were crush injuries sustained?
 - What was the position of the hand at time of injury?
 - Was injury the result of high-pressure grease, water, air, or paint injection?
 - Did a thermal, electric, or chemical injury occur?
 - Was patient wearing any type of jewelry on fingers?

- If so, has it been removed?
- Length of time since initial injury
- Tetanus status if lacerations or abrasions sustained
- Obtain significant medical history. Include documentation of disorders that may compromise healing and record previous hand injury or disability.
- Record medication and allergy history.
- Note other risk factors that may preclude adequate healing, such as tobacco or cocaine use.

Physical

- Physical exam is of vital importance in evaluating the injured hand. Develop a comprehensive routine for examining all hand injuries regardless of mechanism of injury.
- Hand structure
 - Five metacarpal bones are joined to wrist, articulating with the distal carpal row.
 - Thumb articulates chiefly with trapezium, creating a freely movable joint.
 - Remaining metacarpals articulate with trapezoid, capitate, and hamate from radial to ulnar direction.
 - Ring and little fingers have about 20-25° of mobility at articulation in the anteroposterior (AP) plane.
 - Index and middle fingers have no flexion or extension capability at articulation.
 - Thumb consists of proximal and distal phalanges.
 - Remaining fingers consist of proximal, middle, and distal phalanges.
 - Proximal interphalangeal (PIP) joints allow flexion and extension and minimal abduction and adduction.
- Description of function
 - Rotation of hand from neutral position to palm up position is termed supination. Rotation to palm down position is termed pronation.
 - Radial and ulnar deviation correspond to movement of hand to stated direction from anatomic position.
 - Extension of hand refers to dorsal movement and flexion refers to volar movement.
 - Flexion and extension of fingers correspond to dorsal and volar movements, as mentioned above.
 - Abduction of fingers refers to movement of fingers away from an imaginary line drawn through the middle of the third finger. Adduction refers to movement toward this midline.
 - Carpometacarpal joint of thumb is capable of palmar adduction or flexion (toward midline), palmar abduction (away from palmar surface), radial abduction, reposition (extension) adduction, and opposition. Interphalangeal joint of thumb can flex and extend only.
- Neurologic examination
 - Remember to assess nerve integrity prior to instillation of anesthetics.
 - The 3 major nerves of the hand are radial, median, and ulnar nerves.

Differentials

Dislocations, Hand

Dislocations, Wrist

Hand Infections

Hand Injuries, Soft-tissue

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Workup

Imaging Studies

- In ED, plain radiography is diagnostic test of choice to evaluate potential hand fractures.
- Standard radiographs include AP, lateral, and oblique views.
- Special imaging studies, such as MRI, CT, and bone scans, seldom are needed in ED to evaluate hand injuries.

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Treatment

Prehospital Care

- Prehospital care of most orthopedic injuries consists of splinting the hand in the position in which it was found, applying ice, and elevating the extremity, if possible.
- Obtain as much information about mechanism of injury and conditions at scene as possible.

Emergency Department Care

- ED care of hand fractures involves recognition of fracture, appropriate care, and referral. Primary concern is preservation of function.
- Fractures of the phalanges are the most common hand fractures. Fortunately, most are simple fractures and may be treated with padded aluminum splints or buddy taping.

- Distal phalanx fractures
 - The most common distal phalanx injury is comminuted tuft fracture. No angulation or displacement is usually present, because the septa hold fragments in place on the volar surface and the nail acts as a splint along the dorsal surface.
 - Subungual hematoma, a common complication, may be treated by trephination or nail removal and repair of nail bed. Treatment is controversial. Some authors advocate removal if hematoma comprises more than 50% of nail surface, while others recommend removal only if nail is disrupted, as long-term outcome does not improve with removal and repair of nailbed. Regardless of treatment, warn patient of potential nail deformity secondary to nail bed injury.
 - Antibiotics commonly are prescribed if nail is removed; trephination of subungual hematoma does not require antibiotic prophylaxis.
 - Treatment: Open injuries require thorough irrigation. These fractures usually are splinted with a padded aluminum splint extending from the volar proximal phalanx and curving around the fingertip to the proximal dorsal phalanx. This provides optimal protection.
- Transverse fracture of distal phalanx
 - These fractures usually are stable.
 - Transverse fractures may be splinted as above or in a circular cast.
 - If angulation persists after closed reduction, fracture may require surgical fixation with Kirschner wire.
 - Referral to a hand surgeon is necessary.
- Fracture of middle phalanx
 - Middle phalanx fractures have unpredictable stability after reduction.
 - These require splinting.
 - Some advocate buddy taping if the phalanx is stable. Buddy taping allows mobility, may prevent stiffness, and aids quicker return to baseline activity. Buddy taping is not appropriate for any displaced or rotated fracture.
 - Do not use in transverse fractures, as stability is unpredictable.
 - Perform follow-up x-ray in 7 days to assess stability.

- Oblique and spiral fractures

- These fractures frequently cause malrotation of the involved finger.
- Oblique and spiral fractures usually are unstable after reduction.
- These fractures require splinting with either ulnar or radial gutter splints extending out to involve the digit to the distal phalanx.

- Condylar fractures

- Condylar fractures may be noted only on oblique radiograph.
- These fractures usually require open fixation.

- Comminuted fractures of head of middle and proximal phalanges may be treated with closed reduction

and immobilization.

- Intraarticular fractures require orthopedic referral and often open reduction and fixation (ORIF).
 - Splint in the safe position.
 - Wrist should be extended 15-20° and MCP flexed about 70°.
 - Interphalangeal (IP) joints should be flexed 10-20° or the least amount needed to maintain reduction.

- Complications
 - These fractures may result in malrotation, degenerative arthritis, adhesion of tendon to bone (more common in open or widely angulated fractures), and joint stiffness from immobilization.
 - Boutonniere deformity (from rupture of extensor hood apparatus at PIP joint) may result from improperly treated middle phalanx fracture.
 - Flexor tendon rupture is rare.

- Metacarpal fractures
 - Divided below by location (head, shaft, neck, base) for discussion purposes.
 - Applying extension, abduction, and adduction forces to joints tests their integrity.
 - MCP collateral ligaments are taut in flexion and lax in extension, thus stability must be tested in multiple degrees of angulation.

- Metacarpal head fractures
 - These fractures often are severely comminuted and complicated by poor healing.
 - ED management includes splinting.
 - Immediate orthopedic referral is mandatory.
 - Complications include malrotation of finger, extensor tendon injury, posttraumatic arthritis, and avascular necrosis.

- Metacarpal neck fractures
 - These fractures usually occur as a result of a direct blow to the knuckles and are the most common type of metacarpal fracture.
 - Fracture at neck of fifth metacarpal is termed boxer's fracture.
 - Mechanism of injury results in angulation of distal segment toward palm.
 - ED physician must inspect for rotational deformity.
 - Attachments of metacarpals to carpals are different for each finger and require different approaches.
 - Metacarpals of middle and index fingers are fixed at the distal carpal row and do not allow

flexion or extension. Eliminate angulation at the fracture sites of these fingers. Patients cannot tolerate more than 10-15° angulation of these fractures. ED management includes closed reduction, gutter splint, and prompt orthopedic referral. Metacarpal neck fractures often require wire placement to ensure alignment.

- Metacarpals of ring and little fingers allow flexion and extension at carpal attachments. These patients can tolerate greater angulation at fracture without loss of function. Most authors allow 30-40° of angulation. In a satisfactory outcome, the fifth finger can extend to 180° without deformity.

- Metacarpal shaft fractures

- Metacarpal shaft fractures produce dorsal angulation and malrotation.
- Correct index and middle finger angulation. Ring and little fingers may tolerate minimal angulation. Little or no shortening of bones usually takes place, as transverse metacarpal ligaments hold fragments in place. Patient can tolerate 3 mm of shortening if no rotation or angulation is present.
- Treat by splinting for 4-6 weeks.
- Multiple fractures and those with shortening, angulation, or rotation require reduction and, usually, fixation.
- Complications: Malrotation weakens grip and causes pain on grasping. Tendon injury frequently occurs with these fractures, and MCP joint may become stiff if splinted improperly (ie, in extension).

- Metacarpal base fractures

- Intraarticular fractures at base of index and middle fingers are rare and, if present, usually of little clinical significance.
- They may be associated with other fractures.
- Fracture at base of fifth metacarpal is common and often associated with subluxation of metacarpal-hamate joint. Splint this fracture in a gutter splint and immediately refer patient to a hand surgeon.

- Bennett fracture

- Bennett fracture is an oblique, intraarticular fracture at the volar base of the ulnar aspect of the first metacarpal.
- Displacement of the larger fragment occurs from pull of the abductor pollicis longus muscle.
- Emergency department treatment consists of immobilization in a thumb spica splint and orthopedic referral, as this injury requires surgery.
- Complications include traumatic arthritis and malunion (may result in subluxation of metacarpal-trapezium joint).

- Rolando fracture

- This rare fracture is similar to the Bennett fracture, except that in addition to a small palmar fragment a large dorsal fragment creates a T- or Y-shaped fracture at the base of the metacarpal.
- More commonly, base of the metacarpal is severely comminuted.
- ED treatment is thumb spica splint.
- Requires immediate orthopedic follow-up care for ORIF.

Consultations

Hand surgeon

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Medication

Control pain with commonly prescribed medications. Acetaminophen with codeine or hydrocodone usually suffices.

Prescribe antibiotics for open fractures, usually a cephalosporin (ie, cefazolin sodium) with broad-spectrum coverage added for grossly contaminated wounds.

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures.

| | |
|-------------------|---|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tab q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|---|
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |
|-------------|---|

| | |
|-------------------|---|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

Antibiotics

Therapy must cover all likely pathogens in this clinical setting. Antibiotic combinations may be required for broad coverage in grossly contaminated wounds.

| | |
|-------------------|---|
| Drug Name | Cefazolin (Ancef, Kefzol, Zolicef)- First-generation, semisynthetic cephalosporin that, by binding to 1 or more penicillin-binding proteins, arrests bacterial cell wall synthesis and inhibits bacterial replication. Primarily active against skin flora, including <i>Staphylococcus aureus</i> . Typically used alone for skin and skin-structure coverage. Total daily dosages are same for IV/IM routes. |
| Adult Dose | 2 g IV/IM q6-12h depending on severity of infection; not to exceed 12 g/d |
| Pediatric Dose | 25-100 mg/kg/d IV/IM divided q6-8h depending on severity of infection; not to exceed 6 g/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid prolongs effects; aminoglycosides may increase renal toxicity; may yield false-positive urine dip test for glucose |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |

| | |
|-------------|---|
| Precautions | Adjust dose in renal impairment; superinfections and promotion of nonsusceptible organisms may occur with prolonged use or repeated therapy |
|-------------|---|

| | |
|-------------------|---|
| Drug Name | Gentamicin (Gentacidin, Garamycin)- Aminoglycoside antibiotic used for gram-negative bacterial coverage. Commonly used in combination with both an agent against gram-positive organisms and one that covers anaerobes. Used in conjunction with ampicillin or vancomycin for prophylaxis in patients with open fractures. |
| Adult Dose | 1.5 mg/kg IV; not to exceed 80 mg |
| Pediatric Dose | 2 mg/kg IV |
| Contraindications | Documented hypersensitivity; nondialysis-dependent renal insufficiency |
| Interactions | Other aminoglycosides, cephalosporins, penicillins, and amphotericin B may increase nephrotoxicity; aminoglycosides enhance effects of neuromuscular blocking agents, thus prolonged respiratory depression may occur; loop diuretics may increase auditory toxicity of aminoglycosides—possible irreversible hearing loss of varying degrees may occur (monitor regularly) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Narrow therapeutic index (not intended for long-term therapy); caution in renal failure (not on dialysis), myasthenia gravis, hypocalcemia, and conditions that depress neuromuscular transmission; adjust dose in renal impairment |

| | |
|-------------------|---|
| Drug Name | Vancomycin (Vancocin)- Potent antibiotic directed against gram-positive organisms and active against enterococcal species. Useful in treatment of septicemia and skin-structure infections. Used in conjunction with gentamicin for prophylaxis in penicillin-allergic patients with open fractures. May need to adjust dose in patients diagnosed with renal impairment. |
| Adult Dose | 1 g plus 1.5 mg/kg infused over 1 h |
| Pediatric Dose | 1.5 mg/kg infused over 1 h |
| Contraindications | Documented hypersensitivity |
| Interactions | Erythema, histamine-like flushing, and anaphylactic reactions may occur when administered with anesthetic agents; taken concurrently with aminoglycosides, risk of nephrotoxicity may increase above that with aminoglycoside monotherapy; effects in neuromuscular blockade may be enhanced when coadministered with nondepolarizing muscle relaxants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

Precautions

Caution in renal failure, neutropenia; red man syndrome is caused by too rapid IV infusion (dose given over a few minutes) but rarely happens when dose given over 2 h or by PO or IP route; red man syndrome is not an allergic reaction

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Follow-up

Further Inpatient Care

- Care for vast majority of patients with hand fractures as outpatients.
- Reserve inpatient care for those who must go directly to the operating room for ORIF. This is not a common occurrence.

Further Outpatient Care

- As most patients have splints applied in ED, discharge instructions should include signs and symptoms of constrictive splints or casts. Instruct patients of date and time of their follow-up appointment with an orthopedic surgeon or the phone number to call for an appointment.
- Instruct patient to elevate injured hand to reduce swelling and pain.

Transfer

- Transfer of patient with hand fracture seldom is required.
- An exception is the patient with an amputated digit or hand requiring transfer to a hospital capable of emergent reimplantation.

Complications

- Malrotation
- Degenerative arthritis
- Adhesion of tendon to bone (more likely in open or widely angulated fractures)
- Joint stiffness from immobilization
- Boutonniere deformity (may result from improperly treated middle phalanx fracture)
- Nonunion of fractures resulting in prolonged disability

Prognosis

- Prognosis is excellent with good ED management and appropriate, timely referral.
-

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Fractures, Hip

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Introduction

Background

Fracture of the hip can have devastating consequences. This is particularly true in older persons, who often suffer this calamity. Aside from considerable risks of morbidity and death, hip fracture causes loss of mobility and can significantly reduce the patient's quality of life. Athletes rarely return to their pre-morbid level of activity and ability following a hip fracture.

Pathophysiology

The healthy femur, with angular anatomy, is shown in [Picture 1](#). Hip fractures involve fracture of any aspect of proximal femur, from the head to the first 4-5 cm of the subtrochanteric area.

The hip joint is a large ball-and-socket synovial joint. It is enclosed in a thick, synovium-lined articular capsule that extends from the acetabulum to the intertrochanteric line anteriorly. Posteriorly it covers the proximal half to two thirds of the femoral neck. Contiguous with and reinforcing this capsule are 3 strong ligaments: iliofemoral (Y ligament of Bigelow), pubofemoral, and ischiofemoral. The joint is supported further by numerous large muscles that cross it.

Vascular supply

Vascular supply to the hip is tenuous. It consists of perforating branches of the medial and lateral circumflex femoral arteries, the inferior and superior gluteal arteries, and the posterior branch of the obturator artery. These branches form the 3 main vascular structures that supply the femoral neck and head: the vascular ring around the base of the neck, ascending cervical interosseous and intracapsular vessels that emanate either partially or entirely from the vascular ring, and vessels of the ligamentum teres. In most people, the foveal artery that enters the femoral head with the ligamentum teres is insufficient to nourish the entire head.

Fractures of the neck, especially displaced fractures, often lead to avascular necrosis of the femoral head from disruption of interosseous vascular channels and intracapsular vessels, which lie against the periosteum of the femoral neck.

Classifying fractures

Hip fracture classifications most often are based on their anatomic locations: head, neck, intertrochanteric, trochanteric, and subtrochanteric. Subclassification represents significant differences in emergent referral and treatment.

In classifying the fracture, note whether it is intracapsular or extracapsular, as this has a significant impact on healing. Femoral head and neck fractures are considered intracapsular, while trochanteric, intertrochanteric, and subtrochanteric fractures are considered extracapsular. Intracapsular hip fractures, like all other intracapsular fractures, frequently have complicated healing. Surrounded by the thick capsule, these fractures are separated from adjacent soft tissue and its abundant capillaries and have impaired callous formation. Thus, nonunion is an added complication of these fractures.

Femoral head fractures

These usually are associated with hip dislocations. Superior femoral head fractures normally are associated with anterior dislocations, while inferior femoral head fractures are associated with posterior dislocations.

- Type 1 - Single fragment fractures (see [Picture 2](#))
- Type 2 - Comminuted fractures (see [Picture 2](#))

Femoral neck fractures

- Type 1 - Stress fractures or incomplete fractures (see [Picture 3](#))
- Type 2 - Impacted fractures (see [Picture 3](#))
- Type 3 - Partially displaced fractures (see [Picture 4](#))
- Type 4 - Completely displaced or comminuted fractures (see [Picture 5](#))

Intertrochanteric fractures

- Type 1 - Single fracture line; no displacement; considered stable (see [Picture 6](#))
- Type 2 - Multiple fracture lines or comminution; displacement; unstable (see [Picture 6](#))

Trochanteric fractures

- Type 1 - Nondisplaced fractures (see [Picture 7](#))
- Type 2 - Displaced fracture; greater than 1 mm displacement for greater trochanteric fractures and greater than 2 mm displacement for lesser trochanteric fractures (see [Picture 7](#))

Subtrochanteric fractures

- Stable - Bony contact of medial and posterior femoral cortices
- Unstable

Frequency

- **In the US:** Hip fracture occurs in approximately 80 per 100,000 persons or approximately 250,000 persons each year. Incidence of hip fracture increases with age; with current population projections, the number of fractures per year is expected to double by 2040. US frequency, when age and sex are adjusted, is highest in the world.
- **Internationally:** Western Europe and New Zealand also have reported high rates. Lowest rates occur in South African Bantu people and in East Asian countries.

Mortality/Morbidity

- Reported overall mortality rate of hip fractures is 15-20%, yet in older persons that can increase to 36% over the year following hip fracture. Rate of mortality is greatest in the first few months following injury but remains high for up to 1 year. It then returns to the same rate for age- and sex-matched people without hip fracture.
- Morbidity associated with hip fracture is staggering, especially in older persons. Morbidity from immobilization includes development of deep vein thrombosis, pulmonary embolism, pneumonia, muscular atrophy, and associated rehabilitation problems. Morbidity from surgical procedures includes anesthetic morbidity, postoperative infection, loss of fixation, malunion or nonunion, and all possible complications associated with immobilization as outlined above.
- Hip fracture resulting from major trauma often is associated with other bone and soft-tissue injuries, intraabdominal and intrapelvic injuries, major blood loss, head and neck injuries, and other extremity injuries. Morbidity associated with an inability to return to a prefracture level of mobility results in a loss of independence, reduction in quality of life, and depression, particularly in older persons.

Race

Incidence of hip fracture is 2-3 times greater in whites than in nonwhites, primarily because of the increased rate of osteoporosis in whites.

Sex

- Rate of hip fracture is 2-3 times greater in women than in men. At least 75% of all hip fractures occur in women.
- Lifetime risks of hip fracture in white women and men are 15% and 5%, respectively.
- Femoral neck fractures are more frequent in women than men by about 4:1. Intertrochanteric fractures are more frequent in women than men by about 5:1.

Age

Rate of hip fracture increases with age; after age 50 years, it doubles with each decade. Nearly 50% of all hip fractures occur in adults older than 80 years. Hip fracture at a young age is not common and is usually the result of a major traumatic event or, rarely, is related to bone pathology.

- Femoral head fractures are more common in younger patients, because the same mechanism of injury is more likely to cause femoral neck fracture in older persons.
- Trochanteric fractures are uncommon and affect younger patients more often than older persons.
- Femoral neck fractures are rare in younger patients; the average age is 74-78 years.
- Intertrochanteric fractures also are rare in younger patients; the average age is 75-81 years.
- Subtrochanteric fractures have a bimodal age distribution and are seen most often in those aged 20-40 years in association with high-energy trauma and in patients older than 60 years.

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Clinical

History

- In older persons, hip fracture most often results from a simple fall; in a small percentage, it occurs spontaneously in the absence of a fall.
- Patient complains of pain and inability to move the hip.
- With stress fractures in young athletes and nondisplaced fractures, patient may complain of pain in hip or knee and may be ambulatory.

- Patient may have a history of other osteoporotic fractures, such as Colles or vertebral fractures.

Physical

- In patients who experienced trauma, perform a primary survey and stabilize as needed.
- Take a detailed secondary survey because of the high likelihood of other associated injuries. As many as 69% of patients with femoral head fracture-dislocations had major associated injuries, including other extremity injuries, intraabdominal or intrapelvic injuries, neck injuries, and head injuries.
- Pay particular attention to vital signs and secondary manifestations of shock such as changes in skin, mental status, and urine output. Hip fractures are associated with blood volume losses of up to 1500 cc.
- Inspect and palpate for deformity, hematoma formation, laceration, and asymmetry.
- Observe the natural position of the extremity, as this alone often indicates the type of injury the patient has sustained.
- Femoral head fracture: Most often, this occurs as a result of hip dislocation; therefore, the position of the extremity is abduction, external rotation, and flexion or extension for anterior dislocation. With posterior dislocation (most common type), the extremity is held in an adducted and internally rotated position.
- Femoral neck fracture: Extremity is held in a slightly shortened, abducted, and externally rotated position, unless the fracture is only a stress fracture or severely impacted. In this case, the hip is held in a natural position.
- Intertrochanteric fracture: Extremity is held in a markedly shortened and externally rotated position.
- Subtrochanteric fracture: Proximal femur usually is held in a flexed and externally rotated position.
- Trochanteric fracture
 - No deformities are noted on observation.
 - Apply lateral to medial pressure on hips through greater trochanters.
- Perform a detailed distal neurovascular exam.
- If patient is a trauma victim, assess for pelvic fracture by stressing pelvis anteriorly to posteriorly through iliac crests and symphysis pubis, and laterally to medially through iliac crests.

Causes

- In young persons, trauma associated with significant kinetic energy is required to cause a hip fracture. For example, 75% of all femoral head fractures, more common among young patients, occur as a result of motor vehicle accidents.
- In older persons, more than 90% of these fractures result from trauma or torsion associated with a minor fall. They also can occur in the absence of an obvious traumatic event.
- Osteoporosis greatly increases risk of fracture.
- Other risk factors for hip fracture include the following:
 - Dementia

- Cigarette smoking
- Institutional living
- Maternal history of hip fracture
- Previous hip fracture
- Previous Colles or vertebral fracture attributed to osteoporosis
- Physical inactivity
- Low body weight
- Tall stature
- Alcohol abuse
- Impaired vision
- Use of psychotropic medications and drugs that decrease bone mass, including furosemide, corticosteroids, thyroid hormone, phenobarbital, and phenytoin

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Differentials

Dislocations, Hip
Fractures, Pelvic

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Workup

Lab Studies

- Laboratory studies are not useful in diagnosis of fracture. Preoperative laboratory studies usually are drawn.

Imaging Studies

- Anteroposterior (AP) and lateral views demonstrate most fractures.
 - If a fracture is not obvious, look for alteration of the Shenton line and compare to the other hip. In addition, check neck-shaft angle, which is determined by measuring the angle created by lines drawn through the centers of the femoral shaft and femoral neck. This should be approximately 120-130°.
 - For patients in whom femoral neck fracture is strongly suspected but standard x-ray findings are negative, an AP view with hip internally rotated provides a better view of the

femoral neck.

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Treatment

Prehospital Care

- Prehospital treatment of a patient who complains of hip pain should include immobilization on a stretcher.
- If patient is a victim of multiple traumas, address ABCs and immobilize cervical spine as appropriate.
- If fracture or deformity of femur is obvious, apply traction splint and place an intravenous (IV) line for hydration.
- If patient is hypotensive or tachycardic, initiate crystalloid fluid bolus and place patient on supplemental oxygen.

Emergency Department Care

- If patient is a victim of trauma, attend to ABCs first and conduct a thorough search for other possible injuries.
- In cases of obvious femur fracture, immobilize patient, start an IV line to hydrate, restrict patient's oral intake to nothing by mouth (NPO), and obtain specimens for preoperative labs if necessary.
- Orthopedic treatment decisions vary significantly among different practitioners, thus early consultation for all hip fractures is recommended.
- Initiate appropriate parenteral analgesia as soon as possible.
- Femoral head fractures
 - Type 1: Reduce dislocated femoral head and fracture fragment as soon as possible to avoid avascular necrosis of fracture fragment. Early orthopedic consultation is a must. Small fracture fragments may need to be removed.
 - Type 2: Early orthopedic consultation for admission and arthroplasty is recommended.
- Intertrochanteric fractures
 - Apply traction or traction splint.
 - Note potential for significant blood loss. IV fluid resuscitation may be necessary.
 - Stable and unstable fractures usually are treated with open reduction and internal fixation unless patient is not an operative candidate for other reasons.
 - Early orthopedic consultation is recommended.
- Subtrochanteric fractures

- Significant hemorrhage is common, and IV fluid resuscitation is frequently necessary.
- ED application of traction or traction splint is necessary.
- Consult orthopedic surgeon for admission and open reduction with internal fixation for most patients.

Consultations

Orthopedic surgery

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Medication

Parenteral analgesia is strongly recommended. A muscle relaxant also may be necessary. Administer antibiotics to cover skin flora (ie, cefazolin sodium) and tetanus immunization, as necessary, in open fractures.

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures.

| | |
|-------------------|--|
| Drug Name | Morphine sulfate (Duramorph, Astramorph, MS Contin)- DOC for narcotic analgesia due to its reliable and predictable effects, safety, and ease of reversibility with naloxone. Morphine sulfate administered IV may be dosed in a number of ways and commonly is titrated until desired effect attained. |
| Adult Dose | Starting dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h Relatively hypovolemic patients: Start with 2 mg IV/IM/SC and reassess hemodynamic effects of dose |
| Pediatric Dose | Neonates: 0.05-0.2 mg/kg IV/IM/SC prn Children: 0.1-0.2 mg/kg IV/IM/SC q2-4h prn |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects |

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| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

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|-------------------|---|
| Drug Name | Fentanyl citrate (Duragesic, Sublimaze)- More potent narcotic analgesic than morphine sulfate with much shorter half-life. DOC for conscious sedation analgesia. Ideal for analgesic action of short duration during anesthesia (premedication, induction, maintenance), and in immediate postoperative period. With short duration (30-60 min) that is easy to titrate, excellent choice for pain management and sedation. Easily and quickly reversed by naloxone. After initial dose, do not titrate subsequent doses more frequently than q3h or q6h. When using transdermal dosage form, pain is controlled in most patients with 72-h dosing intervals. However, a small number of patients require dosing intervals of 48 h. |
| Adult Dose | 0.5-1 mcg/kg/dose IV/IM q30-60min Transdermal: Apply 25 mcg/h system q48-72h |
| Pediatric Dose | <2 years: 2-3 mcg/kg/dose IV/IM q30-60min 2-12 years: 1-2 mcg/kg/dose q60min >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hypotension, respiratory depression, constipation, nausea, emesis, and urinary retention; idiosyncratic reaction, known as chest wall rigidity syndrome, may require neuromuscular blockade to increase ventilation |

Antibiotics

Therapy must cover all likely pathogens in the context of the clinical setting.

| | |
|-------------------|--|
| Drug Name | Cefazolin (Ancef, Kefzol, Zolicef)- First-generation, semisynthetic cephalosporin that by binding to 1 or more penicillin-binding proteins arrests bacterial cell wall synthesis and inhibits bacterial replication. Primarily active against skin flora, including <i>Staphylococcus aureus</i> . Typically use alone for skin and skin-structure coverage. Total daily dosages are same for IV/IM routes. |
| Adult Dose | 2 g IV/IM q6-12h; not to exceed 12 g/d |
| Pediatric Dose | 25-100 mg/kg/d IV/IM divided q6-8h; not to exceed 6 g/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid prolongs effects; coadministration with aminoglycosides may increase renal toxicity; may yield false-positive urine dip test for glucose |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal impairment; superinfections and promotion of nonsusceptible organisms may occur with prolonged use or repeated therapy |

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| Drug Name | Gentamicin (Gentacidin, Garamycin)- Aminoglycoside antibiotic used for gram-negative bacterial coverage. Commonly used in combination with both an agent against gram-positive organisms and one that covers anaerobes. Used in conjunction with ampicillin or vancomycin for prophylaxis in patients with open fractures. |
| Adult Dose | 1.5 mg/kg IV; not to exceed 80 mg |
| Pediatric Dose | 2 mg/kg IV |
| Contraindications | Documented hypersensitivity; nondialysis-dependent renal insufficiency |
| Interactions | Other aminoglycosides, cephalosporins, penicillins, and amphotericin B may increase nephrotoxicity; aminoglycosides enhance effects of neuromuscular blocking agents, thus prolonged respiratory depression may occur; loop diuretics may increase auditory toxicity of aminoglycosides—possible irreversible hearing loss of varying degrees may occur (monitor regularly) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Narrow therapeutic index (not intended for long-term therapy); caution in renal failure (not on dialysis), myasthenia gravis, hypocalcemia, and conditions that depress neuromuscular transmission; adjust dose in renal impairment |

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|-----------|---|
| Drug Name | Ampicillin (Omnipen, Marcillin)- Used along with gentamicin for prophylaxis in patients with open fractures. Interferes with bacterial cell wall synthesis during active multiplication, causing bactericidal activity against susceptible organisms. Given in place of amoxicillin in patients unable to take medication orally. |
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| Adult Dose | 2 g IV/IM |
| Pediatric Dose | 50 mg/kg IV/IM |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid and disulfiram elevate levels; allopurinol decreases effects and has additive effects on ampicillin rash; may decrease effects of oral contraceptives |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal failure; evaluate rash and differentiate from hypersensitivity reaction |

| | |
|-------------------|--|
| Drug Name | <p>Vancomycin (Vancocin)- Potent antibiotic directed against gram-positive organisms and active against <i>Enterococcus</i> species. Also useful in treatment of septicemia and skin-structure infections.</p> <p>Used in conjunction with gentamicin for prophylaxis in penicillin-allergic patients with open fractures.</p> <p>May need to adjust dose in patients with renal impairment.</p> |
| Adult Dose | 1 g IV infused over 1 h |
| Pediatric Dose | 1.5 mg/kg IV infused over 1 h |
| Contraindications | Documented hypersensitivity |
| Interactions | Erythema, histaminelike flushing, and anaphylactic reactions may occur when administered with anesthetic agents; taken concurrently with aminoglycosides, risk of nephrotoxicity may increase above that with aminoglycoside monotherapy; effects in neuromuscular blockade may be enhanced when coadministered with nondepolarizing muscle relaxants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in renal failure, neutropenia; red man syndrome caused by too rapid IV infusion (dose given over a few minutes) but rarely happens when dose given over 2 h or by PO or IP route; red man syndrome not an allergic reaction |

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Follow-up

Further Inpatient Care

- Most patients should be admitted to the hospital under care of an orthopedic surgeon. If operative repair is planned, the patient should be cleared medically by his or her primary care physician or

internist.

- Patients with multiple medical problems can be admitted to the primary care service with orthopedic consultation.
- Patients who have sustained multiple traumas should be admitted to the trauma service or general/trauma surgeon.

Further Outpatient Care

- Few patients are eligible for discharge; those who are sent home usually require prolonged bed rest.
- Consultation with an orthopedist is imperative because of the variety of treatment options and preferences.

Deterrence/Prevention

- Avoid risk factors (see [Causes](#)) and falls in older persons. An older patient who presents after a fall should undergo a risk assessment to prevent further falls.
- Calcium supplementation and estrogen replacement therapy decrease risk of hip fracture.

Complications

- Infection
- Nonunion
- Gait disturbance

Prognosis

- Outcome of hip fractures varies considerably depending upon patient's age, premorbid level of conditioning, type of fracture, and numerous other factors.
- In general, young patients almost always regain ability to ambulate, yet depending on type of fracture sustained, probably will not return to their premorbid level of activity.
- Most older patients do not regain the ability to ambulate or are able to do so only with assistance. This profoundly affects their ability to live independently.
- Almost 20% of patients never regain the ability to ambulate, and a similar percentage are unable to ambulate outside their homes.
- Only 50-65% regain their premorbid ambulatory status.

Patient Education

- Prevention of hip fracture is vastly superior to current treatment modalities. Gear patient education toward identification of avoidable risk factors in the patient's life.

- In young persons, stress avoidance of tobacco and alcohol abuse and safe, responsible use of automobiles.
- Counsel older persons on ways to make their home environments safe from falls. Encourage them to consult with their primary physician regarding calcium and hormonal replacement therapies.

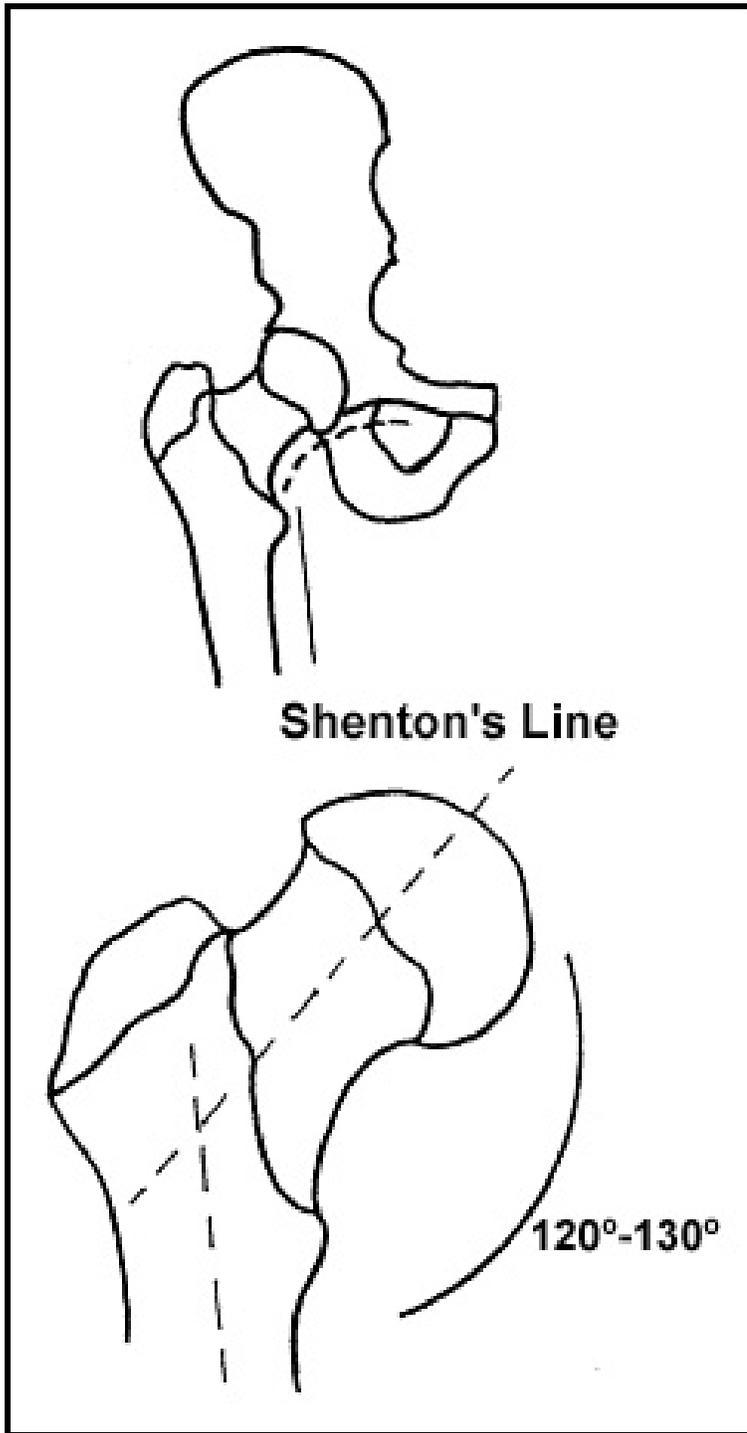
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Miscellaneous

Medical/Legal Pitfalls

- Failure to prevent patient with a stress or incomplete femoral neck fracture from ambulating, thus creating a complete or displaced fracture
 - Failure to consider diagnosis of stress fracture of the femoral neck in a young patient with chronic hip or knee pain
 - Failure to consider diagnosis of incomplete femoral neck fracture in an older patient with hip pain and nondiagnostic standard x-ray views. Consider an AP view with hip internally rotated, MRI, or bone scan.
-

Pictures

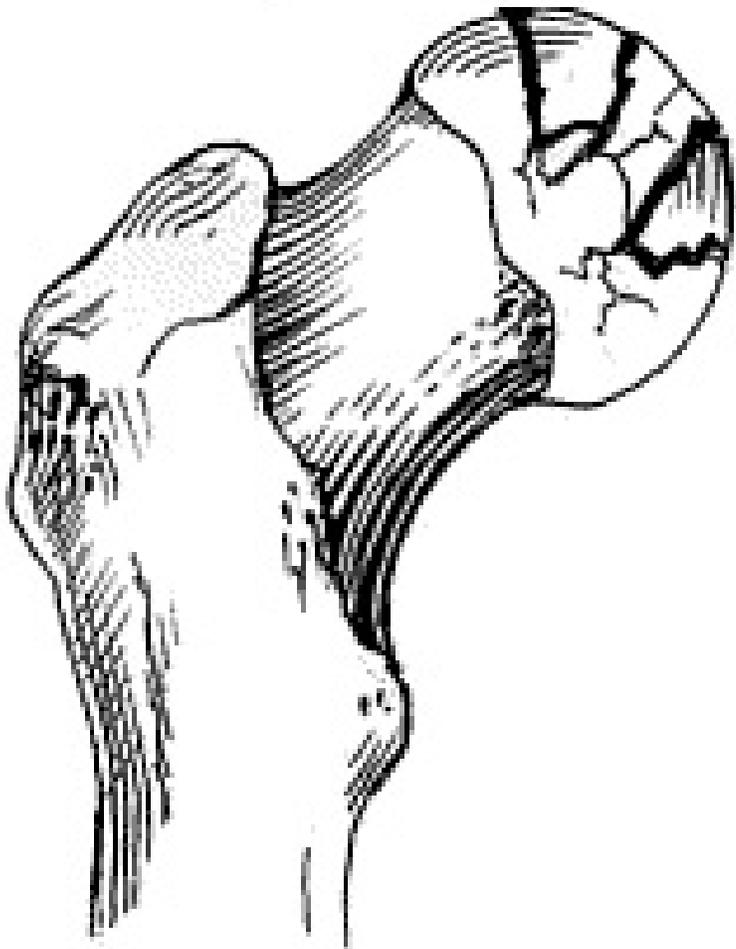
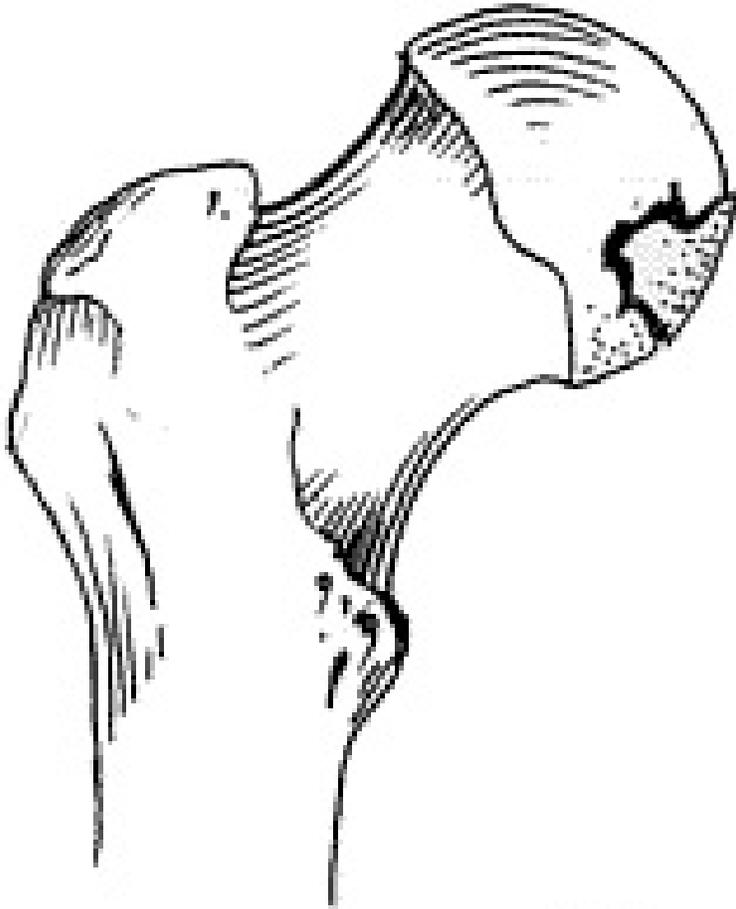


Shenton's Line

120°-130°

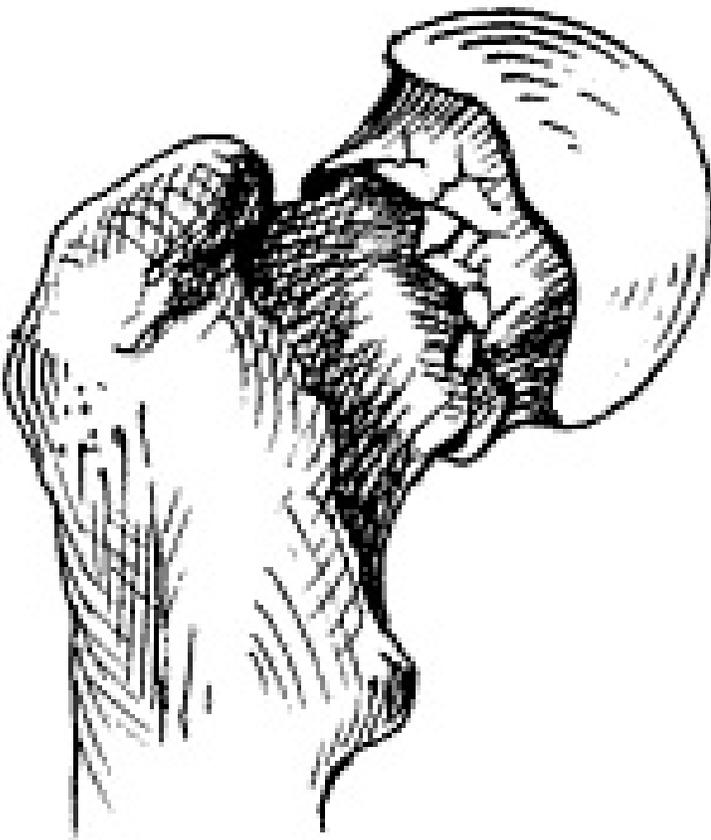
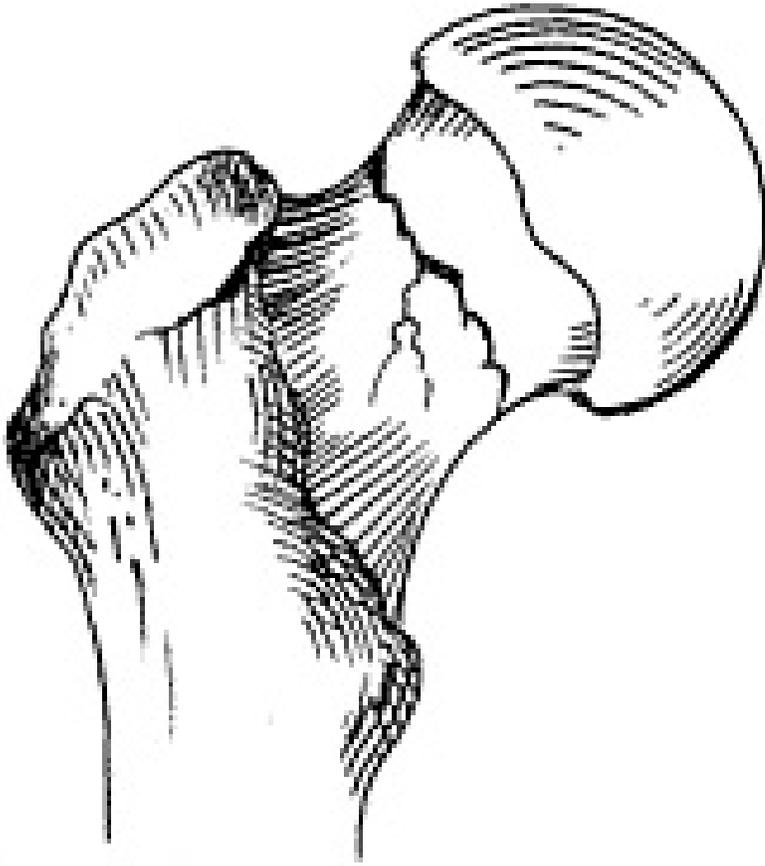
Picture 1: Shenton line and angular anatomy of the femur

Picture type: Photo



Picture 2: Femoral head fractures. Top diagram is a single-fragment femoral head fracture. Bottom diagram is a comminuted femoral head fracture.

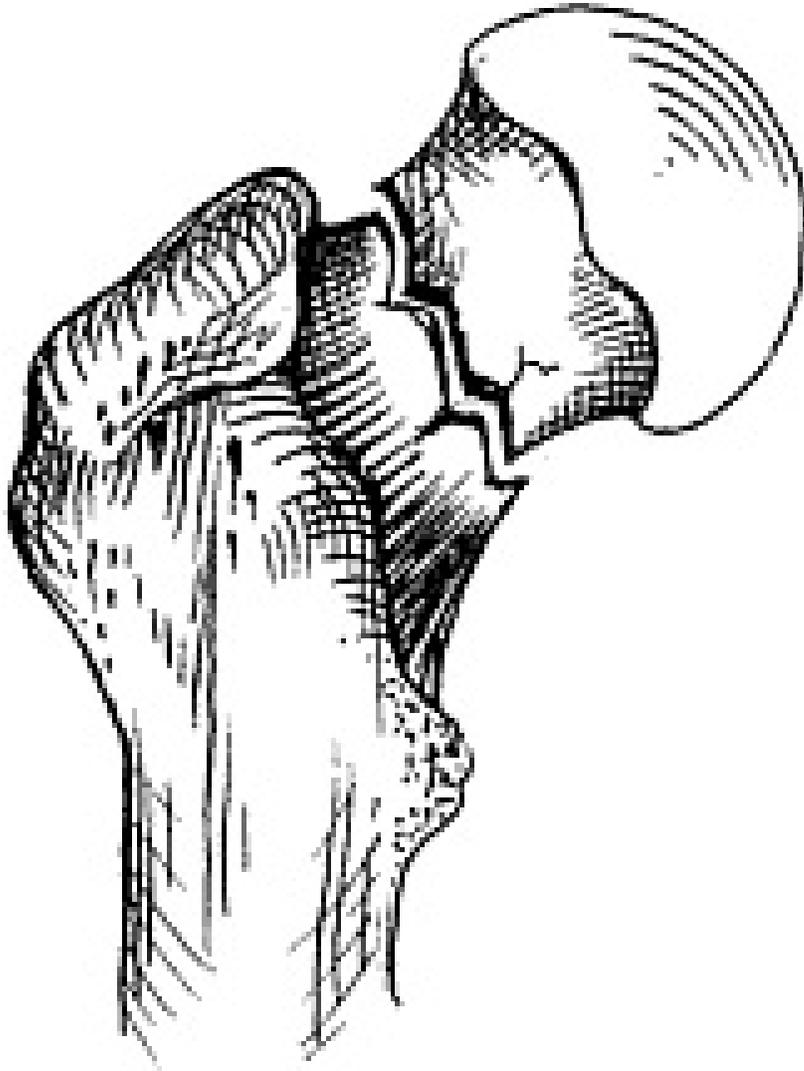
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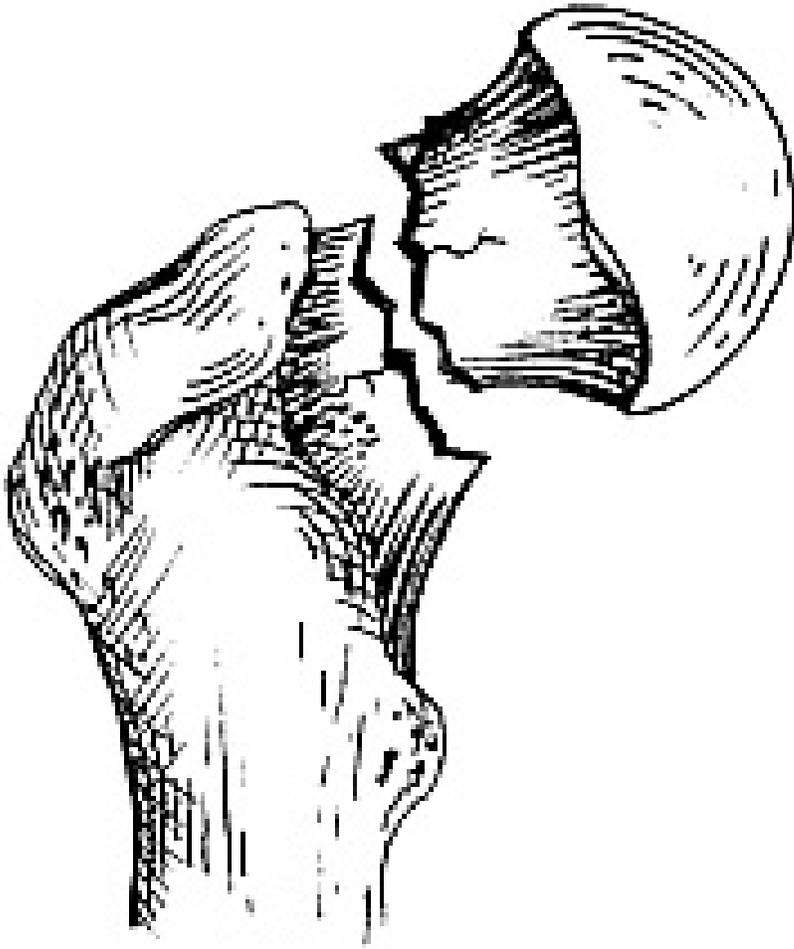
Picture 3: Femoral neck fractures. Top diagram is a nondisplaced, or incomplete, femoral neck fracture. Bottom diagram is an impacted femoral neck fracture.

Picture type: Photo

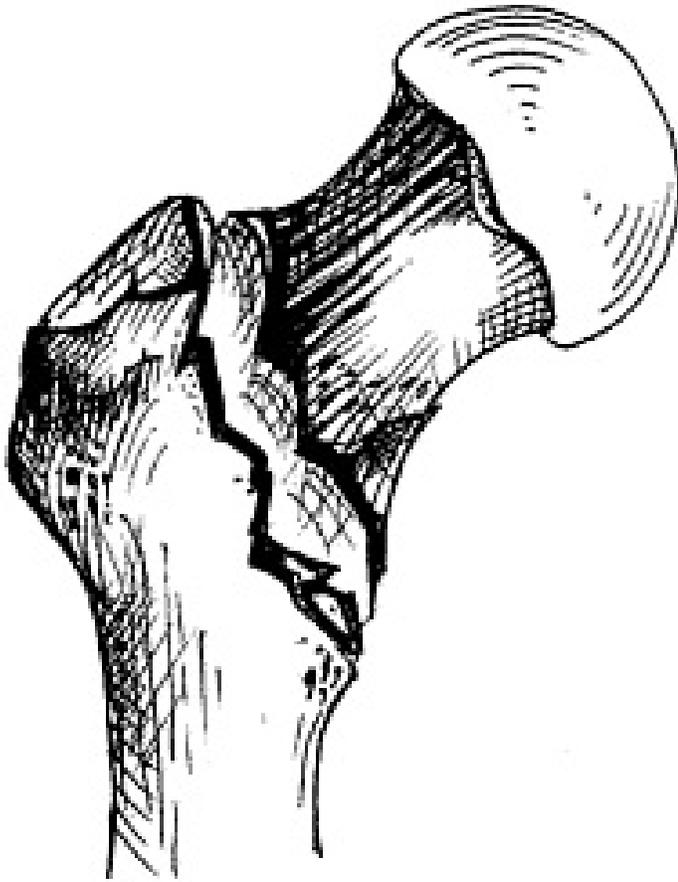
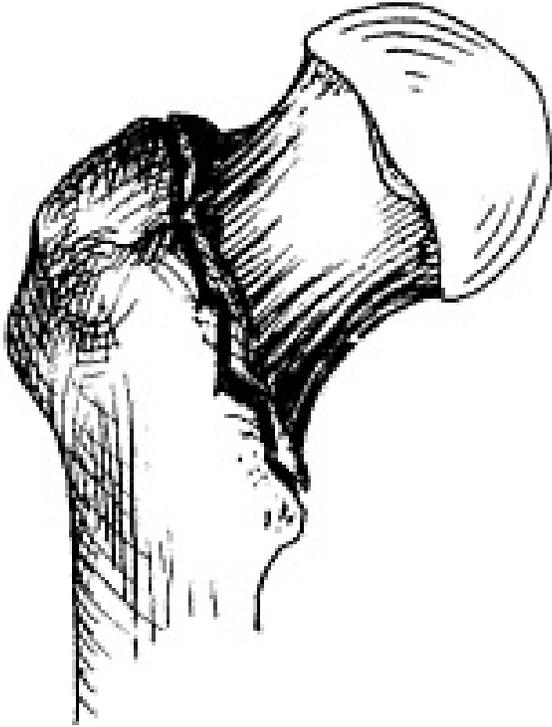


Picture 4: Partially displaced femoral neck fracture

Picture type: Photo

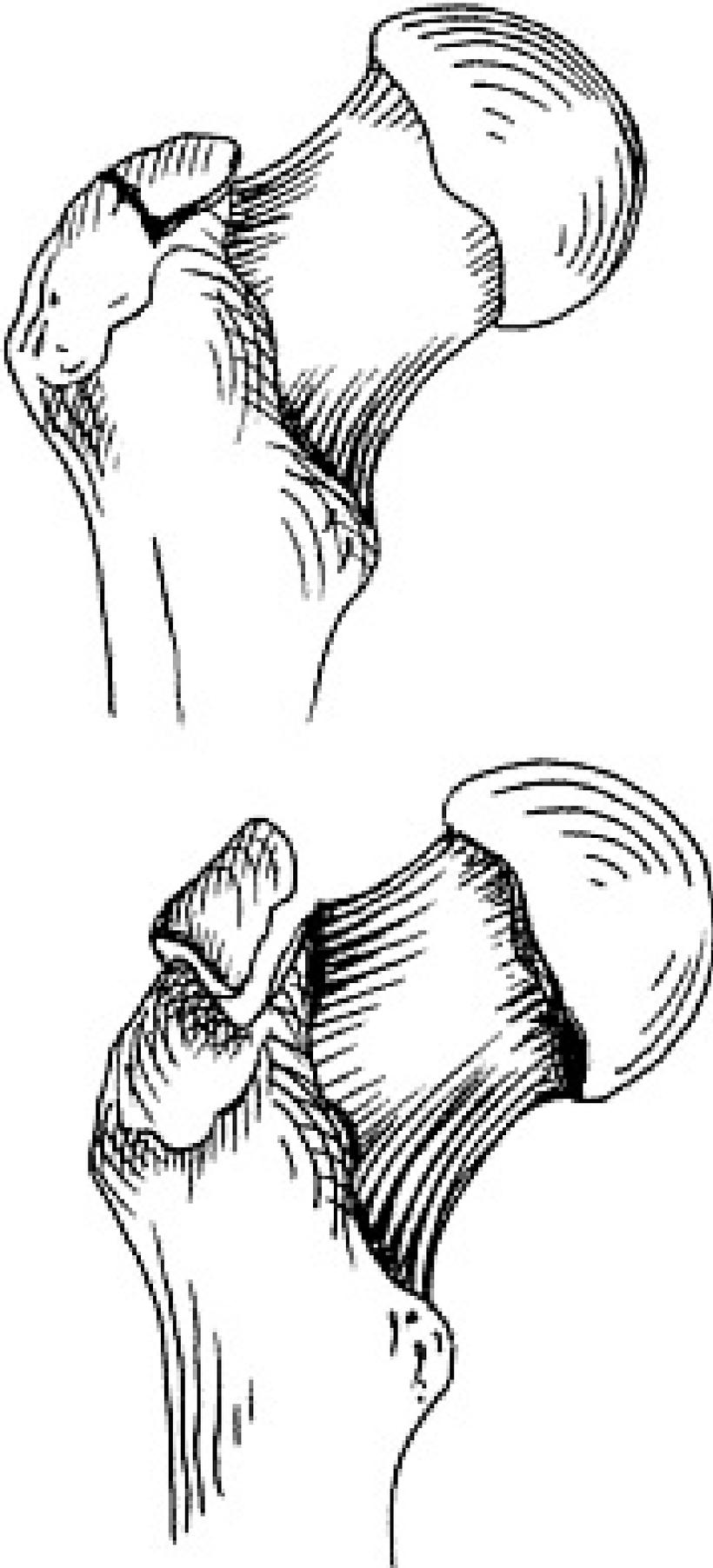


Picture 5: Completely displaced femoral neck fracture
Picture type: Photo



Picture 6: Intertrochanteric fractures. Top diagram is a single fracture line intertrochanteric fracture. Bottom diagram is a displaced, or multiple fracture line, intertrochanteric fracture.

Picture type: Photo



Picture 7: Trochanteric fractures. Top diagram is a nondisplaced trochanteric fracture. Bottom diagram is a displaced trochanteric fracture.

Picture type: Photo

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Fractures, Humerus

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Introduction

Background

Humerus fractures, particularly fractures of the proximal humerus, are encountered commonly in the ED. Rarely do these injuries represent surgical emergencies; however, the emergency physician must recognize which fractures require urgent, versus emergent, orthopedic referral.

Pathophysiology

Humerus fractures typically are caused by direct trauma to the arm or shoulder or axial loading transmitted through the elbow. Attachments from pectoralis major, deltoid, and rotator cuff muscles influence the degree of displacement of proximal humerus fractures.

Frequency

- **In the US:** Humerus fractures represent 4-5% of all fractures.

Age

Fracture patterns are similar across all ages, though older persons are more prone to fracture because of osteoporosis.

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Clinical

History

- Most patients convey blunt trauma to the arm or shoulder or axial loading through the elbow. Typical history involves a fall on an outstretched, abducted arm.
- Pathologic fractures of the humerus may occur with minimal trauma. Suspect these in patients with the following history:
 - Cancer metastatic to bone
 - Paget disease
 - Bone cyst
 - Pain
 - Edema
 - Decreased range of motion (ROM)

Physical

- Pain occurs with palpation or movement of shoulder or elbow.
- Ecchymosis and edema usually are present.
- Perform a careful neurovascular exam. Radial nerve injury following humerus shaft fractures is relatively common.

Causes

- A humerus fracture in a child with an inconsistent injury mechanism should raise suspicion for abuse and trigger a legal and social service investigation.
- Fractures that occur spontaneously, without apparent injury, suggest pathologic fracture. Causes include cancer metastatic to the bone, Paget disease, and bone cyst.

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Differentials

Dislocations, Shoulder
Fractures, Clavicle
Fractures, Elbow
Fractures, Scapular

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Workup

Imaging Studies

- Anteroposterior and lateral views of the humerus, as well as transthoracic and axillary views of the shoulder, should be adequate to visualize a fracture.
- Proximal humerus fracture
 - Proximal humerus has 4 parts: articulating surface, greater tuberosity, lesser tuberosity, and humeral shaft.
 - Neer classification system describes how many of these parts are fractured, displaced, and/or angulated (not just fractured).
 - Operative treatment decisions are based primarily on the number of segments involved and degree of displacement. Most fractures are displaced minimally and treated conservatively. Three- and 4-part fractures often need operative repair.

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Treatment

Prehospital Care

Immobilize the fracture.

Emergency Department Care

- Minimize patient movement and provide adequate analgesia to make patient comfortable in the ED.
- Proximal humerus fracture: Sling and swathe application is primary treatment in ED.
- Humerus shaft fracture

- It is best stabilized using a coaptation splint.
- Wrap splinting material snugly from axilla to nape of neck, creating a stirrup around elbow.
- Fracture reduction is unnecessary because maintaining a reduction is difficult once achieved.

Consultations

- Most isolated proximal and diaphyseal humeral fractures can be managed by an orthopedist in an outpatient setting. Even patients with fractures that may eventually require surgery generally may be discharged with early follow-up care if fracture is otherwise uncomplicated.
- Open fractures represent a surgical emergency; obtain an immediate orthopedic consult.
- Penetrating trauma requires particular neurovascular scrutiny.
- Glenohumeral dislocation in conjunction with a proximal humerus fracture requires orthopedic evaluation.
- Floating elbow (an ipsilateral humerus and forearm fracture) requires operative repair.

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Medication

Drugs used to treat fractures are generally NSAIDs, analgesics, and anxiolytics.

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

These agents are used most commonly for the relief of mild to moderately severe pain. Effects of NSAIDs in the treatment of pain tend to be patient specific, yet ibuprofen is usually DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

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|----------------|---|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually DOC for treatment of mild to moderately severe pain, if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, which inhibits prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: Administer as in adults |

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| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ €monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
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| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for relief of mild to moderately severe pain and inflammation. Administer small dosages initially to patients with small bodies, older persons, and those with renal or liver disease. Doses higher than 75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe patient for response. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 14 years: 0.1â€1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ €monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
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| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Relieves mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, which decreases prostaglandin synthesis. |
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| Adult Dose | 500 mg PO followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ €monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

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|-------------------|--|
| Drug Name | Flurbiprofen (Ansaid)- Has analgesic, antipyretic, and anti-inflammatory effects. May inhibit cyclooxygenase enzyme, inhibiting prostaglandin biosynthesis. |
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ €monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures.

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|-------------------|---|
| Drug Name | Acetaminophen (Tylenol, Panadol, aspirin-free Anacin)- DOC for treatment of pain in patients with documented hypersensitivity to aspirin or NSAIDs and in those with upper GI disease or taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; known G-6-P deficiency |
| Interactions | Rifampin can reduce analgesic effects; barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; APAP is contained in many OTC products and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

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| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tab q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

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| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |

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| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

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|-------------------|---|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in the elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

| | |
|----------------|--|
| Drug Name | Morphine sulfate (Duramorph, Astramorph, MS Contin)- DOC for narcotic analgesia because of its reliable and predictable effects, safety, and ease of reversibility with naloxone. Morphine sulfate administered IV may be dosed in a number of ways and commonly is titrated until desired effect obtained. |
| Adult Dose | Starting dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h Relatively hypovolemic patients: Start with 2 mg IV/IM/SC and reassess hemodynamic effects of dose |
| Pediatric Dose | Neonates: 0.05-0.2 mg/kg IV/IM/SC prn Children: 0.1-0.2 mg/kg dose q2-4h prn |

| | |
|-------------------|---|
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

Anxiolytics

Patients with painful injuries usually experience significant anxiety. Anxiolytics allow a smaller analgesic dose to achieve same effect.

| | |
|-------------------|---|
| Drug Name | Lorazepam (Ativan)- Sedative hypnotic in benzodiazepine class with short onset of effect and relatively long half-life. By increasing action of GABA, a major inhibitory neurotransmitter, may depress all levels of CNS, including limbic and reticular formation. Excellent for sedating patients for >24 h. Monitor patient's BP after administering dose and adjust as necessary. |
| Adult Dose | Initial dose: 2 mg total or 0.044 mg/kg IV, whichever is smaller For greater lack of recall: 0.05 mg/kg IV; not to exceed 4 mg/dose |
| Pediatric Dose | 0.05 - 0.1 mg/kg IV slowly over 2-5 min; may repeat dose of 0.05 mg/kg IV slowly |
| Contraindications | Documented hypersensitivity; preexisting CNS depression; hypotension; narrow-angle glaucoma |
| Interactions | Alcohol, phenothiazines, barbiturates, and MAOIs increase toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in renal or hepatic impairment, myasthenia gravis, organic brain syndrome, or Parkinson disease |

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Follow-up

Further Inpatient Care

- Open fractures
 - These require extensive irrigation.
 - Administer prophylactic antibiotics, such as cephalexin and gentamicin.

Further Outpatient Care

- Proximal humerus fracture
 - Displaced 3- or 4-part fractures frequently require surgical fixation.
 - Perform open reduction and internal fixation in young patients.
 - Perform humeral arthroplasty in older patients.
 - For nonsurgical fractures, continue sling for comfort and institute early ROM exercises.
 - Schedule initial follow-up visit within 1 week.

in/Out Patient Meds

- As with all fractures, provide adequate outpatient analgesia especially during first few days. Narcotic analgesia may be appropriate.

Complications

- Radial nerve injury
 - Nerve transection is rare.
 - Transient neuropraxia from stretching is more common.
 - Complete return to function is the norm.

Prognosis

- Proximal humeral fractures
 - Complete union is expected at 6-8 weeks.
 - Older patients often exhibit functional decrease in shoulder ROM.

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Miscellaneous

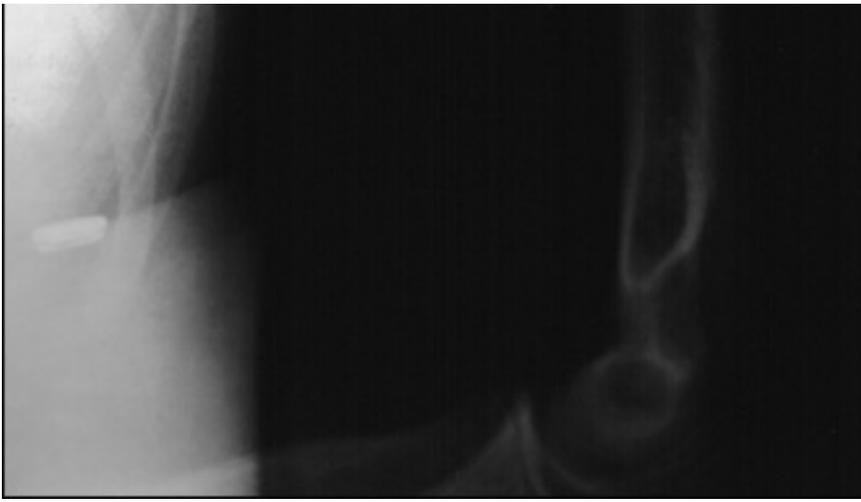
Medical/Legal Pitfalls

- Failure to assess and document radial nerve function in humerus shaft fracture

- Failure to recognize a glenohumeral dislocation associated with a proximal humerus fracture. This may increase risk of avascular necrosis of humeral head.
-

Pictures





Picture 1: Diaphyseal humerus fracture

Picture type: X-RAY

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Fractures, Knee

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Introduction

Background

Fractures of the knee include fractures of the patella, femoral condyles, tibial eminence, tibial tuberosity, and tibial plateau. Direct and indirect forces can cause these fractures.

Frequency

- **In the US:** Patellar and tibial plateau fractures each account for 1% of all skeletal fractures. Distal femoral condyle fractures account for 4% of all femur fractures.

Mortality/Morbidity

- Fractures of the knee can result in neurovascular compromise or compartment syndrome, with resultant risk of limb loss. Soft-tissue infection or osteomyelitis can occur with open fractures.
- Other postfracture complications that can result in morbidity or mortality include fat embolism, thrombophlebitis, delayed union, nonunion, and osteoarthritis.

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Clinical

History

Patients with knee fractures may have a history of the following:

- Direct or indirect trauma with resultant pain and edema
- Patella injury: Caused by a direct blow, such as a collision with a dashboard in a motor vehicle accident or a fall on a flexed knee or forceful quadriceps contraction, such as with stumbling or falling. Ambulation may be possible with nondisplaced fractures.
- Femoral condyle injury due to axial loading with valgus or varus stress
- Tibial spine fracture due to a direct blow to the proximal tibia with the knee flexed or in rotation or hyperextension with varus or valgus stress, such as in motor vehicle collisions or athletic accidents
- Tibial eminence (tubercle) fracture
 - Caused by a force to a flexed knee with the quadriceps contracted
 - More common in males than in females
 - More common in adolescents, infrequent in adults
- Caused by axial loading with valgus or varus forces, such as in a fall from a height or collision with the bumper of a car
- Patient generally unable to ambulate

Physical

Ask the patient to perform a straight leg raise against gravity to check the integrity of the extensor mechanism, which commonly is disrupted with transverse patellar fractures caused by indirect forces.

Joint stability may be present. As many as one third of tibial plateau fractures are associated with knee ligamentous injuries (medial collateral or anterior cruciate ligaments with lateral plateau fractures, lateral collateral or posterior cruciate ligaments with medial plateau fractures). Instability also is frequently present with tibial spine and/or intercondylar eminence fractures.

Findings at physical examination may include the following:

- Neurovascular status
- Edema
- Open wounds
- Ecchymosis
- Deformity

- Limb shortening or rotation
- Point tenderness
- Crepitus
- Effusion

Causes

Knee fractures may be caused by the following:

- Trauma (direct or indirect)
- Chronic stress
- Pathologic conditions

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Differentials

Dislocations, Knee

Fractures, Femur

Fractures, Tibia and Fibula

Osgood-Schlatter Disease

Trauma, Peripheral Vascular Injuries

Other Problems to be Considered

Bipartite patella (superolateral corner, smooth cortical margins, generally bilateral)

Stress fractures

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Workup

Imaging Studies

- Radiographs

- Obtain anteroposterior, lateral, and oblique radiographs of the knee.
- The fat-fluid level (lipohemarthrosis) may be identified on a lateral view of the knee; this finding indicates an intraarticular fracture.
- Oblique views are particularly useful in detecting subtle tibial plateau fractures (internal oblique profiles lateral plateau, external oblique profiles medial plateau). The lateral tibial plateau is fractured more commonly than is the medial plateau. A tunnel or intercondylar view provides a clearer view of the intercondylar region.
- Additionally, a sunrise (ie, skyline, axial, tangential) view of the patella is useful for detecting vertical patellar fractures, which frequently are missed and nondisplaced. Transverse fractures are most common, followed by comminuted and avulsion-fractures.
- Radiographic evidence of ligamentous injury may be present.
- An avulsion fracture at the site of attachment of the lateral capsular ligament on the lateral tibial condyle (Segond fracture) is a marker for anterior cruciate ligament rupture.
- Cortical avulsion fracture of medial tibial plateau (uncommon) is associated with tears of the posterior cruciate ligament and medial meniscus.
- Use of the Ottawa rules for obtaining knee radiographs have proven sensitive for fracture and have reduced ED waiting times and costs. The rules include the following patient findings:
 - Age 55 years or older
 - Tenderness at head of fibula
 - Isolated tenderness of patella
 - Inability to flex knee to 90°
 - Inability to bear weight (4 steps) immediately after injury and in ED

Procedures

- Arthrocentesis may be of diagnostic and therapeutic benefit for tense effusions. Presence of blood and glistening fat globules indicates lipohemarthrosis, which is pathognomonic for intraarticular knee fracture.

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Treatment

Prehospital Care

- Document the neurovascular status.
- Apply a sterile dressing to open wounds.
- Splint the injury.

- Administer parenteral analgesics for isolated extremity injury.

Emergency Department Care

Care for various fractures is as follows:

- Patellar fracture
 - Nondisplaced transverse fractures with an intact extensor mechanism are treated with a knee immobilizer, crutches, restriction to only partial weight bearing, and 6 weeks of immobilization.
 - Displaced fractures, or fractures associated with a disrupted extensor mechanism, are referred to orthopedics for possible open reduction and internal fixation.
 - Refer patients with open fractures to orthopedics for emergent irrigation and debridement.
- Tibial spine fracture
 - For a nondisplaced fracture, immobilize the knee with a stabilized knee joint.
 - Obtain an orthopedic consultation for an unstable knee, a complete avulsion of the tibial spine, or a displaced fracture for possible surgical fixation.
- Tibial plateau fracture
 - Immobilize nondisplaced fractures.
 - Obtain an orthopedic consultation for displaced (depressed) fractures, which require open reduction and internal fixation.
 - The goal of treatment is a stable, aligned, mobile, and painless knee joint to minimize risk of posttraumatic osteoarthritis.

Consultations

- Orthopedic referral is recommended for all knee fractures.
- Nondisplaced fractures may be splinted, with orthopedic follow-up care within a few days.
- Displaced or open fractures require prompt orthopedic consultation.

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Medication

Opioid analgesics and nonsteroidal anti-inflammatory agents are the DOCs for pain associated with fractures.

Nonsteroidal Anti-Inflammatory Drugs (NsAIDs)

Most commonly used for relief of mild to moderate pain. Effects of NSAIDs in the treatment of pain tend to be patient specific, yet ibuprofen usually is the DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually DOC for treatment of mild to moderate pain, if no contraindications exist; inhibits inflammatory reactions and pain, probably by decreasing cyclooxygenase activity, which results in prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderate pain; inhibits inflammatory reactions and pain by decreasing cyclooxygenase activity, which decreases prostaglandin synthesis. |
| Adult Dose | 500 mg PO followed by 250 mg PO q6-8h; not to exceed 1.25 g/d; may increase to 1.5 g/d for limited periods |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |

| | |
|-------------|---|
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion may be at risk for acute renal failure; leukopenia occurs rarely and is transient, and condition usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation |

| | |
|-------------------|---|
| Drug Name | Flurbiprofen (Ansaid)- Has analgesic, antipyretic, and anti-inflammatory effects; may inhibit cyclooxygenase, inhibiting prostaglandin biosynthesis that may result in analgesic and anti-inflammatory activities. |
| Adult Dose | 200-300 mg/d PO bid/qid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion, risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

| | |
|-------------------|---|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for relief of mild to moderate pain and inflammation. Administer small dosages initially to smaller patients, older persons, and those with renal or liver disease. Doses >75 mg do not increase its therapeutic effects. Administer high doses with caution and closely observe patient for response. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 12 years: 0.1-1 mg/kg PO q6-8h prn >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |

| | |
|--------------|--|
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures.

| | |
|-------------------|---|
| Drug Name | Acetaminophen (Tylenol, Panadol, Aspirin Free Anacin)- DOC for treatment of pain in patients with documented hypersensitivity to aspirin or NSAIDs and in those with upper GI disease or those taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg PO tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg PO q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; known G-6-P deficiency |
| Interactions | Rifampin can reduce analgesic effects; coadministration with barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in persons with chronic alcoholism, with various dose levels; severe or recurrent pain or high or continued fever may indicate serious illness; acetaminophen is in many OTC products, and combined use with these products may result in cumulative doses exceeding the recommended maximum dose |

| | |
|------------|--|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderate pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tab PO q4h; not to exceed 12 tab/d |

| | |
|-------------------|---|
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose PO based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | Toxicity increases with use of CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for the relief of moderate to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose based on acetaminophen content PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen PO q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Coadministration with phenothiazines may decrease analgesic effects; toxicity increases with CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite, which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|--|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderate to severe pain; DOC for aspirin-sensitive patients. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose based on oxycodone content PO; not to exceed 5 mg/dose of oxycodone PO q4-6h prn |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; toxicity increases with coadministration of either CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|--|
| Precautions | Duration of action may increase in elderly patients; be aware of total daily dose of acetaminophen; do not exceed 4,000 mg/d of acetaminophen; higher doses may cause liver toxicity |
|-------------|--|

| | |
|-------------------|---|
| Drug Name | Oxycodone and aspirin (Percodan)- Drug combination indicated for relief of moderate to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO; not to exceed 5 mg/dose of oxycodone PO q4-6h prn |
| Contraindications | Documented hypersensitivity; liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma; <16 y with flu (because of association of aspirin with Reye syndrome) |
| Interactions | Phenothiazines may decrease analgesic effects; conversely, toxicity increases when administered with CNS depressants or tricyclic antidepressants; may potentiate anticoagulant effects of warfarin |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Duration of action may increase in elderly patients; caution in renal or liver impairment, peptic ulcer disease, and erosive gastritis |

| | |
|-------------------|---|
| Drug Name | Morphine Sulfate (Duramorph, Astramorph, MS Contin)- DOC for narcotic analgesia due to its reliable and predictable effects, safety, and ease of reversibility with naloxone; IV doses vary and commonly are titrated until desired effect is obtained. |
| Adult Dose | Starting dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h Relatively hypovolemic patients: Start with 2 mg IV/IM/SC, and reassess hemodynamic effects of dose |
| Pediatric Dose | Infants and children: 0.1-0.2 mg/kg/dose IV/IM/SC q2-4h prn; not to exceed 15 mg/dose; may initiate at 0.05 mg/kg/dose |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway where rapid establishment of airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects of opiate agonists; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

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Follow-up

Transfer

- Transfer is appropriate if inpatient beds or operating rooms are unavailable or if an orthopedic specialist's services are indicated.

Complications

- Complications of knee fracture may include the following:
- Neurovascular injury
 - Popliteal artery injury due to displaced distal femur or tibial plateau fractures
 - Peroneal nerve injury due to proximal fibula fractures
- Soft-tissue infection or osteomyelitis secondary to an open fracture
- Delayed union or nonunion
- Fat embolism
- Thrombophlebitis
- Posttraumatic arthritis or knee stiffness
- Chondromalacia patella

Prognosis

- A good prognosis is expected with patellar and tibial spine or tubercle fractures.
- A fair prognosis is expected with tibial plateau and femoral condyle fractures.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to identify neurovascular injury
- Failure to identify open fractures and obtain urgent orthopedic consultation

- Failure to check the integrity of and to identify injuries to extensor mechanism of the knee
-

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Fractures, Mandible

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Introduction

Background

Hippocrates described an array of facial injuries as long ago as 400 BCE. In 1823, von Graeffe described the use of an elastic tube placed in the nares to maintain an open airway. During the early 20th century, Sir Harold Gilles, father of plastic surgery, taught army personnel about breathing problems in patients with facial injuries. He recommended positioning them supine to maintain an airway. Frenchman René Le Fort studied cadavers in 1901 and described 3 basic types of facial fractures. Endotracheal anesthesia and radiography, developed during the First World War, led to a better understanding and treatment of facial fractures. During the Second World War, a multidisciplinary approach to treatment of facial fractures continued to improve outcomes of severely injured soldiers. Advent of CT-guided reconstruction, along with new surgical techniques, has improved the final appearance of patients with bony injuries immensely.

Pathophysiology

Maxillofacial fractures are the result of blunt or penetrating trauma. Most are blunt injuries caused by vehicular crashes, altercations, sporting-related trauma, occupational injuries, and falls. Penetrating injuries are mainly the result of gunshot wounds, stabbings, and explosions.

Determinants of type of injury (ie, soft tissue alone vs bony) are shape and velocity of the striking object.

The amount of force needed to fracture different bones of the face has been studied, and these bones have been divided into those that require high impact to fracture (greater than 50 times the force of gravity [g]) and those that require only low impact to fracture (less than 50 g).

- High impact
 - Supraorbital rim - 200 g
 - Symphysis of the mandible - 100 g
 - Frontal-glabella - 100 g
 - Angle of mandible - 70 g

Mandibular fractures usually occur in 2 or more locations because of the bone's U shape and articulations at the temporomandibular joints. Fractures also may occur at a site apart from the site of direct trauma. A large percentage of mandibular fractures are open, as they often fracture between teeth and communicate with the oral cavity.

Different mechanisms are associated with varying locations. Fractures from automobile crashes most frequently occur at the condyle and symphysis, those from motorcycle accidents at the symphysis and alveolus, and those from altercations mostly at the condyles, angles, and body.

Frequency

- **In the US:** The mandible is the third most fractured bone of the face. Of these fractures, approximately 20-35% are at the condyle and ramus, 20-30% at the angle, 15-30% at the body, 8-20% at the symphysis, and 1-5% at the alveolar ridge.
- One study placed the incidence of severe maxillofacial injury (fractures, lacerations) at 0.04-0.09% for motor vehicle crashes. Incidence of fractures due to motor vehicle injuries is higher in rural areas; altercation-related injuries are more frequent in inner cities.

Mortality/Morbidity

- Incidence of other major injuries is as high as 50% in high-impact mandibular fractures, whereas it is 21% in low-impact fractures. Mortality rate in high-impact fractures is as high as 12%, yet death rarely results directly from maxillofacial injury.
- Patients who are involved in motor vehicle crashes are more likely to have additional injuries than patients with violence-related injuries.
- Incidence of associated cervical spine injuries ranges from 0.2-6%.

Sex

Adult male-to-female ratio is 3:1.

Age

Male predominance is reduced to 3:2 in children.

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Clinical

History

- Since maxillofacial fractures are the result of trauma, primary survey and attention to airway, breathing, and circulation takes priority.
- Focus primary evaluation on patency of airway, control of cervical spine, breathing and circulatory impairment, and loss of consciousness if patient is experiencing neurologic impairment.
- Once life threats are addressed, obtain a thorough history.
 - Allergies
 - Medications
 - Medical history
 - Last meal
 - Events leading to injury

Physical

- Complete exam of the face is necessary, since multiple injuries can occur easily. Portions of the exam specific to the mandible are marked with an asterisk (*).
 - Inspect face for asymmetry, performed while looking down from head of bed.
 - Inspect open wounds for foreign bodies and palpate for bony injury.
 - Palpate bony structures of supraorbital ridge and frontal bone for step-off fracture.
 - Thoroughly examine eyes for injury, abnormal ocular movements, and visual acuity.
 - Inspect nares for telecanthus and widening of nasal bridge, then palpate for tenderness and crepitus.
 - Inspect nasal septum for septal hematoma and clear rhinorrhea, which may suggest cerebrospinal fluid (CSF) leak.
 - Palpate zygoma along its arch as well as along its articulations with the frontal bone, temporal bone, and maxillae.
 - Check facial stability by grasping teeth and hard palate and gently pushing back and forth then up and down, feeling for movement or instability of midface.
 - *Test teeth for stability and inspect for bleeding at gumline, a sign of fracture through the

alveolar bone.

- *Check teeth for malocclusion and step-off.
- *Palpate mandible for tenderness, swelling, and step-off along its symphysis, body, angle, and coronoid process anterior to the ear canal.
- *Check for localized edema or ecchymosis in the floor of the mouth.
- Evaluate distributions of the supraorbital, infraorbital, *inferior alveolar, and *mental nerves for anesthesia.
- *If teeth are missing, account for them to ensure they have not been aspirated.
- *Inspect area just anterior to the meatus of the ear for ecchymosis and palpate for tenderness. This is the condyle of the mandible and site of an often-missed fracture. Plain radiographs are not good at visualizing the condyle, thus maintain a high level of suspicion if physical exam is suggestive.
- *Mandibular fracture is suggested by inability to open mouth, trismus, malocclusion of teeth, or palpable step-offs of bone along symphysis, angles, or body. Gingival bleeding at the base of a tooth suggests fracture, especially if teeth are malaligned. Edema or ecchymosis may be present in the floor of the mouth. Neurologic findings may include hypesthesia in distribution of inferior alveolar or mental nerves.

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Differentials

Dislocations, Mandible
Foreign Bodies, Trachea
Fractures, Face
Neck Trauma
Pediatrics, Child Abuse

Other Problems to be Considered

Airway obstruction

Aspiration of avulsed teeth

Other major traumatic injuries

Dentate, avulsed

Dentate, displaced

Dentate, fractures

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Workup

Lab Studies

- Direct laboratory studies toward workup of a trauma patient. If this is an isolated injury, laboratory studies may not be required.
- If fracture is an isolated injury, obtain preoperative labs if surgery is planned.

Imaging Studies

- Radiographs
 - Best plain film to assess the mandible is a panorama view (ie, Panorex), which shows the mandible in its entirety in a single view. Panoramic view is not always available, as it requires a special radiographic machine. If panorama view is not available or patient is unable to sit for film, obtain routine mandible films.
 - Routine views include bilateral lateral oblique projections to look at the angle, body, and to a lesser extent, symphysis, and Townes view to look at the condyles.
 - Submental view can be helpful in evaluating the symphysis.
 - Obtain chest films of patients with unaccounted missing teeth to rule out aspiration.
 - Cervical spine radiographs may be indicated with severe facial injuries or in patients with a consistent mechanism and neck pain.

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Treatment

Prehospital Care

- Airway, breathing, and circulation are the first priority. Hold airway open by jaw thrust or airway adjuncts, including endotracheal intubation.
- Treat hypoventilation with intubation and bag ventilation. Nasotracheal intubation is considered a relative contraindication with severe maxillofacial trauma because of concern for intracranial

placement of endotracheal tubes.

- Suction usually is needed to keep airway free of blood and debris.
- Place patient on a backboard with a collar if cervical spine injury is a possibility.
- Control actively bleeding wounds by applying direct pressure with a bandage.

Emergency Department Care

- Airway, breathing, and circulation
 - Frequently assess airway. Isolated mandible fracture from a blunt mechanism usually does not require intubation, but frequent suctioning is mandatory.
 - Early intubation before swelling occurs makes airway control much easier, rather than waiting until a problem arises from obstruction. This is usually a clinical decision based on projected course.
 - Do not focus on obvious deformity, thereby forgetting to perform a complete primary survey. Rapidly diagnose other life threats and undertake appropriate resuscitation.

Consultations

- Provide care for the multiple-injured patient in conjunction with a surgeon who has experience in trauma care.
- Definitive treatment of mandibular fractures is performed by an oral-maxillofacial surgeon or an ENT specialist.

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Medication

When airway control is needed, rapid sequence induction often is the preferred method. Rapid sequence induction utilizes medications to induce unconsciousness and muscle paralysis to facilitate intubation. Cricothyroidotomy kit should be at the bedside in case problems arise.

Medication for pain control is appropriate, including NSAIDs, narcotics, and local anesthetics.

Patients with open fractures, which are the majority, should be given IV antibiotics. Current choices are penicillin or a cephalosporin. In penicillin-allergic patients, clindamycin is a good alternative. If the patient has an open wound, administer tetanus toxoid if the patient is not current.

Nonsteroidal Anti-Inflammatory Drugs (NsAIDs)

These agents are used most commonly for relief of mild to moderately severe pain. Effects of NSAIDs in treatment of pain tend to be patient specific, yet ibuprofen is usually the DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually DOC for treatment of mild to moderately severe pain, if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, which inhibits prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- For relief of mild to moderately severe pain and inflammation. Administer small dosages initially to patients with small bodies, older persons, and those with renal or liver disease. Doses higher than 75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe patient for response. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 12 years: 0.1â€1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |

| | |
|--------------|--|
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, which decreases prostaglandin synthesis. |
| Adult Dose | 500 mg PO followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

| | |
|----------------|---|
| Drug Name | Flurbiprofen (Ansaid)- Has analgesic, antipyretic, and anti-inflammatory effects. May inhibit cyclooxygenase enzyme, decreasing prostaglandin biosynthesis. |
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |

| | |
|-------------------|--|
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures.

| | |
|-------------------|---|
| Drug Name | Acetaminophen (Tylenol, Panadol, aspirin-free Anacin)- DOC for treatment of pain in patients with documented hypersensitivity to aspirin or NSAIDs or in those with upper GI disease or taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg q4h; not to exceed 5 doses q24h |
| Contraindications | Documented hypersensitivity; known G-6-P deficiency |
| Interactions | Rifampin can reduce analgesic effects; barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; APAP is contained in many OTC products and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

| | |
|-------------------|---|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tabs q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |

| | |
|--------------|---|
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in the elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

| | |
|-------------------|---|
| Drug Name | Morphine sulfate (Duramorph, Astramorph, MS Contin)- DOC for narcotic analgesia because of its reliable and predictable effects, safety, and ease of reversibility with naloxone. Administered IV, may be dosed in a number of ways and commonly is titrated until desired effect obtained. |
| Adult Dose | Starting dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h Relatively hypovolemic patients: Start with 2 mg IV/IM/SC and reassess hemodynamic effects of dose |
| Pediatric Dose | Neonates: 0.05-0.2 mg/kg IV/IM/SC prn Children: 0.1-0.2 mg/kg q2-4h prn |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

Antibiotics

Prophylaxis is given to patients with open fractures. Therapy must cover all likely pathogens in the context of the clinical setting.

| | |
|-------------------|--|
| Drug Name | Penicillin G (Pfizerpen)- Interferes with synthesis of cell wall mucopeptide during active replication, resulting in bactericidal activity against susceptible microorganisms. |
| Adult Dose | 2.4 million U IM as single dose in 2 injection sites |
| Pediatric Dose | 50,000 U/kg IM |
| Contraindications | Documented hypersensitivity |

| | |
|--------------|---|
| Interactions | Probenecid can increase effects; tetracyclines can decrease effects |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Caution in impaired renal function |

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|-------------------|---|
| Drug Name | Ceftriaxone (Rocephin)- Third-generation cephalosporin that has broad-spectrum activity against gram-negative organisms, lower efficacy against gram-positive organisms, and higher efficacy against resistant organisms. By binding to one or more penicillin-binding proteins, arrests bacterial cell wall synthesis and inhibits bacterial growth. |
| Adult Dose | 1-2 g IV qd/bid for 5-7 d; not to exceed 4 g/d |
| Pediatric Dose | >7 days: 25-50 mg/kg/d IV; not to exceed 125 mg/d Infants and children: 50-75 mg/kg/d IV q12h; not to exceed 2 g/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid may increase levels; ethacrynic acid, furosemide, and aminoglycosides may increase nephrotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal impairment; caution in breastfeeding women and allergy to penicillin |

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|-------------------|--|
| Drug Name | Clindamycin (Cleocin)- Lincosamide useful as a treatment against serious skin and soft-tissue infections caused by most staphylococci strains. Also effective against aerobic and anaerobic streptococci, except enterococci. Inhibits bacterial protein synthesis by inhibiting peptide chain initiation at bacterial ribosome, where it preferentially binds to 50S ribosomal subunit, inhibiting bacterial replication. |
| Adult Dose | 600-1200 mg/d IV/IM q6-8h depending on degree of infection |
| Pediatric Dose | 20-40 mg/kg/d IV/IM tid/qid |
| Contraindications | Documented hypersensitivity; regional enteritis; ulcerative colitis; hepatic impairment; antibiotic-associated colitis |
| Interactions | Increases duration of neuromuscular blockade induced by tubocurarine and pancuronium; erythromycin may antagonize effects; antidiarrheals may delay absorption |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in severe hepatic dysfunction; no adjustment necessary in renal insufficiency; associated with severe and possibly fatal colitis |

Tetanus Toxoid

This agent is used for tetanus immunization. Booster injection in previously immunized individuals is recommended to prevent this potentially lethal syndrome.

| | |
|-------------------|---|
| Drug Name | <p>Tetanus toxoid- Used to induce active immunity against tetanus in selected patients. Tetanus and diphtheria toxoids are immunizing DOC for most adults and children >7 y. Necessary to administer booster doses to maintain tetanus immunity throughout life.</p> <p>Pregnant patients should receive only tetanus toxoid, not diphtheria antigen-containing product.</p> <p>In children and adults, may administer into deltoid or midlateral thigh muscles. In infants, preferred site of administration is midhigh laterally.</p> |
| Adult Dose | <p>Primary immunization: 0.5 mL IM, give 2 injections 4-8 wk apart and third dose 6-12 mo after second injection</p> <p>Booster dose: 0.5 mL q10y</p> |
| Pediatric Dose | Administer as in adults |
| Contraindications | <p>Documented hypersensitivity; history of any type of neurological symptoms or signs following administration of this product</p> <p>FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are an important cause of provocative poliomyelitis</p> |
| Interactions | <p>Patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization due to poor immune response; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent use of medication with systemic chloramphenicol since it may impair anesthetic response to tetanus toxoid; concurrent use of tetanus immune globulin may delay development of active immunity by several days (interaction is nevertheless clinically insignificant and does not preclude its concurrent use)</p> |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | <p>Do not use to treat actual tetanus infections, or for immediate prophylaxis of unimmunized individuals (use instead tetanus antitoxin, preferably human tetanus immune globulin) diminished antibody response to active immunization may be seen in patients receiving immunosuppressive therapy; better to defer primary diphtheria immunization until immunosuppressive therapy discontinued; routine immunization of symptomatic and asymptomatic HIV-infected persons recommended</p> |

Immunoglobulins

Patients who may not have been immunized against *Clostridium tetani* products should receive tetanus

immune globulin.

| | |
|-------------------|---|
| Drug Name | Tetanus immune globulin (Hyper-Tet)- Used for passive immunization of any patient with a wound that may be contaminated with tetanus spores. |
| Adult Dose | Prophylaxis: 250-500 U IM in opposite extremity to tetanus toxoid Clinical tetanus: 3,000-10,000 U IM |
| Pediatric Dose | Prophylaxis: 250 U IM in opposite extremity to tetanus toxoid Clinical tetanus: 3,000-10,000 U IM |
| Contraindications | Since antibodies in globulin preparation may interfere with immune response to vaccination, do not administer within 3 mo of live virus immune globulin administration; may be necessary to revaccinate persons who received immune globulin shortly after live virus vaccination |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Persons with isolated IgA deficiency have potential for developing antibodies to IgA and could have anaphylactic reactions to subsequent administration of blood products that contain IgA; do not perform skin testing since intradermal injection of concentrated gamma globulin may cause localized area of inflammation and can be misinterpreted, causing the medication to be withheld from a patient not allergic to this material; true allergic responses to human gamma globulin given in prescribed IM manner are extremely rare; do not admix with other medications since usually incompatible |

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Follow-up

Further Inpatient Care

- Fractures of the mandible can be stable (favorable) or unstable (unfavorable) depending on how the fracture line courses in the bone. Muscles attached to the mandible continue to exert their forces. Elevators of the mandible are the masseter, temporalis, and medial pterygoid, while depressors and retractors are the mylohyoid, geniohyoid, and anterior belly of the digastric. Lateral pterygoid is the protruder of the mandible.
- Direction of fracture determines whether it is stable or unstable. Fractures running from posterior downward to anterior (favorable) generally are stable, because muscles pull the fragments together and can be treated with soft diet and arch wires if fragments are not aligned.
- Fractures of the body of the mandible running from anterior to posterior in a downward direction

(unfavorable) usually are displaced and can be stabilized with wire bar fixation of upper and lower teeth. Unstable fractures may require open reduction and internal fixation if they are not reduced by wire fixation or if they are markedly unstable.

- An edentulous mandible usually is unfavorable, because the patient has no teeth to stabilize the fracture. A stable nondisplaced fracture in an edentulous patient may be splinted with his or her denture and the patient restricted to a diet of soft food. An unstable fracture usually requires internal fixation to maintain reduction.
- All open fractures and unstable fractures require admission. Depending on institution, some patients with stable fractures that require arch band fixation are treated and released from ED, while others are treated on an inpatient basis.

Further Outpatient Care

- Place patient on a diet of soft or pureed food.
- Instruct patient to return if any signs of infection are noted.
- If arch wires are in place, instruct patient on release of interwire bands and give proper tools. Inability to release bands can be fatal if the patient vomits or has an airway problem.

in/Out Patient Meds

- Medications such as NSAIDs, acetaminophen, and a short course of narcotics can be used for pain control.
- Liquid preparations of medications are preferable.

Transfer

- If appropriate specialists are not available in the receiving institution, arrange transfer to a higher-level hospital.

Deterrence/Prevention

- Use of seat belts and airbags can reduce incidence of facial injuries in motor vehicle crashes.
- Use of helmet with facial guards can reduce injury in motorcycle accidents and accidents in such sports as skiing, snowboarding, hockey, and football.

Complications

- Loss of airway
- Aspiration of avulsed teeth
- Infection
- Nonunion

- Malnutrition and weight loss if teeth are banded together
- Injury to inferior alveolar or, more distally, mental nerve

Prognosis

- Prognosis is generally favorable with proper treatment.

Patient Education

- Instruct patient on how to release Erich arch wire if he or she has problems with airway.
- Place patient on a diet of soft or pureed food.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to diagnose associated intracranial or cervical spine injuries because focus was on obvious injury
- Failure to account for missing teeth, which may have been aspirated

Special Concerns

- Always consider potential for loss of airway and intrathoracic, intraabdominal, and intracranial injuries.
-

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Fractures, Orbital

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Introduction

Background

Orbit is composed of 6 facial bones.

- Superior orbital ridge and upper medial orbital ridge are part of the frontal bone.
- Lateral orbital rim is part of the zygoma.
- Inferior and lower medial rims are part of the maxilla. Floor of the orbit is made of the upper border of the maxillary sinus.
- Medial rim separating orbit from nares is the lacrimal bone.
- Medial wall and part of the posterior wall of the orbit are formed by the ethmoid bone.
- Most of the posterior of the orbit is formed by the 2 wings of the sphenoid bone.

Optic nerve exits the optic foramen in the lesser wing of the sphenoid bone. Globe of the eye sits within the orbit surrounded with periorbital fat and the extraocular muscles that control its movement. Inferior orbital nerve courses through the maxilla in the orbital floor. Weakest portion of the orbit is the thin orbital floor (maxilla) and the lamina papyracea (ethmoid bone) medially and inferiorly.

Pathophysiology

Blow-out fractures occur when a blow to the eye increases pressure in the orbit, causing the weak floor or lamina papyracea to "blow out" into the maxillary sinus or ethmoid bone. This results in a fracture, though it often prevents globe rupture and loss of the eye. Periorbital fat and extraocular muscles can become entrapped in the fracture, leading to problems of ocular movement. When the medial wall (lamina papyracea) is fractured, the medial rectus becomes entrapped, leading to lateral gaze dysfunction.

In maxillary fracture, the orbit floor blows out and inferior rectus entrapment leads to problems in upward gaze. The eye can be injured during compression before the ethmoid bone or the maxillary sinus fractures. About one third of blow-out fractures have an associated eye injury. Superior orbital rim fracture is a frontal bone fracture that is associated with high-impact injuries to the brain, face, and cervical spine. Tripod fractures and zygomaticomaxillary complex fractures occur from high-impact injury to the cheek's malar eminence (zygoma). Often these fractures are associated with eye and inferior orbital nerve injuries.

Mortality/Morbidity

The principal morbidity associated with orbital fractures is eye injury. Associated injuries include corneal abrasion, lens dislocation, iris disruption, choroid tear, scleral tear, ciliary body tear or bruise, retinal detachment and tear, hyphema, ocular muscle entrapment, and globe rupture.

Sex

Males are at higher risk of eye injuries because of their increased incidence of trauma.

Age

For all eye injuries, age distribution has 2 peaks: 10-40 years and older than 70 years.

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Clinical

History

- Since orbital fractures are the result of trauma, primary survey and attention to ABCs take priority. Focus questions on patency of airway, control of cervical spine, breathing difficulties, and symptoms of shock or neurologic impairment such as loss of consciousness.
- Once life threats have been addressed, obtain a thorough history.
 - Allergies

- Medications
- Medical history
- Last meal
- Events leading to injury
- Ask questions specific to the eye.
 - Does patient have diplopia, especially on lateral and upward gaze, indicating possible entrapment or lens dislocation?
 - Does patient have pain with eye motion indicating possible entrapment or periorbital edema?
 - Does patient have photophobia (iritis)?
 - Has patient experienced flashes of light (retinal detachment)?
 - Does patient have blurred vision (hyphema, retinal detachment, vitreous hemorrhage)?

Physical

- Perform a complete exam of the face. An asterisk (*) designates portions of the exam that are involved specifically with orbital fracture or associated eye injuries.
- Inspect the face for asymmetry while looking down from the head of the bed. From this position it is easiest to see enophthalmos (sunken eye) or proptosis (protruding eye).*
- Examine lids for lacerations. If present, consider the possibility of globe penetration.*
- Palpate bony structures of the supraorbital ridge and frontal bone for step-off fractures.*
- Examine ocular movements, especially in upward and lateral gaze, and test for diplopia.*
- Check visual acuity.*
- Check cornea, using fluorescein if needed, for abrasion (uptake of dye) or lacerations (streaming of fluid in dye).*
- Check pupils for roundness and reactivity, both direct and consensual.*
- Examine anterior chamber for presence of blood (flaring on slit-lamp exam) or hyphema (blood layering in inferior aspect of anterior chamber).*
- Examine limbus for signs of laceration (teardrop sign) or deformity.*
- Perform a funduscopic exam to check for blood in the posterior chamber, and examine retina for signs of detachment.*
- Inspect nares for telecanthus (widening of the nasal bridge), then palpate for tenderness and crepitus.
- Inspect nasal septum for clear rhinorrhea, indicating cerebrospinal fluid (CSF) leak, and for septal hematoma.
- Check facial stability by grasping the teeth and hard palate and gently pushing horizontally then vertically, feeling for movement or instability of midface.
- Test teeth for stability and inspect for bleeding at the gum line, a sign of fracture through the alveolar bone.
- Check teeth for malocclusion and step-off.
- Palpate mandible along its symphysis, body, angle, and coronoid process (anterior to ear canal) to check for tenderness, swelling, and step-off.
- Evaluate supraorbital, infraorbital*, inferior alveolar, and mental nerve distributions for

anesthesia.

- Palpate zygoma along its arch, as well as its articulations with the frontal bone, temporal bone, and maxillae.

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Differentials

Retinal Detachment

Other Problems to be Considered

Choroid tear

Ciliary body tear or bruise

Hyphema

Iris disruption

Lens dislocation

Ocular muscle entrapment

Scleral tear

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Workup

Lab Studies

- Direct lab studies toward workup of trauma patient.

Imaging Studies

- Radiographs
 - Obtain routine facial views, including Waters, Caldwell, and lateral projections.
 - Waters view best displays inferior orbital rims, nasoethmoidal bones, and maxillary sinuses. If patient is upright when film is taken, an air-fluid level often can be seen in the maxillary sinus, which may indicate fracture of the maxillary sinus (orbital floor).
 - If patient is immobilized on a backboard when film is taken, blood layers in the posterior of the sinus, making it appear clouded. Another sign of orbital blow-out fracture is the teardrop sign, an opacification in the upper maxillary sinus, which represents periorbital fat and possibly an entrapped extraocular muscle in the maxillary sinus.
 - Caldwell projection provides the best view of the lateral orbital rim and ethmoid bone.
 - Lateral views are the least helpful, but if patient is lying supine on the backboard, they may show air-fluid levels in the posterior of the maxillary sinus.
 - Cervical spine radiographs may be indicated in patients with severe facial injuries or with a consistent mechanism and/or neck pain.

Other Tests

- Perform slit-lamp exam of the eye to exclude eye injury.

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Treatment

Prehospital Care

- Airway, breathing, and circulation are the first priorities. Hold the airway open by jaw thrust or airway adjuncts, including endotracheal intubation. Because of the concern with intracranial placement of endotracheal tubes, severe facial injury is considered a relative contraindication to using the nasotracheal route of intubation.
- Place patient on a backboard with a collar if cervical spine injury is a possibility.
- Treat hypoventilation with intubation and bag ventilation.
- Control actively bleeding wounds by applying direct pressure with a bandage.
- If globe is open, cover it with a protective shield.

Emergency Department Care

- Airway, breathing, and circulation are the first priorities. Reassess airway frequently. Intubation performed early on, before swelling occurs, makes airway control much easier than waiting until a

problem arises from obstruction.

- Do not focus on the obvious deformity, thereby neglecting to perform a complete primary survey. Rapidly diagnose other life threats and undertake appropriate resuscitation.
- Diagnosis of orbital fracture in the ED is part of the secondary survey. Diagnose other injuries to the eye as well by performing a complete slit-lamp examination of the eye and tests for visual acuity.

Consultations

- Depending on the institution, orbital fractures are cared for by an eye, ear, nose, throat (EENT) surgeon, oromaxillofacial surgeon, ophthalmologist, or plastic surgeon.
- Patients with serious eye injuries and decreased visual acuity should have an ophthalmology consultation. Monitor minor injuries, such as corneal abrasions, on an outpatient basis.
- Provide care for the patient with multiple injuries in conjunction with a surgeon with experience in trauma care.

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Medication

When airway control is needed, facilitate intubation using drugs for rapid sequence induction. A cricothyrotomy kit should be at the bedside in case problems arise.

Medication for pain control is appropriate, including NSAIDs, narcotics, and local anesthetics.

Complete exam of the eye may require dilation of the pupil using mydriatic solutions.

Administer tetanus toxoid for open wounds if patient is not current on vaccinations.

Nonsteroidal Anti-Inflammatory Drugs (NsAIDs)

These agents are used most commonly for relief of mild to moderately severe pain. Effects of NSAIDs in the treatment of pain tend to be patient specific, yet ibuprofen is usually DOC for the initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually DOC for treatment of mild to moderately severe pain, if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, which inhibits prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ €monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for relief of mild to moderately severe pain and inflammation. Administer small dosages initially to patients with small bodies, older persons, and those with renal or liver disease. Doses higher than 75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 12 years: 0.1â€1 mg/kg PO q6-8h >12 years: 25-50 mg q6-8h prn; not to exceed 300 mg/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ €monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |

| | |
|-------------|---|
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |
|-------------|---|

| | |
|-------------------|--|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, which decreases prostaglandin synthesis. |
| Adult Dose | 500 mg PO followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

| | |
|-------------------|--|
| Drug Name | Flurbiprofen (Ansaid, Ocufer)- Has analgesic, antipyretic, and anti-inflammatory effects. May inhibit cyclooxygenase enzyme, inhibiting prostaglandin biosynthesis. |
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |

| | |
|-------------|--|
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures.

| | |
|-------------------|---|
| Drug Name | Acetaminophen (Tylenol, Panadol, aspirin-free Anacin)- DOC for treatment of pain in patients with documented hypersensitivity to aspirin or NSAIDs or those with upper GI disease or taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; known G-6-P deficiency |
| Interactions | Rifampin can reduce analgesic effects; barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; APAP is contained in many OTC products and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

| | |
|-------------------|---|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tabs q4h; not to exceed 12 tabs/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants increase toxicity |

| | |
|-------------|---|
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in the elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

| | |
|-------------------|---|
| Drug Name | Morphine sulfate (Duramorph, Astramorph, MS Contin)- DOC for narcotic analgesia due to its reliable and predictable effects, safety, and ease of reversibility with naloxone. Administered IV, may be dosed in a number of ways and commonly is titrated until desired effect obtained. |
| Adult Dose | Starting dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h Relatively hypovolemic patients: Start with 2 mg IV/IM/SC and reassess hemodynamic effects of dose |
| Pediatric Dose | Neonates: 0.05-0.2 mg/kg IV/IM/SC prn Children: 0.1-0.2 mg/kg q2-4h prn |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

Tetanus Toxoid

This agent is used for tetanus immunization. Booster injection in previously immunized individuals is recommended to prevent this potentially lethal syndrome.

| | |
|----------------|--|
| Drug Name | Tetanus toxoid- Used to induce active immunity against tetanus in selected patients. Tetanus and diphtheria toxoids are immunizing DOC for most adults and children >7 y. Necessary to administer booster doses to maintain tetanus immunity throughout life. Pregnant patients should receive only tetanus toxoid, not a diphtheria antigen-containing product. In children and adults, may administer into deltoid or midlateral thigh muscles. In infants, preferred site of administration is midhigh lateral. |
| Adult Dose | Primary immunization: 0.5 mL IM Give 2 injections 4-8 wk apart and third dose 6-12 mo after second injection Booster dose: 0.5 mL q10y |
| Pediatric Dose | Primary immunization: 0.5 mL IM Give 2 injections 4-8 wk apart and third dose 6-12 mo after second injection Booster dose: 0.5 mL q10y |

| | |
|-------------------|---|
| Contraindications | Documented hypersensitivity; history of any type of neurological symptoms or signs following administration of this product FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are an important cause of provocative poliomyelitis |
| Interactions | Patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization due to poor immune response; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent use of chloramphenicol since it may impair amnestic response to tetanus toxoid; concurrent use of tetanus immune globulin may delay development of active immunity by several days (interaction is nevertheless clinically insignificant and does not preclude its concurrent use) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Do not use to treat actual tetanus infections, or for immediate prophylaxis of unimmunized individuals (use instead tetanus antitoxin, preferably human tetanus immune globulin) diminished antibody response to active immunization may be seen in patients receiving immunosuppressive therapy; better to defer primary diphtheria immunization until immunosuppressive therapy discontinued; routine immunization of symptomatic and asymptomatic HIV-infected persons recommended |

Immunoglobulins

Patients who may not have been immunized against *Clostridium tetani* products should receive tetanus immune globulin.

| | |
|-------------------|---|
| Drug Name | Tetanus immune globulins (Hyper-Tet)- Used for passive immunization of any person with a wound that may be contaminated with tetanus spores. |
| Adult Dose | For prophylaxis: 250-500 U IM in opposite extremity to tetanus toxoid For clinical tetanus: 3,000-10,000 U IM |
| Pediatric Dose | For prophylaxis: 250 U IM in opposite extremity as tetanus toxoid For clinical tetanus: 3,000-10,000 U IM |
| Contraindications | Since antibodies in globulin preparation may interfere with immune response to vaccination, do not administer within 3 mo of live virus immune globulin administration; may be necessary to revaccinate persons who received immune globulin shortly after live virus vaccination |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|---|
| Precautions | Persons with isolated IgA deficiency have potential for developing antibodies to IgA and could have anaphylactic reactions to subsequent administration of blood products that contain IgA; do not perform skin testing since intradermal injection of concentrated gamma globulin may cause localized area of inflammation and can be misinterpreted, causing medication to be withheld from patient not allergic to this material; true allergic responses to human gamma globulin given in prescribed IM manner are extremely rare; do not admix with other medications since usually incompatible |
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Follow-up

Further Inpatient Care

- Blow-out fractures without associated serious eye injury do not require admission.
- Admit patient with serious eye injury to ophthalmology service for further care, unless other significant injuries mandate admission to trauma service.

Further Outpatient Care

- Patient with simple blow-out fracture without eye injury can be discharged home, even if patient has signs of entrapment, because most resolve as swelling goes down. Instruct patient to return if he or she notes a change in visual acuity, increasing pain, or flashing lights.
- Follow-up exam in 2 weeks allows for swelling to resolve. If entrapment is confirmed at that time, open reduction of fracture with bone graft may be needed.

Transfer

- If appropriate specialists are not available in the receiving institution, arrange transfer to a higher level hospital.

Deterrence/Prevention

- Use safety glasses at work and while participating in sports that use balls or pucks to reduce incidence of blow-out fractures.

Complications

- Corneal abrasion
- Lens dislocation
- Iris disruption
- Choroid tear
- Scleral tear
- Ciliary body tear or bruise
- Retinal detachment and tear
- Hyphema
- Ocular muscle entrapment
- Globe rupture

Patient Education

- Instruct patients to use ice to reduce edema.
- Instruct patient to return if visual problems develop.
- If injury occurred at work or in a sporting accident, instruct patient to wear safety glasses or goggles.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to diagnose orbital fracture
- Failure to diagnose associated intracranial or cervical spine injuries
- Failure to diagnose an eye injury secondary to poor patient cooperation, extensive edema, or lack of familiarity with eye exam

Special Concerns

- Always consider and check for loss of airway, intraabdominal injury, and intracranial injury.
 - Consult an ophthalmologist in the ED if the patient has experienced loss of vision or significant decrease in visual acuity or signs of entrapment, retinal detachment, hemorrhage, or retrobulbar swelling.
-

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Fractures, Pelvic

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Introduction

Background

Pelvic fracture is a disruption of the bony structure of the pelvis. This generally requires substantial force, such as that from a motor vehicle accident or a fall from a significant height.

Pathophysiology

Pelvis consists of the ilium (ie, iliac wings), ischium, and pubis, which form an anatomic ring with the sacrum. Disruption of this ring requires significant energy. Because of the forces involved, pelvic fractures frequently involve injury to organs contained within the bony pelvis. In addition, as the pelvis is supplied with a rich venous plexus as well as major arteries, fractures may produce significant bleeding.

The Young classification system incorporates anatomic mechanism of injury and identifies 4 types of ring disruption. Acetabular fractures, with or without ring disruption, also may occur.

Lateral compression (LC) fractures involve transverse fractures of the pubic rami, either ipsilateral or contralateral to a posterior injury.

- Grade I - Associated sacral compression on side of impact

- Grade II - Associated crescent (iliac wing) fracture on side of impact
- Grade III - Associated contralateral "open book" injury

Anterior-posterior compression (APC) fractures involve symphyseal diastasis or longitudinal rami fractures.

- Grade I - Associated widening (slight) of pubic symphysis or of the anterior sacroiliac (SI) joint, while sacrotuberous, sacrospinous, and posterior SI ligaments remain intact
- Grade II - Associated widening of the anterior SI joint caused by disruption of the anterior SI, sacrotuberous, and sacrospinous ligaments, while posterior SI ligaments remain intact
- Grade III (open book) - Complete SI joint disruption with lateral displacement and disrupted anterior SI, sacrotuberous, sacrospinous, and posterior SI ligaments

Vertical shear (VS) involves symphyseal diastasis or vertical displacement anteriorly and posteriorly, which is usually through the SI joint, though occasionally through the iliac wing or sacrum.

Combined mechanical (CM) fractures involve a combination of these injury patterns, with LC/VS the most common.

Acetabular fractures most commonly involve disruption of the acetabular socket when the hip is driven backward in a motor vehicle accident. Occasionally, they will occur in a pedestrian struck by a vehicle moving at a significant rate of speed.

Frequency

- **In the US:** Pelvic fractures represent 3% of all skeletal fractures, with single pubic rami and avulsion fractures the most common.

Mortality/Morbidity

- The complication rate is significant and is related to injury of underlying organs and bleeding. Because of the tremendous force necessary to cause most pelvic fractures, concomitant severe injuries are common and are associated with high morbidity and mortality rates.
- Overall mortality rate in adults is approximately 10% and in children, 5%. Pelvic hemorrhage is the direct cause of death in fewer than half of patients with pelvic fractures who die. Retroperitoneal hemorrhage and secondary infection are the main causes of death in children and adults with pelvic fractures.
- If hypotension is present on presentation, mortality rate approaches 50%. If fracture is open, mortality rate reaches 30%.

Sex

- Associated genitourinary (GU) injuries vary greatly between men and women and are discussed in other chapters.

Age

- Age distribution largely matches that of motor vehicle crashes, with car-car injuries more prevalent in adults, especially younger adults, and car-pedestrian injuries more likely to cause injury in children.

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Clinical

History

- Basic mechanism of significant blunt trauma should prompt consideration of a pelvic fracture.

Physical

- Tenderness over the pelvis that can be appreciated with pelvic springing indicates fracture. Pelvic springing involves applying alternating gentle compression and distortion over the iliac wings.
- Palpable instability of the pelvis on bimanual compression or distraction of the iliac wings also indicates fracture. Be very gentle when pelvic tenderness is appreciated. Do not rock or apply great force until radiographs exclude skeletally unstable pelvic fractures, since an overly aggressive exam can increase hemorrhage unnecessarily.
- Instability on hip adduction and pain on hip motion suggests an acetabular fracture (in addition to possible hip fracture).
- Signs of urethral injury in males include a high-riding or boggy prostate on rectal exam, scrotal hematoma, or blood at the urethral meatus.
- Vaginal bleeding or palpable fracture line on careful bimanual exam suggests pelvic fracture in females.
- Other signs of pelvic fracture include the following:
 - Hematuria
 - Rectal bleeding or Earle sign, the appreciation of a large hematoma or palpable fracture line on careful rectal exam
 - Destot sign, a hematoma above the inguinal ligament, on the proximal thigh, or over the perineum
 - Grey Turner sign, a flank ecchymosis associated with retroperitoneal bleeding

- Roux sign, a bilateral asymmetry in the distances between the greater trochanter and the pubic spine on each side (indicating an overriding fracture of the anterior pelvic ring)
- Neurovascular deficits of the lower extremities

Causes

- Adults
 - Motor vehicle crash (50-60%)
 - Motorcycle crash (10-20%)
 - Pedestrian versus car (10-20%)
 - Falls (8-10%)
 - Crush (3-6%)

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Differentials

Abdominal Trauma, Blunt
Dislocations, Hip
Fractures, Hip
Pregnancy, Trauma
Shock, Hemorrhagic
Trauma, Lower Genitourinary

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Workup

Lab Studies

- Serial hemoglobin and hematocrit measurements monitor ongoing blood loss.
- Urinalysis may reveal gross or microscopic hematuria.

Imaging Studies

- Radiographs
 - Anteroposterior pelvic radiograph is the basic screening test and uncovers 90% of pelvic

injuries.

- Additional views include outlet (40 degrees cephalad) and inlet (40 degrees caudad) views.
- Judet (oblique) views show better detail of the acetabulum.
- Computed tomography
 - If a fracture is present or suspected and the patient is medically stable, order a pelvic CT scan, in addition to other necessary CT scans, to determine whether concomitant injury is present.
 - CT scan is the best imaging study for evaluation of pelvic anatomy and degree of pelvic, retroperitoneal, and intraperitoneal bleeding. CT scan also confirms hip dislocation associated with an acetabular fracture.
- Cystogram: Obtain cystogram for patients with gross hematuria.
- Arteriogram
 - This study allows diagnosis of the bleeding site and treatment by embolization.
 - Consider in hemodynamically unstable patients when ultrasound, CT scan, or peritoneal tap excludes significant intraperitoneal bleeding and after external pelvis is stabilized.

Procedures

- Use a suprapubic catheter for patients in whom urethral injuries are suspected but a urethrogram cannot be obtained.
- Use a pneumatic antishock garment (PASG) to control bleeding and temporarily stabilize pelvis.
- External pelvic fixation may be necessary to decrease bleeding and prevent further damage.

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Treatment

Prehospital Care

- Address acute life-threatening conditions. Be very aware that the amount of force necessary to cause a pelvic fracture is likely to have caused other significant injuries.
- Use PASG to mechanically stabilize the pelvis.
- Avoid excessive movement of pelvis.
- Establish large-bore intravenous (IV) access and administer fluids as needed.
- Closely monitor vital signs.

Emergency Department Care

- Investigate associated intraabdominal and intrapelvic injuries.

- Avoid excessive movement of pelvis.
- Consider PSAG or external fixation for skeletally unstable fractures.
- Administer fluid replacement and analgesics as needed.
- Do not place urinary catheter until urethral injury has been ruled out by physical exam or retrograde urethrography.
- Obtain CT scan of pelvis as soon as practical.

Consultations

- Consult an orthopedic surgeon when a pelvic fracture is diagnosed.
- Hemodynamically unstable patients (with unstable pelvic fractures) require emergent orthopedic consultation for possible external fixation.

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Medication

Primary treatment is for pain with narcotic analgesics. Administer antibiotics whenever disruption of bowel, vagina, or urinary tract is suspected. Since bleeding is the major life-threatening complication of pelvic fractures, avoid nonsteroidal anti-inflammatory drugs in initial treatment. They may be considered later if inflammation is a concern.

Analgesics

Narcotic analgesics are the treatment of choice in the acute setting. Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures. Adequate pain control helps keep the patient quiet and avoids movement of the pelvis.

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| Drug Name | Morphine sulfate (Duramorph, Astramorph, MS Contin)- DOC for narcotic analgesia because of its reliable and predictable effects, safety, and ease of reversibility with naloxone. Administered IV, may be dosed in a number of ways and commonly is titrated until desired effect obtained. Titrated doses especially useful in trauma patients to avoid oversedation or hypotension. Caution in hypotensive patients as may worsen hypotension because of histamine release. Consider fentanyl in this setting. |
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| Adult Dose | Starting dose: 0.1 mg/kg IV Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h Relatively hypovolemic patients: Start with 2 mg IV and reassess hemodynamic effects of dose |
| Pediatric Dose | Neonates: 0.05-0.2 mg/kg IV/IM/SC prn Children: 0.1-0.2 mg/kg q2-4h prn |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

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| Drug Name | Fentanyl (Sublimaze, Duragesic)- Excellent drug for analgesia in patients with hypotension or whose cardiovascular condition is unstable. Does not release histamine. Short-acting acute action becomes longer with repetitive dosing. |
| Adult Dose | 1-2 mcg/kg IV then titrate to pain relief |
| Pediatric Dose | 2-3 mcg/kg IV then titrate to pain relief |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hypotension, respiratory depression, constipation, nausea, emesis, and urinary retention; idiosyncratic reaction, known as chest wall rigidity syndrome, may require neuromuscular blockade to increase ventilation |

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| Drug Name | Meperidine (Demerol)- No more effective than morphine or fentanyl, shorter acting and more adverse effects. Useful in patients allergic to morphine and fentanyl. |
| Adult Dose | 0.25-1 mg/kg IV titrate to pain relief |
| Pediatric Dose | 0.25-1 mg/kg IV titrate to pain relief |
| Contraindications | Documented hypersensitivity; concurrent MAOIs; upper airway obstruction or significant respiratory depression; during labor when delivery of premature infant is anticipated |

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| Interactions | Concurrent cimetidine requires monitoring for increased respiratory and CNS depression; hydantoin may decrease effects; avoid with protease inhibitors |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients with head injuries since may increase respiratory depression and CSF pressure (use only if absolutely necessary); caution when using postoperatively and with history of pulmonary disease (suppresses cough reflex); substantially increased dose levels, due to tolerance, may aggravate or cause seizures even if no prior history of convulsive disorders; monitor closely for morphine-induced seizure activity if prior seizure history |

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| Drug Name | Acetaminophen (Tylenol, Panadol, aspirin-free Anacin)- DOC for treatment of pain in patients with documented hypersensitivity to aspirin or NSAIDs or those at high risk of bleeding, with upper GI disease, or taking oral anticoagulants. DOC for pain relief in noninflammatory conditions. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; known G-6-P deficiency |
| Interactions | Rifampin can reduce analgesic effects; barbiturates, carbamazepine, hydantoin, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; APAP is contained in many OTC products and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

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| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. Relatively poor choice in most situations as better analgesics with fewer adverse effects exist. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tabs q4h; not to exceed 12 tabs/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

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| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |
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| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn based on hydrocodone content 5-10 mg dosage |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

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| Drug Name | Oxycodone and acetaminophen (Percocet, Tylox, Roxicet, Roxilox)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in the elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

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| Drug Name | Oxycodone and aspirin (Percodan, Roxiprin)- Drug combination indicated for relief of moderately severe to severe pain. Avoid in early treatment because of platelet inhibition from aspirin and increased risk of bleeding. See discussion under NSAIDs above. |
| Adult Dose | 1-2 tabs/caps PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity; liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma Because of association with Reye syndrome, do not use in children (<16 y) who have flu |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity; may potentiate anticoagulant effects of warfarin |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Duration of action may increase in the elderly; caution in renal or liver impairment, peptic ulcer disease, and erosive gastritis |

Antibiotics

Empiric antimicrobial therapy must be comprehensive and should cover all likely pathogens in the clinical setting.

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| Drug Name | Gentamicin (Gentacidin, Garamycin)- Aminoglycoside antibiotic used for gram-negative bacterial coverage. Commonly used in combination with both an agent against gram-positive organisms and one that covers anaerobes. Used in conjunction with ampicillin or vancomycin and/or metronidazole for prophylaxis in patients with suspected disruption of bowel, vagina, or urinary tract. Dosing regimens are numerous and are adjusted based on CrCl and changes in volume of distribution. Gentamicin may be given IV/IM. |
| Adult Dose | Older patients: 4 mg/kg Younger patients: 5 mg/kg |
| Pediatric Dose | 2 mg/kg IV |
| Contraindications | Duration of action may increase in the elderly; caution in renal or liver impairment, peptic ulcer disease, and erosive gastritis |

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| Interactions | Other aminoglycosides, cephalosporins, penicillins, and amphotericin B may increase nephrotoxicity; aminoglycosides enhance effects of neuromuscular blocking agents, thus prolonged respiratory depression may occur; coadministration with loop diuretics may increase auditory toxicity of aminoglycosides—possible irreversible hearing loss of varying degrees may occur (monitor regularly) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Narrow therapeutic index (not intended for long-term therapy); caution in renal failure (not on dialysis), myasthenia gravis, hypocalcemia, and conditions that depress neuromuscular transmission; adjust dose in renal impairment |

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| Drug Name | Ampicillin (Omnipen, Marcillin)- Interferes with bacterial cell wall synthesis during active replication, causing bactericidal activity against susceptible organisms. |
| Adult Dose | 2 g IV/IM |
| Pediatric Dose | 50 mg/kg IV/IM |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid and disulfiram elevate ampicillin levels; allopurinol decreases effects and has additive effects on ampicillin rash; may decrease effects of oral contraceptives |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal failure; evaluate rash and differentiate from hypersensitivity reaction |

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| Drug Name | Vancomycin (Vancocin)- Potent antibiotic directed against gram-positive organisms and active against enterococcal species. Also useful in treatment of septicemia and skin-structure infections. Used in conjunction with gentamicin for prophylaxis in patients with GI or GU trauma. |
| Adult Dose | 1 g IV infused over 1 h |
| Pediatric Dose | 1 g IV infused over 1 h |
| Contraindications | Documented hypersensitivity |
| Interactions | Erythema, histaminelike flushing, and anaphylactic reactions may occur when administered with anesthetic agents; taken concurrently with aminoglycosides, risk of nephrotoxicity may increase above that with aminoglycoside monotherapy; effects in neuromuscular blockade may be enhanced when coadministered with nondepolarizing muscle relaxants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

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| Precautions | Caution in renal failure, neutropenia; red man syndrome caused by too rapid IV infusion (dose given over a few minutes) but rarely happens when dose given over 2 h or by PO or IP route; red man syndrome not an allergic reaction |
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| Drug Name | Metronidazole (Flagyl)- Provides coverage against anaerobic bacteria (important if bowel contents contaminate peritoneal cavity). |
| Adult Dose | Loading dose: 15 mg/kg IV (approximately 1 g); followed by 7.5 mg/kg IV q6h |
| Pediatric Dose | Not recommended |
| Contraindications | Documented hypersensitivity |
| Interactions | May increase toxicity of anticoagulants, lithium, and phenytoin; cimetidine may increase toxicity; disulfiram reaction may occur with orally ingested ethanol |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in hepatic disease; monitor for seizures and development of peripheral neuropathy |

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Follow-up

Further Inpatient Care

- Monitor patient for signs of ongoing blood loss and signs of infection.
- Monitor for development of neurovascular problems in lower extremities.
- Consider deep venous thrombosis (DVT) prophylaxis in all patients.
- Pain management is very important to facilitate early mobilization and reduce risk of thrombophlebitis.
- Degree and timing of mobilization depends on the exact injury as well as associated injuries and should be determined by orthopedic and trauma surgeons.

Transfer

- Achieve hemodynamic stabilization and consider pelvic stabilization before transfer.
- Transfer all patients except those with minor pelvic fractures to a trauma center.

Deterrence/Prevention

- Encourage use of seat belts, airbags, and other protective gear.
- Promote anti–drunk-driving programs and laws.

Complications

- Increased incidence of thrombophlebitis
- "Intrapelvic compartment syndrome"
- Continued bleeding from fracture or injury to pelvic vasculature
- GU problems from bladder, urethral, prostate, or vaginal injuries. Sexual dysfunction may be a long-term problem.
- Infections from disruption of bowel or urinary system

Prognosis

- Prognosis varies depending on severity of fracture and associated injuries.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to diagnose an underlying injury, especially urethral disruption
- Failure to clinically (or radiographically) exclude urethral injury prior to attempting to insert a urinary catheter in a male
- Failure to cease attempted Foley catheterization in a female after encountering resistance
- Failure to document presence or absence of vaginal bleeding in a female with pelvic fracture
- Failure to diagnose hip dislocation associated with an acetabular fracture
- Failure to appreciate ongoing blood loss
- Failure to diagnose concomitant intraabdominal or retroperitoneal injuries
- Failure to obtain prompt orthopedic consultation for an unstable pelvic fracture

Special Concerns

- Pregnant patients
 - Patients in later stages of pregnancy are at increased risk for complications.
 - Risk of placental abruption and uterine rupture is great.

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Fractures, Rib

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Introduction

Background

Rib fractures account for more than half of thoracic injuries from nonpenetrating trauma. Motor vehicle crashes are the most common mechanism.

Pathophysiology

The chest wall is designed to protect underlying viscera by surrounding internal organs with bony structures, such as the ribs, clavicles, sternum, and scapulae. An intact chest wall is necessary for normal ventilation.

Rib fractures may compromise ventilation by a variety of mechanisms. Pain from rib fractures causes splinting, resulting in atelectasis and pneumonia. Multiple contiguous rib fractures (ie, flail chest) interfere with normal costovertebral and diaphragmatic muscle excursion, causing ventilatory insufficiency. Fractured ribs can act as a penetrating object causing hemothorax or pneumothorax. Ribs commonly fracture at the point of impact or at the posterior angle, structurally their weakest area. Middle ribs (4-9) are most commonly injured.

Mortality/Morbidity

- Morbidity and mortality from rib fractures correlate with injury of underlying structures. In one study of patients with rib fractures, mortality rate reached 12%; of these, 94% had associated injuries, and 32% had hemothorax or pneumothorax. More than half of all patients required either operative or ICU management. Average blood loss per fractured rib is reportedly 100-150 mL.
- Position of the fractured rib in the thorax helps identify potential injury to specific underlying organs. Fracture of the left lower ribs usually is associated with injury to abdominal organs rather than to lung parenchyma. Fracture of the left lower ribs is associated with splenic injuries and the right with liver injuries. Fracture of the floating ribs (ribs 11, 12) is associated with renal injuries.
- First rib fractures are a harbinger of severe trauma, since the first rib is very well protected by the shoulder, lower neck musculature, and clavicle and requires a much higher impact force to fracture than other ribs. Mortality rates as high as 36% have been reported with first rib fractures, which are associated with injury to the lung, ascending aorta, subclavian artery, and brachial plexus.

Age

- Since children have more elastic ribs, they are less likely than adults to sustain fractures following blunt chest trauma. Children present more frequently with trauma to underlying chest and abdominal organs without the associated rib fractures commonly seen in adults. This makes rib fractures in children even more of an ominous sign.
- Consider child abuse in children who lack a significant mechanism for multiple rib fractures or have fractures in different stages of healing. Recent studies of children younger than 2 years with rib fractures show the prevalence of child abuse to be as high as 82-83%.
- Older persons are more prone than younger adults to rib fractures and their pulmonary sequelae such as atelectasis, pneumonia, and respiratory arrest. The presence of cardiopulmonary disease also significantly increases morbidity and mortality rates in patients older than 65 years.

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Clinical

History

- Description of the prehospital scene by paramedics can provide important clues to the possibility of rib fractures.
- Deformation of the steering wheel and activation of seat belts and airbags are associated with rib injuries.

- Patients with rib fracture frequently complain of pain on inspiration and dyspnea.

Physical

- Tenderness on palpation, crepitus, and chest wall deformity are associated with rib fracture.
- Paradoxical chest wall excursion with inspiration is seen with flail chest.
- Specific signs of ventilatory insufficiency include cyanosis, tachypnea, and use of accessory muscles for ventilation.
- Less specific signs include anxiety and agitation.

Causes

- Motor vehicle crashes (most common mechanism)
- Blunt trauma

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Differentials

Abdominal Pain in Elderly Persons
Abdominal Trauma, Blunt
Dissection, Aortic
Pneumothorax, Tension and Traumatic
Trauma, Upper Genitourinary

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Workup

Imaging Studies

- Chest radiographs
 - Anteroposterior (AP) and lateral chest films are used routinely to assist in the diagnosis of rib fractures, yet sensitivity of only 50% has been reported.
 - Chest radiographs are much more useful in the diagnosis of underlying injuries, including hemothorax, pneumothorax, lung contusion, atelectasis, pneumonia, and vascular injuries.
- Angiography

- Since first and second rib fractures are closely associated with vascular injury, ED physicians should consider angiography for such patients, especially if symptoms and signs of neurovascular compromise are present.
- This is particularly important with posteriorly displaced fractures of the first 2 ribs, which have a much higher degree of association with abnormal findings than other rib fractures.
- While first rib fractures previously were considered a strong risk factor for aortic injury, most authorities now believe that aortography and/or CT scan are not indicated without other evidence of injury, such as abnormal mediastinum.

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Treatment

Prehospital Care

- Focus care on airway maintenance and supplemental oxygen.

Emergency Department Care

- Goal of initial ED care is stabilization and multisystem evaluation.
- Primary focus of treatment for rib fractures is pain relief and adequate clearing of pulmonary secretions.
- Isolated rib fractures, without associated injuries, may be managed on an outpatient basis with oral analgesics.
- Other options include parenterally administered narcotics titrated to prevent respiratory depression.
- Patient-controlled anesthesia allows adequate pain relief with minimal inhibition of respiratory drive.
- Intercostal nerve blocks provide pain relief without affecting respiratory function, although risks include intravascular injection and pneumothorax.
- For hospitalized patients, consider epidural and intrapleural catheter placement for delivery of anesthetics.
- While rib belts or binders do control pain, they have been linked to hypoventilation, atelectasis, and pneumonia. As a result, their use is no longer recommended.

Consultations

- Because of the close association of rib fractures with injury to underlying structures, the ED physician may need to consult the trauma service.

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Medication

Pain control remains the mainstay of treatment, usually with nonsteroidal anti-inflammatory or oral narcotic agents.

Nonsteroidal Anti-Inflammatory Drugs (NsAIDs)

These agents are used most commonly for the relief of mild to moderately severe pain. Effects of NSAIDs in the treatment of pain tend to be patient specific, yet ibuprofen is usually DOC for initial therapy. Other options include fenoprofen, flurbiprofen, mefenamic acid, ketoprofen, indomethacin, and piroxicam.

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| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually DOC for treatment of mild to moderately severe pain, if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, which in turn decreases prostaglandin synthesis. |
| Adult Dose | 600-800 mg PO q6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants—monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | A - Safe in pregnancy |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

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| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- For relief of mild to moderately severe pain and inflammation. Administer small dosages initially to patients with small bodies, older persons, and those with renal or liver disease. Doses higher than 75 mg do not increase therapeutic effects. Administer high doses with caution and observe closely. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 14 years: 0.1-1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants—monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

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| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, which decreases prostaglandin synthesis. |
| Adult Dose | 500 mg PO followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants—monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |

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| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |
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| Drug Name | Flurbiprofen (Ansaid)- Has analgesic, antipyretic, and anti-inflammatory effects. May inhibit cyclooxygenase enzyme, inhibiting prostaglandin biosynthesis, which may be mechanism of analgesic and anti-inflammatory activities. |
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants—monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures.

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| Drug Name | Acetaminophen (Tylenol, Panadol, aspirin-free Anacin)- DOC for treatment of pain in patients with documented hypersensitivity to aspirin or NSAIDs, with upper GI disease, or who are taking oral anticoagulants. Reduces fever by direct action on hypothalamic heat-regulating centers, which increases dissipation of body heat via vasodilation and sweating. |
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| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; known G-6-P deficiency |
| Interactions | Rifampin can reduce analgesic effects; barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; contained in many OTC products, and combined use with these products may result in cumulative doses exceeding recommended maximum |

| | |
|-------------------|---|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tabs q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|---|
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |
|-------------|---|

| | |
|-------------------|---|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in the elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

| | |
|-------------------|--|
| Drug Name | Oxycodone and aspirin (Percodan)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity; liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma Because of association with Reye syndrome, do not use in children (<16 y) who have flu |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity; may potentiate anticoagulant effects of warfarin |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Duration of action may increase in the elderly; caution in renal or liver impairment, peptic ulcer disease, and erosive gastritis |

| | |
|-----------|--|
| Drug Name | Morphine sulfate (Duramorph, Astramorph, MS Contin)- DOC for narcotic analgesia because of its reliable and predictable effects, safety, and ease of reversibility with naloxone. Administered IV, may be dosed in number of ways and commonly titrated until desired effect obtained. |
|-----------|--|

| | |
|-------------------|---|
| Adult Dose | Starting dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg q4h IV/IM/SC Relatively hypovolemic patients: Start with 2 mg IV/IM/SC and reassess hemodynamic effects of dose |
| Pediatric Dose | Neonates: 0.05-0.2 mg/kg dose prn Children: 0.1-0.2 mg/kg dose q2-4h prn |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

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Follow-up

Further Inpatient Care

- Patients with isolated rib fractures who are unable to cough and clear secretions adequately should be admitted for at least 24 hours.
- A lower threshold for admission of older persons with isolated rib fractures is warranted, because of their higher incidence of hypoventilation, hypercapnia, atelectasis, and pneumonia.
- Admission allows observation for occult intraabdominal organ injury.

Further Outpatient Care

- Patients with minor rib injuries who are able to cough and clear secretions may be discharged with adequate analgesic medications.
- Consider an incentive spirometer, especially with multiple fractures, as it may help avoid complications and remind the patient to avoid splinting and to take deep breaths.

Transfer

- Currently, no published guidelines exist for transfer of patients with rib fracture to a regional

trauma center.

- Lee and Bass concluded that the presence of 3 or more rib fractures identifies a subgroup of adult patients who may require tertiary care.

Complications

- Hypoventilation
- Hypercapnia
- Hypoxia
- Atelectasis
- Pneumonia

Prognosis

- Isolated rib fractures in younger patients have a good prognosis.
- Older patients have a higher incidence of significant pulmonary complications.

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Fractures, Scapular

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Introduction

Background

The primary function of the scapula is to attach the upper extremity to the thorax and provide a stabilized platform for upper extremity movement. The scapula is attached to the clavicle by the acromioclavicular and coracoclavicular ligaments and articulates with the humerus. The scapula is protected by its surrounding musculature (supraspinatus, infraspinatus, subscapularis) and its ability to move along the wall of the thorax; the body and spine of the scapula are most protected. Fractures to scapular structures typically require significant force. These factors explain the infrequent occurrence of scapular fractures.

Pathophysiology

The primary anatomic features of the scapula provide insight into the mechanisms of injury and offer a convenient classification system. Injuries to the body or spine of the scapula typically result from a direct blow with significant force, as in motor vehicle accident or fall.

Scapular fractures are caused by different mechanisms. Acromion injuries usually result from a direct downward force to the shoulder. Scapular neck fractures most frequently result from an anterior or posterior force applied to the shoulder. Glenoid rim fractures most often result from force transmitted along the humerus after a fall onto a flexed elbow. Stellate glenoid fractures usually follow a direct blow to the lateral shoulder. Finally, coracoid process fractures may result from either a direct blow to the

superior aspect of the shoulder or a forceful muscular contraction that causes an avulsion fracture.

Frequency

- **In the US:** Scapular fractures occur infrequently. They account for approximately 1% of all fractures and fewer than 5% of shoulder girdle injuries.

Mortality/Morbidity

- Morbidity and mortality result primarily from associated injuries.
- Traditional wisdom holds that scapular fractures serve as markers of increased morbidity and mortality in patients with blunt trauma. A recent retrospective study comparing patients with scapular fractures due to blunt trauma with control subjects matched for age, sex, and mechanism of injury demonstrated an increase in associated thoracic injuries yet revealed no difference in mortality or neurovascular injury.
- A large force is usually required to fracture the scapula, particularly the body or spine; however, suspect scapular fractures and thoroughly search for associated injuries.

Sex

Scapular fractures are more common among men than among women because of their increased incidence of significant blunt trauma.

Age

Scapular fractures predominate in those aged 25-40 years because of the increased occurrence of significant blunt trauma in this population.

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Clinical

History

The mechanisms of injury for various scapular fractures include the following:

- **Body or spine fracture (40-75%):** Fractures of the body or spine of the scapula usually result from a severe direct blow, as in a fall or motor vehicle accident.
- **Acromion fracture (8-16%):** Acromion fractures typically result from a downward blow to the

shoulder. Superiorly displaced fractures may occur as the result of a superior dislocation of the shoulder.

- Neck fracture (5-32%): A direct anterior or posterior blow to the shoulder is the typical mechanism for a scapular neck fracture.
- Glenoid fracture (10-25%): Glenoid rim fractures often result from a fall onto a flexed elbow. A direct lateral blow is the common mechanism for a stellate fracture of the glenoid.
- Coracoid fracture (3-13%): Coracoid process fractures usually result from 1 of 2 mechanisms.
 - A coracoid process fracture is the result of a direct blow to the superior point of the shoulder or humeral head in an anterior shoulder dislocation.
 - An avulsion fracture may result from abrupt contractions of the coracoacromial muscle, short head of the biceps, or coracohumeral muscle.

Physical

Findings at physical examination may include the following:

- Body or spine fracture
 - Most common findings are tenderness, edema, and ecchymosis over the affected area.
 - The upper extremity is held in adduction, and any attempt to abduct the extremity (which results in scapular rotation) increases pain.
- Neck fracture
 - A patient with a scapular neck fracture resists all movement of the shoulder and holds the extremity in adduction.
 - Maximal tenderness occurs at the lateral humeral head.
- Coracoid fracture
 - Patients with coracoid process fractures present with tenderness over the coracoid.

Causes

- Scapular fractures usually are the result of significant blunt trauma.

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Differentials

Abdominal Pain in Elderly Persons

Acromioclavicular Injury

Back Pain, Mechanical

Dislocations, Shoulder

Fractures, Clavicle

Fractures, Rib

Fractures, Scapular

Pneumothorax, Tension and Traumatic

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Workup

Lab Studies

- Tailor the laboratory evaluation of a patient with scapular fracture to the likelihood of associated injuries.

Imaging Studies

- Radiographs
 - An anteroposterior shoulder view, along with a lateral scapular view, demonstrates the vast majority of scapular fractures.
 - A lateral scapular (trans-scapular) view, combined with an anteroposterior shoulder view, provides the necessary 2-plane assessment of the scapula.
 - A lateral axillary view isolates the coracoid process and helps to delineate associated shoulder dislocations.
 - Tangential oblique views aid in the evaluation of small or subtle scapular body fractures.

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Treatment

Prehospital Care

- Prehospital care involves transport, with immobilization of affected extremity.
- Because of the significant forces involved in producing a scapular fracture, consider life-threatening associated injuries.

Emergency Department Care

The following discussion of the ED treatment of scapular fractures assumes that a prudent search for associated injuries revealed negative findings.

- Body or spine fracture
 - Use of ice, analgesics, and sling and swath immobilization suffice for most fractures to the body or spine of the scapula.
 - Early range-of-motion exercises are recommended.
- Neck fracture
 - Manage nondisplaced scapular neck fractures with a sling, ice, analgesics, and early range-of-motion exercises.
 - Displaced neck fractures require urgent orthopedic consultation for traction or surgical reduction.
- Coracoid fracture: Coracoid fractures respond well to conservative therapy with sling immobilization, ice, analgesics, and early mobilization.

Consultations

- Follow-up care with an orthopedic surgeon is advised in all cases because of the possibility of long-term complications such as bursitis and posttraumatic arthritis.

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Medication

Nonsteroidal anti-inflammatory agents and opioid analgesics typically are required for scapular fractures.

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

These agents are most commonly used for the relief of mild to moderate pain. Effects of NSAIDs in the treatment of pain tend to be patient specific, yet ibuprofen is usually the DOC for initial therapy. Other options include naproxen, flurbiprofen, and ketoprofen.

| | |
|------------|--|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually DOC for the treatment of mild to moderate pain, if no contraindications exist; inhibits inflammatory reactions and pain, probably by decreasing cyclooxygenase activity, which results in prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |

| | |
|-------------------|--|
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for the relief of mild to moderate pain and inflammation. Administer small doses initially to smaller patients and older persons. Doses of >75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe patient. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 12 years: 0.1-1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

| | |
|------------|--|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderate pain; inhibits inflammatory reactions and pain by decreasing cyclooxygenase activity, which decreases prostaglandin synthesis. |
| Adult Dose | 500 mg followed by 250 mg PO q6-8h; not to exceed 1.25 g/d |

| | |
|-------------------|---|
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion may be at risk for acute renal failure; leukopenia occurs rarely and is transient, and condition usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

| | |
|-------------------|--|
| Drug Name | Flurbiprofen (Ansaid)- Has analgesic, antipyretic, and anti-inflammatory effects; may inhibit cyclooxygenase, causing inhibition of prostaglandin biosynthesis that may result in analgesic and anti-inflammatory activities. |
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion, risk acute renal failure; leukopenia occurs rarely and is transient, and condition usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have fractures.

| | |
|-------------------|--|
| Drug Name | Acetaminophen (Tylenol, Panadol, Aspirin-Free Anacin)- DOC for treatment of pain in patients with documented hypersensitivity to aspirin or NSAIDs or in those with upper GI disease or taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg PO q4-6h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; known G-6-P deficiency |
| Interactions | Rifampin can reduce analgesic effects; coadministration with barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in persons with chronic alcoholism at various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; many OTC products contain acetaminophen, and combined use of these products may result in cumulative doses exceeding the recommended maximum dose |

| | |
|-------------------|---|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for the treatment of mild to moderate pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tab PO q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | Toxicity increases with CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-----------|--|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for the relief of moderate-to-severe pain. |
|-----------|--|

| | |
|-------------------|--|
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen PO q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Coadministration with phenothiazines may decrease analgesic effects; toxicity increases with CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite, which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|--|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for the relief of moderate to severe pain; DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose based on oxycodone content PO; not to exceed 5 mg/dose of oxycodone q4-6h prn |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; toxicity increases with coadministration of CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in elderly patients; be aware of the total daily dose of acetaminophen that the patient is receiving; do not exceed 4,000 mg/d of acetaminophen; higher doses may cause liver toxicity |

| | |
|-------------------|--|
| Drug Name | Oxycodone and aspirin (Percodan)- Drug combination indicated for relief of moderate to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose based on oxycodone content PO; not to exceed 5 mg/dose of oxycodone PO q4-6h prn |
| Contraindications | Documented hypersensitivity; liver damage, hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma; children <16 y with flu (due to the association of aspirin with Reye syndrome) |

| | |
|--------------|--|
| Interactions | Phenothiazines may decrease analgesic effects; conversely, toxicity increases when administered concurrently with, CNS depressants or tricyclic antidepressants; may also potentiate anticoagulant effects of warfarin |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Duration of action may increase in elderly patients; caution in renal or liver impairment, peptic ulcer disease, and erosive gastritis |

| | |
|-------------------|---|
| Drug Name | Morphine Sulfate (Duramorph, Astramorph, MS Contin)- DOC for narcotic analgesia because of its reliable and predictable effects, safety, and ease of reversibility with naloxone. IV doses vary and commonly are titrated until desired effect is obtained. |
| Adult Dose | Starting dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h Relatively hypovolemic patients: Start with 2 mg IV/IM/SC and reassess hemodynamic effects of dose |
| Pediatric Dose | Neonates: 0.05-0.2 mg/kg/dose IV/IM/SC q2-4h prn Children: 0.1-0.2 mg/kg/dose IV/IM/SC q2-4h prn |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway where rapidly establishing airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects of opiate agonists; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects of morphine |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

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Follow-up

Transfer

- Transfer the patient with a scapular fracture when evaluation or treatment of associated injuries or surgical repair of the fracture is necessary and when neither is available at the initial institution.

Deterrence/Prevention

- Enforcement of traffic safety laws and injury prevention education are the 2 most productive measures for reducing scapular fractures.

Complications

- After associated injuries are excluded, the most common complication of an isolated scapular fracture is posttraumatic arthritis or bursitis.

Prognosis

- If no significant associated injury exists, the prognosis for complete or near complete recovery is excellent.

Patient Education

- Patient education about the use of sling immobilization, ice, and analgesics is important.
- A critical part of most treatment regimens is early range-of-motion exercises.

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Miscellaneous

Medical/Legal Pitfalls

- Missed diagnosis
 - Failure to consider associated injuries
-

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Fractures, Sternal

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Introduction

Background

Sternal fractures were once thought to be high-morbidity injuries, with a mortality rate of 25-45% from associated injuries. Recent literature reveals that the morbidity rate may be lower, yet caution is warranted when evaluating and treating patients with this injury.

Pathophysiology

Most sternal fractures are caused by blunt anterior chest trauma, although stress fractures have been noted in golfers, weight lifters, and other participants in noncontact sports. Insufficiency fractures can occur spontaneously in patients with osteoporosis or osteopenia (particularly in older persons), those on long-term steroid therapy, or those with severe thoracic kyphosis.

Frequency

- **In the US:** Motor vehicle collisions account for 60-90% of sternal fractures. Most of these patients used appropriate restraints. Those who were unrestrained generally sustained injury from ejection from the vehicle or impact with the steering wheel or dashboard. Direct impact sports, falls, vehicle-to-pedestrian accidents, and assaults account for most of the rest. Spontaneous

fractures and stress fractures are rare.

Mortality/Morbidity

Mortality rate from isolated sternal fracture is extremely low. Death and morbidity are related almost entirely to associated injuries such as aortic disruption, cardiac contusion, and pulmonary contusion, or unrelated injuries to the abdomen or head sustained in the accident.

Race

No racial predilection is known.

Sex

Sternal fractures are more common in females, possibly due to shoulder restraint positioning.

Age

Sternal fractures are more common in patients older than 50 years, possibly because of a weaker or inelastic bony thorax.

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Clinical

History

- In most cases, consider diagnosis based on the mechanism of injury. The injuries of a patient with spontaneous insufficiency or stress fracture are a greater diagnostic challenge unless the diagnosis is considered carefully.
- Almost all patients complain of localized sternal pain. Pain may be more diffuse in patients with insufficiency fractures and may lead to a more extensive differential diagnosis for chest pain in an older population.
- Dyspnea is present in 15-20% of these patients and may indicate associated cardiopulmonary contusion.
- Palpitations may be noted only if dysrhythmia occurs, which is unusual in isolated sternal injury without associated cardiac contusion.

Physical

- Carefully assess for signs of other potentially associated injuries. These may include rib fractures, flail chest, pneumothorax, hemothorax, pulmonary contusion, blunt cardiac injury (dysrhythmias or murmurs), pericardial tamponade, or vascular injury, as well as head, neck, abdominal, or extremity trauma.
- Pain usually is localized over the fracture site and readily reproducible, though patients with insufficiency fractures may have more diffuse pain.
- Crepitation or displacement is not often palpable unless the sternum is disrupted completely with significant instability of the fragments.
- Only 40-55% of patients have overlying soft-tissue edema or ecchymosis.
- Patients with insufficiency fractures usually exhibit an exaggerated dorsal kyphosis.

Causes

- With increased use of seat belts and shoulder restraints, incidence has increased, but overall severity of injuries has decreased.
- Presumably, incidence has increased because all of the deceleration forces are concentrated into a nonelastic 2-inch strap that transmits this force directly to the sternum.
- Effects of airbags on incidence of sternal fractures have not been studied.

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Differentials

Dissection, Aortic

Other Problems to be Considered

Cardiac tamponade

Flail chest

Cardiac contusion

Pulmonary contusion

Thoracic spine injury

Workup

Lab Studies

- In general, laboratory studies are not indicated for evaluation of isolated sternal injuries, though consider appropriate laboratory studies in evaluation of potential associated injuries.
- Creatine kinase (CK)-MB index and other enzyme markers of cardiac injury are helpful if cardiac contusion is suspected. The routine use of this test is not indicated, however. Remember that total CK may be elevated from other noncardiac muscle injuries.

Imaging Studies

- Radiographs
 - Plain radiography remains the diagnostic tool of choice.
 - Though standard posteroanterior and lateral chest x-rays may reveal fracture, sternal views are necessary if injury is suspected from physical examination.
 - Be aware of normal ossification centers that normally close by the late teenage years, though sternomanubrial and sternoxiphoid centers may never fuse in 10-30% of patients.
 - Sternal views enhance visualization of the sternum, since they change the angle and focus of the exposure. Order these views if highly suspicious for injury and no fracture is seen on chest radiograph.
- Computed tomography
 - CT scanning may reveal this injury yet is less sensitive than plain radiography, as the fracture may be positioned between image cuts.
 - CT scans may demonstrate retrosternal hematoma; although its specificity is high, its sensitivity is poor.
 - Suspicion for other chest injuries warrants CT scans.

Other Tests

- Obtain an ECG in all patients with significant blunt injury to chest. Findings indicative of cardiac contusion include dysrhythmia, conduction disturbances, or ST segment changes consistent with myocardial injury.
- Perform cardiac monitoring as workup proceeds until making a disposition decision for patient.
- Obtain pulse oximetry on all patients during their evaluation.
- Do not routinely consider echocardiography in patients with isolated sternal injury. Studies have shown that up to 25% of patients with sternal fracture have small pericardial effusions, yet in

absence of hemodynamic compromise, this requires no further intervention.

Procedures

- No procedures are indicated in patients with isolated sternal fracture, yet consider interventions for associated injuries.

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Treatment

Prehospital Care

- Initiate basic or advanced trauma care based upon level of training of ambulance crew and initial assessment.
- Care should include the following steps:
 - Supplemental oxygen
 - Cardiac monitoring
 - Intravenous access
 - Consideration of an analgesic
 - Trauma care as warranted by protocol for any suspected associated injuries

Emergency Department Care

- After immediate stabilization, evaluate patient by obtaining complete history and physical examination.
- Taping or splinting of sternal fractures is relatively contraindicated, as restriction of normal chest expansion during respiration can lead to atelectasis and pulmonary insufficiency.
- Adequate analgesia is the treatment of choice, both during initial care and subsequently during the recovery period.

Consultations

- Consult a trauma surgeon when serious associated injury is diagnosed or suspected.
- Surgical fixation for sternal fractures is generally unnecessary.

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Medication

Primary treatment is adequate analgesia with nonsteroidal anti-inflammatory drugs and opiates. Select these on the basis of relative indications and contraindications for each patient and administer in standard doses and routes. Since sternal fractures can take weeks to heal, do not hesitate to offer adequate analgesia for this recovery period. No other pharmacologic therapies are indicated specifically for treatment of sternal fractures.

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

These agents are used most commonly for relief of mild to moderately severe pain. Effects of NSAIDs in the treatment of pain tend to be patient specific, yet ibuprofen is usually the drug of choice for initial therapy. Other options include fenoprofen, flurbiprofen, ketoprofen, and naproxen.

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually DOC for treatment of mild to moderately severe pain, if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, which inhibits prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for relief of mild to moderately severe pain and inflammation. Administer small dosages initially to patients with small bodies, older persons, and those with renal or liver disease. Doses higher than 75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe for response. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 12 years: 0.1-1 mg/kg PO q6-8h >12 years: 25-50 mg q6-8h prn; not to exceed 300 mg/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ €monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, which inhibits prostaglandin synthesis. |
| Adult Dose | 500 mg PO followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ €monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |

| | |
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| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |
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Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures.

| | |
|-------------------|---|
| Drug Name | Acetaminophen (Tylenol, Panadol, aspirin-free Anacin)- DOC for treatment of pain in patients with documented hypersensitivity to aspirin or NSAIDs or those with upper GI disease or taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 g q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; known G-6-P deficiency |
| Interactions | Rifampin can reduce analgesic effects; barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate serious illness; APAP is contained in many OTC products and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

| | |
|-------------------|---|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tab q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

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| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |
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| | |
|-------------------|---|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in the elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

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| Drug Name | Morphine sulfate (Duramorph, Astramorph, MS Contin)- DOC for narcotic analgesia because of its reliable and predictable effects, safety, and ease of reversibility with naloxone. Administered IV, may be dosed in a number of ways and commonly is titrated until desired effect obtained. |
|-----------|---|

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| Adult Dose | Starting dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h Relatively hypovolemic patients: Start with 2 mg IV/IM/SC and reassess hemodynamic effects of dose |
| Pediatric Dose | Neonates: 0.05-0.2 mg/kg IV/IM/SC prn Children: 0.1-0.2 mg/kg q2-4h prn |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

Acetylsalicylic Acids

These agents are effective in reducing pain and inflammation.

| | |
|-------------------|---|
| Drug Name | Aspirin (Anacin, Ascriptin, Bayer aspirin)- Used for treatment of mild to moderately severe pain and headache. Blocks prostaglandin synthetase action, which inhibits prostaglandin synthesis and prevents formation of platelet-aggregating thromboxane A ₂ . Also acts on hypothalamus heat-regulating center to reduce fever. |
| Adult Dose | 325-650 mg PO q4-6h; not to exceed 4 g/d |
| Pediatric Dose | 10-15 mg/kg/dose PO q4-6h; not to exceed 60-80 mg/kg/d |
| Contraindications | Documented hypersensitivity; liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma Because of association with Reye syndrome, do not use in children (<16 y) with flu |
| Interactions | Antacids and urinary alkalinizers may decrease effects; corticosteroids decrease serum levels; anticoagulants may cause additive hypoprothrombinemic effects and increased bleeding times; may antagonize uricosuric effects of probenecid and increase toxicity of phenytoin and valproic acid; doses >2 g/d may potentiate glucose-lowering effects of sulfonylurea drugs |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | May cause transient decrease in renal function and aggravate chronic kidney disease; avoid use in patients with severe anemia, with history of blood coagulation defects, or taking anticoagulants |

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Follow-up

Further Inpatient Care

- Numerous studies demonstrate that admission for isolated sternal fracture is not necessary unless associated injuries or social situations require such considerations.
- Consider admission for cardiac monitoring when ECG abnormalities are found or serum markers indicate cardiac injury.

Further Outpatient Care

- Follow-up outpatient care is suggested to ensure adequacy of analgesia and to monitor healing; however, no specific interventions or diagnostic testing are indicated in the outpatient care of patients with isolated sternal fractures.

in/Out Patient Meds

- Sternal fractures take weeks to heal and provisions must be made for limited physical activity and adequate pain control during this recovery period.
- Medications include NSAIDs, opiates, or opiate and nonopiate combinations in the usual doses unless specific patient conditions contraindicate their usage.

Transfer

- Since most patients with isolated sternal fracture are candidates for discharge from the ED, no indications for transfer exist unless associated injuries dictate otherwise.

Deterrence/Prevention

- Sternal fractures are more prevalent now with the increased use of seat belt restraints; however, the injuries that such restraints prevent more than support their continued usage at all times in motor vehicles. Proper positioning of these restraints is important.

Complications

- Complications may arise from associated injuries. During evaluation of these patients, carefully assess for cardiac, pulmonary, mediastinal, and thoracic spine injuries, as well as associated injuries unrelated to chest trauma.
- Cardiac contusion is much less common than once thought; its incidence currently ranges from 6-18% based upon severity of trauma.
- Traumatic aortic injury occurs in fewer than 2% of sternal fractures, a rate similar to that in patients with blunt chest trauma without sternal fracture.
- Nonunion of sternal fractures is very rare. Painful pseudoarthroses or overlap deformities may require delayed surgical repair.
- A posttraumatic mediastinal abscess is very uncommon. Risk factors include the presence of a large hematoma, intravenous drug abuse, and another source of a staphylococcal infection. Treatment is open debridement.

Prognosis

- Prognosis is excellent for isolated sternal fractures. Most patients recover completely over a period of several weeks.

Patient Education

- Though seat belts contribute to sternal fractures, they prevent more serious injuries. Reinforce their use.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to consider and treat other associated injuries
- Failure to provide adequate analgesia

Special Concerns

- Pregnant patients
 - During pregnancy, shield the abdomen and pelvis with a lead apron prior to obtaining required chest radiographs.
 - NSAIDs for analgesia are contraindicated, though several category B opiate combinations

exist for pain management.

- Children: No specific pediatric concerns are noted.

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Fractures, Tibia and Fibula

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Introduction

Background

Lower leg fractures include fractures of the tibia and fibula. These fractures may result from direct or indirect trauma. Fractures of the tibia generally are associated with fibula fracture, because the force is transmitted along the interosseous membrane to the fibula. Significant numbers of these injuries are open, because the skin and subcutaneous tissue are very thin over the anterior tibia.

Frequency

- **In the US:** Fractures of the tibia are the most common long bone fractures. Isolated midshaft or proximal fibula fractures are uncommon.

Mortality/Morbidity

- Limb loss may occur as a result of severe soft-tissue trauma, neurovascular compromise, popliteal artery injury, compartment syndrome, or infection such as gangrene or osteomyelitis. Popliteal artery injury is a particularly serious injury that threatens the limb and is easily overlooked.
- Injury to the peroneal nerve, resulting in foot drop, can occur with fractures of the fibular neck.
- Delayed union, nonunion, and arthritis may occur. Among the long bones, the tibia is the most

common site of fracture nonunion.

Age

Toddler fracture (distal spiral fracture of the tibia) is most common in children aged 9 months to 3 years.

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Clinical

History

- Patient may report a history of direct (motor vehicle crash [MVC]) or indirect (twisting) trauma.
- Patient complains of pain, swelling, and inability to ambulate with tibia fracture.
- Ambulation is possible with isolated fibula fracture.

Physical

- Neurovascular status
- Open wound
- Deformity
- Ecchymosis
- Point tenderness
- Edema
- Crepitus

Causes

- Direct forces such as those caused by falls and MVCs
- Indirect or rotational forces

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Differentials

Ankle, Soft-tissue Injuries

Compartment Syndrome, Extremity

Fractures, Ankle

Fractures, Knee

Knee, Soft-tissue Injuries

Pediatrics, Child Abuse

Pediatrics, Limp

Peripheral Vascular Injuries

Trauma, Peripheral Vascular Injuries

Other Problems to be Considered

Maisonneuve fracture

Shin splints

Stress fracture

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Workup

Imaging Studies

- Perform anteroposterior and lateral radiographs of tibia and fibula as indicated.
- Technetium bone scan is more sensitive for detection of stress fractures.

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Treatment

Prehospital Care

- Address airway, breathing, and circulation.
- Check and document neurovascular status.
- Apply sterile dressing to open wounds.
- Apply gentle traction to reduce gross deformities; splint the extremity.

- Administer parenteral analgesics for an isolated extremity injury in a hemodynamically stable patient.

Emergency Department Care

- Tibia and fibula fractures
 - Perform closed reduction and apply long leg posterior splint with bulky dressing.
 - Provide tetanus prophylaxis and antistaphylococcal antibiotic coverage for open fractures.
 - Open fractures require immediate orthopedic referral for irrigation and debridement in operating room (restrict patient's intake to nothing by mouth [NPO] in ED).

Consultations

- Tibia and fibula fractures
 - Obtain emergent orthopedic referral for open fractures.
 - Emergent referral also is indicated generally for closed fractures.

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Medication

Drugs used to treat fractures include analgesics and anxiolytics. In addition, administer proper antibiotics and tetanus prophylaxis for open fractures.

Anxiolytics

Patients with painful injuries usually experience significant anxiety. Anxiolytics allow clinician to administer a smaller analgesic dose to achieve the same effect.

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|------------|--|
| Drug Name | Lorazepam (Ativan)- Sedative-hypnotic in benzodiazepine class that has short onset of effect and relatively long half-life. By increasing action of GABA, major inhibitory neurotransmitter, may depress all levels of CNS, including limbic and reticular formation. Excellent for sedating patient for >24 h. Commonly used prophylactically to prevent delirium tremens. Monitor BP after administering dose and adjust as necessary. |
| Adult Dose | 2 mg initially or 0.044 mg/kg IV, whichever is smaller; not to exceed 4 mg/dose |

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| Pediatric Dose | 0.05-0.1 mg/kg IV slowly over 2-5 min; may repeat dose of 0.05 mg/kg IV slowly |
| Contraindications | Documented hypersensitivity; preexisting CNS depression; hypotension; narrow-angle glaucoma |
| Interactions | Alcohol, phenothiazines, barbiturates, and MAOIs increase CNS toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in renal or hepatic impairment, myasthenia gravis, organic brain syndrome, or Parkinson disease |

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

These drugs have analgesic and antipyretic activities. Their mechanism of action is not known, but they may inhibit cyclooxygenase activity and prostaglandin synthesis. Other mechanisms may involve inhibition of leukotriene synthesis, lysosomal enzyme release, lipoxygenase activity, neutrophil aggregation, and various cell-membrane functions.

| | |
|-------------------|---|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually DOC for treatment of mild to moderately severe pain, if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, which decreases prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h prn; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ €monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

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| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for relief of mild to moderately severe pain and inflammation. Administer small dosages initially to patients with small bodies, older persons, and those with renal or liver disease. Doses higher than 75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 12 years: 0.1-1 mg/kg PO q6-8h >12 years: 25-50 mg q6-8h prn; not to exceed 300 mg/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

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| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, decreasing prostaglandin synthesis. |
| Adult Dose | 500 mg PO followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 5-7 mg/kg/dose PO q8-12h |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |

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| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |
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Acetylsalicylic Acids

These drugs have anti-inflammatory and analgesic properties.

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| Drug Name | Aspirin (Anacin, Ascriptin, Bayer aspirin)- Used for treatment of mild to moderately severe pain. Block prostaglandin synthetase action, which inhibits prostaglandin synthesis, decreasing pain. |
| Adult Dose | 325-650 mg PO q4-6h; not to exceed 4 g/d |
| Pediatric Dose | 10-15 mg/kg/dose PO q4-6h; not to exceed 60-80 mg/kg/d |
| Contraindications | Documented hypersensitivity; liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma Because of association with Reye syndrome, do not use in children (<16 y) with flu |
| Interactions | Antacids and urinary alkalinizers may decrease effects; corticosteroids decrease serum levels; anticoagulants may cause additive hypoprothrombinemic effects and increased bleeding time; may antagonize uricosuric effects of probenecid and increase toxicity of phenytoin and valproic acid; doses >2 g/d may potentiate glucose-lowering effect of sulfonylurea drugs |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | May cause transient decrease in renal function and aggravate chronic kidney disease; avoid use in patients with severe anemia, with history of blood coagulation defects, or taking anticoagulants |

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained fractures.

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| Drug Name | Acetaminophen (Tylenol, Panadol, aspirin-free Anacin)- DOC for treatment of pain in patients with documented hypersensitivity to aspirin or NSAIDs, with upper GI disease, or taking oral anticoagulants. |
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| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg PO q4h; not to exceed 5 doses in 24 h |
| Contraindications | Documented hypersensitivity; known G-6-P deficiency |
| Interactions | Rifampin can reduce analgesic effects; barbiturates, carbamazepine, hydantoin, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; APAP is contained in many OTC products and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

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| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tab q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

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|-------------------|---|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg acetaminophen q4h; single dose not to exceed 10 mg of hydrocodone bitartrate; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |

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| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

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| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relief of moderately severe to severe pain. DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tabs/caps PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in the elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/24h of acetaminophen; higher doses may cause liver toxicity |

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| Drug Name | Morphine sulfate (Duramorph, Astramorph, MS Contin)- DOC for narcotic analgesia because of its reliable and predictable effects, safety, and ease of reversibility with naloxone. Administered IV, may be dosed in a number of ways and commonly is titrated until desired effect obtained. |
| Adult Dose | Starting dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h Relatively hypovolemic patients: Start with 2 mg IV/IM/SC, and reassess hemodynamic effects of dose |
| Pediatric Dose | Neonates: 0.05-0.2 mg/kg IV/IM/SC prn Children: 0.1-0.2 mg/kg q2-4h prn |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

Antibiotics

Prophylaxis is given to patients with open fractures.

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| Drug Name | Penicillin G (Pfizerpen)- Interferes with synthesis of cell wall mucopeptide during active replication, resulting in bactericidal activity against susceptible microorganisms. |
| Adult Dose | 2.4 million U IM as single dose in 2 injection sites |
| Pediatric Dose | 50,000 U/kg IM to maximum of 2.4 million U |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid can increase effects; tetracyclines can decrease effects |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Caution in impaired renal function |

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| Drug Name | Clindamycin (Cleocin)- Lincosamide useful as prophylactic against serious skin and soft-tissue infections caused by most staphylococci strains. Effective against aerobic and anaerobic streptococci, except enterococci. Inhibits bacterial protein synthesis by inhibiting peptide chain initiation at bacterial ribosome, where it preferentially binds to 50S ribosomal subunit, causing bacterial growth inhibition. |
| Adult Dose | 600 mg/d PO for 5-7 d |
| Pediatric Dose | 20-40 mg/kg/d PO divided tid/qid for 5-7 d |
| Contraindications | Documented hypersensitivity; regional enteritis; ulcerative colitis; hepatic impairment; antibiotic-associated colitis |
| Interactions | Increases duration of neuromuscular blockade induced by tubocurarine and pancuronium; erythromycin may antagonize effects; antidiarrheals may delay absorption |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in severe hepatic dysfunction; no adjustment necessary in renal insufficiency; associated with severe and possibly fatal colitis |

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| Drug Name | Ceftriaxone (Rocephin)- Third-generation cephalosporin with broad-spectrum activity against gram-negative organisms, lower efficacy against gram-positive organisms, and higher efficacy against resistant organisms. By binding to one or more penicillin-binding proteins, arrests bacterial cell wall synthesis and inhibits bacterial replication. |
| Adult Dose | 1-2 g IV qd or divided bid for 5-7 d; not to exceed 4 g/d |

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| Pediatric Dose | 50-75 mg/kg/d IV divided q12h for 5-7 d; not to exceed 2 g/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid may increase levels; ethacrynic acid, furosemide, or aminoglycosides may increase nephrotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal impairment; caution in breastfeeding women and allergy to penicillin |

Immunoglobulins

Patients who may not have been immunized against *Clostridium tetani* products should receive tetanus immune globulin.

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| Drug Name | Tetanus immune globulin (Hyper-Tet)- Used for passive immunization of any person with a wound that may be contaminated with tetanus spores. |
| Adult Dose | Prophylaxis: 250-500 U IM in opposite extremity to tetanus toxoid Clinical tetanus: 3,000-10,000 U IM |
| Pediatric Dose | Prophylaxis: 250 U IM in opposite extremity to tetanus toxoid Clinical tetanus: Administer as in adults |
| Contraindications | Since antibodies in globulin preparation may interfere with immune response to vaccination, do not administer within 3 mo of live virus immune globulin administration; may be necessary to revaccinate persons who received immune globulin shortly after live virus vaccination |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Persons with isolated IgA deficiency have potential for developing antibodies to IgA and could have anaphylactic reactions to subsequent administration of blood products that contain IgA; do not perform skin testing since intradermal injection of concentrated gamma globulin may cause localized area of inflammation and can be misinterpreted, causing medication to be withheld from patient not allergic to this material; true allergic responses to human gamma globulin given in prescribed IM manner are extremely rare; do not admix with other medications since usually incompatible |

Toxoids

This agent is used for tetanus immunization. Booster injection in previously immunized individuals is

recommended to prevent this potentially lethal syndrome.

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| Drug Name | <p>Tetanus toxoid- Used to induce active immunity against tetanus in selected patients. Tetanus and diphtheria toxoids are immunizing AOC for most adults and children >7 y. Necessary to administer booster doses to maintain tetanus immunity throughout life.</p> <p>Pregnant patients should receive only tetanus toxoid, not a diphtheria antigen-containing product.</p> <p>In children and adults, may administer into the deltoid or midlateral thigh muscles. In infants, preferred site of administration is midthigh laterally.</p> |
| Adult Dose | <p>Primary immunization: 0.5 mL IM; give 2 injections 4-8 wk apart and a third dose 6-12 mo after second injection</p> <p>Booster dose: 0.5 mL q10y</p> |
| Pediatric Dose | Administer as in adults |
| Contraindications | <p>Documented hypersensitivity; history of any type of neurological symptoms or signs following administration of this product</p> <p>FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are important cause of provocative poliomyelitis</p> |
| Interactions | <p>Patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization due to poor immune response; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent use of chloramphenicol since it may impair amnestic response to tetanus toxoid; concurrent use of tetanus immune globulin may delay development of active immunity by several days (interaction is nevertheless clinically insignificant and does not preclude its concurrent use)</p> |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | <p>Do not use to treat actual tetanus infections, or for immediate prophylaxis of unimmunized individuals (use instead tetanus antitoxin, preferably human tetanus immune globulin); diminished antibody response to active immunization may be seen in patients receiving immunosuppressive therapy; better to defer primary diphtheria immunization until immunosuppressive therapy discontinued; routine immunization of symptomatic and asymptomatic HIV-infected persons recommended</p> |

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Follow-up

Further Inpatient Care

- Tibia and fibula fractures
 - Open fractures require debridement and irrigation in operating room.
 - Inpatient admission may be advised to observe development of compartment syndrome.

Further Outpatient Care

- Patient should see primary care physician or be referred to an orthopedic surgeon within 1 week for further evaluation and treatment of isolated fibula fractures.

Transfer

- Transfer is reasonable if approved by patient (for insurance or other reasons) or if a hospital bed or an orthopedic surgeon is unavailable at receiving institution.

Complications

- Neurovascular compromise
- Compartment syndrome
- Peroneal nerve injury
- Infection
- Gangrene
- Osteomyelitis
- Delayed union, nonunion, or malunion
- Amputation or skin loss
- Posttraumatic arthritis
- Fat embolism

Prognosis

- Tibia and fibula fractures
 - Prognosis is generally good yet is dependent on degree of soft-tissue injury and bony comminution.
 - Prognosis is good for isolated fibula fractures.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to recognize and treat associated life-threatening injuries
- Failure to consider ankle injury with proximal fibula fracture (Maisonneuve fracture)
- Failure to recognize open injuries and obtain timely orthopedic consultation
- Failure to recognize compartment syndrome

Special Concerns

- Toddler fracture
 - Typically, this type of fracture is nondisplaced spiral fracture of distal tibia unrelated to child abuse.
 - Midshaft tibial fractures, unrelated to a history of major trauma, should alert emergency physician to possibility of child abuse.
-

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Fractures, Wrist

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Synonyms, Key Words, and Related Terms

carpal bone fracture

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Introduction

Background

The wrist is the most commonly injured region of the upper extremity. Fractures of the distal radius and ulna account for three fourths of wrist injuries. The carpal bones themselves are injured much less frequently but account for up to 10% of injuries to the structures of the hand. Not only are these injuries frequently encountered in the emergency department setting, but the mobility and delicate functional requirements of the hand make accurate diagnosis and treatment crucial to avoiding long-term loss of function and disability.

Pathophysiology

Anatomic considerations

The wrist or carpus is a highly mobile structure composed of a large number of small bones and joints. This complex system of articulations works in unison to provide a global range of motion for the wrist joint. Motion at the wrist joint occurs between the radius and the carpal bones, which function as a single unit, and between the carpals and metacarpals.

Carpal bones

The 8 carpal bones are arranged in 2 rows to form a compact, powerful unit. Each is cuboid with 6 surfaces, 4 covered with cartilage to articulate with the adjacent bones and 2 roughened for ligamentous attachment. The proximal carpal row contains the scaphoid (also called the navicular), lunate, triquetrum, and pisiform. It articulates proximally with the radius and the triangular cartilage. The ulna does not articulate directly with the carpus but is separated from the triquetrum by a triangular fibrocartilage, which acts as a stabilizing structure. The distal carpal row contains the trapezium, trapezoid, capitate, and hamate and articulates with the 5 metacarpals.

Joints and ligaments

The wrist includes 5 large joint cavities in addition to the intercarpal joint spaces: the radiocarpal joint, the distal radio-ulnar joint (DRUJ), the midcarpal joint, the large carpometacarpal joint (between the carpus and the second, third, fourth, and fifth metacarpals), and the small carpometacarpal joint (between the first metacarpal and the trapezium).

The strength of the wrist is dependent upon the integrity of the ligamentous network, which links the carpus together. The volar carpal ligament extends from the trapezium to the hook of the hamate and forms the anterior roof of the osseous/fibrous tunnel. Within this tunnel lie the tendons for the finger flexors and the median nerve. Encroachment on this space results in median nerve compression. The second and third metacarpals are fixed at their bases and are immobile.

The muscles of the hand originate primarily in the forearm and pass over the wrist. The only muscle with insertion into the wrist is the flexor carpi ulnaris, which inserts into the pisiform, a small sesamoid bone.

Movement of the wrist is 80° in flexion, 70° in extension, 30° in ulnar deviation, and 20° in radial deviation. Pronation and supination occur at the radial-ulnar articulation in the forearm, not at the wrist.

Neurovascular anatomy

The wrist comprises several important neurovascular structures. The deep branches of the ulnar nerve and the ulnar artery run deep to the flexor carpi ulnaris tendon through the Guyon canal. They pass in close proximity to the hamate and capitate and can be involved with injuries to these structures. The

ulnar nerve innervates the intrinsic muscles of the hand, including the hypothenar muscles, interossei, ulnar lumbricals, and adductor pollicis.

The median nerve lies between the flexor carpi radialis and the palmaris longus tendon in the carpal tunnel. The median nerve innervates the thenar compartment and provides sensation to the radial portion of the hand. Any displacement of the normal anatomic alignment of the wrist can injure this nerve.

The blood supply to the hand is via the radial and ulnar arteries, which form the dorsal palmar arch. The scaphoid bone receives its blood supply from the distal part of this arch, which is prone to injury.

Surface anatomy

Several anatomic landmarks are important in performing an accurate and thorough examination of an injured wrist. The anatomic snuffbox lies on the radial aspect of the dorsum of the wrist. It is defined in the ulnar aspect by the tendon of the extensor pollicis longus and radially by the tendons of the extensor pollicis brevis and abductor pollicis longus. The floor is composed proximally of the scaphoid and distally of the trapezium. The anatomic snuffbox is seen most easily with the thumb held in a position of extension with the wrist slightly deviated in the radial aspect.

The next landmark is the Lister tubercle, a bony prominence over the dorsum of the distal radius. With the hand held in neutral, a line drawn between the third metacarpal and the Lister tubercle will cut through the capitate distally and the lunate proximally. Just distal to the ulnar styloid, the triquetrum can be palpated. At the base of the hypothenar eminence on the volar aspect of the wrist lies the pisiform. The hook of the hamate can be felt with deep palpation of the palm approximately 1 cm distant from the pisiform along a line pointing to the index metacarpophalangeal (MCP) joint.

Carpal fractures and dislocations

Scaphoid fracture

The scaphoid bone is based in the proximal row of carpal bones but extends into the distal row, making it more vulnerable to injury than the other carpal bones. It is the most frequently injured carpal bone, accounting for 60-70% of all carpal fractures. It is also a frequently missed injury, as approximately 10-15% of fractures are not demonstrated on routine x-rays. More than three fourths of all fractures occur at the narrow midportion or waist of the scaphoid. Because blood is supplied to the scaphoid along its dorsal surface near its waist, fractures at this location potentially compromise flow to the proximal portion of the bone. As a result, avascular necrosis is a serious complication of this injury.

Mechanism of injury: Hyperextension of the wrist is the most common mechanism of scaphoid fracture, either by a fall on an outstretched hand or by a direct blow to the palm. Often the wrist has some degree of radial deviation. Hyperextension causes the radial styloid to impinge on the waist of the scaphoid as it crosses between the two rows of carpal bones. Scaphoid fractures often are associated with other injuries

of the wrist, including dislocation of the radiocarpal joint, dislocation between the 2 rows of carpal bones, fracture-dislocation of the distal end of the radius, fracture at the base of the thumb metacarpal, and dislocation of the lunate.

Lunate fracture

Although a relatively uncommon injury, fracture of the lunate is the third most frequent carpal bone fracture. The lunate is located in the center of the proximal carpal row and articulates with the radius. Fractures can occur in any orientation, and diagnosis often requires a high degree of clinical suspicion.

Fractures of the lunate most often result from hyperextension of the wrist or impact of the heel of the hand on a hard surface. This injury also can occur from a fall on the outstretched hand. Patients usually present with weakness of the wrist and pain aggravated with compression along the third digital ray.

Triquetrum fracture

The triquetrum is one of the more commonly injured carpal bones. It lies on the ulnar aspect of the proximal row of carpal bones. Strong ligaments attach the triquetrum to the lunate, which adjoins its radial aspect. In addition, the triquetrum is connected to the distal ulna by a triangular fibrocartilage complex.

The most common mechanism of injury is forced hyperextension of the wrist with ulnar deviation. In this position the triquetrum is forced against the ulnar styloid, generating a shearing force that results in avulsion of ligaments and a dorsal chip fracture of the triquetrum. A second, less common, mechanism is a direct blow to the dorsum of the hand, which causes a transverse fracture through the body of the triquetrum. This is a high-energy injury and frequently is associated with injury to other carpal bones.

Capitate fracture

The capitate is the largest carpal bone and articulates with 7 other bones, including the second, third, and fourth metacarpals. It is located in the center of the distal row of carpal bones. Axial motion of the third metacarpal depends upon a functional articulation with the capitate. These fractures account for fewer than 10% of carpal bone injuries and usually are transversely oriented. Blood supply to the capitate enters its dorsal segment and often is disrupted following fracture, resulting in avascular necrosis.

Mechanism of injury: Two mechanisms of injury are common in capitate fractures. Like most carpal bones, the capitate can be injured by a fall on an outstretched hand with forced dorsiflexion and a degree of radial deviation of the wrist. In this position the dorsal lip of the radius is able to strike the body of the capitate. A second mechanism is a direct blow or crush injury to the dorsum of the wrist.

Hamate fracture

The hamate occupies the ulnar aspect of the distal row of carpal bones. It is an unusually shaped bone, with a hook that protrudes toward the palmar surface and serves as an attachment site for several ligaments. The most common injury pattern is a fracture through the base of this hook. This is an uncommon injury.

The hook of the hamate typically is fractured by a direct blow when the hand is held slightly dorsiflexed and with some degree of ulnar deviation. A common history is that a golf club, racket, or bat struck a stationary object during full swing resulting in immediate pain over the hypothenar eminence. This pain is exacerbated by any type of gripping activity.

Trapezium fracture

Fractures of the trapezium are rare, comprising no more than 5% of fractures of the carpal bones. Fractures of the body of the trapezium result when an adducted thumb is forced onto the articular surface of the carpal bone. In addition, forced radial deviation of the thumb may result in small avulsion fractures due to capsular strain.

Trapezoid fracture

Fractures of the trapezoid are quite rare, accounting for fewer than 1% of carpal bone fractures. The mechanism of injury is axial loading along the line of the second metacarpal.

Pisiform fracture

The pisiform is a sesamoid bone within the tendon of the flexor carpi ulnaris. It articulates only with the triquetrum and lies in close proximity to the deep ulnar nerve and artery. Fractures are quite rare. The pisiform typically is injured by a fall on an outstretched hand in a dorsiflexed position, with the impact on the hypothenar eminence.

Lunate and perilunate dislocation

Dislocations of the carpal bones are usually the result of extreme flexion or extension of the wrist. The type of dislocation or fracture-dislocation produced by these mechanisms depends upon the direction and intensity of the injuring force and the position of the hand in relation to the forearm at the moment of impact. The integrity of the lunate-capitate relationship is the most crucial factor in all dislocations of the wrist. The resulting lesions are related directly to disruption or preservation of this articulation.

These rare injuries may have a poor outcome if not recognized in a timely fashion. The exact diagnosis often can be difficult by x-ray. Four specific projections can help when taking comparison radiographs: anteroposterior (AP), lateral, 45° of pronation, and 45° of supination. An accurate history can be a clue to the diagnosis. Knowledge of the exact mechanism of injury can allow prediction of the resulting dislocation.

Extension injuries

Dorsal perilunate or volar lunate dislocation is caused when the hand is forced into extension, such as in a fall on the outstretched hand. Commonly, a fracture or fracture-dislocation of the scaphoid complicates the dorsal perilunate dislocation.

Flexion injuries

Dorsal dislocation of the lunate can occur when the hand and carpus are hyperflexed, as occurs with a fall onto the back of the hand. The upward force generated when the hand contacts the ground, together with the downward force acting through the radius, forces the capitate to rotate anteriorly and drive the lunate backward into a dorsal position.

With the volar perilunate dislocation, the lunate remains in its normal position relative to the radius and the rest of the carpus dislocates anteriorly. This often is associated with a scaphoid fracture.

Fractures of the distal radius and ulna

Fractures of the distal radius and/or ulna account for approximately three quarters of bony injuries of the wrist. The radius articulates directly with the carpal bones; the ulna has attachments to the triangular fibrocartilage, which is interposed between the distal ulna and the triquetrum in the proximal row of carpal bones. The radius and ulna themselves articulate at the DRUJ, about which occurs the movements of supination and pronation at the wrist. They are enveloped in a common joint capsule and share multiple ligamentous attachments. Along the midshaft of both bones is the interosseus membrane. Several muscle groups attach on the distal aspect of both bones and contribute to the displacement of fracture fragments.

Extension fractures of the distal radius

Multiple classification schemes have been developed for extension injuries of the distal radius. These tend to be complex and cumbersome. In general, however, the greater the degree of displacement and comminution, the more severe the injury. Extension of a fracture into the radiocarpal or the DRUJ is also a marker for a more severe injury. More complex fractures tend to be more unstable.

Extension fractures result from a fall on an outstretched pronated hand with the impact on the palm and subsequent forced dorsiflexion or hyperextension. On striking a hard surface, the hand becomes fixed while the momentum of the body produces the following 2 forces:

- Twisting force that causes excessive supination of the forearm
- Compression force that acts vertically through the carpus to the radius

The lunate acts as the apex of a wedge against the articular surface of the radius and causes injuries that vary by the age of the patient. Very young children usually sustain a greenstick fracture of the distal radius, with or without an associated fracture of the distal ulna. In adolescents, the lower epiphysis separates, with dorsal displacement or crushing. In adults, fracture occurs within 1 inch of the carpus. The distal fragment usually is displaced upward and backward. In all age groups, the fracture may be complicated by injury to the median nerve or the sensory branch of the radial nerve and/or by fracture of the scaphoid or dislocation of the lunate.

If a concomitant supinating force is applied, often the distal ulna also fractures. Approximately 60% of distal radius fractures are associated with fracture of the ulnar styloid. Approximately 60% of ulnar styloid fractures also have an associated fracture of the ulnar neck.

Colles fracture is the most common extension fracture pattern. The term classically is used to describe a fracture through the distal metaphysis approximately 4 centimeters proximal to the articular surface of the radius. However, now the term tends to be used loosely to describe any fracture of the distal radius, with or without involvement of the ulna, that has dorsal displacement of the fracture fragments.

Colles fractures occur in all age groups, although certain patterns follow an age distribution. In the elderly, because of the relatively weaker cortex, the fracture is more often extraarticular. Younger individuals tend to require a relatively higher energy force to cause the fracture and tend to have more complex intraarticular fractures. In children with open physes, an equivalent fracture is the epiphyseal slip. This is a Salter I or II fracture with the deforming forces directed through the weaker epiphyseal plate.

Flexion fractures of the distal radius (reverse Colles fracture/Smith fracture)

Smith fracture is relatively uncommon compared with the Colles fracture. This term is used loosely to refer to any fracture of the distal radius, with or without involvement of the ulna, that has volar displacement of the distal fragments. A true Smith fracture comprises a fracture of the entire thickness of the distal radius, 1-2 cm above the wrist. The lower end of the radius is displaced forward and upward.

This fracture typically is caused by a fall onto a supinated forearm/hand with generation of a hyperflexion force. On striking the ground, the hand locks in supination while the body's momentum forces the hand into hyperpronation. A direct blow to the dorsum of the wrist with the hand in flexion and forearm pronated also can produce a similar fracture pattern. Another mechanism is punching with the wrist in a slightly flexed position.

Pseudocarpal injuries

Pseudocarpal injuries are those that involve the distal end of the radius and ulna just proximal to the carpus and manifest with clinical signs that mimic carpal bone injuries. Specifically, these include articular disk injuries of the wrist, dislocations of the inferior radioulnar joint, and traumatic dislocation

of the distal end of the ulna. These are rare injuries and require orthopedic consultation for definitive management. Recognition of these injuries in the ED is important if functional outcome is to be optimized.

Wrist articular injuries

Injury to the articular disk of the wrist occurs from multiple mechanisms. It usually coexists with other more common injuries, but isolated injuries to the articular disk can occur. The most common pathologic defect is tearing of the disk from its attachment at the margin of the ulnar notch of the radius. The primary function of the triangular disk of the wrist is to prevent lateral displacement of the ulna. The most common mechanism of injury is dorsiflexion and pronation of the hand. Less frequently, extreme hyperextension and supination may cause injury. Volar or dorsal dislocation of the head of the radius may coexist.

The Barton or push-off fracture is an intraarticular injury involving either the dorsal or volar articular surface of the radius. It is an uncommon fracture pattern. This type of fracture generally is seen with extreme dorsiflexion of the wrist with concomitant exertion of a pronating force.

Traumatic dislocation of the distal ulna

Dislocation or subluxation of the distal ulna most often is associated with fractures of the radius. However, acute traumatic dislocation/subluxation of the head of the ulna without fracture can occur and often is not recognized in the ED.

The ulnar head may be displaced anteriorly or posteriorly, depending on the mechanism of injury. Extreme extension and pronation of the hand produces a dorsal dislocation of the head of the ulna. Extreme extension and supination of the hand produces a volar dislocation of the ulnar head.

Radial styloid fracture

Hutchinson fracture, an isolated fracture of the radial styloid, typically is caused by a direct blow to the radial aspect of the wrist. It also may be referred to as "chauffeur's fracture" or "backfire fracture" as it initially was described in individuals struck by the hand crank on early automobiles when the engine suddenly backfired during starting.

Frequency

- **In the US:** Fractures of the distal radius account for one sixth of all fractures seen and treated in the ED.

Mortality/Morbidity

Little or no risk of death is associated with isolated wrist fractures. The potential does exist for substantial morbidity, including primarily arthritis, chronic pain, limitation of motion, and physical deformity. Morbidity also may be related to associated injuries, including those of the median and ulnar nerves and the radial and ulnar arteries.

Age

Patients aged 6-10 years and those aged 60-69 years have the greatest frequency of distal radius fractures. Injuries to the carpal bones are common in all age groups but more common in adolescents.

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Clinical

History

- Fall onto an outstretched hand
- Direct trauma

Physical

Physical examination should begin with inspection of the injured extremity using the uninjured as a comparison. The site of injury may be identified by ecchymosis or swelling. Fractures of the distal radius may have characteristic deformities. Look for any evidence of a break in the skin indicating an open fracture. Palpation with localization of the point of maximal tenderness further defines the injury.

- With scaphoid fractures, the point of maximal tenderness lies in the anatomic snuffbox, which lies between the tendons of the extensor pollicis brevis and abductor pollicis longus. Radial deviation of the wrist or axial loading of the first metacarpal may increase pain.
- The lunate can be localized just distal to the Lister tubercle, which is palpable on the dorsal radius. Axial loading of the third metacarpal may increase pain with a lunate injury. In addition, lunate fractures may be associated with point tenderness over the lunate fossa (located distal to the radius at the base of the long finger metacarpal).
- The classic finding in a Colles fracture is the so-called dinner fork deformity, which is produced by dorsal displacement of the distal fracture fragments. A Smith fracture may show an obvious volar displacement of the wrist relative to the forearm, known as a garden spade deformity.
- Examine the remainder of the injured extremity for tenderness or other signs of injury to exclude an associated injury to the elbow, upper arm, or shoulder. Particularly with injuries to the lunate, capitate, and pisiform, which represent high-energy mechanisms, maintain a high suspicion for

concomitant injury to other structures of the wrist. A practical piece of advice is to examine last the region identified by the patient as the most painful; this prevents additional pain from the physical examination from masking more subtle injuries to other structures.

- Next, assess the neurovascular integrity of the injured extremity. Evaluate pulses in the brachial and radial arteries. Look for any evidence of impaired circulation such as cyanosis or pallor. Injuries to the ulnar aspect of the hand, particularly those involving the pisiform, hamate, and triquetrum, may place the deep branch of the ulnar artery at risk as it travels beneath the hook of the hamate. The radial artery can be jeopardized with any significant displacement of the distal radius.
- The hand is innervated by 3 nerves, the radial, ulnar, and median. Assess their integrity in all injuries. The deep branch of the ulnar nerve, which supplies most of the intrinsic muscles of the hand, runs with the ulnar artery beneath the hook of the hamate and is vulnerable with injuries to the pisiform, hamate, and triquetrum. Injuries at this point spare the sensory function of the ulnar nerve, which branches more proximally. The median nerve is particularly vulnerable with injuries to the lunate and the distal radius. It may be compromised by swelling, resulting in an acute carpal tunnel syndrome, or may be injured directly. The sensory branch of the radial nerve may be compromised with a dorsally displaced Barton fracture.

Causes

- Distal radius, scaphoid, and lunate fractures usually are the result of a fall on an outstretched hand.
- Wrist fractures may be caused by hyperflexion mechanisms and by direct blows to the wrist.

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Differentials

Dislocations, Wrist

Fractures, Forearm

Fractures, Hand

Tendonitis

Tenosynovitis

Other Problems to be Considered

Falls in older persons

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Workup

Lab Studies

- No lab studies are indicated in patients with isolated wrist injury.

Imaging Studies

- Radiographs
 - Routine radiographs of the wrist include AP, lateral, and oblique views. These are adequate to identify most fractures. Look for evidence of displacement of carpal bone fractures, as this often indicates the need for operative intervention.
 - When evaluating a fracture of the distal radius or ulna, carefully check the normal anatomic alignments. The radiocarpal joint viewed on the lateral film normally has 11° of palmar angulation with a range of 1° to 23°. Ulnar angulation on the AP film is normally 15- 30°. The radial length, which is the distance between the ulnar aspect of the distal radius and the tip of radial styloid, normally measures 11-12 mm.
 - Look for an associated ulnar styloid fracture and involvement of the radiocarpal joint or DRUJ. If the radius appears to be angulated and/or displaced significantly, maintain a high degree of suspicion for a concomitant fracture of the ulna.
 - Scaphoid fractures often are not seen on routine x-rays. Scaphoid views taken with the wrist deviated toward the ulna and slightly supinated may help to demonstrate a fracture. The approximately 10-15% of fractures that are occult may be apparent on plain films after 10-14 days as bony reabsorption occurs at the fracture site. While not appropriate for ED workups, CT scans and bone scans as early as 3 days after injury also may aid in the diagnosis.
 - Injuries to the hamate and trapezium can be visualized best with a carpal tunnel view.
 - Like scaphoid injuries, injuries to the lunate and capitate may not be well visualized on plain films, and CT scan may be required.

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Treatment

Prehospital Care

- The injured extremity should be splinted gently from above the elbow to the hand to prevent additional injury from inadvertent manipulation.
- As with all trauma, address the possibility of additional injuries. Attend to ABCs, and use spine precautions if indicated by history and mechanism.
- Urgent reduction of fractures may be necessary when neurovascular status has been compromised. This should be done in the prehospital setting only when estimated ED arrival is more than 6 hours after the time of injury.

Emergency Department Care

In the ED, obtain a thorough history. Exclude additional injuries, and, if warranted, provide a full trauma evaluation. Maintain gentle, temporary splinting when not directly examining the injured wrist.

- These fractures are managed by reduction and immobilization following administration of adequate anesthesia and analgesia.
- Prior to closed reduction and fixation, but after a careful neurovascular exam, administer proper sedation/anesthesia for the following 2 reasons:
 - Reduce or eliminate discomfort to the patient
 - Reduce muscle spasm and splinting, which allows easier reduction and stabilization
- Reduction and immobilization: Always assess and document neurovascular status before starting reduction. Accurate reduction of the fracture is essential to obtaining good functional results. Early reduction lessens morbidity and improves patient comfort. Anatomic reduction is obtained by manipulation and plaster fixation and confirmed by repeat radiographs. The method of immobilization varies with the specific injury involved.
- Colles fracture
 - The 2 keys to successful reduction of the typical Colles fracture are as follows:
 - Obtain postreduction x-rays; assess and document neurovascular status of the extremity after reduction. Document function of the median nerve and the sensory branch of the radial nerve.
- Volar and dorsal dislocations
 - For volar dislocations, the hand is hyperpronated. For dorsal dislocations, it is hypersupinated. A sugar tong splint is then placed. For volar dislocations, the hand is splinted fully pronated, while for dorsal dislocations, the hand is splinted in supination.
 - Appropriate consultation by an orthopedist must follow within the next 48 hours.
- Other carpal fractures
 - Lunate fractures require a short-arm spica cast or splint with thumb immobilization.
 - Emergency treatment of capitate, trapezium, and trapezoid fractures consists of position of function and orthopedic consultation.
 - Fractures of the pisiform can be immobilized with a volar splint.
 - Injuries to the triquetrum are best treated with a sugar tong splint.
 - Treatment of a hamate fracture involves a short-arm cast with the fourth and fifth MCP joints held in flexion.

- Nerve injury
 - Upon presentation and after treatment, the ED physician must evaluate the neurovascular status of the extremity. Careful note must be taken of ulnar and median nerve function.
 - The ulnar nerve often is injured with closed fractures of the pisiform, triquetrum, hamate, and fourth and fifth metacarpals.
 - The motor branch of the ulnar nerve is the chief motor nerve of the hand.
 - The sensory branch rarely is affected.
 - Blunt trauma to the hypothenar eminence may result in contusion to the ulnar nerve, with resulting neuropraxia.
 - If a large hematoma is present, it may be aspirated or surgically removed after appropriate consultation.

Consultations

- Obtain immediate consultation with a hand specialist or orthopedic surgeon for open or unstable fractures and those requiring fixation.
- All other fractures should have adequate orthopedic follow-up care to insure proper wrist function.

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Medication

Drugs used to treat fractures include analgesics and anxiolytics. In addition, proper antibiotics must be administered for open fractures.

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Most analgesics have sedating properties that benefit patients who have sustained traumatic injuries.

| | |
|----------------|--|
| Drug Name | Fentanyl (Duragesic)- Short duration (30-60 min), ease of titration, and rapid and easy reversal by naloxone make this an excellent choice for pain management and sedation. |
| Adult Dose | 2-3 mcg/kg IV/IM |
| Pediatric Dose | 1-2 mcg/kg/dose IV/IM q30-60 min; not to exceed 3 mcg/kg/h |

| | |
|-------------------|--|
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hypotension, respiratory depression, constipation, nausea, emesis, and urinary retention; idiosyncratic reaction, known as chest wall rigidity syndrome, may require neuromuscular blockade to increase ventilation |

| | |
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| Drug Name | Morphine sulfate (Duramorph, Astramorph, MS Contin)- DOC for narcotic analgesia because of its reliable and predictable effects, safety, and ease of reversibility with naloxone. Administered IV, may be dosed in a number of ways and commonly is titrated until desired effect obtained. |
| Adult Dose | Starting dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h Relatively hypovolemic patients: Start with 2 mg IV/IM/SC and reassess hemodynamic effects of dose |
| Pediatric Dose | Neonates: 0.05-0.2 mg/kg IV/IM/SC prn; not to exceed 15 mg/dose IV Children: 0.1-0.2 mg/kg q2-4h prn |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

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| Drug Name | Propoxyphene and acetaminophen (Darvocet N-100)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 1-2 tab PO q4h prn; not to exceed 600 mg/d |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | May increase serum concentrations of MAOIs, tricyclic antidepressants, carbamazepine, phenobarbital, and warfarin |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

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|-------------|---|
| Precautions | Caution in patients dependent on opiates, substitution may result in acute opiate withdrawal symptoms; caution in severe renal or hepatic dysfunction |
|-------------|---|

| | |
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| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tabs q4h; not to exceed 12 tabs/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine content PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|---|
| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab/cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen PO q4-6h prn; not to exceed 2.6 g/d acetaminophen >12 years: 750 mg acetaminophen PO q4h; not to exceed 10 mg hydrocodone bitartrate per dose or 5 doses/24 h |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

Anxiolytics

Patients with painful injuries usually experience significant anxiety. Anxiolytics allow a smaller analgesic dose to achieve the same effect.

| | |
|-------------------|---|
| Drug Name | Lorazepam (Ativan)- Sedative hypnotic in benzodiazepine class that has short onset of effect and relatively long half-life. By increasing action of GABA, a major inhibitory neurotransmitter, may depress all levels of CNS, including limbic and reticular formation. |
| Adult Dose | 1-10 mg/d PO/IV/IM divided bid/tid |
| Pediatric Dose | 0.05-0.1 mg/kg IV slowly over 2-5 min; may repeat dose of 0.05 mg/kg IV slowly |
| Contraindications | Documented hypersensitivity; preexisting CNS depression; hypotension; narrow-angle glaucoma |
| Interactions | Alcohol, phenothiazines, barbiturates, and MAOIs increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in renal or hepatic impairment, myasthenia gravis, organic brain syndrome, or Parkinson disease |

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|-------------------|---|
| Drug Name | Alprazolam (Xanax)- Indicated for treatment of anxiety and management of panic attacks. |
| Adult Dose | 0.25-0.5 mg PO tid; average dose proven effective is 0.5-4 mg/d |
| Pediatric Dose | < 18 years: Not established |
| Contraindications | Documented hypersensitivity; severe respiratory depression; narrow-angle glaucoma; preexisting hypotension |
| Interactions | Carbamazepine and disulfiram decrease effects; cimetidine, lithium, contraceptives, and CNS depressants (including alcohol) increase toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Withdrawal symptoms, including seizures, may occur upon abrupt discontinuation of drug |

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|-------------------|--|
| Drug Name | Midazolam (Versed)- DOC for acute sedation/anxiety as adjuvant for reduction of acute fracture/dislocations. Titratable effect and anterograde amnesia for 1-2 h make this ideal agent. Onset of action within 2 min and effective duration of action 30 min IV and 45 min IM. |
| Adult Dose | 0.15 mg/kg IV/IM; titrate IV dosage to effect in 0.02 mg/kg increments; 0.1 mg/kg IM supplementation |
| Pediatric Dose | 0.1-0.15 IM mg/kg IV initial dose: 0.05-0.1 mg/kg |
| Contraindications | Documented hypersensitivity; preexisting hypotension; narrow-angle glaucoma; sensitivity to propylene glycol (diluent) |

| | |
|--------------|---|
| Interactions | Sedative effects may be antagonized by theophyllines; narcotics and erythromycin may accentuate sedative effects due to decreased clearance |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in congestive heart failure, pulmonary disease, renal impairment, and hepatic failure |

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Follow-up

Further Inpatient Care

- Open fracture and/or joint capsule injury require the following treatments:
 - Extensive irrigation (2-3 L)
 - Administration of antibiotics (eg, cephalexin, gentamicin)
 - Emergent operative treatment and hospital admission

Further Outpatient Care

- Distal radius fracture
 - Once swelling has subsided, uncomplicated fractures require conversion from a splint to a short-arm cast for 6-8 weeks.
 - An orthopedic specialist should provide follow-up to assess for adequate alignment and the need for operative intervention.
 - Patient may require physical therapy to regain baseline range of motion.
- Lunate fracture: Most heal in a spica cast for 10-12 weeks.

in/Out Patient Meds

- Oral analgesics should provide sufficient pain relief.
- To reduce pain and edema, apply ice to the injured region for the first 48 hours.

Transfer

- When proper orthopedic care is not available at a site, transfer the patient to a higher level care facility once neurovascular stability has been addressed adequately.

Deterrence/Prevention

- Since a large number of wrist fractures occur secondary to in-line skating accidents and other sporting activities, encourage wrist protection during these sports.

Complications

- The anatomy of the scaphoid bone makes it vulnerable to secondary injury. It is supplied by a single blood vessel that penetrates the cortex near the waist of the scaphoid. Scaphoid fractures are prone to delayed healing and avascular necrosis. The more proximal the fracture, the more common these complications. Missed diagnosis and lack of appropriate immobilization increase this risk. Missed diagnosis or nonunion predisposes an individual to development of potentially debilitating radiocarpal arthritis.
- Kienbock disease is osteonecrosis and subsequent collapse of the proximal portion of the lunate resulting in pain, loss of function, and carpal bone instability. The exact mechanism for development of this condition is disputed, with theories ranging from repetitive microtrauma to avascular necrosis from a single injury. As the lunate receives its blood supply from a single distal blood vessel in 20% of individuals, these patients may be predisposed to avascular necrosis and nonunions. Younger patients, typically those younger than 16 years, tend to have better functional outcomes from lunate injuries than older patients.
- Complications from a capitate fracture include nonunion and avascular necrosis as, like the scaphoid, it is dependent on a single blood vessel, which enters from its distal aspect. Posttraumatic arthritis is a frequent complication. Fibrosis of surrounding tissues after injury may result in carpal tunnel syndrome.
- Fractures through the base of the hook of the hamate frequently are displaced by the forces of the hook's multiple ligamentous attachment. Nonunion is a frequent complication and may necessitate surgical excision of the hook to relieve pain from grasping activities.
- Acutely, a Colles fracture has several potential complications. These include compression or contusion of the median and/or ulnar nerves. An acute carpal tunnel syndrome may result from swelling. The flexor tendons may be injured by the bony fragments. Excessive swelling can result in compartment syndromes. Comminuted or severely displaced fractures may be unstable, resulting in a loss of reduction and requiring repeated attempts or surgical intervention.
- Long term, the wrist may have radial shortening and angulation deformity, limiting range of motion. Some individuals experience chronic pain, particularly with supination. Adhesions may limit mobility of the flexor tendons. As with all fractures, malunions or nonunions may complicate healing. With comminuted intraarticular fractures, more than two thirds may be complicated by the late development of arthritis.
- Reflex sympathetic dystrophy complicates some 3% of distal radius fractures. This controversial diagnosis is a syndrome of paresthesias, pain, stiffness, and changes in skin temperature and color.
- Smith (reverse Colles) fracture may result in complications similar to those of Colles fracture.
- Radiocarpal fracture-dislocation may cause entrapment of tendons or of the ulnar nerve and/or artery.

- Hutchinson fracture may result in scapholunate dislocation, osteoarthritis, or ligament damage.
- Ulnar styloid fracture often results in nonunion.

Prognosis

- Prognosis depends on many variables, including the following:
 - The outcome of injuries to the distal radius and ulna is determined largely by the degree to which normal anatomic relationships can be restored. Shortening of the radius is a key determinant of prognosis. In general, the more complex the fracture pattern, the worse the outcome. This often takes the form of loss of mobility and debilitating early onset arthritis.
 - Open fractures with large soft-tissue injuries have a much poorer prognosis.
 - Timely and appropriate care can improve the prognosis.
 - Appropriate follow-up and aggressive rehabilitation are extremely important.
 - With appropriate immobilization, 95% of scaphoid fractures heal with casting for 8-12 weeks.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to check for neurologic compromise before and after splinting
 - Failure to meet standard of care for suspected scaphoid fracture. The ED physician must apply a thumb spica splint to avoid the complication of avascular necrosis.
 - Failure to test for pain in the anatomic snuff box as a sign of a possible scaphoid fracture. If pain is present, splint and refer for specialty consultation and/or further imaging techniques.
-

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Gamekeeper Thumb

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Synonyms, Key Words, and Related Terms

skier thumb, injury to ulnar collateral ligament (UCL), hyperabduction of thumb

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Introduction

Background

Gamekeeper thumb originates from Scottish gamekeepers and is a chronic injury to the ulnar collateral ligament (UCL) resulting in laxity and instability of the thumb. Today, this injury usually occurs acutely from extreme abduction of the thumb (observed in snow skiers). If not diagnosed and treated properly, laxity may result in persistent pain or a weak grasp.

Pathophysiology

Injury occurs at the metacarpophalangeal (MCP) thumb joint on the ulnar side. This joint allows flexion, extension, abduction, and adduction, but limited rotation. On the ulnar aspect, the adductor pollicis

inserts into the volar plate and the proximal phalanx. An aponeurosis, an extension of the dorsal aponeurosis, overlies the ulnar collateral ligament. This collateral ligament provides passive stability of the joint, while the aponeurosis provides active stabilization.

Table 1. Classification Scheme for Injuries to the UCL

| | |
|--------|------------------------------------|
| Type 1 | Avulsion fracture, nondisplaced |
| Type 2 | Avulsion fracture, displaced |
| Type 3 | Torn ligament, stable in flexion |
| Type 4 | Torn ligament, unstable in flexion |

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Clinical

History

- Mechanism of injury usually is a fall onto an outstretched hand with the thumb in abduction.
- Fall can occur while holding a ski pole or in other sporting events (eg, motorcycling, football).

Physical

- Examination reveals tenderness along the ulnar collateral ligament and possible edema and ecchymosis.
- Radiographs generally are obtained prior to stress testing the ulnar collateral ligament. Presence of a fracture negates the need for stress testing.
- Stress testing
 - Stressing a partially injured ligament elicits pain; consider using local or regional anesthesia prior to testing.
 - Stress the MCP joint in extension, then in 30° of flexion.
 - Positive result (somewhat controversial) is normally greater than 35° in flexion or greater than 10° more mobile than the opposite side.
 - Patient often is unable to hold a piece of paper securely between the thumb and index finger.
- Carefully document neurovascular function and examine the hand and wrist for associated injury, particularly of the scaphoid bone. Injuries to this area in children may have false laxity as result of Salter-Harris fracture.

Causes

Fall onto outstretched hand with thumb in abduction

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Differentials

Arthritis, Rheumatoid

Fractures, Hand

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Workup

Imaging Studies

- Obtain plain radiographs prior to stress testing.

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Treatment

Emergency Department Care

- Adult cases
 - Immobilize thumb with a thumb spica cast or splint.
 - A palmar splint does not protect the metacarpophalangeal joint from further injury.

Consultations

Orthopedic or hand surgery: Injuries that require operative fixation generally are best discussed directly with an orthopedic surgeon or a hand surgeon to ensure timely repair.

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Medication

The goal of pharmacotherapy is to reduce pain and morbidity. Analgesics, including narcotic analgesics or NSAIDs, can be given as necessary.

Analgesics

Pain control is essential to quality patient care. Analgesics ensure patient comfort and have sedating properties, which are beneficial for patients who have sustained trauma.

| | |
|-------------------|---|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually the DOC for treating of mild to moderate pain, if no contraindications exist. Inhibits inflammatory reactions and pain probably by decreasing the activity of the enzyme cyclo-oxygenase, which results in prostaglandin synthesis. |
| Adult Dose | 400 mg q4-6h, 600 mg q6h, or 800 mg q8h PO while symptoms persist; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid |
| Contraindications | Documented hypersensitivity; peptic ulcer disease, recent GI bleeding or perforation, renal insufficiency, or high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

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| Drug Name | Acetaminophen (Tylenol, Panadol, Aspirin-free Anacin)- DOC for treating mild pain in patients with documented hypersensitivity to aspirin or NSAIDs, diagnosed with upper GI disease, or who take oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d Alternative: 1000 mg PO tid/qid up to a maximum of 4 g/d |

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| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg PO q4h; not to exceed 5 doses/d |
| Contraindications | Documented hypersensitivity; known G-6-P deficiency |
| Interactions | Rifampin can reduce analgesic effects of acetaminophen; coadministration with barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; APAP is contained in many OTC products and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

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| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderate pain. Inhibits inflammatory reactions and pain by decreasing activity of the enzyme cyclooxygenase, which results in prostaglandin synthesis. |
| Adult Dose | 500 mg PO followed by 250 mg q6-8h; not to exceed 1.25 g/d; may increase to 1.5 g/d for limited periods |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

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| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used to relieve mild to moderate pain and inflammation. Initially administer small dosages to patients with a small body size, elderly patients, and those with renal or liver disease. Doses higher than 75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe patients for response. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 14 years: 0.1-1 mg/kg q6-8h <12 years: 25-50 mg q6-8h prn; not to exceed 300 mg/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

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| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderate pain. |
| Adult Dose | Based on codeine content: 30-60 mg/dose q4-6h or 1-2 tab q4h; not to exceed 12 tab/d |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | Toxicity increases with CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates because this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

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| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated to relieve moderate to severe pain; the DOC for aspirin-hypersensitive patients. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone; not to exceed 5 mg/dose of oxycodone q4-6h prn |

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| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects of this medication; toxicity increases with coadministration of either CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in elderly patients; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/d of acetaminophen; higher doses may cause liver toxicity |

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| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin)- Drug combination indicated to relieve moderate to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen q4-6h prn; not to exceed 2.6 g/d of acetaminophen or 5 mg of hydrocodone bitartrate/dose >12 years: 750 mg acetaminophen q4h; not to exceed 5 doses/d acetaminophen or 10 mg of hydrocodone bitartrate/dose |
| Contraindications | Documented hypersensitivity; high altitude cerebral edema (HACE) or elevated intracranial pressure (ICP) |
| Interactions | Coadministration with phenothiazines may decrease analgesic effects; toxicity increases with CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

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Follow-up

Further Outpatient Care

- Provide early outpatient follow-up for injuries requiring casting, if casting is not performed in the ED.
- Arrange follow-up care with an orthopedic or a hand surgeon.
- Refer for surgical repair if a displaced avulsion fracture or a fracture involving more than 25% of the joint space is present. Surgical treatment of isolated ligamentous rupture is sometimes preferred.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to diagnose this ligamentous injury, especially in the dominant hand results in significant future morbidity
 - Failure to conduct ligamentous testing in patients with injuries to the thumb and normal radiographs
-

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Hand Injuries, High-Pressure

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Introduction

Background

A high-pressure injection injury should be considered a potential surgical emergency. Immediate decompression and thorough cleansing of the offending material from the tissue is required to preserve optimal function.

Pathophysiology

The acute injury is due to introduction of a foreign material, under high pressure between 2,000 and 10,000 psi, into the poorly distensible digital or palmar tissues. The pathophysiology involves acute and chronic inflammation and foreign body granuloma formation. Damage results from the impact, ischemia resulting from vascular compression, chemical inflammation, and secondary infection. Highly viscous substances (eg, grease) require higher injection pressures than paint or solvents.

Fuel and paint injections lead to the most severe inflammatory response with a high incidence of subsequent amputation. Grease- and oil-based compounds may lead to oleogranulomas with chronic fistula formation, scarring, and eventual loss of digit function.

Mortality/Morbidity

Overall incidence of amputation approaches 48%. Morbidity is dependent to a large degree upon the material injected. Paint solvents appear to cause the greatest damage and result in amputation in 60-80% of the cases. Grease, the more common injectant, causes a less severe inflammatory response. Amputation is necessary in about 25% of these patients.

Sex

These injuries are rare in women.

Age

This injury usually occurs in young men while working, most often to their nondominant index finger. The average age at time of injury in one large review was 35 years (range 16-65 y). These injuries occurred to the nondominant hand 76% of the time.

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Clinical

History

- The injection typically occurs to the fingertip when the operator is trying to wipe clear a blocked nozzle or to the palm when the operator is attempting to steady the gun with a free hand during the testing or operation of equipment.
- The left hand (usually nondominant) is involved in about two thirds of cases.
- The most common site of injury is the index finger.
- The palm and long finger are the next most frequently injured.

Physical

- The innocuous appearance of the wound may hide the severity of the injury.
- With time, edema and intense pain develop and the digit may appear erythematous or cold.

Causes

Most injuries have resulted from grease guns, paint sprayers, or diesel fuel injectors.

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Workup

Lab Studies

- Standard preoperative labs

Imaging Studies

- Preoperative roentgenograms may facilitate the surgical strategy by localizing subcutaneous air, debris, or unanticipated fractures.

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Treatment

Emergency Department Care

- Obtain roentgenograms.
- Prescribe prophylactic, broad-spectrum antibiotics.
- Update tetanus and administer parenteral analgesics.
- Splint the extremity and keep it elevated.
- Several authors report that steroids may be beneficial in selected cases, especially when the patient develops an intense inflammatory response or treatment is delayed.

Consultations

Refer these patients emergently to an experienced hand or orthopedic surgeon. Prompt surgical debridement optimizes tissue salvage.

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Medication

The goal of therapy is to prevent infections. Prophylactic broad-spectrum antibiotics are indicated.

Antibiotics

Therapy must cover all likely pathogens in the context of the clinical setting.

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| Drug Name | Cefazolin (Ancef, Kefzol, Zolicef)- DOC; first-generation semisynthetic cephalosporin which, by binding to one or more penicillin-binding proteins, arrests bacterial cell wall synthesis and inhibits bacterial growth. Primarily active against skin flora, including <i>Staphylococcus aureus</i> . |
| Adult Dose | 1 g IV/IM q6-8h for 5-7 d |
| Pediatric Dose | 25-50 mg/kg/d IV/IM divided tid/qid for 5-7 d |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid prolongs effect; aminoglycosides may increase renal toxicity; may yield false-positive urine dip test for glucose |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal impairment; superinfections and promotion of nonsusceptible organisms may occur with prolonged use or repeated therapy |

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| Drug Name | Trimethoprim/sulfamethoxazole (Bactrim)- Inhibits bacterial synthesis of dihydrofolic acid by competing with para-aminobenzoic acid, inhibiting folic acid synthesis and thus bacterial growth. Antibacterial activity of TMP-SMZ includes common urinary tract pathogens except <i>Pseudomonas aeruginosa</i> . |
| Adult Dose | 160 mg TMP or 800 mg SMZ PO q12h for 5-7 d |
| Pediatric Dose | <2 months: Not recommended Infants and children >2 months: 15-20 mg/kg/d (TMP dose) PO divided tid/qid for 5-7 d |
| Contraindications | Documented hypersensitivity; megaloblastic anemia due to folate deficiency Do not administer to infants <2 mo |
| Interactions | May increase PT when used with warfarin (perform coagulation tests and adjust dose accordingly); coadministration with dapsone may increase blood levels of both drugs; diuretics increase incidence of thrombocytopenia purpura in elderly; may increase phenytoin levels; may potentiate effects of methotrexate in bone marrow depression; may increase hypoglycemic response to sulfonylureas; may increase levels of zidovudine |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

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| Precautions | Discontinue at first appearance of skin rash or sign of adverse reaction; obtain CBCs frequently; discontinue therapy if significant hematologic changes occur; goiter, diuresis, and hypoglycemia may occur with sulfonamides; prolonged IV infusions or high doses may cause bone marrow depression (if signs occur, give 5-15 mg/d leucovorin); caution in folate deficiency (eg, chronic alcoholics, elderly, those receiving anticonvulsant therapy, or those with malabsorption syndrome); hemolysis may occur in G-6-PD-deficient individuals; AIDS patients may not tolerate or respond; caution in renal or hepatic impairment (perform urinalyses and renal function tests during therapy); give fluids to prevent crystalluria and stone formation |
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| Drug Name | Clindamycin (Cleocin)- Lincosamide useful as treatment against serious skin and soft-tissue infections caused by most staphylococci strains. Also effective against aerobic and anaerobic streptococci, except enterococci. |
| Adult Dose | 600-1200 mg/d IV/IM divided q6-8h for 5-7 d |
| Pediatric Dose | 20-40 mg/kg/d IV/IM divided tid/qid |
| Contraindications | Documented hypersensitivity; regional enteritis; ulcerative colitis; hepatic impairment; antibiotic-associated colitis |
| Interactions | Increases duration of neuromuscular blockade induced by tubocurarine and pancuronium; erythromycin may antagonize effects; antidiarrheals may delay absorption |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in severe hepatic dysfunction; no adjustment necessary in renal insufficiency; associated with severe and possibly fatal colitis |

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| Drug Name | Tetracycline (Sumycin)- Treats susceptible bacterial infections of both gram-positive and gram-negative organisms, as well as infections caused by <i>Mycoplasma</i> , <i>Chlamydia</i> , and <i>Rickettsia</i> species. Inhibits bacterial protein synthesis by binding with 30S and possibly 50S ribosomal subunit(s) of susceptible bacteria. |
| Adult Dose | 250-500 mg PO q6h |
| Pediatric Dose | 25-50 mg/kg/d PO divided q6h |
| Contraindications | Documented hypersensitivity; severe hepatic dysfunction |
| Interactions | Bioavailability decreases with antacids containing aluminum, calcium, magnesium, iron, or bismuth subsalicylate; can decrease effects of oral contraceptives, causing breakthrough bleeding and increased risk of pregnancy; can increase hypoprothrombinemic effects of anticoagulants |
| Pregnancy | D - Unsafe in pregnancy |

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| Precautions | Photosensitivity may occur with prolonged exposure to sunlight or tanning equipment; reduce dose in renal impairment; consider drug serum level determinations in prolonged therapy; use during tooth development (last half of pregnancy through age 8 y) can cause permanent discoloration of teeth; Fanconilike syndrome may occur with outdated tetracyclines |
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| Drug Name | Amoxicillin (Amoxil, Biomox, Trimox)- Interferes with synthesis of cell wall mucopeptide during active multiplication, resulting in bactericidal activity against susceptible bacteria. |
| Adult Dose | 250-500 mg PO q8h; not to exceed 3 g/d |
| Pediatric Dose | 20-50 mg/kg/d PO divided q8h |
| Contraindications | Documented hypersensitivity |
| Interactions | Reduces efficacy of oral contraceptives |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal impairment |

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Follow-up

Further Inpatient Care

- Extravasation of the injected material may further jeopardize the limb.
- Prompt decompression and directed debridement of the nonviable tissue is an important strategy to prevent further tissue damage.
- Less aggressive therapy may have a role in injection injuries with less irritating substances (eg, freon).

Further Outpatient Care

- Outpatient management is contraindicated.

Transfer

- Transfer patient to a facility with a hand specialist if none is available at the receiving hospital.

Complications

- Amputation is more likely if debridement is delayed more than 10 hours, especially with low viscosity substances.
- Tissues that survive the initial injection injury but still contain grease, paint, or oil heal slowly and may develop multiple oleogranulomas of varying sizes.
- In time, the oleomas drain through sinuses or open directly through the skin.

Prognosis

- Factors that determine the severity of the injury
 - Type and viscosity of the material injected
 - Time interval between injury and treatment
 - Amount of material injected and velocity of the injectant
 - Pressure of the appliance
 - Anatomy and distensibility of the site of injection
 - Presence of secondary infection

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Miscellaneous

Medical/Legal Pitfalls

- This injury is a common cause of litigation when evaluated by the unwary physician.
- The innocuous appearance of the wound obscures the potential severity of the injury.
- Without diagnosis and treatment, a compartment syndrome with subsequent necrosis usually destroys tissue viability.

Special Concerns

- A digital block for pain control, via injection of the finger, is contraindicated. Unlike a metacarpal block, this technique may increase tissue distention and vascular insufficiency.
-

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Hand Injuries, Soft-Tissue

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Introduction

Background

Hand injuries occur commonly, accounting for 5-10% of emergency department (ED) visits nationwide. The complexity of the hand and the similarities in clinical presentation of different injuries make understanding of hand anatomy and function, good physical examination skills, and knowledge of indications for treatment indispensable for the emergency physician.

Terminology

Thorough knowledge of the anatomy and functions of the hand is required for proper diagnosis and treatment. Use of proper terminology prevents confusion that may compromise the care of patients with hand injuries. The following is a brief review of standard terminology and key anatomic structures.

- The hand and digits have palmar (volar) and dorsal surfaces and radial and ulnar borders.
- The digits are best described using their standard names rather than numbers.
- The proper names of the 5 digits beginning radially are thumb, index finger, long or middle finger, ring finger, and little finger.

Motion and position: Standard terminology also applies to motions and positions of the hand and digits.

- Supination of the forearm positions the hand with the palmar surface superior. Pronation places the palmar surface inferior.
- Lateral motion of the hand, relative to the forearm, is described as radial or ulnar deviation.
- Anterior and posterior motions of the hand, relative to the forearm, in its anatomic position are described as flexion and extension, respectively.
- Abduction of the digits refers to motion away from the middle finger and adduction to motions toward the middle finger.
- The fingers are in extension when held in the anatomic position. Movement of the digits dorsally is hyperextension, while movement toward the palm is flexion. In addition to flexion and extension, the thumb may move in toward the other digits (opposition) or away from them (retroposition).

Anatomy

Surface anatomy: Three creases are present on the palmar surface of the digits. The distal and middle palmar creases correspond to the distal interphalangeal (DIP) and proximal interphalangeal (PIP) joints, respectively. The proximal digital palmar crease does not overlie a joint. The long thenar crease partially encircles the thenar eminence and overlies the metacarpophalangeal (MCP) joint.

Bony anatomy: The metacarpal bones articulate with the wrist at the carpometacarpal joint. The MCP joint is formed by the union of the metacarpal bones and proximal phalanges (see [Picture 1](#)). The heads of the metacarpals form the knuckle, which is seen in the closed fist. The thumb has only 1 interphalangeal (IP) joint, while the rest of the digits have PIP and DIP joints.

Each of the MCP, PIP, and DIP joints has collateral ligaments, which provide lateral stability, and a volar plate, which prevents hyperextension. The volar plate is damaged frequently in subluxation and dislocation injuries. The wrist is composed of 8 bones that are arranged in 2 rows of 4. The flexor retinaculum, together with the carpal bones, forms the carpal tunnel. The median nerve passes through the carpal tunnel with the tendons of the flexor digitorum profundus and superficialis. The ulnar nerve enters the hand passing between the hook of the hamate bone and the pisiform bone.

Blood supply: The blood supply to the hand is derived from the ulnar and radial arteries, which form the superficial and deep palmar arterial arches by anastomosis. In the absence of vascular disease, either artery alone is sufficient to perfuse the entire hand in most of the population.

Intrinsic and extrinsic muscles: The muscles of the hand are designated intrinsic or extrinsic. Extrinsic muscle bellies are in the forearm and their tendons insert into the hand, while intrinsic muscles both arise in and insert in the hand. The muscles of the hand and digits also are named according to their function as either flexors or extensors.

Forearm flexors: The forearm flexors are extrinsic muscles of the hand. These muscles arise from the

medial epicondyle of the humerus and include the following:

- Flexor carpi radialis
- Palmaris longus
- Flexor carpi ulnaris
- Flexor digitorum profundus
- Flexor digitorum superficialis

The tendons of flexor carpi radialis, palmaris longus, and flexor carpi ulnaris are visible in the forearm (see [Picture 2](#)). The palmaris longus is absent in about 14% of the population. The median nerve lies between the palmaris longus and the flexor carpi radialis. The flexor carpi ulnaris is a good landmark to locate the ulnar nerve and artery.

Flexion of the fingers is controlled by the flexor digitorum profundus and superficialis muscles. Both of the finger flexors lie on the ulnar side of the wrist with the median and ulnar nerves and the ulnar artery. The flexor carpi ulnaris and radialis flex the wrist when acting together and cause deviation to the active side when contracting alone.

Intrinsic muscles of the hand: Branches of the median and ulnar nerves innervate all the intrinsic muscles of the hand. They can be divided into 3 groups as follows: thenar (thumb), hypothenar (little finger), and lumbricals.

The thenar eminence is formed by the extensor pollicis brevis and the 3 short thenar muscles: the abductor pollicis brevis, flexor pollicis brevis, and opponens pollicis. These muscles have short tendons that insert onto the proximal phalanges. They are innervated by the recurrent branch of the median nerve. The superficial location of this branch renders it vulnerable to seemingly trivial trauma to the thenar eminence.

- The adductor pollicis adducts the thumb and by doing so, provides grip. It is innervated by the ulnar nerve.
- The lumbricals flex the digits at the MCP joints and extend the IP joints. They place the fingers in the writing position.
- Seven interosseous muscles are located between the metacarpal bones; 3 are palmar and 4 are dorsal. The palmar interossei adduct, while the dorsal interossei abduct. The lumbricals also assist with flexion at the MCP joints and extension of the IP joints.

Forearm extensors: Eleven muscles extend the wrist, hand, and digits (see [Picture 3](#)). The forearm extensors pass into the hand in 6 compartments. All forearm extensors arise from the lateral epicondyle.

Innervation: The median, ulnar, and radial nerves supply all of the sensory and motor innervation to the hand. The superficial volar and dorsal distributions of the sensory nerves are shown in [Picture 4](#) and [Picture 5](#). The median nerve enters the hand via the carpal tunnel and often is involved in carpal tunnel

syndrome. The median nerve sends motor fibers to the 3 short thenar muscles and the first and second lumbricals. The ulnar nerve sends motor fibers to the hypothenar muscles, the ulnar 2 lumbricals, the adductor pollicis, and all of the interosseous muscles. The radial nerve sends no motor branches to the intrinsic muscles of the hand.

Pathophysiology

The pathophysiology of soft-tissue injuries of the hand is diverse. The most common mechanisms of injury are blunt trauma (eg, crush injury, contusions, abrasions), laceration, avulsion, ring avulsion, and burns. Besides skin and superficial tissues, the many muscles, ligaments, and tendons of the hand are vulnerable to injury, as are the nerves and blood vessels that supply these structures. Damage to these structures may create permanent functional and/or sensory deficits specific to the site of injury.

Nerve injuries

Blunt, penetrating, and crush injuries to the hand result in nerve damage. Nerve injury is divided into 3 types, as follows:

- Neuropraxial injury occurs when a nerve is bruised or stunned but remains essentially intact.
- Axonotmesis describes a partial injury in which the axonal core of a nerve is damaged but the myelin sheath remains intact. These injuries usually regenerate at a rate of 1-3 mm per day.
- Neurotmesis is complete disruption of both axons and myelin sheath. It requires re-approximation of the nerve endings for healing to occur.

Sprains

Joints of the digits are stabilized by the combination of collateral ligaments and the volar plate. Stretching or partial tearing of the ligaments results in a sprain. The volar plate may be injured alone or in combination with the collateral ligaments. The common mechanism for an isolated volar plate injury is hyperextension during an axial load.

A sprain produces pain and swelling but little or no instability. Pain location is a good indicator of the site of injury. For example, lateral pain suggests collateral ligament injury, while pain on the palmar surface of the joint suggests volar plate injury. Loss of stability more commonly is associated with joint dislocation.

Sprains of the PIP and MCP joints are classified as first, second, or third degree. If the joint does not open at all but has pain with stressing of a ligament, the injury is first degree. A joint that is opened slightly in the ulnar or radial direction is defined as having a second-degree injury. This finding suggests a unilateral collateral ligament tear. A joint that is opened by at least 3-5 mm must have damage to at least 2 of the 3 structures stabilizing the joint (ie, volar plate, 2 collateral ligaments). This is referred to as a third-degree sprain or an unstable joint.

Sprains of the MCP joint are rare because of the anatomy of the joint, the laxity of the collateral ligaments, and the protection afforded the joints by surrounding structures. Hyperextension of the extended digit is the most common mechanism causing sprains. Diagnosis is indicated by a stable but painful edematous joint.

Dislocations

DIP: The DIP joint is stabilized not only by collateral ligaments but by adjacent flexor and extensor tendons, making dislocations of this joint uncommon. If dislocation does occur, it usually is directed dorsally and often is associated with an open wound. DIP joint dislocations are detected easily by physical examination.

PIP: The ligaments of the PIP joints are the most commonly injured in the hand. Dorsal dislocations are the most common and usually are the result of a blow to the extended digit, causing a combination of axial loading and dorsal deviation. Volar dislocations are uncommon because the joint does not resist motion in this direction. Lateral dislocation is the result of a tangential load applied to the extended digit that ruptures a collateral ligament and disrupts the volar plate. Ulnar deviation, with rupture of the radial collateral ligament, is more common than radial deviation.

MCP: Dislocation of the MCP joint is uncommon, but when it occurs deviation is usually dorsal. The common mechanism of injury is the application of a dorsally directed force that is sufficient to rupture the volar plate. Dorsal dislocations that are in 60-90° of hyperextension and are without intervening soft tissue are simple dislocations. Complex dislocations have the volar plate entrapped between the metacarpal and the proximal phalanx. Complex dislocations are less striking in their clinical presentation but are more serious injuries.

Thumb: The IP joint of the thumb is very stable and seldom injured. Dislocations usually are dorsal and often open. The MCP joint of the thumb is one of the most frequently injured joints. Injury most commonly is caused by hyperextension force sufficient to rupture the volar plate and cause dorsal dislocation. As in MCP joints of the other digits, dorsal dislocation of the MCP joint may be a simple subluxation or complex dislocation. The complex dislocation is complicated by entrapment of the proximal phalanx.

Ligament injuries

Ligamentous injuries occur frequently and often are misdiagnosed because a mild sprain may have a similar presentation. Sequelae from missed ligamentous injuries range from chronically painful to unstable or chronically deformed joints. The ability to hold objects between the thumb and 4 fingers is an essential function of the hand and depends upon an intact ulnar collateral ligament (UCL).

Injury to the UCL is known as the gamekeeper's thumb. This is because Scottish gamekeepers frequently

damaged their UCLs killing game. The head of a small animal was placed between the thumb and index finger and a hyperextension/longitudinal traction force applied to the animal's cervical spinal cord by abruptly yanking the lower extremities. In a certain percentage of these procedures, the UCL of the gamekeeper was disrupted.

In modern times, skiing is the activity that most often causes UCL injury. However, a history of a missed punch, a fall onto the thumb, or the forceful removal of an object from the flexed hand also should be considered suggestive of UCL injury.

The common mechanism of injury is the forceful abduction of the thumb. Any patient with pain in the distribution of the UCL or inability to forcefully oppose the thumb has an injury of the UCL until proven otherwise.

Rupture of the radial collateral ligament is much less common than UCL rupture. The mechanism of injury is forceful adduction of the thumb in any position.

Tendon injuries

The extensor tendons' superficial location predisposes them to injury from seemingly trivial lacerations as well as avulsions, crushes, and burns. Tendon injuries also may be sustained as the result of forced hyperextension or forced flexion of an extended digit. Injuries may include complete or partial transection, avulsion, or maceration. Whenever a tendon is damaged, particularly with an open injury, the vessels and nerves that are in close proximity may be injured.

Understanding that a tendon may be 70-90% lacerated and still functional is critical. Damage to these tendons may result in such findings as boutonnière deformity (see [Picture 6](#), [Picture 7](#)) and mallet finger (see [Picture 8](#)).

Mortality/Morbidity

Soft-tissue injuries of the hand rarely are life threatening. However, the high incidence of disability from chronically painful or unstable joints is reflected by the fact that hand derangements account for 9% of all worker compensation claims.

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Clinical

History

- General
 - Age
 - Hand dominance
 - Occupation/hobbies
 - History of previous hand problems
- How was the trauma sustained? This gives clues to the most likely injury. For example, the water skier who injured a hand when the towing line was removed forcefully from that hand is likely to have an injury to the flexor tendon mechanism.
- What was the posture of the hand at the time of the injury? Structures in the hand slide with movement. The tissue under a bruise or laceration may not be the same tissue that was present when the injury was sustained because of movement of structures in the hand (eg, extensor tendons injured with the digits in flexion may not be visible in the wound when digits are extended).
- Past history of treatment or surgery in the hand

Physical

- The entire upper extremity should be exposed. Note any of the following findings:
 - Muscle wasting
 - Color change
 - Surgical or nonsurgical scars
 - Asymmetry
 - Deformities that suggest dislocation
 - Differences in flexion/extension in the relaxed hand: The relaxed hand is in moderate flexion. The digits on both sides should be in about the same amount of flexion. The little finger usually is in more flexion than the others fingers. If a digit has a marked difference in flexion, the examiner should be suspicious for tendon injury. This finding may be useful in identifying the injury of the patient who is a poor historian.
 - Dry patches of skin may indicate loss of innervation.
 - Dimpling over the thenar eminence suggests complex dislocation of the MCP joint of the thumb.
 - Check range of motion in every joint in the hand, shoulder, and elbow. Ability to pronate and supinate the forearm should be tested actively and passively.
- Examination of extrinsic flexors
 - Each of these tests is performed with and without resistance.
 - Flexor pollicis longus: Instruct the patient to bend the tip of the thumb against resistance.
 - Flexor digitorum superficialis: While stabilizing the rest of the fingers to block the action of flexor digitorum profundus, instruct the patient to bend the middle joint of the finger. Palpate the tendons of flexor carpi ulnaris, flexor carpi radialis, and palmaris longus (which is not present in 20% of individuals) while the patient holds their wrist and fingers in hyperflexion.
- Extrinsic extensors may form adhesions secondary to old trauma. This phenomenon is referred to

as extensor tightness.

- Extensor tightness is evaluated by passive extension of the MCP joint and flexion of the PIP joint with the wrist in anatomical position. The PIP joint should flex.
- Repeat the test with the MCP joint in passive flexion.
- If the PIP joint will flex when the MCP joint is extended but not when it is flexed, adhesions are present in the extensors, stopping the simultaneous flexion of the finger MCP and PIP joints.
- Joints
 - The stability of a joint is assessed by active and passive motion.
 - Pain causes some patients to consciously or unconsciously limit the range of motion of an injured joint. Therefore, administering a digital block may be necessary prior to assessing joint stability.
 - Evaluate stability by applying anterior, posterior, radial, and ulnar stress to each IP and MCP joint in the extended and flexed positions. Evaluation in the flexed and extended positions is necessary as the volar plate may stabilize a dislocated or subluxed joint in certain positions.
- Circulation
 - Look for color changes in the nails and skin of the hand.
 - The Allen test has variable sensitivity, but it may be used to help assess perfusion to the hand.
 - Compress radial and ulnar arteries at the wrist.
 - Instruct the patient to open and close the fist to exsanguinate the hand.
 - Have the patient open the hand.
 - Release the radial artery.
 - If the hand fills with blood within 5 seconds, the radial artery is patent. Repeat the test for the ulnar artery.

Causes

Trauma accounts for the majority of these injuries. However, patients also present with complaints that are secondary to infection, burns, or overuse.

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Differentials

Dislocations, Hand

Fractures, Hand

Hand Infections

Hand Injuries, High-pressure

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Workup

Imaging Studies

- Plain films of the hand or wrist should be obtained when a patient presents with a soft-tissue injury suggestive of fracture or an occult foreign body. Of particular concern are lacerations from glass.
 - Radiographs are indicated in dislocation of a DIP joint to rule out fracture.
 - In dislocation of an MCP joint, posterior-anterior view shows marked widening of the joint. Sesamoid bones seen inside the joint space on radiographs are diagnostic of complex MCP joint dislocation.
- MRI has been shown to have a high sensitivity in detecting ruptured tendons. However, it does not have a role in emergent management of hand wounds.

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Treatment

Prehospital Care

Prehospital care of soft-tissue hand injuries consists of application of sterile dressings and splinting, as needed.

Emergency Department Care

The prevalence and acute nature of soft-tissue injuries to the hand require that the emergency physician understand the principles of evaluation and treatment.

- Time considerations: The time that a patient was injured and the time of presentation to the ED should be recorded. Certain types of injuries require rapid response to prevent unfavorable outcomes. The following injuries require immediate treatment after diagnosis:
 - Vascular injuries that cause hemorrhage
 - Vascular injuries that compromise perfusion
 - Compartment syndromes

- Amputations with potential for reimplantation
- Hydrofluoric acid burns
- High-pressure injuries
- Self-inflicted injuries: Multiple lacerations that are partial thickness and parallel to each other are known as hesitation marks. Hesitation marks are indicative of self-inflicted injury. Psychiatric consultation is recommended for any intentionally self-inflicted wound.
- Nerve injury
 - A completely disrupted nerve should be repaired microsurgically. However, in the acute setting, distinguishing the severity of nerve damage often is impossible. Consultation with a hand surgeon is advised.
 - Primary repair is optimal, but if delayed repair is advised because of a dirty wound, multiple injuries, or logistic constraints, the finger/hand involved should be splinted and the patient should receive prompt follow-up with a hand surgeon.
- Dislocations
 - Distal interphalangeal joints
 - Radiographic studies are indicated to rule out fractures.
 - Reduce the dislocation after administering a digital or metacarpal block. While holding the phalanx proximal to the injury, apply a distracting force along the longitudinal axis of the digit. While maintaining traction, hyperextend the phalanx (for dorsal dislocations) and bring it back to its normal anatomic position.
 - Examine the joint thoroughly after reduction. Then immobilize the finger with an aluminum splint. If the joint is irreducible, consultation with a hand surgeon is required. Inability to reduce a digit may be the result of entrapment of the volar plate or an avulsion fracture in the joint space.
 - Irrigation, debridement, bacterial prophylaxis, and wound closure are indicated for open wounds.
 - Metacarpophalangeal joints
 - The recommended treatment of complex and volar dislocations is a gentle compression dressing and urgent consultation with a hand surgeon, because they are likely to require open reduction.
 - Reduction of simple dislocations of the MCP joint may be attempted by an emergency physician, although reduction often is unsuccessful. Entrapment of the metacarpal head between muscles and tendon on the palmar side of the hand often prevents closed reduction. After administration of a metacarpal or wrist block, flex the wrist to relax the flexor tendons. Flex the proximal phalanx while applying mild longitudinal traction. Use care to avoid hyperextension or excessive longitudinal force, which may open the joint space and allow entrapment of the volar plate.
 - Following successful reduction, immobilize the hand in a planar splint and refer the patient to a hand surgeon.
 - Metacarpophalangeal joint of the thumb
 - Simple dislocations may be reduced following administration of a median nerve block. Flex and abduct the MCP joint and apply longitudinal force to the base of the proximal phalanx.

- If this method is unsuccessful, flexion of the IP joint and wrist will relax the flexor pollicis longus tendon, which may be complicating the reduction.
- Thorough examination is necessary after reduction. If the joint is stable, immobilization of the MCP joint in 20° of flexion for 3 weeks is indicated.
- Tendon injuries
 - Extensor tendon injuries
 - The superficial location facilitates evaluation and permits repair in the ED.
 - Partial tendon injuries (<40-50% of the tendon width) usually do not require repair. They should be splinted and follow-up arranged with a hand surgeon.
 - Complete extensor tendon injuries can be repaired using 4.0 nonabsorbable suture material and a figure 8 or modified Kessler suture, with the knot buried on the palmar aspect of the tendon. However, this procedure does not need to be performed urgently, and closure of the skin, splinting of the hand, and referral to a hand surgeon for delayed repair often is the best option.
 - The hand should be splinted in 30° of extension at the wrist with the MCP in a neutral position.

Consultations

Orthopedic surgeon or plastic surgeon with expertise in hand injuries

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Follow-up

Complications

- Pain
- Stiffness
- Abscess
- Decreased range of motion
- Scar formation
- Loss of digit/hand (rare)

Prognosis

- With early and appropriate treatment, prognosis is good.

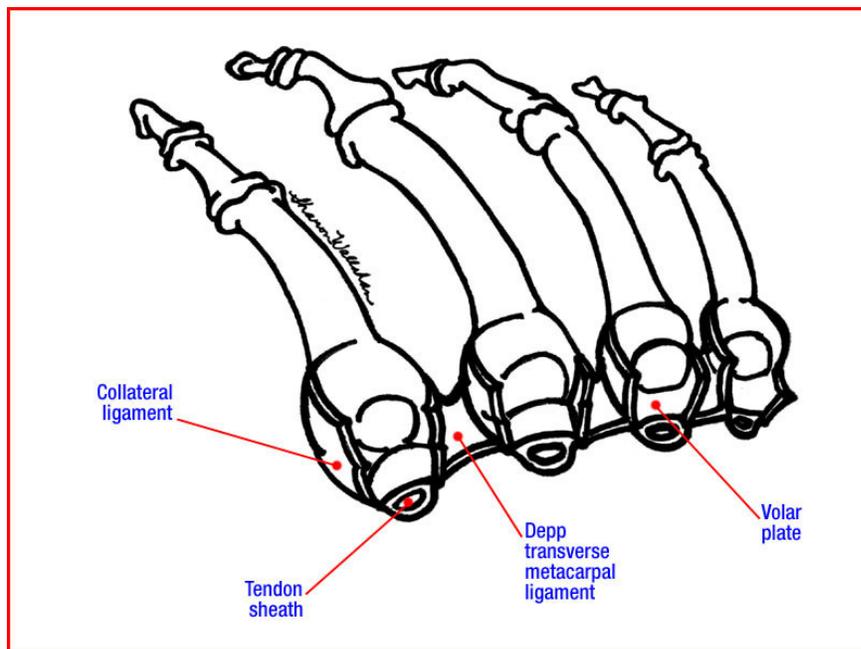
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Miscellaneous

Medical/Legal Pitfalls

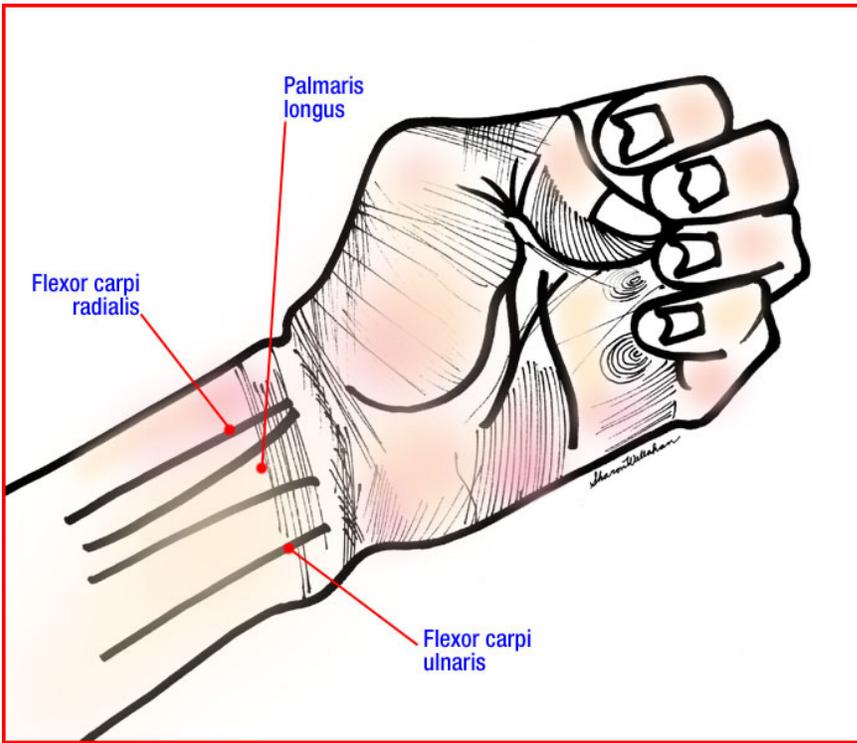
- Complications of traumatic wounds account for the highest number of medicolegal actions against emergency physicians in the US. Be sure to consider the possibility of retained foreign bodies or deep tissue injury in all open wounds.
- Clearly inform all patients with hand injuries of the possibility of complications and instruct them on the potential signs and symptoms of complications. Patients should be informed that pain, limitation of mobility, and stiffness commonly occur after hand injuries even with optimal care.
- Finally, carefully document the care, procedures, and follow-up plan for all patients.

Pictures



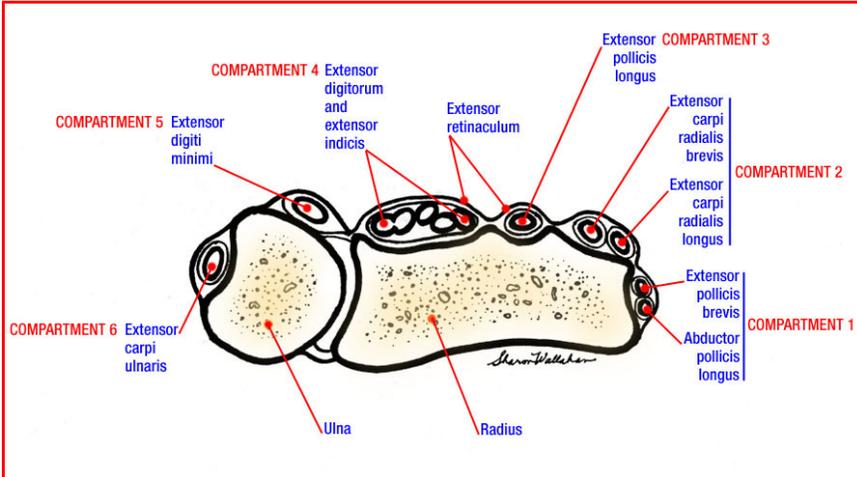
Picture 1: Metacarpophalangeal joints of the digits

Picture type: Photo



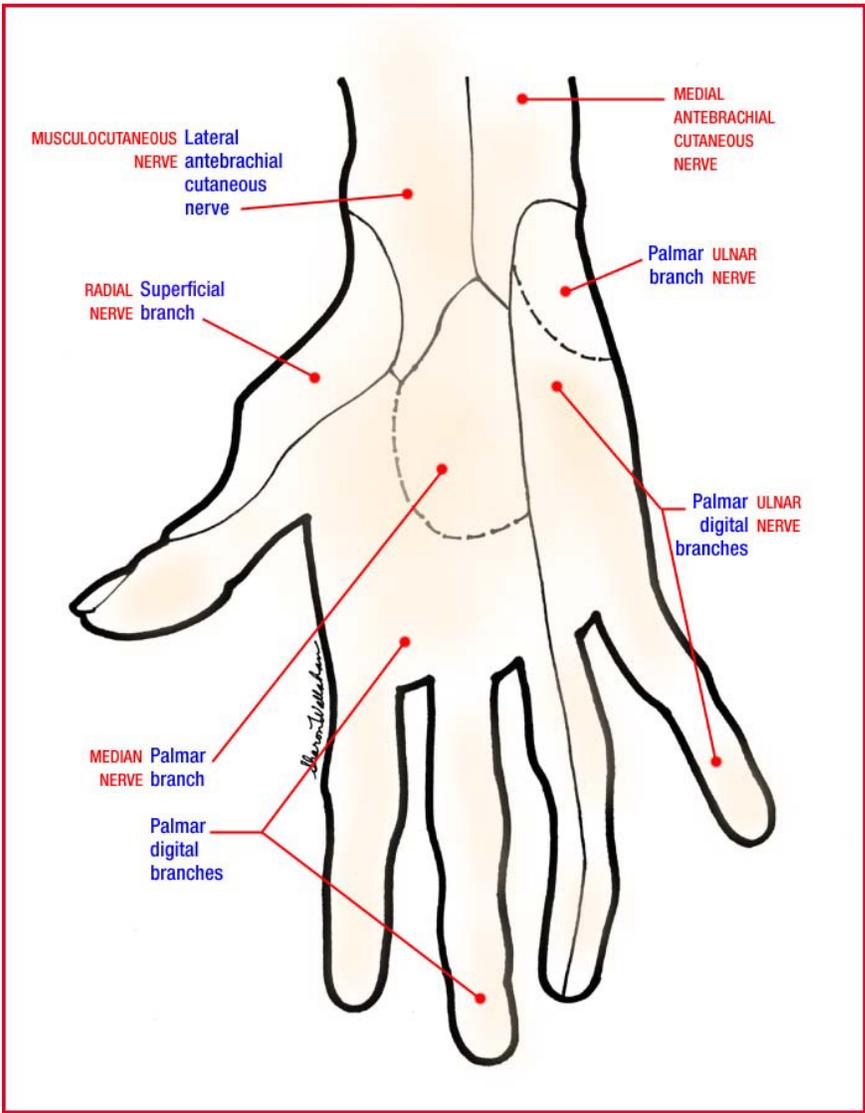
Picture 2: Volar tendons at the wrist. These can be used as landmarks for injections.

Picture type: Photo



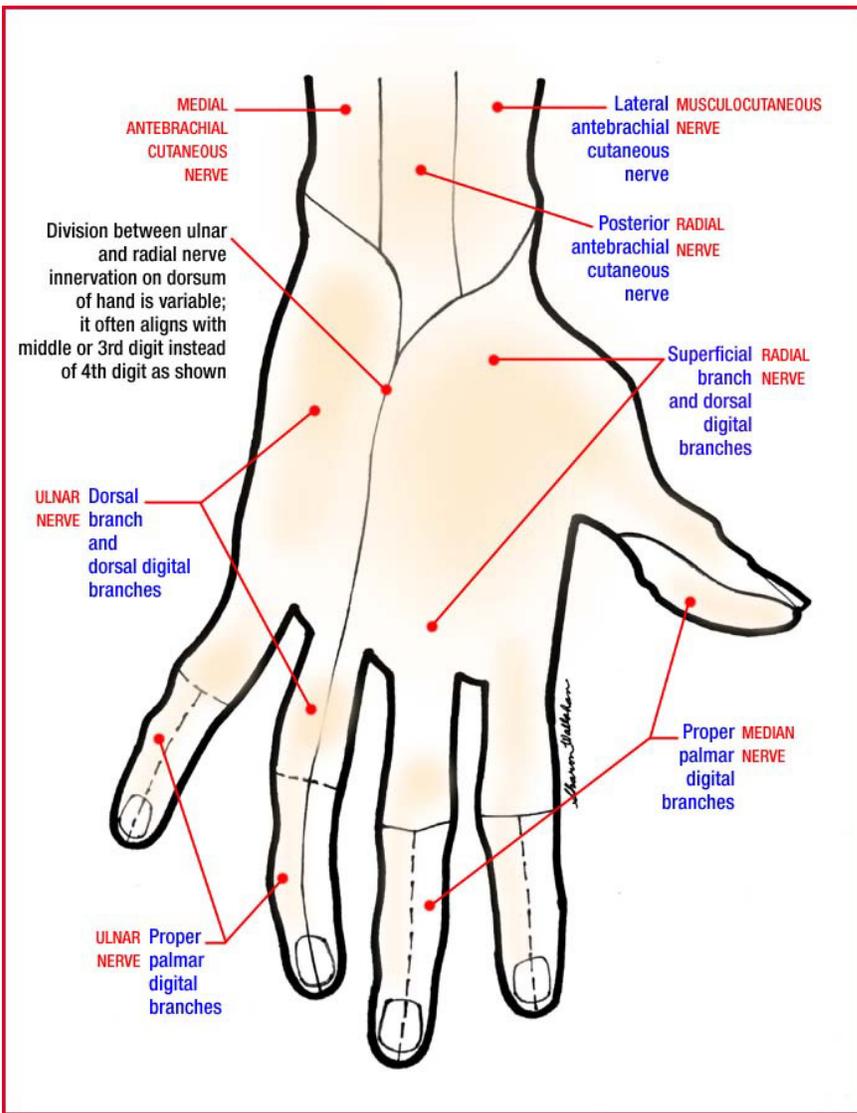
Picture 3: Sagittal section of extensor compartments

Picture type: Photo



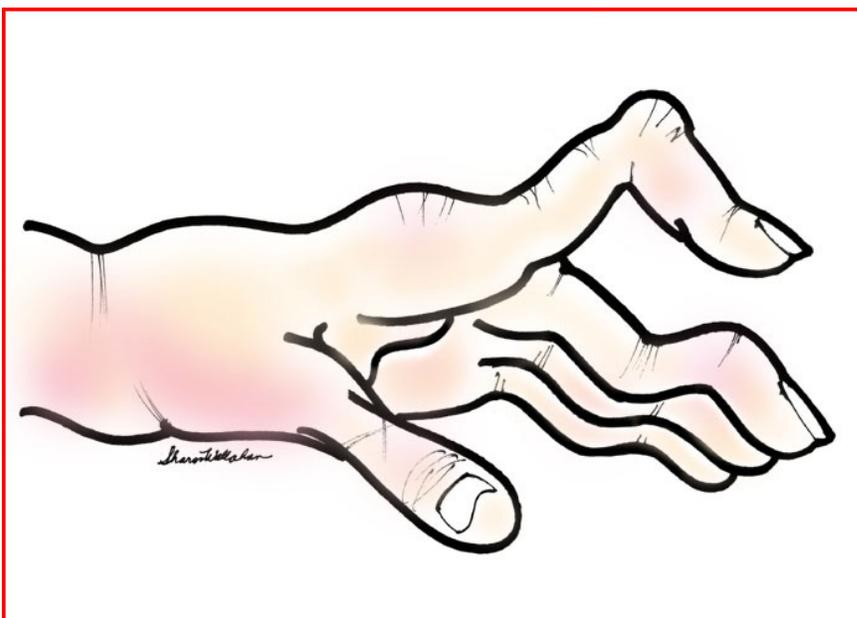
Picture 4: Superficial volar sensation of the hand

Picture type: Photo



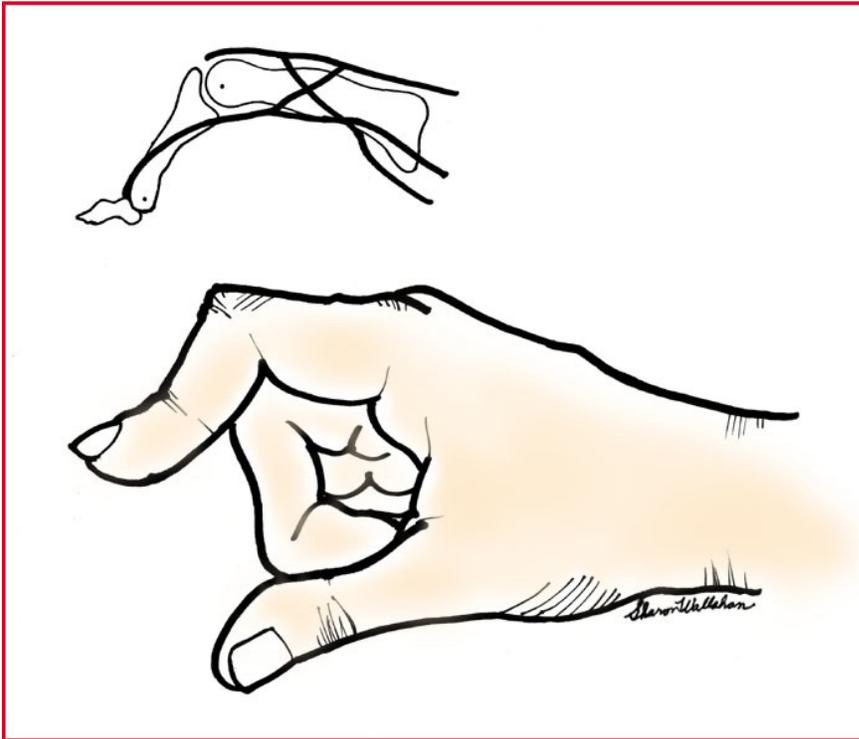
Picture 5: Superficial dorsal sensation of the hand

Picture type: Photo



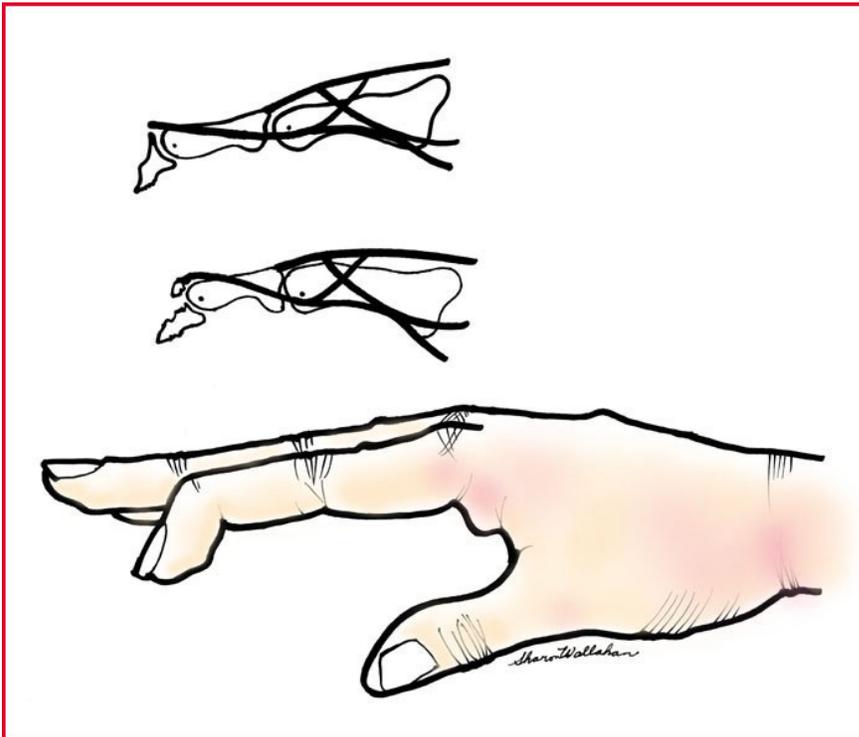
Picture 6: Boutonnière deformity due to closed central tendon rupture

Picture type: Photo



Picture 7: Boutonnière deformity

Picture type: Photo



Picture 8: Mallet finger due to loss of central extensor tendon to the distal phalanx

Picture type: Photo

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Hanging Injuries and Strangulation

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Introduction

Background

With its relatively small diameter, lack of bony shielding, and close association of the airway, spinal cord, and major vessels, the human neck is uniquely vulnerable to life-threatening compression injuries. Throughout recorded history, assailants and the penal systems that have received them have used various methods of strangulation to injure or kill.

Hangings can be classified as complete or incomplete. When the whole body is suspended and does not touch the floor or platform at the end of the drop, the hanging is said to be complete. Incomplete hangings imply that some part of the body is touching the ground. Hangings also may be classified by intent (eg, homicidal, suicidal, autoerotic, accidental).

Pathophysiology

The pathophysiology of strangulation injuries is controversial, except for classical judicial hangings. In judicial hangings, the drop is at least as long as the height of the victim and the hanging is complete. In such cases, the mechanism of death is effectively decapitation, with distraction of the head from the neck and torso, fracture of the upper cervical spine (typically traumatic spondylolysis of C2 in the classic hangman fracture), and transection of the spinal cord.

In all other choking injuries, by hand or application of tool or ligature, pathophysiologic theories to account for observed symptoms include the following:

- Venous obstruction, leading to cerebral stagnation, hypoxia, and unconsciousness, which allows muscle tone relaxation and final arterial and airway obstruction
- Arterial spasm due to carotid pressure, leading to low cerebral blood flow and collapse
- Vagal collapse, caused by pressure to the carotid sinuses and increased parasympathetic tone

Interestingly, none of the proposed mechanisms emphasize airway compromise alone. In fact, although delayed mechanical airway compromise occurs and often complicates patient management, it appears to play a minimal role in the immediate death of victims of successful strangulation. Many classical jujitsu and aikido strangle holds are applied to the vascular structures of the neck and not the trachea. However, in judicial hangings and many suicidal hangings, direct spinal cord injury is the cause of death. Several reports exist of suicidal posttracheostomy patients who successfully hung themselves with ligatures well above the tracheostomy, where death did not appear to be related to spinal cord injury. Regardless of disagreement on theories, most experts agree that death ultimately occurs from cerebral hypoxia and ischemic neuronal death.

Frequency

- **In the US:** In 1996, hangings and strangulations accounted for 5330 deaths, the majority of which were suicidal. Strangling injuries are common, as the necessary weapons are as close as the attacker's own hands. Perhaps 5-10% of urban assaults involve strangulations or ligature assaults.
- **Internationally:** Judicial hangings are uncommon worldwide. However, autoerotic injuries are becoming more prevalent in urban centers (see [Causes](#)).

Mortality/Morbidity

- If death is not immediate, the patient's risk of delayed presentation of airway obstruction is significant.
- Tracheal intubation is difficult because of edema of the laryngeal structures.
- Strangulation injuries account for approximately 2.5% of all traumatic deaths worldwide.

Sex

- Women are victims of strangulation assault more frequently than men.
- In contrast, nearly all reported autoerotic strangulation deaths involve men.
- Suicidal hangings are overwhelmingly more common among men.

Age

Commonly affected groups include toddlers (eg, due to ill-constructed cribs), adolescents (eg, depression leading to suicide attempts), and young adults (eg, autoerotic injuries, assault, suicidal depression, prison inmates).

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Clinical

History

Varies by underlying cause

- Patients may present after an assault in which they were manually strangled or garroted by a ligature.
- Patients in whom a suicide attempt has failed can present to the emergency department (ED) after being found by family or acquaintances and brought by Emergency Medical Services (EMS).
- Certain autoerotic experiments with erotic asphyxiation can be misidentified as suicide attempts (see [Causes](#)).
- Infants present after accidents in which they were caught between crib or fence slats or on objects in their environment. This is most common in infants younger than 3 months, whose limb strength is insufficient to extricate themselves.
- Attempt to determine the height of the drop in near-hanging victims because this may influence management decisions.

Physical

- Abrasions, lacerations, contusions, or edema to the neck, depending on how the patient was strangled
- Subconjunctival and skin petechiae cephalad to the site of choking (Tardieu spots)
- Severe pain on gentle palpation of the larynx
- Mild cough
- Stridor
- Muffled voice
- Respiratory distress
- Hypoxia (usually a late finding)
- Mental status changes

Causes

Many different causes can lead to strangulation injuries.

- Assaults can result in strangulation. Risk factors follow other types of assault, with the exception that women are more likely than men to be victims of strangulation.
- Depression can lead to hangings, especially in subgroups (eg, prisons, where hangings are part of tradition or culture).
- Those who explore variant erotic practices occasionally experiment with hypoxia as a means of intensifying orgasm. Marquis de Sade first described this practice in his writings. Autoerotic play using a ligature or noose to produce hypoxia during masturbation can result in accidental hanging.

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Differentials

Anaphylaxis
Angioedema
Asthma
Chronic Obstructive Pulmonary Disease and Emphysema
Depression and Suicide
Domestic Violence
Epiglottitis, Adult
Neck Trauma
Sexual Assault
Spinal Cord Injuries

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Workup

Lab Studies

- Laboratory tests should not be drawn until after the airway has been assessed and secured, if necessary.
- Arterial blood gases (ABGs) should be evaluated in all patients requiring definitive airway intervention.

- Given the ready availability of pulse oximetry, ABGs are probably unnecessary in patients who do not require tracheal intubation.

Imaging Studies

- In nonjudicial hangings, cervical spine injury is rare. The head hyperextends in hanging victims who drop the distance of their height or greater. Classically, the result is bilateral fracture through the pedicles of C2 with the C2 vertebral body displaced anterior to the vertebral body of C3. Hangman fractures are seen best on the lateral radiograph of the cervical spine (C spine).
 - Soft-tissue neck x-rays should be ordered in nearly all strangulation patients.
 - Generally, a fractured hyoid bone indicates a severe occult soft-tissue injury, even in a patient whose medical condition is otherwise stable.
- CT imaging of the head is indicated when the neurologic status is compromised. Clinically subtle injuries to the laryngeal cartilage may not be apparent on plain x-rays and may require a CT scan of the neck for definition.
- Doppler vascular imaging or arteriography of the carotids should be considered in cases of garroting. The thin wires or cords used in these assaults often produce deep vascular thrombosis.

Procedures

- In less immediately severe cases, direct fiberoptic laryngoscopy and microlaryngoscopy may play a role in establishing the full pattern of injuries. Ear, nose, and throat (ENT) consultation can establish both the need for, and the timing of, these studies.

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Treatment

Prehospital Care

- C-spine stabilization and airway assessment are the primary concerns.
- Do not attempt intubation unless airway is obstructed acutely.
- If respiratory failure or airway obstruction is present and EMS is unable to intubate the patient, the clinician should consider cricothyroidotomy, or percutaneous translaryngeal or transtracheal ventilation.

Emergency Department Care

- Assessment and treatment of the ABCs is paramount.
 - Generally, strict enforcement of C spine immobilization is not imperative unless the fall is significant (ie, a drop equal to or greater than the height of the victim).
 - Unless the patient suffers from volume loss, fluid restriction is prudent to help prevent acute respiratory distress syndrome (ARDS) and cerebral edema.
- Tracheal intubation may be required emergently with little warning, and clinicians should be prepared.
- Cricothyroidotomy may be necessary for any patient with airway deterioration in the event that tracheal intubation fails.
- Percutaneous translaryngeal ventilation also may be necessary to temporarily ventilate a patient.

Consultations

- Consider early consultation with an ENT, trauma, or general surgeon for strangulation injuries.
- Psychiatric consultation should be obtained when strangulation intent was suicidal or autoerotic.

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Follow-up

Further Inpatient Care

- Prudence dictates admitting all near-hanging victims for 24 hours to safeguard against delayed airway and pulmonary complications.

Further Outpatient Care

- Psychiatric support for near-hanging victims is recommended, as these patients are prone to depression and violence.

in/Out Patient Meds

- Phenytoin may help prevent additional insult from cerebral ischemia and in treating hanging-induced seizures.

Transfer

- Once stabilized, patients who have sustained a spinal cord injury should be transferred to a

designated spinal cord center.

Deterrence/Prevention

- Caution parents about the dangers of postural asphyxiation in toddlers. This occurs when the child places his or her neck over an object and the body weight produces compression.

Complications

- Respiratory complications: These are the major cause of delayed mortality in near-hanging victims. Both aspiration pneumonia and ARDS may develop, complicating the clinical course.
- Tracheal stenosis
- Neurologic sequelae: A wide array may occur in survivors of strangulations and near-hangings, including muscle spasms, transient hemiplegia, central cord syndrome, and seizures. Spinal cord injury also can cause long-term paraplegia or quadriplegia and short-term autonomic dysfunction.
- Scarring of neck tissue
- Psychiatric disturbances: A multitude of these may declare in survivors of strangulation and near-hanging injuries. Psychosis, Korsakoff syndrome, amnesia, and progressive dementia all have been reported. Nearly all patients who have undergone strangulation or near-hanging demonstrate restlessness and a propensity for violence.

Prognosis

- Varies depending on the development of complications

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Miscellaneous

Medical/Legal Pitfalls

- Failure to adequately stabilize or address associated C-spine injuries in near-hanging victims
- Failure to refer near-hanging victims for psychiatric evaluation
- Failure to obtain soft-tissue neck x-rays when evaluating strangulation victims
- Failure to address the potential for delayed airway compromise in strangulation victims
- Failure to obtain consultation for evaluation of suspected laryngeal injuries
- Failure to seek other injuries or illnesses in the potentially suicidal hanging patient
- Failure to consider carotid artery injury in patients with neurologic sequelae

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Knee, Soft-Tissue Injuries

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Introduction

Background

Soft-tissue injuries of the knee represent one of the more common, yet perplexing, musculoskeletal disorders presenting to the ED. Establishing clear-cut diagnostic and therapeutic objectives is important.

Knee pain and related symptoms may derive from damage to one or more of the soft-tissue structures that stabilize and cushion the knee joint, including the ligaments, muscles, tendons, and menisci. Try to differentiate knee symptoms due to an acute traumatic event from complaints secondary to an exacerbation of a chronic overuse syndrome.

Correct and timely diagnosis increases a patient's chances of restoring normal pain-free use of the affected knee. Generally, the severity of the acute injury can be determined from a focused history, an attentive physical examination, and thoughtfully ordered radiographs.

Pathophysiology

Mechanically, the knee performs like a rolling cam, rather than as a simple hinged (ginglymus) joint. As the knee proceeds from flexion to extension, a complex screw-home motion takes place, with the femoral

condyles locking into the tibial plateau as the femur rotates internally. Full knee extension increases the tautness of the major bracing ligaments, transforming the knee into a mechanically rigid structure. Flexion loosens the knee joint by unlocking and disengaging the bracing structures, thereby enhancing ligamentous laxity and allowing an increase in the joint range of motion (ROM). Retraction of the menisci also takes place with knee flexion.

The 2 separate but interdependent joints that comprise the knee are the tibiofemoral articulation and the patellofemoral coupling. Weight-bearing forces, up to 5 times an individual's body weight, transmit through the opposing condyles of the femur and the tibia. Two shock-absorbing cartilaginous menisci interpose between the femur and the tibia, forming the largest synovial joint in the body. A fibrous capsule lined by a synovial membrane also surrounds and bolsters the knee joint; however, this design lacks inherent stability.

Fitness of the knee joint depends largely on the fortifying ligaments binding the femur, tibia, and patella. Two sets of knee ligaments frequently are affected. Lying outside of the knee joint proper are the extracapsular collateral ligaments, consisting of the medial collateral ligament (MCL), which opposes extreme abductive/valgus forces, and its counterpart, the lateral collateral ligament (LCL), which limits excessive adductive/varus pressures. Crisscrossing within the knee joint are the anterior cruciate ligament (ACL) and the posterior cruciate ligament (PCL), individually bracing against excessive translation in the anteroposterior plane.

Formed primarily by the quadriceps muscles, the extensor apparatus envelops and stabilizes the patella. Distally, the quadriceps consolidate into the patellar ligament, ultimately inserting onto the tibial tubercle. Diverse bursae combat friction between susceptible structures. Fixed in the back of the knee joint, in the popliteal fossa, reside vital neurovascular structures, in particular the popliteal artery.

Sprains are characterized by stretching or tearing of noncontractile segments, such as ligaments or the joint capsule, whereas strain refers to stretching or severing along the course of muscles or tendons. Collateral ligament sprains are relatively common. Ligamentous (sprain) and muscle (strain) injuries may be classified according to the degree of impairment.

- Grade I sprain - Stretching but no tearing of the ligament, local tenderness, minimal edema, no gross instability with stress testing, firm end point
- Grade II sprain - Partial tears of the ligaments, moderate local tenderness, mild instability with stress testing (but firm end point), moderately incapacitating
- Grade III sprain - Complete tear, discomfort with manipulation but less than expected for degree of injury, variable amount of edema (ranging from negligible to grossly conspicuous), clear instability with stress testing (expressing a mushy end point), severe disability

ACL tears: Rupture of the ACL is among the most serious of the common knee injuries and results from a variety of mechanisms. Most victims of ACL damage complain of immediate and profound pain, exacerbated with motion, and inability to ambulate. Disruption of the ACL may occur alone or with other

knee injuries, especially a meniscal or MCL tear.

PCL tears: Patients typically report falling on a flexed knee or sustaining a direct blow to the anterior aspect of the knee (eg, when the knee strikes the dashboard in a motor vehicle accident [MVA]). PCL harm signifies a major injury and rarely occurs as an isolated injury.

Frequency

- **In the US:** Knee injuries afflict over 3 million Americans per year.
- Trauma to the knee is the second most common occupational accident.
- The MCL is the most frequently injured ligament in the knee, while ACL damage causes the highest incidence of pathological joint instability.
- In National Collegiate Athletic Association (NCAA) football, one major knee injury occurs per team every year. Sports-related activity accounts for approximately 60% of knee injuries producing ligamentous laxity.
- **Internationally:** As in the US, incidence of knee injuries is increased in countries where sporting activities, such as skiing, soccer, and basketball, are popular.

Mortality/Morbidity

- Undue morbidity (eg, amputation) may befall the patient subjected to inappropriate or ill-timed care of knee dislocations.
- Oversight of the magnitude of soft-tissue injuries of the knee may result in a failure to expeditiously consider compartment syndrome and its resultant complications, including loss of limb.
- Misdiagnosis or mismanagement of damage to supporting structures of the knee may lead to chronic knee instability with subsequent development of degenerative joint disease and loss of knee function, including an inability to bear weight or ambulate.

Sex

- Disorders of the patella and lateral meniscus generally are more common in females.
- Specifically, chondromalacia patellae or patellar malalignment syndrome (ie, premature erosion and degeneration of patellar cartilage) predominates in young females.
- Larsen-Johansson disease of the patella, also known as inferior pole patellar chondropathy, is 9 times more prevalent in males.

Age

Generally, knee dislocations arise from high-energy trauma, such as MVA. Therefore, patients tend to be young males.

- Ligamentous and meniscal injuries are more likely in young to middle-aged adults, while children and adolescents remain more susceptible to osseous damage.
- Most patients with a meniscal tear are aged 20-30 years, but a second peak occurs in older patients (>60 y).
- Elderly patients may sustain fractures from minimal disturbances that typically produce only soft-tissue injuries in younger patients.
- The region of the extensor mechanism susceptible to disruption correlates with the patient's age.
 - The older the patient, the more proximal the area ruptured.
 - Disruption of the quadriceps tendon occurs more often in the elderly, while more distal severance of the patellar tendon and the tibial tubercle occurs in younger patients.

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Clinical

History

Confirm that an acute traumatic event occurred. Document mechanism of injury, type and location of pain, associated symptoms, amount of immediate dysfunction, presence and onset of joint edema, and history of past knee problems. Verify that the cause of the knee problem is mechanical by establishing that pain is exacerbated by movement (eg, walking, climbing, jumping).

- Mechanism of injury
 - Knowledge of the mechanism can help predict which structures may be injured.
 - Determine the circumstances, such as the position of the extremity, whether the foot was anchored to the ground, if the forces were direct or indirect, and the direction, magnitude, and torque of impact.
 - Direct blows and valgus or varus contact may provoke injury to the collateral ligaments, epiphyseal fractures (in children with open growth plates), and patellar dislocation.
 - Pure valgus forces, such as those occurring when a football player is struck on the lateral aspect of the knee, are more common than varus-directed contact.
 - The MCL is more prone to injury than the LCL.
 - A combination of valgus or varus stress, whether direct or indirect, delivered to a rotated leg accounts for a wide array of injuries.
 - Vulnerable structures include the collateral and cruciate ligaments, the menisci, and the joint capsule. Consider the intricate damage to the knee of a skier who catches the inside edge of a ski, diverting the tip outward as the body continues to advance forward. This action produces torque, forcing the knee into extreme valgus and external tibial rotation. Depending on the magnitude of the force, this mechanism may tear the MCL, the

posteromedial capsule, and the ACL. Additionally, the menisci are at risk.

- ACL tears: Rupture of the ACL is among the most serious of the common knee injuries and results from a variety of mechanisms.
 - Most victims of ACL damage complain of immediate and profound pain, exacerbated with motion, and inability to ambulate.
 - A majority of patients report a snapping or popping sensation or sound at the time of injury.
 - An acute knee injury heralded by a pop or snap, followed by a rapidly evolving effusion, almost always affirms a rupture of the ACL.
 - Disruption of the ACL may occur alone or with other knee injuries, especially a meniscal or MCL tear.
 - ACL tears are associated with anterior blows that hyperextend the knee, excessive noncontact hyperextension of the knee, and extreme deceleration forces to the knee.
- PCL tears: Patients typically report falling on a flexed knee or sustaining a direct blow to the anterior aspect of the knee (eg, when the knee strikes the dashboard in an MVA).
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- Look for abrasions, contusions, or lacerations over the knee region.
- Onset of edema and pain tends to occur within the first 3 hours after injury.
- PCL harm signifies a major injury and rarely occurs as an isolated injury.
- Rotational movements may cause a meniscal tear.

● Instability

- Decide if the patient is describing true instability, characterized by an aberrant displacement of the osseous components of the knee. (The patella dislocates laterally on the femur or the tibia slides excessively forward in an ACL-deficient knee.)
- Such terms as "giving way" or "slippage" usually denote instability. Buckling tends to be associated with a different phenomenon, often resulting from pain or muscle weakness of the quadriceps.

● Pain

- Determine if the pain is acute or chronic in nature.
- Abrupt onset of anterior knee pain with inability to bear weight indicates damage to the extensor mechanism.
- Acute pain confined to the proximity of medial or lateral regions of the knee joint tends to result from ligamentous and/or meniscal damage.
- Recent onset of pain at the posteromedial corner of the knee indicates a tear of the medial meniscus or an expanding or ruptured Baker cyst.
- Tumors are characterized by chronic pain that is worse at night.
- Discomfort from bursitis/tendinitis is likely to be chronic and bilateral, typically worse with rising or walking after sitting, and provoked by prolonged exercise or use.

- Knee effusion
 - Gauging the rapidity of onset of knee effusion may corroborate a particular diagnosis. Effusion onset within 6 hours supports the assumption of a cruciate ligament tear, articular fracture, or knee dislocation, while delayed edema occurs with meniscal injuries.
 - Nearly one half of patients who sustain an acute ligament rupture experience localized edema at the site of injury.
 - Complete ligamentous or capsular disruption can give rise to a smaller-than-anticipated amount of swelling if fluid exudes through the tear.
 - Localized distension may originate from bursitis (prepatellar bursitis), meniscal cystic changes, outgrowth of a popliteal cyst (Baker cyst), or dilation of an artery (popliteal artery aneurysm).
- Locking
 - True locking manifests as a knee that becomes stuck, usually at 45 degrees of flexion; patients cannot bend further without assistance.
 - True locking may arise from tears in the meniscus, detached tissue lodging in the knee joint, injury of the cruciate ligament(s), or an osteochondral fracture.
 - Pseudolocking may result from pain and muscle spasm secondary to increasing edema.
 - Occasionally, locking of the knee is confused with giving way of the knee. Giving way of the knee may accompany ACL injuries, quadriceps weakness, or patellar disorders.
 - Acute traumatic weakness of the knee often follows a derangement of the extensor mechanism of the knee. Significant atrophy of the quadriceps may appear within 1 week of disuse.
- Amount of immediate dysfunction: Injuries, including meniscal tears, patellar subluxation, or ligament strains or ruptures, may not entirely preclude a patient from bearing weight.
- History of prior knee problems

Physical

- Ensure adequate exposure of both lower extremities, from the groin to the toes.
- Examine the patient supine and compare the symptomatic knee with the contralateral knee.
- Develop a standard routine, so as not to omit important aspects of the examination.
- Try to alleviate the patient's fears and convince the patient to relax as much as possible during palpation and during stress testing of the injured knee joint.
- Examine the uninjured knee first to approximate baseline values and to allow the patient to understand what will be done to the injured knee.
- Focus the initial examination on inspection, palpation, and neurovascular evaluation.
- Begin distal to the injury. Observe the patient, if possible, standing and/or walking.
 - A grossly antalgic gait indicates a significant problem with one or both lower limbs.
 - Normally, when a patient stands with feet together, medial aspects of both knees and

ankles are in contact.

- Ordinarily, the knee appears hollow on either side of the patella and slightly indented just above the patella.
 - Small accumulations of fluid swell these gaps.
 - Larger effusions are most conspicuous above the patella, where the joint cavity is most spacious.
 - Classify the edema as localized (bursal) or generalized (intraarticular).
- Conspicuous atrophy of the quadriceps muscles indicates the presence of a long-standing or preexisting disorder. Atrophy of the vastus medialis muscle commonly follows surgical repair of the knee.
- Inspect and palpate the popliteal fossa, which is performed best with the patient prone.
 - Ordinarily, the only palpable structure within this space is the popliteal artery.
 - Abnormal bulges may derive from popliteal artery aneurysms, popliteal thrombophlebitis, or Baker cysts.
- Range of motion testing
 - Evaluate the knee for active flexion and extension.
 - Inability to extend the knee to any extent implies damage to the extensor mechanism.
 - Significant effusions may hinder complete extension of the knee joint and may be confused with locking.
- McMurray testing substantiates meniscal disorders.
 - With the patient supine and the knee in maximum flexion, palpate the posteromedial margin of the affected knee joint with one hand and support the foot with the opposite hand.
 - Externally rotate the lower leg as far as possible, affix varus pressure, and cautiously extend the knee joint.
 - If a tear is present in the medial meniscus, an audible, palpable, and painful clunk transpires, as the femur passes over the damaged portion of the meniscus.
 - To check the lateral meniscus, repeat the above technique, but place one hand over the posterolateral aspect of the knee joint and internally rotate the lower leg to its maximum extent.
 - Slowly extend the leg again, listening and feeling for a click or pop, and observe the patient for distress.
 - Clicks unassociated with pain or joint-line tenderness, especially during lateral meniscus testing, may represent a normal variant and should not be interpreted as evidence of a meniscal tear.
 - Unfortunately, the McMurray test is neither sensitive nor specific for meniscal damage.

Causes

The intrinsic structural framework of the knee and its exposure to the environment account for particular injuries.

- Harm may arise from direct impact, such as in contact or collision sports or blows to the knee connected to MVAs.
- Most soft-tissue wounds sustained by the knee do not, however, involve direct trauma but arise from actions producing excessive torque on the knee joint, especially those activities involving twisting, rapid deceleration, or landing from a jump.
- If tensile forces placed on the knee exceed the intrinsic tone of the ligaments, injury to the ligaments results.
- Low-intensity forces may provoke a reversible injury, with only transient deformation of the elastic ligament; however, profound loads applied to the knee joint produce irreversible rupture of the ligament fibers.
- Valgus-directed blows sustained by the externally rotated knee occur commonly.
- High-intensity impact to the lateral side of the knee tends to be serious and befalls skiers when catching a ski tip or football players following a blind side clipping collision.
- Valgus contact injuries may cause a series of injuries to the knee, including a tear of the MCL, followed by damage to the posterior medial capsule, and, finally, damage to the ACL. This combination of injuries is referred to as O'Donahue triad.
- An intense varus stress to the knee joint gives rise to a sequence of injuries, depending on the position of the knee.
- Impact to the medial side of the knee in a neutral position causes disruption of the LCL, the iliotibial band, and/or the biceps femoris.
- Profound varus strain to the extended and internally rotated knee harms not only the LCL and the ACL, but also may mar the PCL and the lateral posterior capsule.
- Undue varus stress to the flexed and internally rotated knee brings about LCL injury, proceeding to damage of the lateral posterior capsule and/or lateral meniscus and, if extreme, impairment of the PCL.
- Rotational movements may cause meniscal tears.
- Imposing extreme hyperextension force may disrupt the cruciate ligaments, conceivably anywhere along their span.
- ACL tears
 - Rupture of the ACL is among the most serious of the common knee injuries and results from a variety of mechanisms.
 - ACL tears are associated with anterior blows that hyperextend the knee, excessive noncontact hyperextension of the knee, and extreme deceleration forces to the knee.
 - Disruption of the ACL may occur alone or with other knee injuries, especially a meniscal or MCL tear.

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Differentials

Arthritis, Rheumatoid

Other Problems to be Considered

Anterior cruciate ligament rupture

Baker cyst

Bursitis

Collateral ligament tear, medial or lateral

Knee dislocation

Meniscal tear

Osteochondral fracture (osteochondritis dissecans and osteonecrosis)

Patellar dislocation

Patellar fracture

Patellar tendon rupture

Posterior cruciate ligament rupture

Segond fracture

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Workup

Lab Studies

- For patients with significant injuries or individuals who may require surgery, appropriate studies include type and screen, CBC, electrolytes, serum glucose, BUN, and creatinine.

Imaging Studies

- Plain films of the knee generally include, at the minimum, anteroposterior (AP) and lateral views.
- Some centers add 2 oblique projections that are helpful in detecting tibial plateau fractures.
- Special views, such as the tunnel view, aid in evaluating the tibial and femoral articular surfaces, while the sunrise view assists assessment of the articular surface of the patella.
- In most patients sustaining severe ligamentous or meniscal damage, plain film findings are normal.
- Fewer than 15% of knee radiographs reveal clinically significant findings.
- Recent studies recommend plain film radiographs for the following patients with an acute knee injury:
 - Patients older than 55 years (increased risk of pathological fracture associated with osteoporosis)
 - Patients experiencing tenderness over the fibular head
 - Patients with discomfort confined to the patella upon palpation
 - Patients unable to flex the knee to 90 degrees
 - Patients incapable of bearing weight, immediately and in the ED, for at least 4 steps
- Review particularly the lateral radiograph for fluid within the suprapatellar pouch. The extensor tendon mechanism normally is well outlined.
- As an effusion accumulates in the suprapatellar pouch, the posterior margin of the quadriceps is apt to be obliterated, and, with additional fluid collecting, the space between the prefemoral and the anterior superior suprapatellar fat pads widens.
- A cross-table lateral radiograph may expose a fat-fluid level, also known as a lipohearthrosis, a pathognomonic sign of an intraarticular fracture.
- Avulsion fractures of the tibial spine or the femoral condyles imply ligamentous rupture.
- A Segond fracture or a lateral capsular sign (ie, a small vertical avulsion crack of the proximal lateral tibia) denotes probable injury to the LCL and tears of the ACL.
- Avulsion of the fibular head aligns with LCL or biceps femoris injury.
- Tibial spine fractures suggest potential cruciate ligament impairment.
- Unilateral widening of the joint space may denote ligamentous instability.
- Chronic MCL damage, usually lasting longer than 6 weeks, may heal with calcification, presenting as Pellegrini-Stieda syndrome.
- A bipartite or multipartite patella is a normal variant that may be difficult to distinguish from a patellar fracture.
- An unfused secondary ossification center forming the bipartite patella typically appears in the upper lateral quadrant, tends to occur bilaterally, and reveals well-defined margin lines.
- CT scans prove effective for corroborating areas questionable for fracture in the knee region, particularly tibial plateau fractures in elderly patients.
- Ultrasound simplifies differentiation of a Baker cyst, popliteal artery aneurysm, and thrombophlebitis.
- MRI has supplanted the arthrogram as the procedure of choice for evaluating soft-tissue injuries of the knee; however, while MRI plays an important role in future surgical management of acute knee disorders, rarely does it become an essential part of the ED workup.

Other Tests

- Strong suspicion of a knee dislocation, even in the absence of reliable signs or symptoms of vascular impairment, mandates an arteriogram. Palpation of a regular pulse or Doppler confirmation of pedal pulses does not exclude vascular injury, as intimal tears may be undetectable. The incidence of a concomitant popliteal artery injury ranges from 20-50%. Do not delay emergent surgery for an arteriogram; it can be performed intraoperatively.

Procedures

- Knee joint aspiration
 - Because of its size (the largest synovial joint in the body) and relatively accessible location, the knee joint provides one of the easier sites to achieve arthrocentesis.
 - Indications for knee joint aspiration include confirmation of a diagnosis (the knee is a common site for septic and inflammatory arthritis) and presence of pain arising from a tense effusion.
 - Place the patient supine with the knee joint extended, trying to ease any contraction of the quadriceps muscles.
 - After properly cleansing the skin, infiltrate the skin and underlying dermis with local anesthetic at the point where aspiration will take place.
 - Approach the joint for aspiration from the medial aspect of the knee, with the site of puncture being 1 cm medial to the anteromedial border of the patella.
 - Insert an 18-gauge needle or catheter through the same tract used to inject local anesthetic, at the midpoint or superior position of the patella, aiming for a point between the posterior surface of the patella and the femoral intercondylar notch.
 - Approach the anterolateral border in a similar manner, mirroring the access sites.
 - Occasionally, the examiner perceives a slight give as the needle perforates the joint capsule.
 - Aspirate as much fluid as possible, recalling that the knee may contain 50 cc or more of fluid.
 - When the fluid stops flowing freely, compress the suprapatellar pouch and attempt to push additional fluid into the adjacent pouch.
 - If the bore appears obstructed during the arthrocentesis, try rotating the needle or injecting some aspirate, attempting to clear the needle. If this fails, try reinserting the needle one fourth of an inch deeper.
 - Aspiration of blood indicates a ligamentous tear (eg, ACL, PCL), osteochondral fracture, peripheral meniscus tear, capsular tear, or patellar dislocation.
 - Presence of fat globules in the aspirant is pathognomonic for an intraarticular fracture.
 - Hypertrophied synovial tissue or clot formation may hinder aspiration.
 - Instilling 5-10 cc of 1% lidocaine into the joint and then reexamining the knee may facilitate knee testing.
 - Following withdrawal of the needle, dress the iatrogenic puncture wound with an antibiotic

ointment and appropriate adhesive sterile dressing. Although rare, infection and hemarthrosis may complicate arthrocentesis.

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Treatment

Prehospital Care

- Basic responsibilities in the prehospital setting, if knee trauma is the primary injury, include stabilizing the lower extremity and monitoring the neurovascular status of the limb.
- Endeavor to realign the deformed knee only if associated neurovascular structures are compromised.
- Recheck pulses after splinting or manipulation of the limb.
- If initial efforts meet with resistance, prehospital personnel should not force realignment.
- Cover open wounds with saline-soaked sterile gauze.
- Frequently, the joint reduces spontaneously or is reduced at the scene by trained emergency medical services (EMS) personnel; in such cases, the ED physician must obtain information about the time and mechanism of injury and the original position of the limb.

Emergency Department Care

Adhere to the conventional dictums of emergency and trauma care by first corroborating the absence of life-threatening (primary survey) or limb-threatening (secondary survey) injuries before focusing on soft-tissue damage sustained by the knee. A cardinal error occurs when earliest attention is diverted to an obvious extremity injury, such as a knee dislocation, while neglecting possibly lethal trauma.

- Always determine the mechanism of injury and verify hemodynamic stability.
- Assess vascular perfusion and control any bleeding.
 - Hard signs of vascular injury include absent or diminished pulses, active hemorrhage, and expanding or pulsatile hematoma.
 - Signs of distal ischemia include pain out of proportion to the injury, pallor, paralysis, and paresthesias.
- Popliteal artery injuries must be repaired within 6-8 hours to avert amputation.
- Observe for signs and symptoms of compartment syndrome. Excluding the possibility of compartment syndrome often necessitates a definitive diagnostic test.
- Remove any constricting clothes and bandages.
- Make sure patients ingest nothing by mouth (NPO) until the need for emergent or urgent surgery is ascertained.

- For knee dislocations or grossly malaligned fractures with potential vascular compromise, attempt immediate reduction or realignment of the knee if an orthopedic specialist is not immediately available. Splint all obvious fractures and unstable knee injuries, stabilizing the femur above and the tibia below.
- General treatment principles
 - Aside from the particular injury suffered, treatment plans depend on the patient's age and activity level and the presence of additional injuries.
 - Obtain orthopedic consultation when appropriate.
 - Initial nonpharmaceutical treatment employs rest, ice, compression, and elevation (RICE).
 - Acutely (first 1-3 d), employ therapeutic measures that minimize incipient damage and reduce pain and inflammation.
 - Consider splinting the injured knee to provide support and to prevent further injury.
 - Serviceable devices include commercially available immobilizers and handcrafted compressive dressings, such as the Robert Jones dressing, which incorporates coaptation plaster.
 - Detrimental effects of immobilization include joint stiffness, degenerative changes in articular cartilage, muscle atrophy and weakness, and decreased vascularity.
- Knee dislocation mandates emergent orthopedic consultation for immediate reduction and evaluation of vascular integrity. If expedient orthopedic consultation is not obtainable and signs of vascular compromise are present, the ED physician should undertake maneuvers to restore both integrity of the joint and perfusion. (See [Dislocations, Knee](#) for specific reduction techniques.) Angiography is imperative.
- Reduction of knee dislocation
 - While most knee dislocations reduce spontaneously prior to arrival in the ED, an exam suggesting neurovascular compromise necessitates an attempt via traction and/or reduction to restore circulation, in addition to necessitating an emergent orthopedic consultation.
 - Classify dislocations with respect to the relationship of the tibia on the femur.
 - Anterior dislocations occur most commonly.
 - Ideally, perform reductions in the operating room under general anesthesia, but, if circumstances preclude this scenario, an attempt in the ED is warranted.
 - Barring contraindications, administer conscious sedation.
 - With an assistant providing stabilization and countertraction of the thigh, a second person applies longitudinal traction to the leg. Usually, this maneuver suffices for reduction.
 - Reduction of an anterior knee dislocation may be aided by trying to transpose the femur anteriorly.
 - Avoid affixing pressure over the popliteal space as it may exacerbate arterial damage.
 - For posterior dislocations (where the tibia lies posterior to the femur), attempt to reinstate the tibia anteriorly by gently lifting the tibia forward.
 - Following relocation of the knee, confirm neurovascular status and immobilize the knee in 15 degrees of flexion.
 - Order postreduction films and consultation with the orthopedic surgeon, and obtain an emergent arteriogram.
- Injection therapy

- First, ensure that the joint is not infected.
- Pain secondary to aseptic inflammation of the prepatellar, suprapatellar, and pes anserine bursae may be relieved by instilling 2-4 cc of lidocaine mixed with 15-20 mg of a prednisolone suspension into the affected bursae.

Consultations

Depending on the degree of knee instability, affected ligaments, and age and baseline activity level of the patient, early surgical intervention may be the best option.

- Emergent or urgent orthopedic consultation - Indications include the following:
 - Gross knee dislocation or unstable knee
 - All knee injuries with associated neurologic or vascular injury
 - Complete quadriceps tendon rupture or a complete patellar tendon rupture
 - First-time patellar dislocations and patellar displacements accompanied by an osteochondral fracture

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Medication

In addition to resolving the etiology of the pain, the ED physician needs to make every effort to relieve suffering as completely and expeditiously as possible. Distinguish acute from chronic distress. Consider the intensity of the pain; however, pain tends to be subjective, and therapy should be individualized.

Objective parameters, such as tachycardia, are unreliable. Usually, minor trauma to the knee that involves the ligaments, muscles, and joints causes a mild to moderately severe, self-limiting discomfort. An enormous choice of analgesics is available for use by the ED physician, but pharmacologic agents tend to fall into 2 general categories, nonnarcotic and narcotic analgesics. Furthermore, consider the best route of delivery of the drug.

Nonnarcotic Analgesics

Patients with pain accompanying minor acute soft-tissue injuries of the knee generally benefit from a short course of nonnarcotic analgesics, with acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs) being the most frequently prescribed agents. If inflammation is a component of the injury and no contraindication exists, prescribe NSAIDs, as acetaminophen lacks anti-inflammatory properties. An increasing number of NSAIDs are available for use. NSAIDs share a common mechanism of action, which involves inhibiting the production of pain-mediating prostaglandins. Generally, NSAIDs afford a

comparable degree of pain and inflammatory relief, but they differ in dosing schedule.

The 5 broad categories of marketed NSAIDs are acetic acid derivatives, fenamates, oxicams, propionic acid derivatives, and related compounds. Numerous NSAIDs are obtainable over the counter (OTC). Choosing an NSAID to prescribe can be perplexing; data comparing these agents are meager, and individual responses are inconsistent. Since no individual NSAID is clearly superior, base decisions on personal experience, safety profiles, cost, and convenience.

| | |
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| Drug Name | Acetaminophen (Tylenol, Feverall, Aspirin Free Anacin)- Most commonly ingested pain reliever; marketed in combination with other drugs to provide analgesia. Advantages include availability, cost, and relatively high safety profile. Onset of relief usually within 20-30 min. Extended-release preparations do not appear to offer major benefits (other than dosing convenience) and may increase incidence of toxicity. For children, available as drops (80 mg/0.8 mL), elixir (160 mg/5 mL), tablets (80 mg, 160 mg, 325 mg), and suppositories (125 mg, 325 mg). |
| Adult Dose | 650-1000 mg PO q4h; not to exceed 4000 mg/d PO |
| Pediatric Dose | 10-15 mg/kg/dose PO q4-6h |
| Contraindications | Documented hypersensitivity; known G-6-P deficiency |
| Interactions | Rifampin can interact to reduce analgesic effects; barbiturates, carbamazepine, alcohol, hydantoin, zidovudine, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate serious illness; APAP contained in many OTC products, and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

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| Drug Name | Acetylsalicylic acid (Bayer Aspirin, Bufferin, Anacin, Ecotrin)- Prototype NSAID; used in combination with many other drugs; available OTC. Offers anti-inflammatory benefits, unlike acetaminophen. Efficacious in PO, suppository, and topical preparations. Therapeutic anti-inflammatory serum level 15-30 mg/dL. |
| Adult Dose | 650 mg PO q4-6h |
| Pediatric Dose | 10-15 mg/kg/dose PO q 4-6h |
| Contraindications | Documented hypersensitivity; liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma Because of association with Reye syndrome, do not use in children (<16 y) with flu |

| | |
|--------------|---|
| Interactions | Effects may decrease with antacids and urinary alkalinizers; corticosteroids decrease serum levels; additive hypoprothrombinemic effects and increased bleeding time may occur with anticoagulants; may antagonize uricosuric effects of probenecid and increase toxicity of phenytoin and valproic acid; doses >2 g/d may potentiate glucose-lowering effect of sulfonylurea drugs |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | May cause transient decrease in renal function and aggravate chronic kidney disease; avoid use in patients with severe anemia, with history of blood coagulation defects, or taking anticoagulants |

| | |
|-------------------|--|
| Drug Name | Ketorolac (Toradol)- Choice of parenteral pain medications dispensed in ED. Frequently overlooked that this medication is NSAID, carrying all of the group's attendant risks, and costs almost 20 times more than morphine (and 140 times more than ibuprofen). Data supporting superiority over other analgesics scarce. |
| Adult Dose | 10 mg PO q6h prn 15-30 mg IV/IM q6h prn; administer IV dose over 15-30 sec; not to exceed 5 d of treatment |
| Pediatric Dose | <16 years: Not established >16 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding Do not administer into CNS |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulants €monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, hyperkalemia, hyponatremia, interstitial nephritis, and renal papillary necrosis may occur; increases risk of acute renal failure in patients with preexisting renal disease or compromised renal perfusion; low WBC counts (rare) usually return to normal during ongoing therapy; discontinue therapy if persistent leukopenia, granulocytopenia, or thrombocytopenia occur |

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Motrin, Advil, Nuprin)- Widely used NSAID, also available OTC, derivative of propionic class of NSAIDs and considered safest of NSAIDs. Available as tablets of 200 mg, 400 mg, 600 mg, and 800 mg; pediatric dosage forms available as both tab and oral suspension (20 mg/mL). Advise taking with food or milk if possible; caution in children with flulike illnesses. |
| Adult Dose | 400-600 mg PO q6h or to 800 mg PO q8h |
| Pediatric Dose | 30-50 mg/kg/d PO divided qid; not to exceed 2400 mg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

Narcotic Analgesics

Patients reporting inadequate pain relief from NSAIDs may benefit from short-term supplementation with an opioid compound. A wide array of products is available. Orally, hydrocodone (Lortab, Lorcet, Vicodin, Anexsia), a schedule III narcotic, and oxycodone (Roxicet, Percodan, Tylox), a schedule II substance, usually provide additional pain relief. Codeine-containing products (schedule III drugs) are not as reliable for alleviating pain. While the relative potencies of oxycodone and hydrocodone are approximately 0.33 compared with parenteral morphine, that of oral codeine is 0.05. Mixed agonist-antagonist oral agents, such as butorphanol, nalbuphine, and pentazocine, offer no real advantages to opioid agents, yet they cause a higher incidence of adverse effects. Common adverse effects include constipation, nausea, respiratory depression, sedation, and urinary retention.

Generally, the approved dosage of hydrocodone is 5-10 mg, combined with 500-750 mg of acetaminophen, administered PO q6h prn. Oxycodone analgesic preparations typically combine 2.5-5 mg of oxycodone with 325 mg of acetaminophen, dosed as 1-2 tabs PO q4h prn for moderate to severe pain. Acetaminophen with codeine (Tylenol #3) contains 30 mg of codeine with 325 mg of acetaminophen. Usually, 1-2 pills q4h prn is recommended.

Elixirs containing hydrocodone (Hycodan) are convenient for children older than 6 years with

moderately severe to severe pain and who are unable to swallow pills. One tsp (5 mL) of Hycodan contains 5 mg of hydrocodone, with 1.25-2.5 mg administered q4h, depending on the child's size and severity of pain. The elixir of Tylenol with codeine for children contains 120 mg of acetaminophen and 12 mg/5mL of codeine in an alcohol base (7%).

Orally administered drugs generally impart a slower onset of action. For patients in severe pain or for those patients who must be kept NPO, parenteral agents may be necessary. Although the IM route may be more convenient for the staff, the IV route offers a number of advantages. Narcotics administered IV provide a rapid and predictable onset of action and are easier to titrate. Morphine and meperidine are the most commonly used parenteral narcotic agents.

| | |
|-------------------|--|
| Drug Name | Hydrocodone and acetaminophen (Vicodin, Lorcet, Lortab, Anexsia)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose of acetaminophen q4-6h prn; not to exceed 2.6 g/d of acetaminophen >12 years: 750 mg of acetaminophen q4h Single dose not to exceed 10 mg of hydrocodone bitartrate; do not exceed 5 doses in 24 h |
| Contraindications | Documented hypersensitivity; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Tablets contain metabisulfite which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|--|
| Drug Name | Oxycodone and acetaminophen (Percocet, Tylox, Roxicet)- Drug combination indicated for relief of moderately severe to severe pain. |
| Adult Dose | 1-2 tab or cap PO q4-6h prn |
| Pediatric Dose | 0.05-0.15 mg/kg/dose oxycodone PO q4-6h prn; not to exceed 5 mg/dose of oxycodone |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|---|
| Precautions | Duration of action may increase in the elderly; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4000 mg/24h of acetaminophen; higher doses may cause liver toxicity |
|-------------|---|

| | |
|-------------------|---|
| Drug Name | Acetaminophen and codeine (Tylenol #3)- Drug combination indicated for treatment of mild to moderately severe pain. |
| Adult Dose | 30-60 mg/dose (based on codeine content) PO q4-6h or 1-2 tab q4h; not to exceed 12 tabs/d |
| Pediatric Dose | 0.5-1 mg/kg/dose (based on codeine content) PO 10-15 mg/kg/dose q4h (based on acetaminophen content); not to exceed 2.6 g/d |
| Contraindications | Documented hypersensitivity |
| Interactions | CNS depressants or tricyclic antidepressants increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|--|
| Drug Name | Morphine sulfate (Oramorph, MS Contin, Duramorph)- Criterion standard for relief of acute severe pain; may be administered in a number of ways; commonly titrated until desired effect obtained. IV morphine demonstrates half-life of 2-3 h; however, half-life may be 50% longer in the elderly. |
| Adult Dose | Loading dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h Relatively hypovolemic patients: Start with 2 mg IV/IM/SC and reassess hemodynamic effects of dose |
| Pediatric Dose | 0.11-0.2 mg/kg/dose IV; then titrate carefully to adequate pain relief |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

| | |
|-------------------|--|
| Drug Name | Meperidine (Demerol)- Narcotic analgesic with multiple actions similar to morphine; however, may cause less constipation, smooth muscle spasm, and depression of cough reflex than similar analgesic doses of morphine. Serum half-life 2 h. Metabolism occurs by hepatic demethylation into normeperidine. Patients with moderately severe to severe liver disease may develop excessive levels of metabolites, precipitating CNS side effects, including tremors and seizures. |
| Adult Dose | 50 mg PO/IV/IM/SC initially; then, titrate up to 150 mg |
| Pediatric Dose | 0.5-1 mg/kg/dose PO/IV/IM/SC; not to exceed 150 mg |
| Contraindications | Documented hypersensitivity; concurrent MAOIs; upper airway obstruction or significant respiratory depression; during labor when delivery of premature infant anticipated |
| Interactions | Monitor for increased respiratory and CNS depression with coadministration of cimetidine; hydantoins may decrease effects; avoid with protease inhibitors |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients with head injuries since meperidine may increase respiratory depression and CSF pressure (use only if absolutely necessary); caution when using postoperatively and with history of pulmonary disease (suppresses cough reflex) Substantially increased dose levels given because of tolerance may aggravate or cause seizures even if no history of convulsive disorders; monitor closely for meperidine-induced seizure activity if prior seizure history |

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Follow-up

Further Inpatient Care

- Consequential soft-tissue damage of the knee with concurrent serious disorder, such as vascular compromise, threatening compartment syndrome, or multiple trauma, warrants admission to the appropriate surgical or orthopedic service for monitoring and potential surgical intervention.
- Injuries requiring timely surgery, such as knee dislocation, complete quadriceps tendon rupture, and total patellar tendon rupture, may necessitate admission to the general orthopedic service.

Further Outpatient Care

- The type and degree of injury dictate the timing and specifics of follow-up care. Arrange checkups, preferably within 24 hours, for any patient sustaining soft-tissue injury of the knee in which (1) muscle spasm precludes adequate knee assessment, (2) the mechanism of injury

suggests a more serious injury, or (3) the patient perceives a snap or pop at the time of the incident, and a hemarthrosis evolves.

- Promote RICE therapy for mild to moderately severe strains/sprains.
 - R - Rest (crutch ambulation without weight bearing for initial 24-48 h)
 - I - Ice (application of ice on injured region for 20 min of each waking hour during the initial 48 h after injury)
 - C - Compression (with knee brace or splint, if necessary)
 - E - Elevation (above the level of the heart)
- The necessity for arthroscopy or MRI to clarify equivocal diagnoses usually is made by the orthopedic consultant when the diagnosis remains in doubt.

Transfer

- Patients with soft-tissue knee injuries complicated by vascular impairment or additional confounding trauma may require transfer to a facility capable of evaluation and treatment of such injuries.

Deterrence/Prevention

- Prophylactic bracing may prevent further injury.
- Conditioning programs strengthen surrounding supporting structures.

Complications

- Specific dislocations and fractures predispose the knee to popliteal artery and/or peroneal nerve damage.
- Significant soft-tissue injuries of the knee and lower leg risk the development of compartment syndrome.
- Knee joint instability may follow unrecognized ligament damage.
- Complications of ACL injuries include occurrence of abnormal knee motion, eventually causing major degenerative changes in the knee joint.
- Recurrent locking, damage to the articular cartilage, and ensuing arthritis may follow missed meniscal injuries.
- Infection may arise from abrasions, lacerations, aspiration, or injection of the knee. If unrecognized, knee joint destruction results.
- Spontaneous rupture of tendons may follow use of intraarticular steroids.

Prognosis

- Sprains
 - Most grade I and grade II collateral ligament sprains heal uneventfully following a 4- to 6-

week course of conservative therapy; however, chronic pain and tendency for recurrent injury may befall the patient.

- Grade III collateral sprains invariably give rise to tears of the posterior capsule, frequently requiring 3 months or more of bracing and physical therapy prior to returning to unrestricted activity.
- A high rate of recurrence follows simple aspiration of a Baker cyst, while these cysts reappear less than 5% of the time following surgical correction.
- Infection and chronic weakness of the extensor apparatus may follow surgical repair.
- Development of recurrent locking, popping, or effusions subsequent to an adequate trial of conservative therapy for meniscal tears may suggest the need for surgical intervention.

Patient Education

- Failure to respond to conservative treatment may indicate a missed or overlooked diagnosis, such as ligamentous or meniscal damage, which is more complicated than first suspected.
- Follow-up care is essential. Inadequate treatment may result in chronic instability and/or degenerative joint disease. Concurrent collateral ligament injuries and meniscal tears often are difficult to diagnose, increasing the importance of follow-up care.
- Physical therapy focuses on quadriceps strengthening and extensor stretching, in conjunction with ultrasound and phonophoresis.
- For patients recovering from a patellar subluxation or dislocation, once the immediate problems are under control, focus further therapy on quadriceps strengthening and use of a patellar cutout brace.

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Miscellaneous

Medical/Legal Pitfalls

- Do not automatically focus on the knee if the patient is complaining of knee pain. Hip pathology and disorders of other anatomic sites may masquerade as knee problems.
- While major injuries to the knee often make weight bearing unattainable, the ability to walk does not exclude serious internal derangement; the absence of joint effusion does not rule out significant internal damage.
- Severe pain, muscle spasm, or expansion of an effusion may mask knee joint instability.
- Do not neglect to test the patient for active knee extension. About one half of patients with a quadriceps tendon rupture initially are misdiagnosed. Delayed diagnosis of extensor apparatus disruption may lead to contracture of the affected muscles, impairing the ability for later surgical

repair of the lesion.

- Failure to properly diagnose or to adequately treat a meniscal tear may result in chronic osteoarthritis in the knee joint.
- Overzealous use of intraarticular corticosteroid injections may produce spontaneous rupture of tendons.
- Documentation
 - Document the neuromuscular evaluation, including verification of full function of the extensor apparatus.
 - Record observations that excluded obvious dislocations and fractures.
 - Record results of valgus and varus stress testing and the Lachman maneuver.

Special Concerns

- Pregnancy
 - Knee problems, especially underlying meniscal and patellar problems, may worsen with pregnancy because of changes in the biomechanics of weight bearing and shifting in the center of gravity with fetal development.
 - Production of relaxin hormone occurring during pregnancy may modify ligaments in addition to those of the pelvis, increasing knee laxity.
 - The use of radiography in pregnancy is always a concern. Although some risk is associated with taking diagnostic radiographs in pregnancy, animal and human data do not reveal an increased risk to the fetus when fetal exposure is limited. After 20 weeks of gestation, risk of radiation exposure causing fetal abnormalities is remote; however, adopt the practice of avoiding unessential radiographs. If x-rays are deemed indispensable, shield the abdomen.
- Geriatric
 - Extensor mechanism rupture may precede trivial trauma in the elderly, especially in individuals with coexisting disorders, such as renal failure, systemic lupus erythematosus, hyperparathyroidism, or diabetes mellitus.
 - Because of inveterate degenerative disease, meniscal tears in older patients may emerge despite a history of minimal or no trauma. For example, simply rising from a squatting position may cause a tear.
 - Discomfort and distention relating to the knee following a fall may arise from a tibial plateau fracture, especially of the lateral plateau.
 - Osteoporosis, which occurs commonly in elderly persons, leaves bones more vulnerable to fracture. Visualization of fractures on plain films proves difficult, and fractures may be overlooked easily. Misdiagnosing this fracture as a soft-tissue injury may permit additional morbidity, as fracture fragments may be displaced.

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Legg-Calve-Perthes Disease

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Synonyms, Key Words, and Related Terms

Legg-Calvé-Perthes disease

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Introduction

Background

Legg-Calvé-Perthes disease (LCPD) is the name given to idiopathic osteonecrosis of the capital femoral epiphysis of the femoral head. The goal of treatment is to avoid severe degenerative arthritis.

Pathophysiology

The capital femoral epiphysis always is involved. In 15-20% of patients with LCPD, involvement is bilateral.

Frequency

- **In the US:** One in 1200 children younger than 15 years is affected by LCPD.

Mortality/Morbidity

- It is a self-limited disease if it is not treated.
- Outcome is extremely variable.

Race

Caucasians are affected more frequently than other races.

Sex

Males are affected 4-5 times more often than females.

Age

LCPD most commonly is seen in persons aged 3-12 years, with a median age of 7 years.

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Clinical

History

Symptoms usually have been present for weeks because the child often does not complain.

- Hip or groin pain, which may be referred to the thigh
- Mild or intermittent pain in anterior thigh or knee
- Limp
- Usually no history of trauma

Physical

- Decreased range of motion (ROM), particularly with internal rotation and abduction

- Painful gait
- Atrophy of thigh muscles secondary to disuse
- Muscle spasm
- Leg length inequality due to collapse
- Thigh atrophy: Thigh circumference on the involved side will be smaller than on the unaffected side secondary to disuse.
- Short stature: Children with LCPD often have delayed bone age.
- Roll test
 - With patient lying in the supine position, the examiner rolls the hip of the affected extremity into external and internal rotation.
 - This test should invoke guarding or spasm, especially with internal rotation.

Causes

The etiology remains unclear; however, the following scenario generally is accepted:

- The blood supply to the capital femoral epiphysis is interrupted.
- Bone infarction occurs, especially in the subchondral cortical bone, while articular cartilage continues to grow. (Articular cartilage grows because its nutrients come from the synovial fluid.)
- Revascularization occurs, and new bone ossification starts.
- At this point, a percentage of patients develop LCPD, while other patients have normal bone growth and development.
- LCPD is present when a subchondral fracture occurs. This is usually the result of normal physical activity, not direct trauma to the area
- Changes to the epiphyseal growth plate occur secondary to the subchondral fracture.

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Differentials

Anemia, Sickle Cell
Arthritis, Rheumatoid
Fractures, Pelvic
Hypothyroidism and Myxedema Coma
Pediatrics, Limp
Tuberculosis

Other Problems to be Considered

Septic hip

Toxic synovitis

Lymphoma

Spondyloepiphyseal dysplasia

Metaphyseal dysplasia

Slipped femoral capital epiphysis

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Workup

Lab Studies

- CBC
- Erythrocyte sedimentation rate - May be elevated if infection present

Imaging Studies

- Plain x-rays of the hip are extremely useful in establishing the diagnosis.
- Frog leg views of the affected hip are very helpful.
- Multiple radiographic classification systems exist, based on the extent of abnormality of the capital femoral epiphysis.
 - Waldenstrom, Catterall, Salter and Thompson, and Herring are the 4 most common classification systems.
 - No agreement has been reached as to the best classification system.
- Technetium 99 bone scan - Helpful in delineating the extent of avascular changes before they are evident on plain radiographs
- Dynamic arthrography - Assesses sphericity of the head of the femur

Procedures

- Hip aspiration if a septic joint is suspected

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Treatment

Emergency Department Care

- Goals of treatment
 - Achieve and maintain ROM
 - Relieve weight bearing
 - Containment of the femoral epiphysis within the confines of the acetabulum
 - Traction

Consultations

- Once the diagnosis of LCPD is suspected, an orthopedic surgeon or a pediatric orthopedic surgeon should be contacted for further management decisions.
- An orthopedic consultant may choose to order more specialized tests (eg, bone scintigraphy, arthrogram, and magnetic resonance imaging), usually on an outpatient basis, to better determine the extent of the disease.

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Medication

Medical treatment does not stop or reverse the bony changes. Appropriate analgesic medication should be given.

Nonsteroidal Anti-Inflammatory Drugs (NsAIDs)

These drugs most commonly are used for the relief of mild to moderately severe pain. Although the effects of NSAIDs in the treatment of pain tend to be patient specific, ibuprofen is usually the DOC for the initial therapy.

| | |
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| Drug Name | Ibuprofen (Advil, Motrin, Nuprin)- Usually DOC for treatment of mild to moderately severe pain if no contraindications. Inhibits inflammatory reactions and pain, probably by decreasing activity of enzyme cyclooxygenase, thus decreasing prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h while symptoms persist; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid or qid; start at lower end of dosing range and titrate upward; not to exceed 2.4 g/d >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|---|
| Drug Name | Acetaminophen (Tylenol, Panadol, Aspirin-Free Anacin)- DOC for treatment of pain in patients with documented hypersensitivity to aspirin and NSAIDs, as well as those with upper GI disease or who are taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg PO q4h; not to exceed 5 doses in 24 h |
| Contraindications | Documented hypersensitivity; known G-6-P deficiency |
| Interactions | Rifampin can reduce analgesic effects; barbiturates, carbamazepine, hydantoin, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate serious illness; APAP contained in many OTC products, and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

Follow-up

Further Outpatient Care

- LCPD does not require emergent inpatient care.
- Treatment may involve observation, usually in children younger than 6 years.
- Bed rest and abduction stretching exercises are recommended.
- Nonsurgical containment allows the femoral head to stay within the acetabulum, where it can be molded. Various casts, braces, and crutches have been used for containment.
- Initially, close follow-up is required to determine the extent of necrosis.
- Once the healing phase has been entered, follow-up can be every 6 months.
- Long-term follow-up is necessary to determine the final outcome.
- Surgical correction of gross deformities of the femoral head may be necessary.

Complications

- LCPD may result in femoral head deformity and degenerative joint disease.
- The femoral head may be distorted permanently.

Prognosis

- The younger the age of onset of LCPD, the better the prognosis.
- Children older than 10 years have a very high risk of developing osteoarthritis.
- Most patients have a favorable outcome.
- Prognosis is proportional to the degree of radiologic involvement.

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Miscellaneous

Medical/Legal Pitfalls

- Radiographs of the hip should always be considered for a child complaining of thigh or knee pain.
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Lumbar (Intervertebral) Disk Disorders

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Introduction

Background

Lumbar disk disease is a frequent source of low back pain.

Sciatica is defined as neuralgia along the course of the sciatic nerve.

Pathophysiology

The intervertebral disks act as shock absorbers and are found between the bodies of the vertebrae. They have a central area composed of a colloidal gel, called the nucleus pulposus, which is surrounded by a fibrous capsule, the annulus fibrosis. This structure is held together by the anterior longitudinal ligament, which is anterior to the vertebral bodies, and the posterior longitudinal ligament, which is posterior to the vertebral bodies and anterior to the spinal cord. The muscles of the trunk provide additional support.

The most common site of disk herniation is at the L5-S1 interspace in the lumbosacral region. This is believed to be due to the thinning of the posterior longitudinal ligament as it extends caudally.

Nomenclature specific to lumbar disk disease

- Disk bulge - Annular fibers intact
- Disk protrusion - Localized bulging with damage of some annular fibers
- Disk extrusion - Extended bulge with loss of annular fibers, but disk remains intact
- Disk sequestration - Fragment of disk broken off from the nucleus pulposus

Frequency

- **In the US:** Sciatica has been reported by various authors to occur in 1-10% of the population.

Mortality/Morbidity

Low back pain usually is self-limited and of short duration.

Sex

The male-to-female ratio is approximately 1:1.

Age

The group most commonly affected is adults aged 25-45 years.

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Clinical

History

The history guides the physician's usage of ancillary testing and consultations, but it often is not enough to differentiate disk disease from other causes of back pain.

- Patients with disk disease usually are not able to give a precise time that the problem began because it usually is preceded by multiple episodes of less severe low back pain.
- Asking the patient the location of the pain is important.
 - Pain that is localized to the lower back and gluteal area often is associated with disk disease.
 - Pain associated with nerve root involvement commonly radiates down the leg, particularly

below the level of the knee.

- Ask the patient about any unusual recent activity, especially if it involved the patient remaining in a flexed or rotated position. Find out if the patient experienced any recent trauma.
- Pain with flexion, rotation, or prolonged sitting or standing, and sharp (rather than dull) pain are suggestive of disk disease.
- The onset of pain may begin suddenly or gradually after injury.
- Typically, the pain is located bilaterally at the posterior belt line.
- The pain pattern usually is referred rather than radicular.
- Back motion, sitting, standing, lifting, bending, and twisting usually aggravate the pain; it often is relieved with rest and a recumbent position.

Physical

Nerve roots exit the spine below the intervertebral disks; thus, herniation of a disk involves the nerve root below it.

- Observe the patient for abnormal gait, which is suggestive of a loss of the normal rhythm. Have ambulatory patients walk on their toes to test the function of S1.
- Observe the patient for abnormal posture, which is suggestive of splinting or guarding from pain.
- Test the patient's ability to dorsiflex the foot while sitting to test the L5 nerve root. Test for sensory loss that corresponds to a dermatomal area.
- Palpation of the lumbar spine and lower back is not helpful in the diagnosis of disk disease, but it should be done to rule out other causes of low back pain.
- A positive straight leg-raising test is indicative of nerve root involvement.
 - This test is performed while the patient is lying supine with one leg either straight or flexed at the knee, with the sole of the foot flat on the stretcher. The other leg is kept straight and lifted by the examiner.
 - If pain occurs when the leg is lifted between 30-70 degrees from horizontal and travels down the leg until below the knee, the test is positive.
- Patients may exhibit decreased lumbar range of motion (ROM).
- The usual motor, sensory, and reflex examinations (including perianal sensation and anal sphincter tone) should be performed.
- A careful abdominal and vascular examination is mandatory in evaluation of these patients.

Causes

- The normal aging process of the musculoskeletal system aggravates acute events.
- Risk factors
 - Age
 - Activity
 - Smoking

- Obesity
- Vibration (eg, driving a car)
- Sedentary lifestyle
- Psychosocial factors

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Differentials

Aneurysms, Abdominal
Arthritis, Rheumatoid
Back Pain, Mechanical
Cauda Equina Syndrome
Epidural and Subdural Infections
Osteomyelitis
Pelvic Inflammatory Disease
Renal Calculi

Other Problems to be Considered

Spinal stenosis

Spondylolisthesis

Fracture

Ankylosing spondylitis

Degenerative joint disease

Metastases

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Workup

Lab Studies

- Laboratory tests generally are not helpful in the diagnosis of lumbar disk disease.
- Indications for screening laboratory examinations include a pain of a nonmechanical nature, atypical pain pattern, persistent symptoms, and age older than 50 years.
 - Complete blood count (CBC) with differential
 - Erythrocyte sedimentation rate (ESR)
 - Alkaline and acid phosphatase
 - Serum calcium
 - Serum protein electrophoresis

Imaging Studies

- Radiographic studies are very helpful in the diagnosis of lumbar disk disease, but several important caveats should be taken into account with the use of these tests.
- Most patients with pain from lumbar disk disease will have resolution of their symptoms with conservative treatment.
- Unless the patient is immobilized completely by the pain and requires admission, or the pain has been present for more than 6 weeks, diagnostic studies are not recommended.
- MRI is the imaging modality of choice in evaluating patients with lumbar disk disease. Studies have shown that as many as 60% of people without back symptoms have disk bulges and protrusions on MRI. Therefore, these findings may not correlate with the patient's symptoms.
- CT scanning is useful for diagnosing disk disease but is less sensitive than MRI. Combining CT scan with myelography can increase its sensitivity.
- Myelography may provide a definitive diagnosis on its own, but this is an invasive test requiring a lumbar puncture and the use of contrast material.
- Plain films of the lumbar spine generally are not helpful in the diagnosis of lumbar disk disease, except to rule out other diseases.
- Bone scan (scintigraphy)
 - Technetium-99m labeled phosphorus indicates active mineralization of bone.
 - A bone scan is indicated to rule out tumors, trauma, or infection.

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Treatment

Prehospital Care

Little is needed in the way of prehospital care. Appropriate spinal immobilization should be considered if

the patient has evidence of trauma; otherwise, simple transportation in a comfortable position is all that is indicated.

Emergency Department Care

- Patients should lie in a position in which they are most comfortable.
- Muscle relaxants are of limited use and clinical studies have not proven their efficacy. This class includes benzodiazepines, methocarbamol, and cyclobenzaprine. Patients should be warned that all of these drugs are sedating.
- Opioids provide very effective acute pain relief, but they should not be used in patients with chronic pain.
- Salicylates, acetaminophen, and nonsteroidal anti-inflammatory drugs (NSAIDs) all have been used in the treatment of pain from lumbar disk disease, but none of these has been shown to be superior to the others. Acetaminophen lacks anti-inflammatory activity.

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Medication

The goals of therapy are to reduce pain and inflammation.

Nonsteroidal Anti-Inflammatory Drugs (NsAIDs)

These agents are used most commonly for the relief of mild to moderately severe pain. Although effects of NSAIDs in the treatment of pain tend to be patient specific, ibuprofen usually is the DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually DOC for treatment of mild to moderately severe pain if no contraindications. |
| Adult Dose | 200-400 mg PO q4-6h while symptoms persist; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid or qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |

| | |
|--------------|--|
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Ketoprofen (Oruvail, Orudis, Actron)- Used for relief of mild to moderately severe pain and inflammation. Administer small dosages initially to patients with small body size, to the elderly, and to those with renal or liver disease. Doses higher than 75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe patient for response. |
| Adult Dose | 25-50 mg q6-8h PO prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 12 years: 0.1-1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in coagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Flurbiprofen (Ansaid)- May inhibit enzyme cyclooxygenase, which in turn inhibits prostaglandin biosynthesis. These effects may be mechanism of its analgesic, antipyretic, and anti-inflammatory activities. |
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |

| | |
|--------------|--|
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

| | |
|-------------------|--|
| Drug Name | Naproxen (Anaprox, Naprelan, Naprosyn)- Used for relief of mild to moderately severe pain. Inhibits inflammatory reactions and pain by decreasing activity of enzyme cyclooxygenase, causing decrease in prostaglandin synthesis. |
| Adult Dose | 500 mg, followed by 250 mg PO q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity; may decrease effects of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT in patients taking anticoagulantsâ€ monitor PT closely and instruct patients to watch for signs of bleeding; may increase risk of methotrexate toxicity; may increase phenytoin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

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Follow-up

Further Inpatient Care

- Inpatient care generally is not required, except for those rare cases of intractable pain or in cases in which the social situation does not allow adequate home care. Further inpatient care mostly consists of continued analgesics, physical therapy, and possible consultation with a spine specialist.

Further Outpatient Care

- The patient's bed does not have to be rock hard. Patients should lie in a position in which they are most comfortable.
- Bed rest is not recommended most of the time. The exception is for patients whose pain is so severe that they cannot ambulate.
 - Prolonged immobilization may worsen pain and extend recovery time.
 - Strict bed rest should never exceed 2 days. Patients should be encouraged to begin limited activity as soon as possible.
- Multiple surgical techniques have been used in patients with disk herniation who have not responded to 6 weeks of conservative therapy. These techniques include discectomy, spinal fusion, and injection of chymopapain. Surgery resolves symptoms in up to 90% of patients; however, repeat operations have higher failure rates.

Deterrence/Prevention

- Smoking cessation
- Weight reduction
- Improve general physical condition
- Avoid aggravating factors

Complications

- Incorrect diagnosis
- Chronic low back pain
- Narcotic addiction
- Persistent psychosocial problems

Prognosis

- Most patients can resume normal activities.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to consider alternate diagnoses, such as acute vascular, neurologic, malignant, or infectious etiologies, of the patient's pain
 - Failure to adequately advise the patient of warning signs of complications from their diagnosis. These may include loss of bowel or bladder control due to cauda equina syndrome, persistent leg numbness, weakness from a further disk herniation, or other complications.
-

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Nailbed Injuries

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Introduction

Background

Injuries to the nailbeds are common. Hand injuries account for approximately 10% of ED visits, and many of these visits involve injury to the nailbeds.

The fingernails protect the fingertips, enable persons to pick up small objects, and contribute to tactile sensation. An abnormality of the nail can cause cosmetic and functional problems.

Pathophysiology

To discuss nailbed injuries, the following definitions must be understood:

- Perionychium - Nailbed and paronychium
- Nail fold - Area into which the proximal nail fits
- Paronychium - Lateral nail folds
- Nail wall - The skin over the dorsum of the nail fold
- Eponychium (cuticle) - The thin wall extending from the nail wall onto the dorsum of the nail
- Lunula - The white, curved opacity, just distal to the eponychium, and located at the junction of

the germinal (intermediate) and the sterile (ventral) matrixes

- Nailbed (matrix) - All of the soft tissue beneath the nail
- Hyponychium - The tissue between the nailbed and the distal nail (very resistant to infection)

The germinal (intermediate) matrix forms the majority of the nail, with some material being added to the undersurface of the nail by the sterile (ventral) matrix. The arterial blood supply comes from 2 terminal branches of the volar digital artery. The venous drainage gathers in both the proximal portion of the nailbed and the skin proximal to the nail fold, and then drains over the dorsum of the finger. Numerous lymphatic vessels are present in the nailbed. The nerve supply arises from the dorsal branch of the volar digital nerve.

Longitudinal growth takes between 70-160 days, with a delay of 21 days in distal growth after injury during which the nail thickens proximal to the injury site. The nail progresses distally because of the confinement of the nail fold. The material produced by the roof of the nail fold is responsible for the shiny surface of the nail. Scar tissue does not produce any nail. Therefore, a scar of the dorsal roof creates a dull streak, a scar of the germinal matrix may cause a split or absent nail, and a scar in the sterile matrix may cause a split or nonadherence of the nail beyond the scar.

Frequency

- **In the US:** Nailbed injuries are very common, but the exact prevalence is unknown.

Mortality/Morbidity

Complications of nailbed injuries include abnormal growth, nonadherence of the new nail, soft tissue infection, and osteomyelitis.

Sex

No sex predilection exists.

Age

Nailbed injuries occur in all ages.

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Clinical

History

- The history should include the patient's general health status, prior hand injuries, tetanus status, and right- or left-handedness.
- The patient's occupation and hobbies also are important.
- The mechanism of the injury must be determined because of the prognostic implications.
- Most frequently, the long finger is injured.
- The middle and distal third of the nail are the most frequent sites of injury.
- Fifty percent of injuries have an associated distal phalanx or tuft fracture.

Physical

Subungual hematomas, simple lacerations, stellate lacerations, crush injuries, avulsion injuries, and disruption of the proximal nail out of the nail fold are among the basic types of nailbed injuries.

- Subungual hematomas are caused by disruptions (ie, lacerations) in the nailbed.
- In general, the prognosis is worse with increasing tissue damage and with more proximal injuries.

Causes

The most common causes of injury are secondary to smashing the finger between 2 hard objects (eg, doors, hammers), saws, knives, and lawnmowers.

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Differentials

Fingertip Injuries

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Workup

Imaging Studies

- A finger x-ray may be indicated for extensive injuries to rule out a concomitant phalangeal fracture.

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Treatment

Emergency Department Care

- If the subungual hematoma involves less than 25% of the visible nail plate, a hole must be made to relieve the pressure of a hematoma. This can be done with a heated paper clip or a handheld ophthalmic cautery.
- If the injury involves more than 25% of the nail plate with a large hematoma, the nail may be removed to examine the injury.
 - Proper anesthesia is required. This is best accomplished with a digital block or a metacarpal block.
 - The nail is elevated using the blades of iris scissors; a cleavage plane is developed between the nailbed matrix and the nail plate.
 - Once the nail plate is elevated, gentle distal traction on the nail plate will separate it from the proximal sulcus.
- Severe stellate lacerations and nailbed avulsion injuries carry a poor prognosis.
- It is important to return all fragments of the nailbed as accurately as possible. Often, a fragment of the nailbed is attached to the undersurface of the avulsed nail. If it is small, the nail and the nailbed may have to be replaced without separating them.
- Scar tissue (ie, granulated tissue) does not form nail, and nail does not adhere to scar tissue. Surgical referral is suggested for severe crush injuries and nailbed avulsions. Occasionally, grafting is necessary.
- When the proximal nail has been displaced out of the nail fold, the nail should be removed, the nailbed should be repaired, and most importantly, the proximal nail should be reinserted into the nail fold. A drainage hole should be placed in the nail prior to replacement. The nail should be sutured in place with 6-0 sutures for 3 weeks.

Consultations

A hand surgeon should be consulted for severe injuries.

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Follow-up

Further Outpatient Care

- In general, except for a simple subungual hematoma, a wound check in 3-5 days is suggested to check for infection and to repack the nail fold, if necessary.
- Tape or sutures should be removed from any replaced nail in approximately 2-3 weeks.
- The old nail will fall off as the new nail grows in.
- The routine use of prophylactic antibiotics has no proven value.

Complications

- Scarring
- Destruction of the nail plate with a lack of new nail growth or disrupted nail growth
- Infection

Prognosis

- Nailbed injuries generally heal well with appropriate treatment.

Patient Education

- All patients should be advised that there is the possibility of a deformed nail.

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Miscellaneous

Medical/Legal Pitfalls

- Careful repair of nailbed lacerations should be performed to lessen the chance of a cosmetically unacceptable result.
 - Patients should be advised of the potential of having a deformed nail.
-

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Neck Trauma

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Introduction

Background

Few emergencies pose as great a challenge as neck trauma. Because a multitude of organ systems (eg, airway, vascular, neurological, gastrointestinal) are compressed into a compact conduit, a single penetrating wound is capable of considerable harm. Furthermore, seemingly innocuous wounds may not manifest clear signs or symptoms, and potentially lethal injuries could be easily overlooked or discounted.

Airway occlusion and exsanguinating hemorrhage pose the most immediate risks to life. From the time when Ambroise Pare successfully treated a neck injury in 1552, debate has continued about the best approach for particular neck wounds. Awareness of the various presentations of neck injuries and the establishment of a well-conceived multidisciplinary plan prior to the traumatic event is critical for improving patient outcome.

Pathophysiology

A clear understanding of the anatomic relationships within the neck and the mechanisms of injury is

critical to devising a rational diagnostic and therapeutic strategy.

Anatomic relationships

Structures at risk

With the neck protected by the spine posteriorly, the head superiorly, and the chest inferiorly, the anterior and lateral regions are most exposed to injury. The larynx and trachea are situated anteriorly and are, thus, readily exposed to harm. The spinal cord lies posteriorly, cushioned by the vertebral bodies, muscles, and ligaments. The esophagus and the major blood vessels are between the airway and spine.

Musculoskeletal structures at risk include the cervical spine; cervical muscles, tendons, and ligaments; clavicles; first and second ribs; and hyoid bone.

Neural structures at risk include the spinal cord, phrenic nerve, brachial plexus, recurrent laryngeal nerve, cranial nerves (specifically IX-XII), and stellate ganglion.

Vascular structures at risk include the carotid (common, internal, external) and vertebral arteries and the vertebral, brachiocephalic, and jugular (internal and external) veins.

Visceral structures at risk include the thoracic duct, esophagus and pharynx, and larynx and trachea.

Glandular structures at risk include the thyroid, parathyroid, submandibular, and parotid glands.

Associated structures at risk of intrathoracic injuries include the esophagus, tracheobronchial tree, lung, heart, and great vessels.

Anatomic zones

Dividing the neck into anatomic zones or regions assists in the evaluation of injury. Serving as the line of demarcation, the sternocleidomastoid separates the neck into anterior and posterior triangles. The majority of the important vascular and visceral organs lie within the anterior triangle bounded by the sternocleidomastoid posteriorly, the midline anteriorly, and the mandible superiorly. Except for individual nerves to specific muscles, few vital structures cross the posterior triangle, which is delineated by the sternocleidomastoid, the trapezius, and the clavicle (with the exception of the region just superior to the clavicle).

For clinical purposes, the neck is partitioned into 3 zones. Zone I is the thoracic inlet to the cricoid cartilage level, which defines the base of the neck. Structures at greatest risk in this zone are the great vessels (subclavian vessels, brachiocephalic veins, common carotid arteries, and jugular veins), aortic arch, trachea, esophagus, lung apices, cervical spine, spinal cord, and cervical nerve roots. Zone II

encompasses the midportion of the neck and the region from the cricoid cartilage to the angle of the mandible. Important structures in this region include the carotid and vertebral arteries, jugular veins, pharynx, larynx, trachea, esophagus, and cervical spine and spinal cord. Zone III characterizes the superior aspect of the neck and is bounded by the angle of the mandible and the base of the skull. Diverse structures, such as the salivary and parotid glands, esophagus, trachea, cervical spine, carotid arteries, jugular veins, and major nerves (including cranial nerves IX-XII), traverse this

zone.

Mechanisms of injury

Neck trauma may be caused by penetrating or blunt trauma.

Penetrating trauma

Penetrating trauma includes gun shot wounds (GSWs) and stab wounds. These involve knives in 50% of cases, guns in 45% of cases, and shotguns in 5% of cases.

Generally, people with GSWs sustain greater injury than those with stab wounds because of a bullet's proclivity to penetrate deeper and cause cavitation, damaging structures lying outside the tract of the missile. Bullets tend to course randomly and follow a more direct pathway. GSWs, particularly those involving high-velocity bullets (>2000-2500 ft/s) produced by military-style weapons or hunting rifles, generate shock waves that devitalize surrounding tissues. High-velocity missiles and their ensuing blast effects may suck debris into the wound tract or cause secondary injuries from bullet or bone fragmentation.

A transcervical GSW is more likely to cause a grave injury than a GSW involving injury to only 1 side of the neck. Close-range GSWs of the neck that produce massive destruction are usually fatal. After a GSW to the neck, surgery is indicated in 75% of cases, while only 50% of neck stab wounds require surgical exploration.

Vascular injuries arising from penetrating trauma may occur directly, causing a partial or complete transection of the vessel or inducing formation of an intimal flap, arteriovenous fistula, or pseudoaneurysm. Blood vessel injury results from external compression, mural contusion, or thrombosis and is the most prevalent disorder arising from penetrating trauma, occurring in 25-40% of patients.

The internal jugular vein (9%) and carotid artery (7%) are the most common sites of vascular injuries. Injury to the pharynx or the esophagus occurs in 5-15% of cases. The larynx or the trachea is injured in 4-12% of cases.

Major nerve injury occurs in 3-8% of patients sustaining penetrating neck trauma. Spinal cord injury occurs infrequently and almost always results from direct injury rather than secondary osseous instability.

Blunt trauma

Blunt trauma to the neck typically results from motor vehicle accidents, but it also occurs with sports-related injuries (eg, clothesline tackle), strangulation, blows from the fists or feet, and excessive manipulation (ie, any manual operation, such as chiropractic treatment or physical realignment or repositioning of the spine).

In motor vehicle accidents in which the driver is not belted, the driver is in danger of thrusting forward with the head extended, forcing the anterior neck against the steering column. Shoulder harnesses appear to offer some, though incomplete, protection against blunt neck trauma; cerebral vessel and laryngeal injuries secondary to shoulder strap compression have occurred.

Nonpenetrating trauma can injure a blood vessel through a multitude of mechanisms. Direct forces can shear the vasculature. Excessive rotation and/or hyperextension of the cervical spine causes distention and stretching of the arteries and veins to the point of rupture. Intraoral trauma may extend to the blood supply. Basilar skull fractures may disrupt the intrapetrous portion of the carotid artery.

Impact to the exposed anterior aspect of the neck may crush the larynx or the trachea, particularly at the cricoid ring, and compress the esophagus against the posterior spinal column. A sudden increase in intratracheal pressure against a closed glottis (eg, improper wearing of a seat belt), a crush bruise (eg, clothesline tackle), or a rapid acceleration-deceleration action may cause a tracheal injury.

Strangulation may result from hanging (partial or complete suspension of the body from the neck), ligature suffocation, manual choking, and postural asphyxiation (eg, seen in children when the neck is placed over an object and the body weight produces compression). Significant cervical spine and spinal cord damage happens in only those hangings that involve a fall from a distance greater than the body height. Simple asphyxiation is not the major cause of death in hanging injuries. Cervical spinal disruption subsequent to strangulation is almost uniformly fatal.

Frequency

- **In the US:** Neck trauma accounts for 5-10% of all serious traumatic injuries. Approximately 3500 people per year die from neck trauma secondary to hanging, suicide, and accidents.

Mortality/Morbidity

- During the Vietnam era, when mandatory exploration and vascular repair was the standard of care for penetrating neck wounds, the mortality rate for the civilian population was 4-7%. Today, the overall mortality rate has decreased to 2-6%.
- Initially missed cervical injuries secondary to neck trauma result in a mortality rate of greater than

15%. Ten percent of neck wounds lead to respiratory compromise. Loss of the airway patency may occur precipitously, resulting in mortality rates as high as 33%.

- Zone I injuries are associated with the highest morbidity and mortality rates.

Race

Minorities experience a disproportionate share of inner city violence.

Sex

Trauma is more common among males.

Age

Most people who experience trauma are adolescents and young adults.

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Clinical

History

Since many critical organs and structures remain at risk from neck trauma, clinical manifestations may vary greatly. The presence or absence of symptoms can be misleading, serving as a poor predictor of underlying damage. For example, only 10% of patients with blunt vascular damage develop symptoms in the first hour.

- Use all available sources when trying to establish the mechanism of injury. Question the patient, involved bystanders, and prehospital personnel. Clarify events surrounding the traumatic event, establish the amount of time that elapsed since the injury, and confirm the patient's baseline condition. Determine the amount of blood that was lost at the scene and whether the patient lost consciousness. Determine if any evidence of recent drug or alcohol ingestion is present.
- When neck trauma results from a motor vehicle accident, inquire about seat belt use, location of the patient in the car (driver or front or back seat passenger), deployment of an air bag, and magnitude of car damage (eg, intrusion, steering column and windshield intact or broken).
- In the event of a penetrating trauma, try to verify details about the weapon used, such as type and size of knife or type and caliber of gun.
- For patients with injuries due to hanging, try to determine the suspension time (when the patient was last seen), drop height, ligature used, history of alcohol or drug abuse, and history of suicide

attempts.

- Characterize pain (eg, location, nature, intensity, onset, radiation), and document the nature and location of all stated injuries.
- Cardiovascular manifestations range from bleeding to symptoms normally associated with a cerebrovascular accident.
- Symptoms relating to the aerodigestive tract include dyspnea, hoarseness, dysphonia, and dysphagia.
- CNS problems include paresthesias, weakness, plegia, and paresis.

Physical

Airway loss may occur precipitously. Determine airway patency, breathing, and adequacy of circulation. Fully expose the patient and note any disabilities. After completion of the primary survey, methodically examine the neck, searching for clues that indicate damage to vital contents. The sensitivity of the physical examination to identify all significant neck damage remains controversial. Some experts in the field of trauma assert that physical examination alone is sufficient to assess zone II for injury, while others believe that diagnostic testing is mandatory. The literature is not definitive. Most importantly, a single examination is never sufficient, since the onset of signs of injury may be delayed with neck trauma.

Search the neck for a breach of the platysma. Invested by the most superficial fascia, the platysma serves as the harbinger for serious penetrating neck wounds. Any violation of the platysmal muscle should alert the physician to the potential for grave damage to the contents of the neck. If the platysma is violated, judge whether the wound lies anterior (anterior triangle) or posterior (posterior triangle) to the sternocleidomastoid muscle, and determine in what zone the injury is found. Try to specify the direction of the wound tract (eg, toward or away from the midline or clavicle). Half of the cases of penetrating neck trauma in which the platysma is violated have no further injury. If the platysma clearly is not violated by a penetrating injury, the patient can be safely cleared of a significant underlying injury.

Consider an arterial injury of the neck in patients manifesting gross bleeding; a hematoma; asymmetry of arterial pulses; a new bruit on auscultation; neurological deficits, especially lateralizing cerebral findings; or hypotension. Do not unnecessarily probe or manipulate the wound or perform any action that may cause the patient to gag, choke, or cough. Any of these reactions may dislodge a clot and provoke a life-threatening hemorrhage.

Hard signs of an arterial injury include a large expanding hematoma, severe active or pulsatile bleeding, shock unresponsive to fluids, signs of cerebral infarction, presence of a bruit or thrill, and diminished distal pulses. Virtually all patients with hard signs of an arterial injury require operative repair.

Soft signs, such as a nonexpanding hematoma and paresthesias, do not improve the predictive value of an arterial injury more than indicating the proximity of the wound to a major vessel. The presence of a pulse does not exclude a vascular injury, nor is absence of a pulse diagnostic of vascular damage. Clinical

findings are lacking initially in almost one third of patients with an arterial injury of the neck. Nearly one third of carotid artery injuries are associated with a central neurological deficit.

Perforation of the pharynx or the esophagus following blunt neck trauma rarely occurs. Initially, the patient may have no complaints, and the physical examination fails to reveal injury. Since the wall of the esophagus is fragile, iatrogenic injury can follow endoscopy, passage of a nasogastric tube, or inadvertent esophageal intubation. Esophageal perforation is the most serious and rapidly fatal trauma-induced perforation of the gastrointestinal tract.

- Signs of spinal cord or brachial plexus injury
 - Brachial plexus injuries sustained from blunt trauma tend to involve the upper nerve roots (C5 to C7), diminishing the capacity of the upper arm while sparing strength and sensation of the lower arm. A radical avulsion of the brachial plexus results in a flaccid, numb extremity.
 - Quadriplegia occurring with complete transection of the spinal cord manifests as an absence of all motor, sensory, and reflex function below the level of injury. Bilateral neurological findings imply a spinal cord injury until proven differently.
 - Pathological reflexes, such as the Babinski reflex (extension of big toe) and Hoffmann sign (overactive muscle-stretch reflex), may be present.
 - Brown-Séquard syndrome results from hemisection of the spinal cord, causing ipsilateral motor paralysis with contralateral sensory deficits.
 - Priapism and loss of the bulbocavernosus reflex may occur, and rectal tone may be poor.
 - Urinary retention, fecal incontinence, and paralytic ileus can occur from spinal cord damage.
 - Horner syndrome (ipsilateral miosis, enophthalmos, anhidrosis) results from disturbances of the stellate ganglion.
 - Neurogenic shock is a diagnosis of exclusion and is characterized by persistent bradycardia despite hypotension.
 - Hypoxia and hypoventilation can follow disruption of phrenic innervation to the diaphragm.
- Signs of penetrating injuries of the heart, aorta, and great vessels
 - Hemorrhage, usually associated with large wounds (eg, GSWs)
 - Massive hemothorax
 - Hypotension
 - Tamponade (if intrapericardial portion of aorta is injured)
 - Weak or absent carotid or brachial pulse
 - Paradoxical pulse (decrease in systolic BP with inspiration)
 - Bruit
 - Cervical or supraclavicular hematoma
 - Bleeding from the entrance wound
 - Upper extremity ischemia
 - Coma
 - Hemiparesis

- Respiratory distress secondary to tracheal compression
- Signs of carotid artery injury
 - Decreased level of consciousness
 - Contralateral hemiparesis
 - Hemorrhage
 - Hematoma
 - Dyspnea secondary to compression of the trachea
 - Thrill
 - Bruit
 - Pulse deficit
- Signs of cranial nerve injuries
 - Facial nerve (cranial nerve VII): Drooping of the corner of the mouth
 - Glossopharyngeal nerve (cranial nerve IX): Dysphagia (altered gag reflex)
 - Vagus nerve (cranial nerve X, recurrent laryngeal): Hoarseness (weak voice)
 - Spinal accessory nerve (cranial nerve XI): Inability to shrug a shoulder and to laterally rotate the chin to the opposite shoulder
 - Hypoglossal nerve (cranial nerve XII): Deviation of the tongue with protrusion
- Signs of esophagus and pharynx injury
 - Dysphagia
 - Bloody saliva
 - Sucking neck wound
 - Bloody nasogastric aspirate
 - Pain and tenderness in the neck
 - Resistance of neck with passive motion testing
 - Crepitus
 - Bleeding from the mouth or nasogastric tube
 - Ligation marks: Examine the patient who has been strangulated. Note location and depth of marks, petechial hemorrhages of the skin and subconjunctival tissue (Tardieu spots), noisy or impaired respiration or phonation (eg, stridor, hoarseness, poor air movement), and palpable crepitus or tenderness over the larynx and trachea. Check for neurological deficits.

Causes

Neck trauma may be caused by penetrating or blunt trauma.

- Penetrating trauma
 - GSWs
 - Stab wounds

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Differentials

Spinal Cord Injuries

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Workup

Lab Studies

- For any patient thought to have a neck injury, order the standard trauma blood studies (CBC, electrolytes, other warranted blood chemistry levels, blood type and cross-matching).
- Generally, a CBC and blood typing suffice in a previously healthy individual, but patients with comorbid disease or those in shock may require additional studies, including a determination of coagulation profiles.
- Order alcohol and toxicology screens, when indicated.

Imaging Studies

- Recognize the delay inherent with any imaging study. Do not delay transport to the operating room when the patient's condition warrants emergent surgery.
- Determining the specific study and order of testing depends on institutional preferences, mechanism of injury, and clinical scenario.
- Detection of pharyngoesophageal injuries poses many problems, and a high index of suspicion is required because failure to diagnose these injuries can lead to significant morbidity and mortality.
- Cervical x-rays
 - Unless indicated otherwise, most patients sustaining significant injury to the neck require plain-film radiography.
 - In general, order a 3-view series of the cervical spine.
 - Review the cervical radiographs for emphysema, fractures, displacement of the trachea, and presence of a foreign body (eg, missile fragments).
- Supplementary tests
 - Order supplementary tests in the stable patient if specific system injuries are suggested by the history, physical, or prior ancillary studies. Additional imaging studies include the following: CT scans, MRIs, color flow Doppler studies, contrast studies of the esophagus, interventional angiograms, and endoscopic images.
 - CT scans prove most useful when bony or soft tissue damage is a consideration. Requesting a CT scan of the neck when a laryngeal fracture is suspected is especially

important because clinically subtle injuries of the larynx often escape detection but become readily identifiable on CT scan.

- Consider an emergent MRI and/or magnetic resonance angiogram for evaluation of the patient exhibiting neurological impairment with minimal or absent abnormalities on plain radiographs of the cervical spine.
- Some institutions now substitute color flow Doppler ultrasonography or use it as a screening test in low-risk patients or those thought to have a carotid injury. However, its sensitivity remains variable and its use, controversial.
- Although a normal Gastrografin study occasionally proves useful in the evaluation of the cervical esophagus, it does not rule out a pharyngoesophageal leak. Deciding which contrast agent to use when studying the esophagus remains a subject of dispute among the experts. Advocates for Gastrografin use note that it is less likely than barium to cause an inflammatory response if extravasation through a breach occurs. However, barium induces less inflammation in the lungs; therefore, it poses less of a risk in the patient predisposed to aspiration.

Procedures

- Endoscopy
 - Laryngoscopy, bronchoscopy, pharyngoscopy, and esophagoscopy may be useful in the assessment of the aerodigestive tract. Rigid endoscopes are superior to flexible scopes.
 - Before inserting any scope, confirm that the airway is patent, intact, and protected (usually ensured by placement of an endotracheal tube). Ecchymosis of the posterior or lateral pharyngeal wall implies concealed neck damage.
 - Endoscopy, especially indirect laryngoscopy, often becomes problematical in the apprehensive trauma patient, and it may be best to defer examination until the airway is protected and the patient is anesthetized.

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Treatment

Prehospital Care

In most urban settings, immediate transport of the patient with neck trauma to the closest level I trauma setting is most appropriate because state-of-the-art care frequently necessitates a multidisciplinary approach.

- Emergency medical personnel should restrict intubation attempts when anticipating a prolonged

transport time, or when the patient appears apneic, pulseless, or moribund and respiratory arrest is imminent. Providing supplemental oxygen and clearing the airway of all secretions and foreign bodies, including unfastened dentures and loose teeth, frequently prove sufficient, practical, and helpful for the conscious patient.

- Injudicious attempts to vigorously insert an endotracheal tube may worsen the patient's state by running the risk of utterly marring the airway. Conversely (although less likely), ventilating the patient with a positive pressure bag-valve-mask device could exacerbate underlying subcutaneous emphysema, conceivably distorting the airway anatomy and impairing breathing and circulation.
- In general, use impregnated gauze to cover sucking neck wounds or lacerations exuding bubbling air.
- Patients sustaining significant blunt trauma require cervical spine precautions including cervical spine immobilization and supine placement of the patient on a backboard. Defer removal of helmets or other headgear until neck stabilization has been ensured.
- Bleeding from the neck is best controlled with direct pressure.
 - Do not extract impaled objects in the field.
 - Establish intravenous access en route to the hospital; preferably place the catheter in the extremity opposite the side of the injury in case disruption of the ipsilateral venous circulation has occurred.

Emergency Department Care

Initial evaluation and stabilization includes securing the airway, controlling bleeding, providing cervical spine precautions, and identifying life-threatening conditions. Most blunt traumatic neck injuries can be managed nonoperatively. Surgical assessment of penetrating neck wounds usually requires a greater resolve for operative intervention, although prior axioms decreeing surgery as the only option are no longer as absolute. However, when a cut violates the platysma, it is sensible and prudent to engage a qualified surgeon, or transfer a stable patient to a trauma center where such care is available.

- Airway
 - ED care of the patient with neck trauma commences with assessment and stabilization of the ABCs, starting with the airway first. Unfortunately, the same conditions compelling active airway management also intensify the obstacles to achieving successful intubation. Nonetheless, a wait-and-see attitude merely invites disaster.
 - Consequently, a preplanned strategy based on the expertise of the available staff, equipment at hand, the patient's clinical condition, and the determined necessity for further testing should be planned before this scenario occurs. An entrenched partnership must exist among all potentially involved departments, especially emergency medicine, surgery, and anesthesiology.
 - Intubating a patient with penetrating neck trauma may incite gagging or coughing, potentially dislodging a clot and setting off massive bleeding from a previously injured blood vessel. Additionally, existent bleeding and edema rapidly distort the surrounding anatomy, making oral intubation difficult, if not impossible. Nevertheless, assessment of the airway takes priority over all other actions, including those procedures that risk

exacerbating hemorrhage. Early preparation by the physician treating the patient is crucial. This includes ensuring ease of access to an acceptable suction apparatus and having multiple-sized endotracheal tubes as well as any tools and supplies necessary to perform the surgical airway procedure close at hand.

- Before intubation, clear the mouth of foreign debris with the fingers or manual suction. Remedy partial airway occlusion originating from the tongue by performing a modified jaw thrust. (Never do a head-tilt chin-lift maneuver in a patient with a suspected cervical spine injury.)
- Perform emergent intubation in patients displaying signs of acute or impending respiratory distress, such as perceptible noisy breathing; an inability to suitably handle blood, vomitus, or other body secretions; and obvious distortion of any neck landmarks, particularly tracheal deviation or existence of massive subcutaneous air. Rapid sequence intubation facilitates securing the airway, but this technique may be ill-advised if assessment indicates likely difficulty in obtaining airway control and/or an inability to establish adequate ventilation if the intubation cannot be achieved. Choice of technique depends on the expertise of the attending staff and the capability to perform a surgical airway procedure. If time permits, procedures such as oral awake intubation, with a carefully sedated and orally anesthetized patient, may be preferred.
- An awareness of potential laryngeal damage is imperative prior to intubation, even when the airway must be emergently secured. A neck hematoma can obscure landmarks, in addition to causing the danger of precipitating life-threatening exsanguination. Overwhelming suspicion for laryngeal injury directs execution of a surgical airway procedure to avoid injudicious endeavors at oral intubation that could sever a tenuously attached trachea or larynx, conceivably causing a catastrophe consequent to complete loss of the airway if the larynx detaches and dislodges into the chest.
- Several large case series (eg, that by Shatney et al) demonstrate the safety of oral intubation with cervical in-line stabilization, provided direct laryngoscopy and intubation are performed in a gentle, atraumatic manner and explicit cervical spine immobilization is maintained. This is the preferred approach for the accomplished intubator in the patient with blunt trauma with suspected cervical spine injury.
- Alternate techniques for securing the airway include fiberoptic intubation, percutaneous transtracheal intubation, and wire-guided retrograde intubation.
 - Fiberoptic intubation is a sensible course of action, especially for patients thought to have sustained a cervical spine injury or who exhibit gross distortion of the airway. Limitations include physician inexperience, lack of necessary equipment, and copious bleeding or secretions.
 - Percutaneous transtracheal intubation, also referred to as translaryngeal ventilation, is a quick and relatively simple technique in which a needle is inserted through the cricothyroid membrane and attached via a Y connector to an oxygen supply of at least 50 psi. This procedure is contraindicated when transection of the trachea or damage to the cricoid cartilage or the larynx is strongly suspected. Barotrauma may occur with percutaneous ventilation.
 - Retrograde tracheal intubation is an invasive procedure that may be suitable when

excessive amounts of blood or secretions preclude fiberoptic intubation or when neck movement must be restricted.

- **Circulation**

- Control bleeding that originates from neck trauma with direct pressure or digitally. Do not blindly clamp a transected vessel, since inadvertent injury to adjacent structures or extension of blood vessel damage may occur. Never probe, cannulate, or locally explore these wounds in the ED because these actions may cause an air embolus or dislodge a clot and provoke bleeding. Do not remove objects protruding from the neck in the ED.
- Concurrent with checking bleeding, establish intravenous access with at least 2 large-bore catheters (14 or 16 gauge). If there is a possibility of injury to the brachiocephalic or subclavian vein, place 1 intravenous access site in a lower extremity site and another access site in the upper extremity on the uninjured side.
- Placing the patient in a mild Trendelenburg position to decrease the risk of air embolization may be advantageous.
- In selected cases, bleeding that cannot be controlled or reached with direct pressure may benefit from balloon tamponade. Insert a Foley catheter into the wound. Direct it toward the site of bleeding, and inflate the balloon until bleeding resolves or moderate resistance is noted. As an example, for zone I injuries, slide in a Foley catheter toward the pleural cavity, and then inflate the balloon with sterile saline and retract it, striving to compress the injured subclavian vessel against the first rib or clavicle.
- On rare occasions, such as with wounds in the pharynx, applying direct pressure to wounds may be impractical. These wounds may necessitate a cricothyroidotomy with subsequent packing of the pharynx as a temporary strategy.

- **Exposure**

- To view the anterior part of a neck that is concealed by a cervical collar, appoint an assistant to maintain the neck in a neutral position, and then remove the anterior aspect of the collar and proceed with the evaluation.
- Expose and observe the patient's entire body to avoid overlooking other potentially lethal injuries.

Consultations

Consult an experienced trauma surgeon emergently once platysmal violation is confirmed. Additional consultants should be prioritized with guidance from the trauma surgeon who will oversee the patient's care.

Ordinarily, urgent surgical exploration of a penetrating wound to the neck is indicated for the following:

- Continued blood loss, expanding hematoma, hypovolemic shock, and/or pulse deficit
- Airway obstruction, impending airway obstruction, open trachea, and/or air bubbling from the wound site
- Neurologic deficit

- Blood in the aerodigestive tract, hemoptysis, and/or hematemesis
- New-onset bruit

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Medication

Infection subsequent to penetrating wounds of the neck is a major cause of death and disability. Administering prophylactic antibiotics, while not decisively validated by scientific studies, should be a consideration. Recommended medications vary from penicillin to those with broad-spectrum coverage. Factors to consider include the physical nature of the injury (eg, simple laceration vs blunt trauma with tearing-type injuries). If prophylactic antibiotics are to be effective, attempt to obtain adequate tissue levels immediately, preferably within 4 h of injury.

Other therapeutic agents to consider are the corticosteroids. Massive doses of steroids are believed to have possible benefit in improving neurological function in selected subsets of patients. In the second phase of a benchmark study (Bracken et al), patients who had sustained blunt spinal injury within a 12-h time frame were given a 30 mg/kg IV bolus of corticosteroids followed by 5.4 mg/kg/h for 23 h. Overall, patients who appropriately received steroids within 8 h revealed slight improvement in function in motor and sensory function at 6 mo. Other experimental agents include naloxone, dimethylsulfoxide, and growth factors. Spinal cooling also has been proposed.

Drugs facilitating endotracheal intubation, especially those used for rapid sequence intubation, should be readily available.

Antibiotics

Therapy must cover all likely pathogens in the context of the clinical setting.

| | |
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| Drug Name | Cefotetan (Cefotan)- Second-generation cephalosporin indicated for infections caused by susceptible gram-positive cocci and gram-negative rods. Dosage and route of administration depends on condition of patient, severity of infection, and susceptibility of causative organism. |
| Adult Dose | 1-2 g IV/IM q12h |
| Pediatric Dose | 20-40 mg/kg/dose IV/IM q12h |
| Contraindications | Documented hypersensitivity |

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| Interactions | Consumption of alcohol within 72 h of cefotetan may produce disulfiramlike reactions; may increase hypoprothrombinemic effects of anticoagulants; coadministration with potent diuretics (eg, loop diuretics) or aminoglycosides may increase nephrotoxicity; monitor renal function |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Reduce dosage by one half if CrCl <10-30 mL/min and by one fourth if CrCl <10 mL/min; bacterial or fungal overgrowth of nonsusceptible organisms may occur with prolonged or repeated therapy |

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| Drug Name | Gentamicin (Garamycin)- Aminoglycoside antibiotic for gram-negative coverage. Used in combination with both an agent against gram-positive organisms and one that covers anaerobes. Not the DOC. Consider if penicillins or other less toxic drugs are contraindicated, when clinically indicated, and in mixed infections caused by susceptible staphylococci and gram-negative organisms. |
| Adult Dose | 3-5 mg/kg/d IV/IM q6-8h Follow each regimen by at least a trough level drawn with the third or fourth dose (0.5 h before dosing); may draw a peak level 0.5 h after 30-min infusion |
| Pediatric Dose | <5 years: 2.5 mg/kg/dose IV/IM q8h >5 years: 1.5-2.5 mg/kg/dose IV/IM q8h or 6-7.5 mg/kg/d divided q8h; not to exceed 300 mg/d; monitor as in adults |
| Contraindications | Documented hypersensitivity; renal insufficiency not dependent on dialysis |
| Interactions | Coadministration with other aminoglycosides, cephalosporins, penicillins, and amphotericin B may increase nephrotoxicity; aminoglycosides enhance effects of neuromuscular blocking agents, prolonged respiratory depression may occur; coadministration with loop diuretics may increase auditory toxicity; possible irreversible hearing loss of varying degrees may occur (monitor regularly) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Narrow therapeutic index (not intended for long-term therapy); caution in renal failure (patient not undergoing dialysis), myasthenia gravis, hypocalcemia, and conditions that depress neuromuscular transmission; adjust dose in renal impairment |

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| Drug Name | Ampicillin (Omnipen)- Bactericidal activity against susceptible organisms. Alternative to amoxicillin when patient unable to take medication orally. |
| Adult Dose | 1-2 g IV/IM q4-6h |
| Pediatric Dose | 50 mg/kg IV/IM q4-6h |
| Contraindications | Documented hypersensitivity |

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| Interactions | Probenecid and disulfiram elevate ampicillin levels; allopurinol decreases ampicillin effects and has additive effects on ampicillin rash; may decrease effects of oral contraceptives |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal failure; evaluate rash and differentiate from hypersensitivity reaction |

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| Drug Name | Clindamycin (Cleocin)- Lincosamide for treatment of serious skin and soft tissue staphylococcal infections. Also effective against aerobic and anaerobic streptococci (except enterococci). Inhibits bacterial growth, possibly by blocking dissociation of peptidyl t-RNA from ribosomes causing RNA-dependent protein synthesis to arrest. |
| Adult Dose | 600-1200 mg/d IV/IM divided q6-8h depending on degree of the infection |
| Pediatric Dose | 20-40 mg/kg/d IV/IM divided tid/qid; may increase dose to 16-20 mg/kg/d divided tid/qid for severe infections |
| Contraindications | Documented hypersensitivity; regional enteritis, ulcerative colitis, hepatic impairment, antibiotic-associated colitis |
| Interactions | Increases duration of neuromuscular blockade, induced by tubocurarine and pancuronium; erythromycin may antagonize effects of clindamycin; antidiarrheals may delay absorption |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in severe hepatic dysfunction; no adjustment necessary in renal insufficiency; associated with severe and possibly fatal colitis |

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| Drug Name | Ceftriaxone (Rocephin)- Third-generation cephalosporin with broad-spectrum, gram-negative activity; lower efficacy against gram-positive organisms; higher efficacy against resistant organisms. Arrests bacterial growth by binding to one or more penicillin binding proteins. |
| Adult Dose | 1-2 g IV qd, or divided bid; not to exceed 4 g/d |
| Pediatric Dose | >7 days: 25-50 mg/kg/d IV/IM; not to exceed 125 mg/d Infants and children: 50-75 mg/kg/d IV/IM divided q12h; not to exceed 2 g/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid may increase ceftriaxone levels; coadministration with ethacrynic acid, furosemide, and aminoglycosides may increase nephrotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal impairment; caution in breastfeeding women and patients with allergy to penicillin |

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Follow-up

Further Inpatient Care

- Standing protocols dictating the treatment of patients with neck trauma must be in place. Such guidelines should indicate which patients require emergent surgery, transfer, or further workup.
- A major disadvantage of exploring all penetrating neck injuries with platysma violation is a nontherapeutic exploration in approximately 50% of cases. This results in unnecessary costs and nonessential invasive procedures. Recent studies (eg, those by Demetriades et al and Ngakane et al) suggest that the majority of patients with penetrating neck trauma can be treated nonoperatively. No definitive recommendation exists, and treatment protocols should be based on a multidisciplinary agreement within the institution. However, because of the prospect of occult injuries with zone I and III wounds, a relatively aggressive workup is warranted.
- Decisions regarding whether to ligate or repair arterial injuries rely on the presence or absence of a major neurological deficit (coma and/or paralysis); some surgeons prefer to avoid the danger of reperfusion injury of the brain.
- Embolization may halt bleeding from a damaged vessel in the neck.
 - If the patient's vital signs are not stabilized, death in the radiological suite is a real risk.
 - Temporary occlusion of the blood vessel may be achieved by insertion of a gelatin sponge (Gelfoam).

Further Outpatient Care

- Observe patients with all but the most trivial of neck wounds for delayed onset of symptoms.
 - Platysma violation usually justifies admission for 24 hours of observation to avoid missing occult injuries, particularly vascular and esophageal wounds.
 - Decisions regarding the need to admit a patient with blunt neck trauma are based on the presence or absence of signs and symptoms, as well as the patient's underlying physiological status and factors such as the availability of care, extent of care warranted, and willingness of responsible parties to participate.
- Care for lacerations superficial to the platysma in an otherwise asymptomatic patient as one would care for cuts elsewhere in the body. Clean lacerations may be sutured as late as 12-18 hours after injury, since, ordinarily, the neck is exceptionally well perfused.

in/Out Patient Meds

- Outpatient treatment for minor neck injuries may include use of various analgesics, such as

acetaminophen or nonsteroidal anti-inflammatory drugs, for mild neck strains or sprains.

- Check the tetanus status on all patients sustaining penetrating neck wounds.

Transfer

- All consequential neck injuries are best managed in a level I trauma center.
- Arrangements to transfer such patients are made concurrently with stabilization of the airway and hemorrhage control.

Deterrence/Prevention

- Establish injury prevention programs.
- Support efforts to limit violence in society.
- Provide instruction on the proper use of seat belts.

Complications

- Airway obstruction may result from evolving tracheal and/or laryngeal edema or stenosis. Vocal cord paralysis and voice change also may follow laryngeal trauma.
- Swallowing dysfunctions may affect patients with neck trauma.
 - Aspiration of material (eg, blood, vomitus) is always a possibility.
 - Patients who survive the initial strangulation injury may succumb to pulmonary edema or bronchopneumonia.
- Soft tissue necrotizing infections caused by mixed bacterial organisms may originate from contamination of the neck or extravasation from oral wounds. Sepsis, mediastinitis, and cervical osteomyelitis may occur.
- Fistulas include tract formation between the trachea and the esophagus (tracheoesophageal fistula), the trachea and the brachiocephalic artery (potential for a catastrophic hemorrhage within the tracheobronchial tree), and the esophagus and the skin (esophagocutaneous fistula).
- Complications associated with arteriography range from arterial wall injuries (eg, intimal flaps, thrombosis, and severe vascular spasm) to neurological impairment, anaphylactic reaction, and groin hematoma (may lead to femoral artery occlusion).
- Air embolism is an infrequent seldom-mentioned complication that arises from tears in the major neck veins. Penetrating neck trauma may precipitate an air embolism. Depending on where the embolus settles, positioning the patient may lessen the chance of embolus propagation. Suspect this entity in patients developing unexpected hypotension and/or arrhythmia, especially in the setting of an increase in central venous pressure.
- Lead intoxication is an unusual problem that occurs subsequent to a bullet remaining lodged in the neck, usually in a joint space. Warning signs and symptoms include abdominal pain, nephropathy, and unexplained anemia.

Prognosis

- As a general rule, zone I injuries have the worst prognosis in regard to imminent morbidity and mortality.
- Zone II injuries are the most prevalent penetrating neck wounds. Because of their accessibility, injuries in zone II have the best prognosis.
- Zone III presents unique therapeutic and diagnostic challenges because of its secluded nature of the critical structures spanning this locale.
- Complete disruption of the spinal cord above C4 is frequently fatal. Preservation of any neurological function, including rectal tone, following a spinal cord injury improves the outlook.
- Vascular injuries arising from blunt trauma are associated with a poor outcome.
- The prognosis is poor when severe neurological deficits (eg, hemiparesis, coma) occur subsequent to carotid artery damage. Early revascularization may improve the outlook.
- Identification of pharyngeal or esophageal injuries is paramount because delayed diagnosis leads to significant morbidity.
- Strangulation patients presenting in cardiac arrest have a dismal prognosis, as do strangulation patients who are successfully resuscitated but who completely lack neurological function.
- If the Glasgow score is greater than 3, the chances are good that the patient with a choking or strangulation injury will eventually be discharged neurologically intact.

Patient Education

- Soft tissue cervical strains and sprains, commonly known as a whiplash injuries, initially may be associated with minimal pain.
 - Subsequent edema and medicolegal considerations may cause worsening of symptoms over the ensuing 24-48 hours.
 - Instruct the patient that complete resolution of symptoms may require 2-12 weeks. Stress the importance of follow-up care.
 - Recommendations regarding the use of alternate modalities (eg, cold, heat, manipulation, massage) depend on what works best for the patient. Discourage the prolonged use of soft collars.

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Miscellaneous

Medical/Legal Pitfalls

- Achievement of a protected airway takes priority over all other actions. When intubating the

patient, do not force the endotracheal tube into the trachea because this can cause complete severance of a partially transected airway or displace the trachea into the mediastinum.

- Coexisting head injury, coma, spinal cord damage, or drug and/or alcohol impairment may distort findings in the neck trauma patient.
- If the condition of a patient with a penetrating neck wound continues to deteriorate despite resuscitation, consider an intrathoracic injury, such as massive hemothorax or tension pneumothorax.
- If an injury to the jugular vein is suspected, position the patient's head lower than the heart to reduce the risk of air embolization.
- Presence of a neurologic deficit, especially one contralateral to the side of injury, should prompt speculation of an injury to the carotid or vertebral artery, in addition to nerve injury.
- Do not remove an impaled object in the ED because the object may be causing tamponade to stop the hemorrhage.
- Never accept suboptimal imaging studies. Ensure that the physician interpreting the image is sufficiently qualified.
- Physical findings in a neck wound tend to be deceptive. An apparently insignificant hematoma may conceal a larger, deeply confined subfascial accumulation of blood. Do not probe neck wounds that penetrate the platysma in the ED; this may dislodge a clot, causing a massive hemothorax or creating an iatrogenic pneumothorax.
- Do not send a patient with a compromised airway to a darkened, nonequipped angiographic suite. Consider prophylactic intubation in a controlled setting.
- Do not blindly clamp blood vessels because other vital structures (eg, nerves) travel alongside and may be irreparably damaged. Direct pressure is usually sufficient.

Special Concerns

- Pregnancy
 - Consider the physiological changes that occur with pregnancy in any gravid patient who sustains trauma. Specifically, blood pressure decreases 10-15 mm Hg systolic and 5-10 mm Hg diastolic, the heart rate increases 10-15 beats per minute, and physiological anemia accompanies pregnancy. Since fetal viability relies completely on satisfactory maternal cardiopulmonary perfusion (the fetus is termed the "yellow canary" for the mother), focus all efforts on resuscitating the mother. Pay particular attention to oxygenation.
 - Maternal cardiac output depends greatly on venous return, which is influenced by the position of the mother during late pregnancy. Supine positioning allows the gravid uterus to compress the inferior vena cava, and cardiac output may be increased by 30% with the patient in the lateral recumbent position. If cervical spine injury remains a concern, the uterus may be manually displaced.
 - After restoration of maternal oxygenation and circulation, evaluate the mother for secondary injuries and rapidly assess the fetus. If the fetal parameters suggest potential viability (eg, >24 wk), obtain an obstetric consultation.
 - Try to limit ionizing radiation exposure to the patient during early pregnancy, and use ultrasound for evaluation, when suitable.

- Geriatric
 - Aging tends to compromise physiological reserves, making it difficult to assess commonly used parameters of hemodynamic status.
 - Degenerative and arthritic changes hinder radiological evaluation.
 - Elderly patients sustaining blunt trauma to the neck are more predisposed to central cord compression, especially after hyperextension injuries in which the spinal cord becomes compressed between osteophytes anteriorly and the buckling ligamentum flavum posteriorly.
 - Elderly patients sustaining carotid artery injury without coexisting neurological deficits may require an extracranial-intracranial bypass.
-

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Osgood-Schlatter Disease

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Synonyms, Key Words, and Related Terms

OS

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Introduction

Background

Osgood-Schlatter (OS) disease is one of the most common causes of knee pain in the adolescent. Consisting of pain and edema of the tibial tubercle (and hence this is an extra-articular disease), OS disease is generally a benign, self-limited knee condition associated with traction apophysitis in adolescent boys and girls.

Paget first described the clinical syndrome in 1891. In 1903, Osgood and Schlatter published separate papers on the subject. Because of a lack of a precise definition, it is difficult to differentiate OS disease from avulsion fractures of the tibial tubercle.

Pathophysiology

Histologic studies suggest a traumatic etiology for OS disease. Bone growth is faster than soft tissue growth, which may result in muscle tendon tightness across the joint and loss of flexibility.

During periods of rapid growth, stress from contraction of the quadriceps is transmitted through the patellar tendon onto a small portion of the partially developed tibial tuberosity. This may result in a partial avulsion fracture through the ossification center. Eventually, secondary heterotopic bone formation occurs in the tendon near its insertion, producing a visible lump. Approximately 25% of patients have bilateral lesions.

Frequency

- **In the US:** Frequency is not known, but condition is uncommon.
- **Internationally:** One Finnish study found that OS disease affected 13% of athletes.

Mortality/Morbidity

OS disease is typically benign and self-limited.

Sex

OS disease occurs more frequently in boys, probably because a greater number of boys participate in sports.

Age

- OS disease usually is seen in the adolescent years after undergoing a rapid growth spurt the previous year.
- Girls who are affected are typically aged 10-11 years.
- Boys who are affected are typically aged 13-14 years.

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Clinical

History

- Pain is the most common presenting complaint.
 - The pain may be reproduced by extending the knee against resistance, stressing the quadriceps, or squatting with the knee in full flexion.
 - Running, jumping, kneeling, squatting, and ascending/descending stairs exacerbate the pain.
 - Relief of symptoms occurs with rest or restriction of activities.
 - Pain usually has been present intermittently for several months before the patient sees the physician.
 - The pain is bilateral in 25% of cases.
 - Approximately 50% of patients give a history of precipitating trauma.

Physical

- A visible soft tissue edema is present over the proximal tibial tuberosity.
- Patient may have tenderness to palpation over the proximal tibial tuberosity at the site of patellar insertion.
- A firm mass may be palpable.
- Pain is reproduced by extension against forced resistance.
- Knee joint examination is normal; OS disease is an extra-articular disease.
- Absence of effusion or condylar tenderness is typical.
- Erythema of the tibial tuberosity may be present.
- Some patients may have quadriceps atrophy.

Causes

- Etiology is controversial, but condition clearly is exacerbated by exercise.
- Approximately 50% of patients relate a history of precipitating trauma.
- Chronic microtrauma to the tibial tuberosity secondary to overuse of the quadriceps muscle is a leading theory of etiology.
- Histologic studies support a traumatic etiology.
- Risk factors
 - Age between 11-18 years
 - Male sex
 - Rapid skeletal growth
 - Repetitive jumping sports

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Differentials

Fractures, Tibia and Fibula

Other Problems to be Considered

- Quadriceps tendon avulsion
- Patellofemoral stress syndrome
- Pes anserinus bursitis
- Chondromalacia patellae
- Osteomyelitis of the proximal tibia
- Patellar tendonitis
- Sinding-Larsen-Johansson syndrome

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Workup

Lab Studies

- Laboratory evaluation is not indicated unless other diagnoses are being entertained.

Imaging Studies

- Knee radiography (AP and lateral)
 - The OS lesion is best seen on the lateral view with the knee in slight internal rotation.
 - Not all patients with OS disease need radiographs since the diagnosis is primarily clinical. Plain films are helpful to rule out other etiologies, such as neoplasm and infection.
 - Superficial ossicle in the patellar tendon
 - Irregular ossification of the proximal tibial tuberosity
 - Calcification within the patellar tendon
 - Thickening of the patellar tendon
 - Soft tissue edema proximal to the tibial tuberosity

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Treatment

Emergency Department Care

- Once the diagnosis is made and other pathologies are ruled out, the patient may be discharged with an orthopedic referral. Therapy is conservative.
- Initial treatment includes the application of ice for 20 minutes every 2-4 hours.
- Analgesics and nonsteroidal anti-inflammatory medications (NSAIDs) may be given for pain relief and reduction of local inflammation.
- Inform the patient to avoid pain-producing activities (eg, sports that involve excess amounts of jumping).
- Use of a knee immobilizer for a few days may improve compliance, especially in more severe cases. Pads or braces also can be used for support.
- Once the acute symptoms have abated, quadriceps-stretching exercises, including hip extension for a complete stretch of the extensor mechanism, should be performed to reduce tension on the tibial tubercle. Stretching exercises for the hamstrings, which are commonly tight, also should be performed.

Consultations

Patients should have follow-up care with an orthopedist.

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Medication

The only medications that need to be prescribed are NSAIDs for pain relief and reduction of local inflammation (any NSAID may be used). However, one author concluded that anti-inflammatory drugs are not particularly beneficial in the management of OS disease.

Nonsteroidal Anti-Inflammatory Agents

These agents are commonly used for relief of mild to moderate pain. Although effects of NSAIDs in treatment of pain tend to be patient specific, ibuprofen is usually the DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

| | |
|------------|--|
| Drug Name | Ibuprofen (Ibuprin, Advil, and Motrin)- DOC for patients with mild to moderate pain. Inhibits inflammatory reactions and pain by decreasing prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h while symptoms persist; not to exceed 3.2 g/d |

| | |
|-------------------|---|
| Pediatric Dose | 6 months to 12 years: 20-40 mg/kg/d PO divided tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease, recent GI bleeding or perforation, renal insufficiency, or high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related side effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|---|
| Drug Name | Ketoprofen (Oruvail, Orudis, and Actron)- For relief of mild to moderate pain and inflammation. Small doses initially are indicated in small and elderly patients and in those with renal or liver disease. Doses >75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe patient for response. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 12 years: 0.1-1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related side effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

| | |
|-----------|---|
| Drug Name | Flurbiprofen (Ansaid)- May inhibit cyclo-oxygenase enzyme, which inhibits prostaglandin biosynthesis. These effects may result in analgesic, antipyretic, and anti-inflammatory activities. |
|-----------|---|

| | |
|-------------------|--|
| Adult Dose | 200-300 mg/d PO divided bid/qid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related side effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently; nephrotoxicity of cyclosporine may be increased |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

| | |
|-------------------|---|
| Drug Name | Naproxen (Anaprox, Naprelan, and Naprosyn)- For relief of mild to moderate pain; inhibits inflammatory reactions and pain by decreasing activity of cyclo-oxygenase, which results in a decrease of prostaglandin synthesis. |
| Adult Dose | 500 mg PO, followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related side effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |

Precautions

Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug

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Follow-up

Further Inpatient Care

- Surgical treatment rarely is indicated and generally is reserved for those patients with recurrent disabling pain unresponsive to conservative therapy.
 - Once the other physes have closed, surgery may be necessary for non-united ossicles.
 - Simple excision of the mobile ossicle may be necessary.
 - A tuberosity thinning procedure followed by ossicle excision may be performed.

Further Outpatient Care

- Conservative therapy is usually all that is needed.
 - Avoid physical activities that require frequent deep knee bending for 2-4 months.
 - Therapeutic exercises to strengthen the quadriceps and the hamstrings are prescribed.
- Analgesics
 - Control pain and inflammation
 - Corticosteroid injections should be avoided because of the risk of degenerative changes and tissue atrophy.

Deterrence/Prevention

- Avoid sports that involve heavy quadriceps loading.
- Increase hamstring and quadriceps flexibility.

Complications

- Nonunion of the tibial tubercle
- Upriding of the patella
- Patellar tendon avulsion

- Genu recurvatum
- Patellofemoral degenerative arthritis
- Patellar subluxation
- Patella alta
- Chondromalacia

Prognosis

- The prognosis is excellent. Symptoms usually resolve spontaneously within 1 year.
- Discomfort may persist for 2-3 years until the tibial growth plate closes.
- Persisting complaints may be from residual enlargement of the tuberosity or from ossicle formation in the patellar tendon.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to consider other diagnoses, such as underlying fracture or tumor
 - Failure to advise of activity restrictions
-

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Peripheral Vascular Injuries

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Introduction

Background

Peripheral vascular injuries may result from penetrating or blunt trauma to the extremities. If not recognized and treated rapidly, injuries to major arteries, veins, and nerves may have disastrous consequences.

Pathophysiology

In the upper extremity, the areas of greatest concern include the axilla and the area from the deltopectoral groove distally across the elbow to the proximal forearm. The axilla, medial/anterior upper arm, and antecubital fossa particularly are considered high-risk areas due to the superficial location of the axillary and brachial arteries in these regions.

Wounds distal to the bifurcation of the brachial artery are less likely to result in serious limb ischemia, as long as the ulnar and radial arteries remain intact. Injuries to a single distal artery often can be managed by ligation if the palmar arches are complete (95% of patients), and if there is no history of prior injuries.

In the lower extremity, the area of greatest concern extends from the top of the leg (marked by the inguinal ligament anteriorly and by the inferior gluteal fold posteriorly), across the knee inferiorly to the

level of the mid calf. The inguinal region, medial thigh, and popliteal fossa particularly are considered high-risk locations.

Below the knee, the popliteal artery trifurcates to form the anterior and posterior tibial arteries and the peroneal artery. Arterial wounds affecting a single vessel distal to the trifurcation do not always produce serious limb ischemia. If distal collateralization is adequate, injuries to a single branch may be managed by ligation.

The highest risk of serious vascular injury is associated with high-energy gunshot wounds such as those produced by military rifles and shotguns. Blunt and penetrating injuries associated with fractures also have a high incidence of associated vascular injury, even in the absence of clinical signs. The likelihood of serious vascular injury is less in patients who have received low-energy wounds such as those produced by handguns and knives.

Frequency

- **In the US:** Peripheral injuries account for 80% of all cases of vascular trauma. In two thirds of patients with vascular injuries, the lower extremities are involved.

Mortality/Morbidity

Death due solely to extremity vascular trauma is uncommon, except by exsanguination or development of a necrotizing myofascial infection.

- Compartment syndrome may result from ischemia of a muscle compartment. Limb survival is threatened by delays in diagnosis and treatment, particularly when limb perfusion is compromised for more than 6 hours.
- Extensive associated musculoskeletal and skin injuries indicate a poor prognosis.
- Crush injuries associated with open tibial fractures are particularly likely to cause loss of the lower leg.

Sex

Ninety percent of patients with vascular trauma are male.

Age

Vascular trauma most often occurs in patients aged 20-40 years.

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Clinical

History

- Mechanism of injury
- Time interval between injury and evaluation
- Previous history of vascular injury or disease
- Extensive or pulsatile external hemorrhage
- Anticoagulant therapy or impaired hemostatic function
- Prior venous thrombosis or embolism in the patient or family

Physical

- The presence of hard signs has a 91-95% sensitivity for injuries requiring intervention. The vast majority of patients exhibiting the following hard signs have such injuries.
 - Bruit or thrill, suggesting an arteriovenous fistula
 - Active or pulsatile hemorrhage
 - Pulsatile or expanding hematoma
 - Signs of limb ischemia
 - Diminished or absent pulses: This is not a sensitive prognostic sign, as up to a quarter of patients with major vascular injuries requiring repair may have normal pulses distal to the injury.

Causes

- Gunshot wounds, particularly high-energy and close-range shotgun wounds, cause 70-80% of all vascular injuries requiring intervention.
- Stab wounds (5-15% of cases)
- Blunt trauma (5-10% of cases): Presence of fracture increases risk.
- Iatrogenic injury (5% of cases): Cardiac catheterization and line placement are the 2 most common iatrogenic causes of vascular injury requiring intervention.

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Differentials

Compartment Syndrome, Extremity
Deep Venous Thrombosis and Thrombophlebitis

Other Problems to be Considered

Embolic phenomena

Vasospasm (eg, due to cocaine or extravasated dopamine)

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Workup

Lab Studies

- The arterial pressure index is useful in detecting patients with major vascular injury who have pulses that appear normal.
 - Systolic blood pressure in the affected extremity is divided by systolic pressure in the normal extremity. A value of less than 90% is abnormal.
 - The sensitivity of the pressure index for injuries requiring intervention ranges from 44-95%, depending on circumstances.

Imaging Studies

- Duplex ultrasonography
 - Ultrasound is an as yet unreliable but very promising noninvasive technique for investigating injuries with a high-risk mechanism or location but without any obvious indication for surgical management.
 - Small prospective studies with highly qualified teams maintaining a high index of suspicion suggest that the sensitivity of ultrasound can be up to 95-100% for diagnosing vascular injuries that lack hard signs but still require intervention.
 - Ultrasound examination is extremely operator dependent. The negative predictive value can be as low as 50% in some settings.

Other Tests

- Ankle: Brachial indices also may be used.
- Allen test: This test is only useful for injuries distal to the brachial artery bifurcation.

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Treatment

Prehospital Care

- Stabilize the extremity in anatomic position.
- Control hemorrhage with direct pressure.

Emergency Department Care

- Immediately reduce displaced or angulated fractures if any evidence or suspicion of vascular compromise exists. Promptly reduce dislocations of the elbow and knee to relieve tension on neurovascular structures.
- External hemorrhage usually can be controlled with direct pressure, but blood pressure–cuff tourniquets may be applied proximal to the injury if compression fails or is not possible.
- Once the patient has been stabilized, identify peripheral vascular injuries and restore normal circulation as rapidly as possible.
- Do not apply clamps or hemostats to vascular structures, since this may make repair more difficult.

Consultations

A vascular surgeon must be consulted whenever there is concern for vascular structures.

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Follow-up

Further Inpatient Care

- Surgical repair is performed as soon as possible for patients with hard signs of vascular injury, refractory hypotension, and obvious limb ischemia. Otherwise, a preoperative angiogram is preferred.
- Patients without hard signs
 - Proximity angiography and/or duplex sonography is performed only for high-risk injuries.

- Low-risk injuries may be observed in the hospital or on an outpatient basis, with a strict schedule of repeat evaluations.

Further Outpatient Care

- Low-risk injuries without hard signs may be managed on an outpatient basis with careful follow-up.
- All other patients should be admitted, either for definitive repair or for observation.

Complications

- Delayed diagnosis and treatment may result in thrombosis, embolization, or rupture.
- Arteriovenous fistula
- Pseudoaneurysm
- Intimal tear
- Segmental narrowing can present with diminished flow but intact pulses. This injury may resolve spontaneously (with fluids and rest) or may require intervention.

Prognosis

- Approximately 50% of nonocclusive injuries presenting without hard signs resolve over time.

Patient Education

- Patients must be given explicit instructions to perform extremity neurovascular checks on a scheduled basis. Instruct patients to return to the ED if they experience increased pain, edema, or active bleeding from the wound or if any weakness, numbness, or tingling develops in the injured extremity.

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Miscellaneous

Medical/Legal Pitfalls

- Major risk - Failure to appreciate the severity of the injury
- Failure to recognize that injuries may require repair even when pulses are intact
- Inappropriate delay in angiography and/or surgical intervention

- Failure to perform an appropriate examination, including objective tests, in all patients, including those who lack hard signs of vascular injury
 - Clamping vascular structures
-

Pictures



Picture 1: Pseudoaneurysm of the axillary artery

Picture type: Photo



Picture 2: Arteriovenous fistula between common femoral artery and vein

Picture type: Photo

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Plantar Fasciitis

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Synonyms, Key Words, and Related Terms

bone spur, heel pain

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Introduction

Background

Plantar fasciitis is the most common cause of inferior heel pain. Pain on the bottom of the heel, most prominent with the first steps that one takes in the morning, is the hallmark. Pain also occurs with the onset of activity such as walking and running. This pain may decrease as activity progresses, but it usually returns after resting and then resuming activity. In severe cases, the pain may occur with any weight bearing. Although the pain usually occurs in the heel, it can radiate throughout the bottom of the foot towards the toes.

Considered a chronic inflammatory syndrome rather than a post-traumatic disorder, plantar fasciitis is common in runners and dancers who use repetitive, maximal plantar flexion of the ankle and dorsiflexion

of the metatarsophalangeal joints. It is common in those who experience sudden weight gain and in overweight individuals who increase their activity level.

Pathophysiology

The plantar fascia is a dense band of tissue that originates from the anteromedial border of the calcaneus (medial calcaneal tuberosity) and fans out from there. It inserts on the flexor mechanism of the toes at the metatarsal heads.

The plantar fascia provides support for the medial longitudinal arch of the foot. Shortly after a heel strike, at the beginning of the stance phase of the gait cycle, the tibia rotates internally and the foot pronates, stretching the plantar fascia as the foot flattens. Since the fascia has no elastic properties, repetitive stretching results in microtears at its origin.

Typical morning pain is caused by the foot resting in equinus (plantar flexion) during the night, allowing the fascia to contract. With the first dorsiflexion steps of the day, the irritated fascia is stretched, resulting in pain.

Biopsy of the inflamed fascial area reveals fibroblastic proliferation and chronic granulomatous tissue, with thickening of the fascia from a normal 3.0 mm to as much as 15 mm. As this tight fascia is pulled and torn away from the tuberosity, calcium is deposited, and an exostosis (bone spur) may form on the inferior calcaneus.

Contrary to popular opinion, the bone spur itself does not cause the pain, but rather, the chronic inflammation of the torn fascia causes the pain. The spur is a sign of fasciitis, not a cause. Half of patients with plantar fasciitis have spurs; however, a bone spur often exists without concomitant fasciitis. Findings from recent studies suggest that bone spurs are more often associated with the flexor digitorum brevis muscle than with the plantar fascia itself.

Frequency

- **In the US:** Eleven percent of the population has bone spurs. However, a bone spur does not necessarily indicate plantar fasciitis. Bilateral symptoms occur in 20-30% of patients with plantar fasciitis.

Sex

Plantar fasciitis is more common in middle-aged women and young male runners. Obesity is present in 90% of affected females and 40% of affected males.

Age

Persons of all ages are affected.

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Clinical

History

- Heel pain, which is worse with the first steps in the morning, is the hallmark of this disorder.
 - Pain may be noted at the beginning of exercise and when weight bearing is resumed after resting.
 - The pain is dull and is similar to that of a toothache.

Physical

No tenderness should be present with medial to lateral heel compression. A finding of tenderness is suggestive of a stress fracture or osteomyelitis. Findings at physical examination may include the following:

- Flat foot, highly-arched foot, or excessively pronated foot
- Gait alteration (supination of foot to redistribute the load laterally or toe-walking to avoid pressure on the heel)
- Tenderness of heel with dorsiflexion of the foot or great toe that resolves with plantar flexion of the foot, which relaxes the fascia
- Decreased dorsiflexion of great toe
- Decreased dorsiflexion of ankle (<90°)
- Possible small granuloma palpable along the medial fascial origin
- Mild edema (Significant edema suggests a different diagnosis such as calcaneal stress fracture.)

Causes

Repetitive stretching of a tight plantar fascial band leads to microtears at its calcaneal origin.

Predisposing factors include the following:

- Obesity or sudden weight gain
- Tight Achilles tendon
- Change in walking or running habits
- Use of shoes with poor cushioning

- Change in running or walking surface
- Occupation with prolonged weight bearing (policeman's heel)
- Excessive pronation, pes planus, or pes cavus

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Differentials

Fractures, Foot
Osteomyelitis
Reiter Syndrome

Other Problems to be Considered

Calcaneus fracture (stress or traumatic)

Heel fat pad syndrome

Atrophy of plantar fat pad

Sever disease (calcaneal apophysitis in children aged 7-15 y)

Foreign body

Entrapment of medial calcaneal branch of posterior tibial nerve

Entrapment of first branch of lateral plantar nerve

Tarsal tunnel syndrome

Ankylosing spondylitis

Psoriatic arthritis

Sacral radiculopathy

Infection

Tumor

Plantar fascial rupture

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Workup

Lab Studies

- If inflammatory arthritis or infection is being considered, determination of the sedimentation rate; CBC; and rheumatoid factor, HLA-B27, uric acid, and antinuclear antibody levels may be helpful. Tests for these might be considered if bilateral symptoms exist, if other joints are involved, or if the clinical picture is atypical.

Imaging Studies

- Lateral radiographs of heel may be indicated to rule out stress fractures or bony erosion if the diagnosis is uncertain. They may or may not show bone spur, which is of no diagnostic value.
- Bone scanning is helpful only if a stress fracture or osteomyelitis is still a possible differential diagnosis. Plantar fasciitis shows increased focal uptake at the medial calcaneal tubercle. Stress fractures show intense uptake throughout the bone
- MRIs and ultrasonographic images may show a thickened, inflamed fascia; however, these generally are not indicated.

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Treatment

Emergency Department Care

- No specific treatment is necessary in the ED other than patient education, as described later.
- In severe cases, the patient may need to use a below-the-knee walking cast for 3-4 weeks.

Consultations

A podiatrist may be consulted if conservative treatment fails after weeks or months. A podiatrist may assist in obtaining splints, arch supports, and other devices.

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Medication

Nonsteroidal anti-inflammatory drugs are the only medications indicated for this disorder. They should be used for 2-4 weeks in conjunction with the treatment regimen.

Nonsteroidal Anti-Inflammatory Drugs (NsAIDs)

Decrease inflammatory responses and systemically interfere with events leading to inflammation.

| | |
|-------------------|---|
| Drug Name | Ibuprofen (Advil, Motrin)- Inhibits inflammatory reactions and pain by decreasing prostaglandin synthesis. |
| Adult Dose | 600 mg PO tid with food |
| Pediatric Dose | 10 mg/kg PO tid with food |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, decreased renal and hepatic function; anticoagulation abnormalities, during anticoagulant therapy |

| | |
|------------|---|
| Drug Name | Naproxen (Naprosyn, Aleve)- Inhibits inflammatory reactions and pain by decreasing activity of cyclo-oxygenase, which results in a decrease of prostaglandin synthesis. Slightly longer half-life than that of ibuprofen (which decreases the frequency of daily dosing). |
| Adult Dose | 220-550 mg (sodium salt) PO bid |

| | |
|-------------------|---|
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Coadministration with aspirin increases risk of serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion may be at risk of acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

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Follow-up

Further Outpatient Care

- In moderate cases, patients may be advised to do the following:
 - Avoid activities that irritate the condition and avoid barefoot walking on hard surfaces.
 - Lose weight.
 - Perform heel cord (Achilles) and plantar fascia stretches (see [Patient Education](#)). Stretches may exacerbate symptoms for 1-3 weeks but should be continued.
 - Tape the heel and arch (eg, with low-dye tape) (see [Picture 1](#)).
 - Use a silastic or felt heel pad with or without a hole cut in the center, or use shoe inserts with arch support and a cushioned heel (see [Picture 2](#)). Medial longitudinal arch supports, extending from the distal medial calcaneal tubercle to 5 mm proximal to the metatarsal heads, are generally recommended. However, findings of a recent cadaveric study suggest that a lateral forefoot wedge might unload the strain on the plantar fascia; use of this device has not been studied in active humans.
 - If limb length discrepancy is an issue, use a quarter-inch heel lift for the shorter leg to help relieve symptoms.
 - Use night splints designed to keep the foot in slight (5°) dorsiflexion to stretch the fascia

while sleeping. Reportedly, the cure rate is 80% at 4 months (see [Picture 3](#)). These are thought to be most helpful in patients with recalcitrant symptoms (duration >1 y).

- Massage the fascia by rolling the foot over a tube (3-4-inch diameter) such as a rolling pin or soup can.
- Strengthen the foot and ankle muscles that support the arch by bunching up a hand towel with the toes or by pulling a towel weighted with a soup can across the floor.
- After exercising, apply a cold pack to heel at the point of maximal tenderness for 20-30 minutes.
- Use other modalities such as iontophoresis with 0.4% dexamethasone 6 times during 2 weeks. Compared with standard treatment, this treatment has resulted in some early symptomatic improvement but no real difference at 4 weeks.
- Use extracorporeal shock wave therapy. In Europe and Canada, experiments have revealed some good results. This procedure is now approved in the US for the treatment of chronic proximal plantar fasciitis that now responds to conventional treatment after 6 months. The treatment is painful and requires 4-6 weeks of relative rest afterwards before pain relief and healing are noted. One center has reported an 80% success rate if the patients are pre-screened and have a plantar fascia that is thickened by more than 4 mm at ultrasonography. The Food and Drug Administration reports that the overall success for this therapy is 47%.

Deterrence/Prevention

- Weight loss
- Stretching the Achilles tendon area 2-3 times per day and before exercising
- Use of proper footwear with arch support and a cushioned heel

Complications

- Fat pad atrophy
- Osteomyelitis
- Rupture of the plantar fascia from steroid injections

Prognosis

- If treatment begins early, most patients can be cured in 6-12 weeks, or at least, symptoms can improve. However, chronic problems that resist all treatment efforts may be frustrating for the patient. Patients aged 45 years or older may have a slower improvement, but improvement eventually occurs; aging fibroblasts may be less adept at connective tissue matrix repair because of slower protein synthesis.

Patient Education

- Patients may be instructed to perform the following stretching exercise.
 - Instructions: Stand on a step with only the toes on a step; the heels should be free. Hold onto a rail and slowly lower the heels. Hold stretch for a count of 10, then rest with the heels back on the step. Repeat this exercise 10 times per set; perform 5 sets per day.
 - The patient should feel stretching in the calf and plantar fascia.

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Miscellaneous

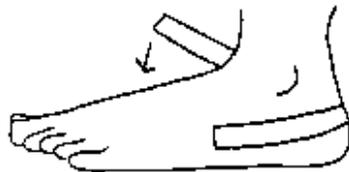
Medical/Legal Pitfalls

- Failure to recognize, consider, or treat fractures, osteomyelitis, or other illnesses listed in the differential diagnosis list
 - Steroid injections may cause Infection and fat pad atrophy
 - Injections introduced through the plantar surface are more apt to cause infection.
 - Excessive steroid in the fat pad area can result in fat pad atrophy and make the problem worse.
-

Pictures

Low-dye Taping

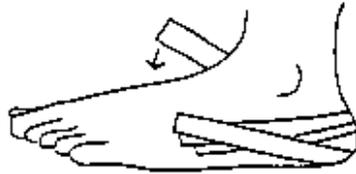
The following sketches illustrate the steps involved in low-dye taping, a technique that provides support for the plantar fascia and helps reduce excessive pronation.



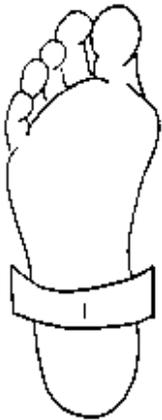
Step 1



Step 2



Step 3



Step 4



Step 5



Step 6

Picture 1: Low-dye taping method (drawing by Dawn Hankins, ATC, MS)

Picture type: Photo



Picture 2: Example of an arch support with a cushioned heel: These are available in three-quarter or full lengths to fit in the shoe.

Picture type: Photo



Picture 3: Example of a night splint: These are intended to prevent shortening of the Achilles tendon and plantar fascia at night.

Picture type: Photo

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Pneumothorax, Tension and Traumatic

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Synonyms, Key Words, and Related Terms

pleural gas

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Introduction

Background

A pneumothorax refers to a collection of gas in the pleural space resulting in collapse of the lung on the affected side. A tension pneumothorax is a life-threatening condition caused by air within the pleural space that is under pressure; displacing mediastinal structures and compromising cardiopulmonary function. A traumatic pneumothorax results from blunt or penetrating injury that disrupts the parietal or visceral pleura. Mechanisms include injuries secondary to medical or surgical procedures.

Pathophysiology

A tension pneumothorax results from any lung parenchymal or bronchial injury that acts as a one-way valve and allows free air to move into an intact pleural space but prevents the free exit of that air. In addition to this mechanism, the positive pressure used with mechanical ventilation therapy can cause air trapping.

As pressure within the intrapleural space increases, the heart and mediastinal structures are pushed to the contralateral side. The mediastinum impinges on and compresses the contralateral lung.

Hypoxia results as the collapsed lung on the affected side and the compressed lung on the contralateral side compromise effective gas exchange. This hypoxia and decreased venous return caused by compression of the relatively thin walls of the atria impair cardiac function. The decrease in cardiac output results in hypotension and, ultimately, in hemodynamic collapse and death, if untreated.

Frequency

- **In the US:** A study conducted from 1959-1978 involving a US community with an average of 60,000 residents demonstrated an incidence of primary spontaneous pneumothorax of 7.4 per 100,000 persons per year for men and 1.2 per 100,000 persons per year for women. When these figures are extrapolated, about 8,600 individuals develop a primary spontaneous pneumothorax in the US per year.

Tension pneumothorax is a complication in approximately 1-2% of the cases of idiopathic spontaneous pneumothorax. Until the late 1800s, tuberculosis was a primary cause of pneumothorax development. A 1962 study showed a frequency of pneumothorax of 1.4% in patients with tuberculosis.

Undoubtedly, the incidence of pneumothorax and/or tension pneumothorax in US hospitals has increased as intensive care treatment modalities have become increasingly dependent on positive-pressure ventilation, central venous catheter placement, and other causes that potentially induce iatrogenic pneumothorax.

Mortality/Morbidity

The clinician should assume that a tension pneumothorax results in hemodynamic instability and death, unless immediately treated.

Sex

The male-to-female ratio is about 6:1 for primary spontaneous pneumothorax development.

- In men, the risk of spontaneous pneumothorax is 102 times higher in heavy smokers than in

nonsmokers. Spontaneous pneumothorax most frequently occurs in tall, thin men aged 20-40 years.

- Catamenial pneumothorax is a rare phenomenon that generally occurs in women aged 30-50 years. It frequently begins 1-3 days after menses onset. Its etiology may be primarily related to associated diaphragmatic defects.

Age

Pneumothorax occurs in 1-2% of all neonates. The incidence of pneumothorax in infants with neonatal respiratory distress syndrome is higher. In one study, 19% of such patients developed a pneumothorax.

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Clinical

History

The signs and symptoms produced by tension pneumothorax are usually more impressive than those seen with a simple pneumothorax. They may include the following:

- Chest pain (90%)
- Dyspnea (80%)
- Anxiety
- Fatigue

Physical

Findings at physical examination may include the following:

- Respiratory distress and/or arrest
- Cyanosis
- Unilaterally decreased or absent lung sounds
- Lung sounds transmitted from the nonaffected hemithorax are minimal with auscultation at the midaxillary line
- Tachypnea
- Hyperresonance of the chest wall on percussion
- Increasing resistance to providing adequate ventilation assistance
- Tachycardia
- Tracheal deviation (relatively late finding due to midline shift with a tension pneumothorax)

- Jugular venous distension (with a tension pneumothorax)
- Hypotension (key sign of a tension pneumothorax)
- Pulsus paradoxus
- Mental status changes, including decreased alertness and/or consciousness
- Abdominal distension (from increased pressure in the thoracic cavity producing caudal deviation of the diaphragm and from secondary pneumoperitoneum produced as air dissects across the diaphragm through the pores of Kohn)

Causes

A wide variety of disease states and circumstances increase the patient's risk of a pneumothorax. If a pneumothorax is complicated by a one-way valve effect, tension pneumothorax may result.

- Infants requiring ventilatory assistance and those with meconium aspiration have a particularly high risk for tension pneumothorax. Aspirated meconium may serve as a one-way valve and produce a tension pneumothorax.
- Trauma may cause a pneumothorax.
 - Tension pneumothorax may be the result of blunt trauma with or without associated rib fractures.
 - Incidents that may cause tension pneumothoraces include unrestrained head-on motor vehicle accidents, falls, and altercations involving laterally directed blows.
 - Any penetrating wound that produces an abnormal passageway for gas exchange into the pleural spaces and that results in air trapping may produce a tension pneumothorax.
 - In a recent study, 12% of patients with asymptomatic chest stab wounds had a delayed pneumothorax or hemothorax.
- Pneumothorax is associated with asthma, chronic obstructive pulmonary disease, pneumonia (especially with *Staphylococcus*, *Klebsiella*, *Pseudomonas*, and *Pneumocystis* species), pertussis, tuberculosis, lung abscess, and cystic fibrosis.
 - In pulmonary disorders such as asthma and emphysema, hyperexpansion disrupts the alveoli.
 - Increased pulmonary pressure due to coughing with a bronchial plug of mucous or phlegm may play a role.
- Individuals may inherit a predisposition for primary spontaneous pneumothorax.

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Differentials

Angina

Anxiety
Asthma
Congestive Heart Failure and Pulmonary Edema
Diaphragmatic Injuries
Dissection, Aortic
Esophageal Perforation, Rupture and Tears
Foreign Bodies, Trachea
Myocardial Infarction
Pediatrics, Pertussis
Pediatrics, Pneumonia
Pericarditis and Cardiac Tamponade
Pneumonia, Aspiration
Pneumonia, Bacterial
Pneumonia, Empyema and Abscess
Pneumonia, Immunocompromised
Pneumonia, Mycoplasma
Pneumonia, Viral
Pneumothorax, Iatrogenic, Spontaneous and Pneumomediastinum
Respiratory Distress Syndrome, Adult
Tuberculosis

Other Problems to be Considered

Airway obstruction

Hemothorax

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Workup

Lab Studies

- ABG analysis does not replace physical diagnosis nor should treatment be delayed while awaiting results if symptomatic pneumothorax is suspected. However, ABG analysis may be useful in evaluating the following:
 - Hypoxia
 - Hypercarbia and respiratory acidosis

Imaging Studies

- Translumination: In neonatal patients, one may notice increased transmission of light through the chest on the affected side.
- Chest radiography
 - Although the initial chest radiograph may show no evidence of pneumothorax, consider the possibility of delayed traumatic pneumothorax development in any penetrating chest wound. Obtain serial chest radiographs every 6 hours the first day after injury to rule this out. Some authors advocate the acquisition of only one or two serial examinations every 4-6 hours.
 - Air in the pleural cavity, with contralateral deviation of mediastinal structures, is evidence of a tension pneumothorax.
 - When evaluating the chest radiograph for pneumothorax, assess rotation. Rotation can obscure a pneumothorax. Rotation can also mimic a mediastinal shift.
 - In evaluating the radiograph for rotation, compare the symmetry and shape of the clavicles. Also, look at the relative lengths of the ribs in the middle lung fields on each side on the anteroposterior or posteroanterior views. On an image with rotation, the ribs on each side often have unequal lengths.
 - In a nonloculated pneumothorax, air rises to the nondependent portion of the pleural cavity. Therefore, carefully examine the apices of an upright chest radiograph, and scrutinize the costophrenic and cardiophrenic angles on a supine chest radiograph.
 - A skin fold can be mistaken for a pneumothorax. Unlike pneumothoraces, skin folds usually continue beyond the chest wall, and lung markings can be seen peripheral to the skin fold line. Viewing the film under the hot lamp may be necessary to discern obscure peripheral lung markings.
 - In evaluating the chest radiograph, first impressions of pneumothorax size can be misleading. To assist in determining the size of pneumothorax on the radiograph, a 2.5-cm margin of gas peripheral to the collapsing lung corresponds to a pneumothorax of about 30%. Complete collapse of the lung is a 100% pneumothorax.

Procedures

- Needle thoracostomy is performed as follows:
 - Locate puncture site. The second intercostal space in the midclavicular line on the affected side immediately superior to the rib is most commonly recommended site.
 - Prepare the puncture site with Betadine and/or alcohol scrubs.
 - Insert a large-bore Angiocath (14-gauge in an adult, 18-gauge or 20-gauge in an infant) into the desired intercostal space over the top of the rib and perpendicular to the chest wall. Listen for a rush of air.
 - Remove the needle.
 - Secure the Angiocath in place, and establish a water seal or flutter valve.
 - Immediately prepare to insert a chest tube.

- Listen for a rush of air on insertion to confirm the diagnosis of tension pneumothorax. Note this finding on the patient's chart. In an area with high ambient noise, the escape of air may not be detected.
- Needle thoracostomy requires follow-up placement of a chest tube.

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Treatment

Prehospital Care

Attention to the ABCs is mandatory for all patients with thoracic trauma. Evaluate the patency of the airway and the adequacy of the ventilatory effort. Assess the circulatory status and the integrity of the chest wall.

- Failure of the emergency medical service personnel and medical control physician to make a correct diagnosis of tension pneumothorax and to promptly perform needle decompression in the prehospital setting likely results in rapid clinical deterioration and cardiac arrest.
- However, if an incorrect diagnosis of tension pneumothorax is made in the prehospital setting, the patient's life is endangered by unnecessary invasive procedures. Close cooperation and accurate communication between the physician and the emergency medical service personnel is of paramount importance.
- To prevent reentry of air into the pleural cavity after needle thoracostomy and decompression in the prehospital setting, a one-way valve should be attached to the distal end of the Angiocath. If available, a Heimlich valve may be used. If a commercially prepared valve is not available, attach a finger condom or the finger of a rubber glove with its tip removed to serve as a makeshift one-way valve device.
- Clothing covering a wound that communicates with the chest cavity can play a role in producing a one-way valve effect, allowing air to enter the pleural cavity but hindering its exit. Removing such clothing items from the wound may facilitate decompression of a tension pneumothorax.
- A tension pneumothorax is a contraindication to the use of military antishock trousers.

Emergency Department Care

For all patients with thoracic injury, immediate and careful attention to the ABCs is vital. Fully assess the patency of the airway and adequacy of the ventilatory effort. Carefully evaluate the cardiovascular system, because a tension pneumothorax and a pericardial tamponade can cause similar findings.

- If a tension pneumothorax is suspected, immediately administer 100% oxygen, and evaluate the

patient for evidence of respiratory compromise, hemodynamic instability, or clinical deterioration. Place large-bore catheters, because hemothorax can be associated with pneumothorax, and the patient may, therefore, require immediate intravenous infusion.

- Immediately perform needle thoracostomy or chest tube placement if the clinical condition warrants such action. Once a needle thoracostomy has been performed, chest tube insertion must follow.
- If a hemothorax is associated with the pneumothorax, additional chest tubes may be needed to assist drainage of blood and clots. If the hemopneumothorax requires insertion of a second chest tube, the second tube should be directed inferiorly and should be posterior to the diaphragm.
- Chest tubes are attached to a vacuum apparatus that continually removes air from the pleural cavity. The collapsed lung re-expands and heals, thereby preventing continued air leakage. After air leaks have ceased for 24 hours, the vacuum may be decreased and the chest tube removed.
- The process of lung re-expansion and healing is not immediate and may be complicated by pulmonary edema. A chest tube is, therefore, usually left in place for at least 3 days unless the clinical condition warrants a longer placement.
- In general, traumatic pneumothoraces should be treated with insertion of a chest tube, particularly if the patient cannot be closely observed.
 - A subset of patients who have a small (<15-20%), minimally symptomatic pneumothorax may be admitted, observed closely, and monitored by using serial chest radiographs.
 - In these patients, administration of 100% oxygen promotes resolution by speeding the absorption of gas from the pleural cavity into the pulmonary vasculature.

Consultations

- Treatment of tension pneumothorax should commence immediately after diagnosis, without waiting for further consultation and/or evaluation.
- A trauma or general surgeon should evaluate patients with trauma, and the patient should be admitted for observation.

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Medication

A tension pneumothorax requires treatment with procedural modalities. Anesthetics and analgesics should be used if the patient is not in distress. Medication may be necessary to treat the pulmonary disorder that caused the pneumothorax. For example, intravenous antibiotics are included in the treatment of a pneumothorax that developed as a sequela of staphylococcal pneumonia. Also, studies suggest that the administration of prophylactic antibiotics after chest tube insertion may reduce the incidence of complications such as emphysema.

Follow-up

Further Inpatient Care

- If the patient has had repeated episodes of pneumothorax or if the lung remains unexpanded after 5 days with a chest tube in place, surgery may be necessary. The surgeon may use treatment options such as thoracoscopy, electrocautery, laser treatment, resection of blebs or pleura, or open thoracotomy.
- In patients with repeated pneumothoraces who are not good candidates for surgery, sclerotherapy with talc or doxycycline may be necessary.

Deterrence/Prevention

- Advise patients to wear safety belts and passive restraint devices while driving.
- Encourage smoking cessation.
- The incidence of iatrogenic tension pneumothorax may be decreased with prophylactic insertion of a chest tube in patients with a simple pneumothorax that requires positive pressure ventilation.
- When subclavian vein cannulation is required, use the supraclavicular approach rather than the infraclavicular approach when possible to help decrease the likelihood of pneumothorax formation.
- Prompt recognition and treatment of bronchopulmonary infections decreases the risk of progression to a pneumothorax.

Complications

- Respiratory distress and/or arrest
- Cardiac arrest
- Pulmonary edema (following lung re-expansion)
- Empyema
- Persistent bronchopulmonary fistula
- Pneumomediastinum
- Pneumopericardium
- Pneumoperitoneum
- Pyopneumothorax
- Hemopneumothorax

Prognosis

- The prognosis is generally good with appropriate therapy, but it varies depending on the etiology.

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Miscellaneous

Medical/Legal Pitfalls

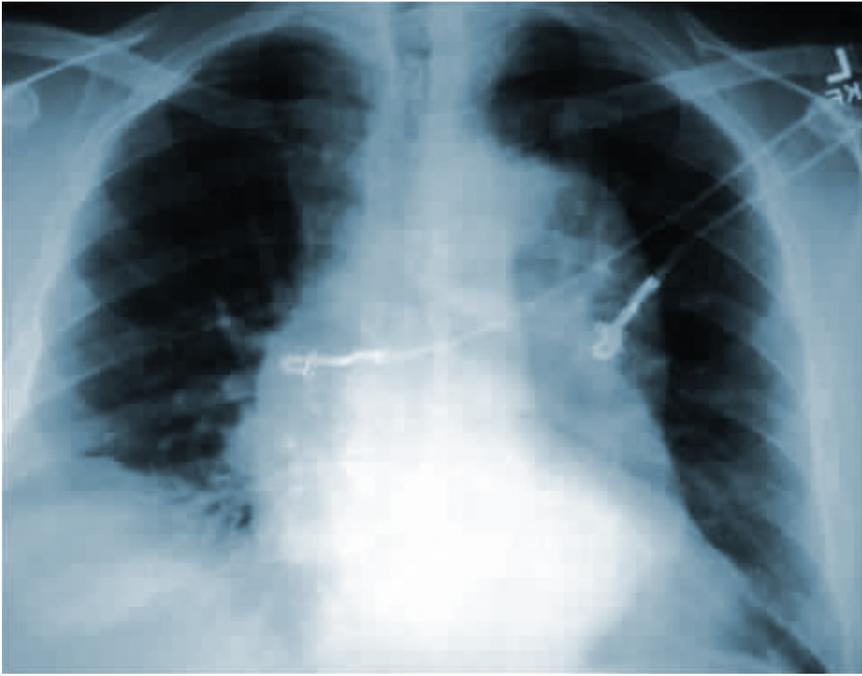
- The diagnosis of a tension pneumothorax should be made based on the history and physical examination findings. A chest radiograph or CT scan should be used only in those instances when the clinician is in doubt regarding the diagnosis and when the patient's clinical condition is sufficiently stable. Obtaining such imaging studies when the diagnosis of tension pneumothorax is not in question causes an unnecessary and potentially lethal delay in treatment.
- A tension pneumothorax is a life-threatening condition and requires immediate action (eg, needle thoracostomy or chest tube insertion). However, the clinician should be wary of prematurely diagnosing a tension pneumothorax in a patient without hypoxia, hypotension, or cardiopulmonary compromise. If the patient's clinical presentation is questionable and if the patient appears stable, the clinician should re-examine the patient and request immediate portable chest radiography. (or the clinician can reexamine the chest radiographs if they have already been obtained) to confirm the diagnosis.
- Consider the diagnosis of a pneumothorax and/or tension pneumothorax with blunt and penetrating trauma. In the patient blunt trauma and mental status changes, hypoxia, and acidosis, symptoms may be attributed to a suspected intracerebral injury rather than a tension pneumothorax. Portable chest radiography should always be included in the initial radiographic evaluation of major trauma.
- When assessing the trauma patient, be aware that clinical presentations of tension pneumothorax and myocardial rupture with tamponade may be similar.
- Maintain a high index of suspicion for a tension pneumothorax in patients using ventilators who have a rapid onset of hemodynamic instability or cardiac arrest, particularly if they require increasing peak inspiratory pressures. Patients at greatest risk of a pneumothorax and/or tension pneumothorax include those with COPD who are using ventilators; those with acute respiratory distress syndrome; and those requiring a tidal volume greater than 12 mL/kg, a peak airway pressure greater than 60 cm H₂O, or a positive end-expiratory pressure greater than 15 cm H₂O.
- Avoid assuming that a patient with a chest tube does not have a tension pneumothorax if he or she has respiratory or hemodynamic instability. Chest tubes can become plugged or malpositioned and cease to function. Also, improper attachment of a one-way valve to the chest tube may produce tension pneumothorax.

Pictures



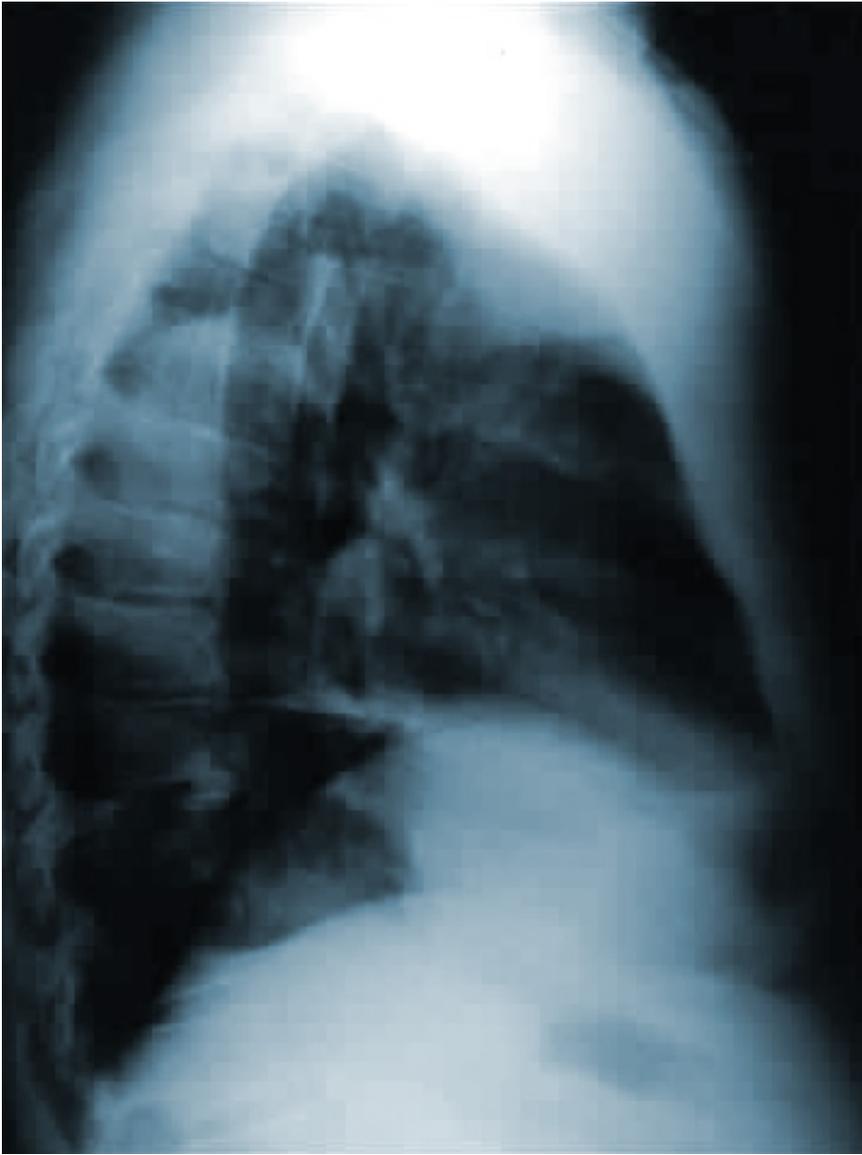
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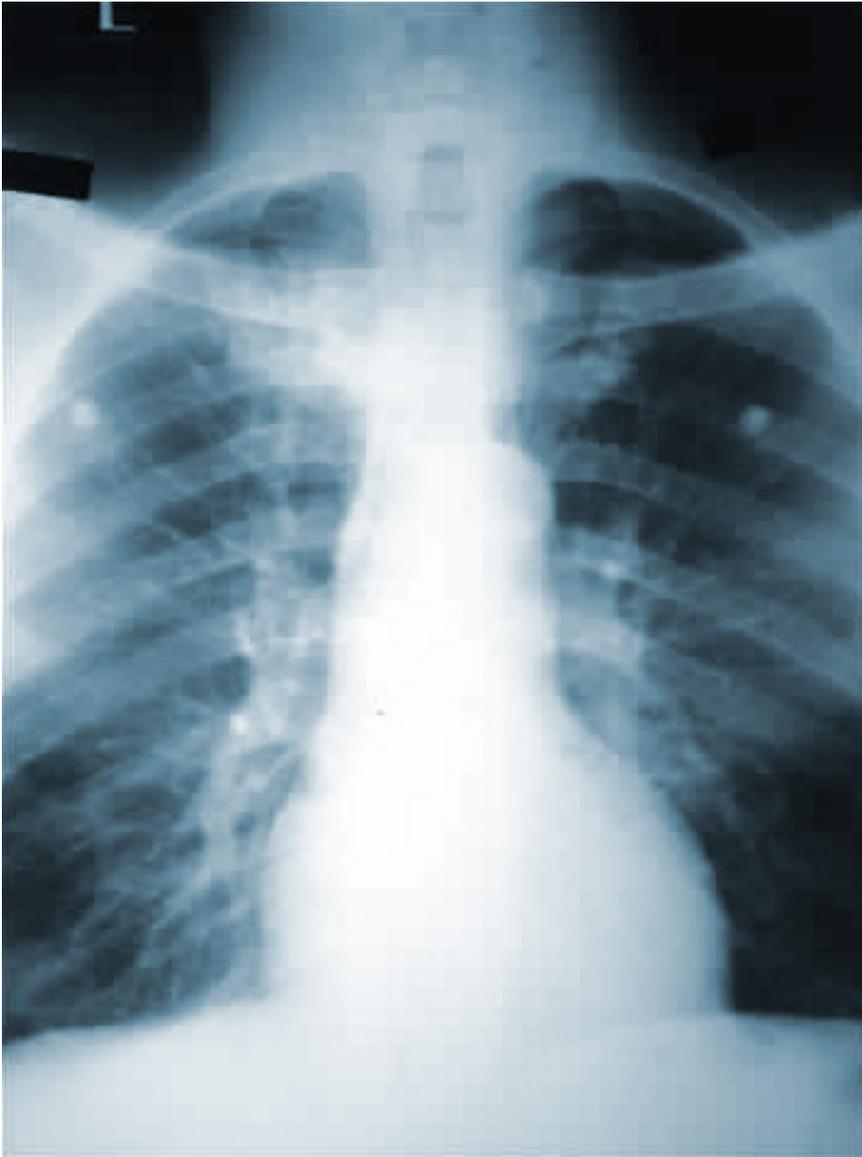
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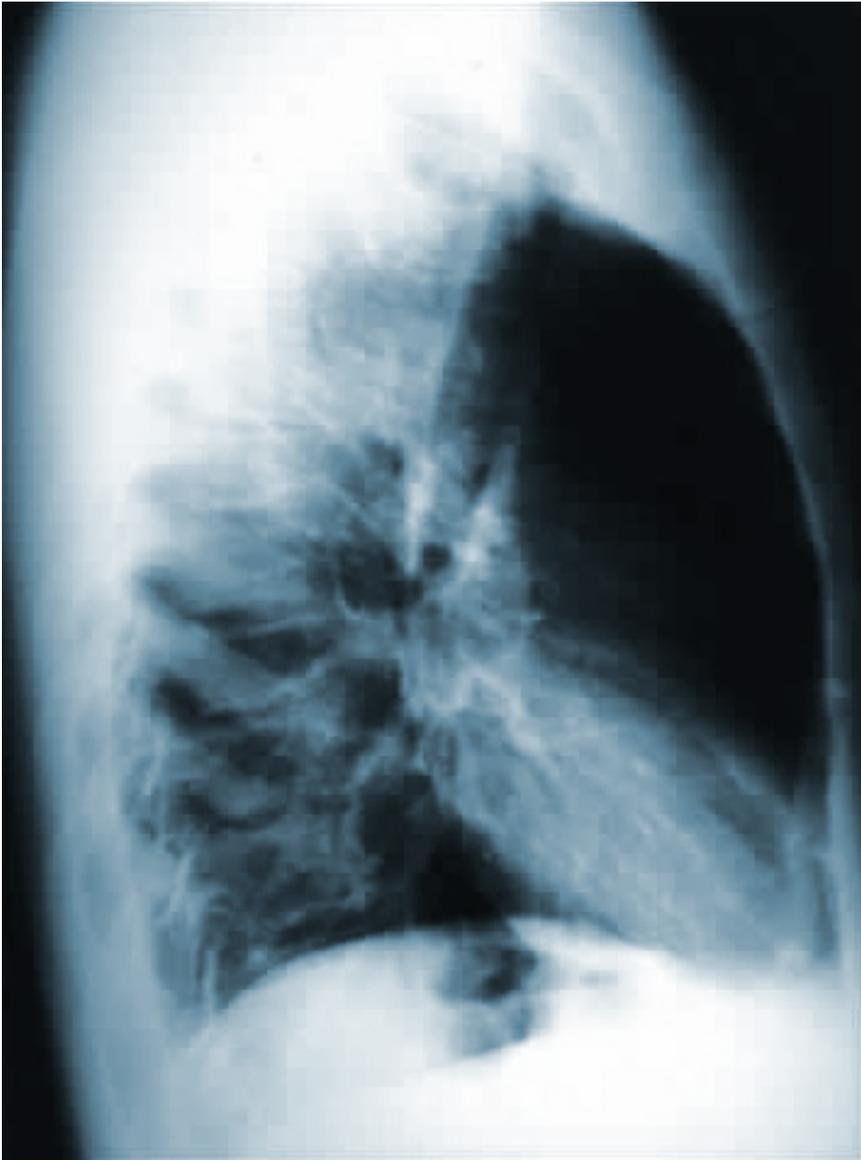
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Picture 4: "
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Picture 5: "
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Picture 6: "

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Postconcussive Syndrome

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Synonyms, Key Words, and Related Terms

PCS, minor head injury, MHI

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Introduction

Background

Postconcussive syndrome (PCS), a sequela of minor head injury (MHI), has been a much-debated topic of recent years. Muddled by conflicting findings regarding symptom duration, an absence of objective neurologic findings, inconsistencies in presentation, and significant methodologic problems in the literature, PCS remains controversial. Depending on the definition and the population examined, 29-90% of patients experience postconcussive symptoms shortly after the traumatic insult.

While no universally accepted definition exists, most of the literature defines the syndrome as the continuation of at least 3 of the following symptoms: headache, dizziness, fatigue, irritability, impaired memory and concentration, insomnia, and lowered tolerance for noise and light. Confusion exists in the

literature, with some authors defining it as symptoms of at least 3 months duration, while others define it as symptoms appearing within the first week. In this article, the syndrome is loosely defined as symptom occurrence and persistence within several weeks after the initial insult.

Pathophysiology

An MHI typically indicates a blow to the head with a brief period of loss of consciousness (LOC) and/or posttraumatic amnesia or disorientation. At presentation, the Glasgow Coma Scale score is 13-15. LOC is thought to result from trauma to the brainstem or from diffuse cerebral injury. Findings at initial neurologic examination may be completely normal, and the patient usually has a normal CT scan of the brain.

An absence of LOC and/or a normal CT scan, however, do not confirm the absence of damage to the brain. Shear strain on the neurons that leads to diffuse axonal injury can occur without CT abnormalities. The soft tissues of the neck may be strained while protecting the brainstem and inhibiting LOC.

Neuropsychological assessments and scanning (eg, CT scanning, MRI, SPECT) used to detect organic causes may reveal impairments associated with PCS. Findings from neuropsychological evaluations demonstrate that symptom severity is not necessarily dependent on neurologic status immediately following injury. However, in some series, the length of LOC or posttraumatic amnesia may be correlated with the probability of developing PCS.

Discrepancies between organic evidence and symptom presentation bring symptom origination into question. Manifestations most commonly occur during the initial weeks and resolve within 3 months after injury, yet symptom persistence in approximately one third of patients with an MHI, often with objective cognitive deficits that have resolved, may make assessment and treatment difficult.

It is not entirely discernible which symptoms are organic and which have a psychological basis. Physical-emotional comorbidity precipitates the development and continuation of symptoms associated with PCS, because recovery is based on injury severity, age, education, vocational abilities, physical well being, psychosocial skills, cognitive functioning, and personality factors.

Frequency

- **In the US:** More than 2 million instances of MHI occur in the United States each year. The overall incidence rate of MHI for persons not hospitalized, with data compiled by the National Hospital Ambulatory Medical Care Survey (1995-1996), was 392 per 100,000 population or 1,027,000 visits per year to hospital EDs in the US. Depending on the definitions used and population examined, approximately 20-90% of patients develop at least 1 symptom of PCS within the first month, and about 40% have at least 3 symptoms at 3 months.

One survey of high school sports revealed that 20% of football players sustained some form of minor head trauma in 1 year. The incidence of PCS in these players is unknown.

Mortality/Morbidity

Morbidity is mainly due to the persistence of symptoms, which make it difficult for patients to resume premorbid functions.

Sex

The incidence of PCS is greater in females than in males.

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Clinical

History

Most patients present shortly after a MHI. Often, patients return after a previous evaluation in the ED because of persistent postconcussive symptoms. Findings may include the following:

- Anxiety
- Depression
- Short-term memory deficits
- Irritability

Physical

In general, the findings at physical examination are normal. The patient may exhibit subtle neurologic findings, but objective focal motor deficits should raise a concern about an undiagnosed intracranial bleed. Other findings may include the following:

- Depressed affect
- Decreased ability to smell and taste
- Neurasthenia or hyperesthesia (but not in a dermatomal distribution)
- Cognitive deficits
 - Neuropsychological testing has revealed that defects can persist 6 months or longer when other symptoms are present.
 - These defects include difficulties with vocabulary, short-term and intermediate-term

memory, object recall, drawing, and mathematics.

- Patients without other subjective symptoms usually perform normally on these tests.
- However, testing also has revealed that these defects resolve when other somatic and neurologic symptoms do not.

Causes

Risk factors for the development of PCS include nonsporting mechanisms, loss of consciousness, amnesia for the event, female sex, and abnormal neurobehavioral testing results after the incident.

- Although a common perception is that people develop PCS from head injury who perceive a source of blame for the injury desire to pursue litigation, one study did not demonstrate a correlation between blame and litigation.
- Some authors have concluded that persons with certain premorbid personality types or poor coping skills may be predisposed to PCS, but the data are conflicting.
- Neck pain after a head injury has not been correlated with the development of PCS.

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Differentials

Epidural Hematoma
Epidural and Subdural Infections
Headache, Migraine
Headache, Tension
Stroke, Hemorrhagic
Subarachnoid Hemorrhage
Subdural Hematoma

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Workup

Lab Studies

- No specific laboratory studies are needed, unless concomitant illness is suspected or unless the diagnosis is unclear and believed to be of toxic or metabolic origin.

Imaging Studies

- CT scanning is used to determine the presence of intracranial abnormalities and skull fractures. Patients with PCS usually do not present immediately after the trauma.
 - If a CT scan has already been obtained, the utility of a repeat scan is minimal in the absence of focal neurologic signs or unless the patient is at risk for delayed hemorrhage.
 - If a CT scan has not been obtained and if the patient has a GCS of 15, the likelihood of finding an operable lesion is extremely limited.
 - CT scanning is still a reasonable, fast, and effective screening test.

Other Tests

- Neuropsychological testing
 - This testing rarely is performed in the acute setting, although it may have some value in predicting the development of symptoms.
 - A series of standardized tests and questionnaires are used to measure attention, language, memory, emotional functioning, and other neurobehavioral parameters.
 - The Rivermead Postconcussion Symptoms Questionnaire is used to quantify PCS symptoms.
 - Neuropsychological assessments may be used. These include the Wechsler Adult Intelligence Scale and specific subtests (digit span and vocabulary), Trail Making Test, complex figure drawings (eg, Rey Osterreith), copy trials and memory trials, category tests, controlled oral word association, and the Paced Auditory Serial Addition Task.
 - The objective personality measure, Minnesota Multiphasic Personality Inventory, Second Edition (MMPI2), may be used.
 - The Hospital Anxiety and Depression Scale, Impact of Even Scale, and assessments of posttraumatic amnesia are used together as prognostic screening instruments for predicting PCS persistence.

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Treatment

Emergency Department Care

No specific care is required in the ED. Patients with the symptom constellation consistent with PCS require thorough physical and neurological examinations. A CT scan should be obtained if significant concern about intracranial hemorrhage exists, although this injury is rare in the patient presenting late

with a nonfocal findings at examination.

- Supportive care may include the use of nonnarcotic analgesics and antiemetics.
- Although some patients may be admitted if symptoms are severe, most can be discharged.
 - Several studies have revealed that patients admitted acutely after an MHI may have a lower incidence of PCS and its attendant social and psychological morbidity.
 - This finding is probably due to active interventions at follow-up.

Consultations

Rarely is consultation warranted in the ED once the diagnosis is made. Outpatient referral is the cornerstone of treatment. One study suggests that findings of early neuropsychological assessment may determine the prognosis; however, this assessment rarely is performed in the ED.

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Follow-up

Further Outpatient Care

- Outpatient care is the cornerstone of treatment and involves multidisciplinary teams that provide testing and treatment, including cognitive rehabilitation, psychotherapy, stress management, vocational counseling, and pain management.
 - No treatments have been proven effective; however, the multidisciplinary approach is believed to be worthwhile.
 - A neurologist, physical medicine specialist, primary care physician, or psychologist specializing in these disorders usually coordinates treatment.

Deterrence/Prevention

- The emergency physician should encourage the use of interventions to decrease the incidence of traumatic brain injury.
 - This approach is particularly important in young adults, who have a higher incidence of head injury than do others.
 - Encourage patients to wear a seatbelt and bicycle and/or motorcycle helmets, as appropriate.

Prognosis

- Most patients recover fully.
- Patients who are not better within 1 year will probably not get better.

Patient Education

- Educate the patient about the usual self-limited nature of PCS.
- Education about the usual symptoms may be helpful.
- Discussions concerning preparation for the graded resumption of vocational and academic routines may lessen PCS persistence.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to recognize the benefits of reassurance and follow-up care.
 - Patients are likely to experience difficulty with symptoms when neurologic examinations, psychological assessments, and physical examinations reveal limited evidence of or explanation for the persistence of the symptoms.
 - Patients' veracity may be questioned; they may be treated as if they are neurotic or merely seeking compensation.
 - Practitioners may be confused about complaints when objective evidence is not present.
 - Follow-up during recovery may aid in preventing symptom persistence.
-

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Replantation

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Synonyms, Key Words, and Related Terms

amputation

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Introduction

Background

In the past 200 years, successful replantation of amputated digits has gradually moved from fantasy to reality. William Balfour performed the first successful fingertip reattachment in 1814; Thomas Hunter is credited with the first thumb replantation performed in the following year.

Little progress was made until the pioneering work of William Steward Halstead and Alexis Carrel, who performed replantation experiments with dog limbs in the 1880s. Dr Carrel won the Nobel Prize in 1912 for his work on vascular anastomoses and for pioneering renal transplantation.

In 1962, Ronald A Malt performed the first successful replantation of an entire limb, in a 12-year-old boy

whose arm had been severed in a train accident. With the development of the operating microscope by Julius Jacobson and Ernesto Suarez in the early 1960s, replantation became easier, and its use began to spread throughout the Western world.

With the advent of microvascular reanastomosis, digit replantation became tenable. In 1965, Shigeo Kmatsu and Susumu Tamai performed the first such procedure. Modern replantation now is available in most large hospitals.

Pathophysiology

Amputation replantation is the reattachment of a completely severed part. This is distinguished from incomplete nonviable amputations, which require revascularization. Revascularization is the reconstruction of the blood supply of an incompletely amputated part.

In general, revascularization usually provides better functional results than replantation itself. Experienced hand surgeons can successfully replant most amputations. However, viability alone is an inadequate measure of success. The goal in replantation is the restoration or reconstruction of a functional limb, not merely the restoration of adequate tissue perfusion.

Degloving injuries are those in which the soft tissue is torn from the underlying bone, as when a glove is removed from the hand. These often are a result of jewelry getting caught in machinery.

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Clinical

History

An adequate history of the injury is important and should include the mechanism, time, and place of injury; condition of the injured part; hand dominance; and general condition of the patient.

- The mechanism of amputation is important; injuries due to sharp mechanisms have a much better chance of successful replantation than those caused by blunt crushing forces.
 - If a narrow zone of crush injury is present, replantation may be possible by excising the crush zone and replanting with clean margins.
 - Avulsion amputations caused by rollers offer a markedly reduced chance of successful, functional replantation, although such repairs are not impossible.
- Determine the patient's dominant hand, although this information is of only relative importance.

- Ask about allergies, immunizations, and chronic active disease processes.
- Ask if any old injury is present. Negative prognostic factors include old age, peripheral vascular disease, congestive heart failure, and diabetes mellitus with complications. In the surgeon's judgment, these factors may make replantation inadvisable.
- Assess the patient's psychiatric history. If the amputation was self-inflicted, replantation may be contraindicated, pending the results of a psychiatric evaluation.

Physical

- Perform a detailed examination of the hand, and describe the injury and neurovascular status.
- Perform a general physical examination, concentrating on cardiovascular disease.
- Perform a rectal examination to ensure that anticoagulation can be accomplished during or after surgery, if necessary, without placing the patient at risk for GI bleeding.

Causes

The 6 mechanisms of amputation injury are the following:

- Sharp cut, as from a knife or meat slicer
- Dull cut, as from a saw or dull edge (eg, fan blade)
- Cut with a narrow segment of crush injury, as from a punch press
- Cut and avulsion, as from a machine that causes partial amputation and subsequent reflexive withdrawal of the hand that completes the amputation
- Avulsion, as from a finger or hand caught in an anchor rope or horse reins
- Crush avulsion, as from a machine (eg, rollers) that crushes the limb then pulls the digits off

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Workup

Lab Studies

- Assess the patient's hemoglobin and/or hematocrit level at baseline, and follow up with serial determinations if significant blood loss is suspected.
- Assess coagulopathy by determining the prothrombin time and platelet count if history suggests a bleeding disorder or liver disease.
- Type and cross-match 2-4 units of packed RBCs if the history suggests significant blood loss.
- Obtain an ECG in patients older than 45 years and in those with a history of cardiac ischemia or

arrhythmia.

- A recent case report suggests that pulse oximetry can be used to document arterial flow to a part that is incompletely amputated when clinical findings of arterial flow with Doppler ultrasonography suggest an absence of arterial perfusion.

Imaging Studies

- Radiographs of the injured part
 - Obtain posteroanterior, lateral, and oblique radiographs of the amputated part and stump.
 - Carefully assess for radio-opaque foreign bodies.
 - Comminution of the fracture implies a crush injury mechanism and is associated with soft-tissue trauma.
 - If the joint is destroyed at the level of amputation, perform arthrodesis (fusion); this results in loss of joint function.
 - If a crush injury is severe, a mosaic of fragments may preclude attempts at replantation.

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Treatment

Prehospital Care

- At the scene, collect and preserve all amputated parts, even those crushed and not thought to be useful. Parts not suitable for replantation can provide tendons or bone.
- Cool the amputated part to 4°C to preserve it; 1 hour of warm ischemia is equal to approximately 6 hours of cold ischemia. Hence, cooling can markedly prolong the window of opportunity for replantation or revascularization.
- Wrap the part in saline-soaked gauze, and place it in a dry plastic bag. Place this bag on ice as soon as possible. This 2-layer approach avoids submersion of the part in ice water, which causes the tissue to freeze and the cells to lyse. Dry ice is too cold and causes tissue freezing and cell destruction.
- Estimate the blood lost at the scene; this information is useful regarding resuscitation prior to surgery. Control bleeding from the amputated stump.

Emergency Department Care

- Uncontrolled arterial bleeding is the only immediately life-threatening complication likely to be encountered in the ED after injury to the upper extremity. Normal hemostasis involves circumferential constriction of affected arteries and their retraction into the amputated stump. The

addition of a pressure dressing usually suffices to control bleeding. With partial arterial lacerations, retraction is prevented, and bleeding control can be more difficult.

- Control hemorrhage in the upper extremities with local direct pressure or a pressure dressing. Use of a proximal tourniquet is acceptable, although not preferred, if direct pressure is not effective. The surgeon can clamp and ligate a bleeding vessel, but this can complicate later repair. In the ED, point control with localized pressure over the bleeding vessel or use of a pressure dressing is preferred.
- Elevate the arm. Ensure that a poorly applied pressure dressing does not become a tourniquet and cause ischemia in the amputated stump. If a tourniquet is used, use it for as briefly as possible, perhaps only during resuscitation for acute hypovolemia. Use of a tourniquet for more than 3 hours may lead to irreversible loss of function. Do not use a tourniquet during an interhospital transfer. A consultant may appropriately use a temporary tourniquet to better identify important structures such as nerves and vessels.
- Blind ligation or clamping of bleeding vessels could lead to greater damage, because neurovascular bundles place ischemia-sensitive nerves near bleeding vessels. Careless clamping also can lead to vessel thrombosis, which requires shortening of a vessel and/or interposition of a vessel graft.
- Do not allow the patient to smoke prior to making the decision to replant or repair the amputation; smoking can cause vasospasm and complicate the procedure.
- For partial amputations, splint the involved extremity to prevent further damage. Reduce any malrotation to limit ischemia. Avoid tension on the tissue bridge, which can damage nerves or vessels. Cooling of a partially amputated part is controversial. If no demonstrable perfusion of the part exists, cool it as if it were completely amputated. If a pulse or bleeding from the capillary bed is present, avoid cooling.
- Bone, tendon, and skin can tolerate approximately 8-12 hours of warm ischemia and as long as 24 hours of cold ischemia. However, muscle necroses after 6 hours of warm ischemia or 12 hours of cold ischemia. Excessive ischemia time reduces muscle function and can result in myoglobinuria on reperfusion, placing renal function at risk. More proximal amputations involving more muscle must, therefore, be treated quickly.
- Amputations are tetanus-prone wounds. Therefore, deoxythymidine 0.5 mL must be administered intramuscularly if the last booster was received more than 5 years earlier. If the patient was not previously immunized or if the immunization status is unclear, administer deoxythymidine and tetanus immune globulin 500 units intramuscularly.
- A digital or regional nerve block is not recommended before a hand or plastic surgeon evaluates the patient because documentation of nerve function prior to surgery is important. Use systemic analgesics with intravascularly administered narcotics.

Consultations

Consult a hand surgeon.

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Medication

Prophylactic antibiotics are indicated with amputation, crush, or degloving injuries. Devitalized tissue is a good culture medium for bacterial contaminants. Common pathogens are *Staphylococcus aureus* (most likely organism) and group A streptococci, while clostridia species and organisms from the Enterobacteriaceae family are less common. Gram-negative and anaerobic bacteria more commonly are found with extensive tissue damage or with wounds grossly contaminated with soil, saliva, or feces. In these cases, perform Gram staining and cultures before initiating antibiotic therapy. If the amputation is from a human bite, treat for streptococci, *Eikenella corrodens*, and anaerobic bacteria, as well as staphylococci should be covered. Use oral amoxicillin and clavulanate for human bites without amputation. Use intravenous ampicillin and sulbactam or ticarcillin and clavulanate for amputations or established infections caused by human bites. A combination of penicillin G and an

antistaphylococcal antibiotic also is acceptable for minor bite wounds.

Antibiotics

Empiric antimicrobial therapy must be comprehensive and should cover all likely pathogens.

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| Drug Name | Cefazolin (Ancef, Kefzol)- First-generation semisynthetic cephalosporin; binds 1 or more penicillin-binding proteins; arrests bacterial cell-wall synthesis and inhibits bacterial growth; primarily active against skin flora, including <i>S aureus</i> . |
| Adult Dose | 250-1000 mg IV/IM q6-8h |
| Pediatric Dose | 25-50 mg/kg/d IV/IM divided tid/qid |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid decreases renal clearance and prolongs effect; concurrent use with aminoglycosides may increase renal toxicity; may yield a false-positive result for glucose with urine dip testing |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in patients with renal impairment |

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| Drug Name | Ampicillin and sulbactam (Unasyn)- Drug combination that involves a beta-lactamase inhibitor with ampicillin; covers skin organisms, enteric flora, and anaerobes; not ideal for nosocomial pathogens. |
| Adult Dose | 1.5 g (1 g ampicillin with 0.5 g sulbactam) to 3 g (2 g ampicillin with 1 g sulbactam) IV/IM q6h; not to exceed 4 g/d sulbactam or 8 g/d ampicillin |

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| Pediatric Dose | 3 months to 12 years: ampicillin 100-200 mg/kg/d (150-300 mg Unasyn) IV divided q6h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid and disulfiram decrease renal excretion of ampicillin and sulbactam and increase levels of the antibiotics; allopurinol increases ampicillin excretion; may potentiate ampicillin rash and decrease the effect of oral contraceptives |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in patients with renal failure; evaluate rash and differentiate from hypersensitivity reaction |

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| Drug Name | Ticarcillin and clavulanic acid (Timentin)- Inhibits biosynthesis of cell wall mucopeptide and is effective during stage of active growth; antipseudomonal penicillin and a beta-lactamase inhibitor covers most gram-positive and gram-negative organisms, as well as anaerobes. |
| Adult Dose | 3.1 g q4-6h IV |
| Pediatric Dose | 100 mg/kg/dose q8h |
| Contraindications | Documented hypersensitivity; do not treat severe pneumonia, bacteremia, pericarditis, emphysema, meningitis, and purulent or septic arthritis with oral penicillin during the acute stage |
| Interactions | Tetracyclines may decrease the effects of ticarcillin; high concentrations of ticarcillin may physically inactivate aminoglycosides if they are administered in same IV line; effects when administered with aminoglycosides are synergistic; probenecid may increase penicillin levels |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Obtain CBCs prior to therapy and at least weekly during therapy; monitor for liver function abnormalities by measuring AST and ALT during therapy; exercise caution in patients with hepatic insufficiencies; perform urinalysis, and determine BUN and creatinine levels during therapy, and adjust dose if values become elevated; monitor blood levels to avoid possible neurotoxic reactions |

Analgesics

Pain control is essential to quality patient care, ensuring patient comfort and promoting pulmonary toilet. Most analgesics have sedating properties, which are beneficial for patients with painful skin lesions.

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| Drug Name | Meperidine (Demerol)- Analgesic with multiple actions similar to those of morphine; may produce less constipation, smooth muscle spasm, and depression of cough reflex than similar analgesic doses of morphine. |
| Adult Dose | 25-50 mg IV q2-3h prn |
| Pediatric Dose | 0.25-0.50 mg/kg q2-3h prn; not to exceed adult dose |
| Contraindications | Documented hypersensitivity; upper airway obstruction; significant respiratory depression; during labor when premature delivery of is anticipated; use of MAOIs |
| Interactions | Monitor for increased respiratory and CNS depression with coadministration of cimetidine; hydantoins may decrease effects; avoid use with protease inhibitors |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients with head injuries, since meperidine may increase respiratory depression and CSF pressure (use only if absolutely necessary); caution with postoperative use and in patients with a history of pulmonary disease (suppresses cough reflex); use of substantially increased doses, due to tolerance, may aggravate or cause seizures even if no prior history of convulsive disorders exists; closely monitor the patient for morphine-induced seizure activity if a prior seizure history exists |

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| Drug Name | Fentanyl (Duragesic)- More potent narcotic analgesic with a much shorter half-life than morphine sulfate; DOC for conscious sedation analgesia; ideal for analgesic action of short duration during anesthesia and in immediate postoperative period. After the initial dose, do not titrate subsequent doses more frequently than q3h or q6h. Pain in most patients is controlled with 72-h dosing intervals; some patients require dosing intervals of 48 h. |
| Adult Dose | 1 mcg (0.001 mg)/kg IV/IM q30min to q2h prn |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; respiratory depression; constipation; nausea; emesis; urinary retention; hypotension; potentially compromised airway that would make it difficult to establish airway control rapidly |
| Interactions | Phenothiazines may antagonize analgesic effects of opiate agonists; tricyclic antidepressants may potentiate adverse effects when both drugs are used concurrently |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hypotension, respiratory depression, constipation, nausea, emesis, or urinary retention; idiosyncratic reaction, known as chest wall rigidity syndrome, may require neuromuscular blockade to increase ventilation |

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|-------------------|---|
| Drug Name | Morphine (Astramorph, MS Contin, Duramorph, Oramorph)- DOC for narcotic analgesia because of its reliable and predictable effects, safety profile, and ease of reversibility with naloxone. Various IV doses are used and are commonly titrated until desired the effect is obtained. |
| Adult Dose | 4-10 mg bolus slow IV; may repeat to maximum of 30 mg for severe pain |
| Pediatric Dose | 0.1-0.2 mg/kg slow IV/IM |
| Contraindications | Documented hypersensitivity; respiratory depression; nausea; emesis; constipation; urinary retention; hypotension; potentially compromised airway that would make it difficult establish airway control rapidly |
| Interactions | Phenothiazines may antagonize analgesic effects of opiate agonists; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in atrial flutter and other supraventricular tachycardias; vagolytic action may increase the ventricular response rate |

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Follow-up

Further Inpatient Care

- Repair may be performed with an axillary nerve block with bupivacaine, which provides anesthesia lasting 12-16 hours. However, children must have general anesthesia, because they do not tolerate axillary block well.
- The surgical sequence for replantation varies slightly with amputations distal and those proximal to the wrist and with the mechanism of injury (clean cut, crush, avulsion). Since injury distal to the wrist is more common, the following surgical sequence is delineated:
 - Surgeons must be skilled at microvascular reanastomosis and be able to achieve a 90% patency rate in a 1-mm-diameter vessel in laboratory animals. Viability of the replanted limb is no longer the sole determinant of success; functional recovery, preoperative and postoperative risks, and duration of treatment are vital factors in making the decision to replant. The duration of treatment, including rehabilitation, should not exceed 2 years; if it does, the replantation is not thought to be worthwhile. Amputation with early fitting of a prosthesis is a viable alternative in these cases.
 - With tourniquet-induced ischemia and use of a microscope, the stump is debrided of all crushed tissue, foreign bodies are removed, and the vessels and nerves are identified and tagged. The amputated part then is similarly debrided, with irrigation of the cut end, while maintaining cooling. Vessels and nerves are identified and tagged.

- If indicated, an intramedullary k wire is inserted into the bone of the amputated part. If needed, the bone is shortened to eliminate tension on repaired arteries, veins, and nerves. Alternatively, a vein graft may be performed to reduce tension on vessels. Bones are fixed with k wires, intramedullary screws or pegs, or small plates with screws.
- The extensor tendon is repaired by using horizontal mattress 4-0 polyester sutures. A tendon graft also may be necessary if a sufficient length of tendon is not available. Finally, if extension is deemed expendable, arthrodesis (joint fusion) may be performed. Then, the flexor tendon is repaired with sutures.
- Arterial repair is performed next. Brisk blood flow from the proximal vessel should be confirmed prior to vascular anastomosis. Restoration of proximal blood flow may require relief of vascular compression, warming of the patient, administration of adequate blood volume, elevation of the patient's blood pressure, irrigation of the proximal part with warmed lactated Ringer solution, intraluminal flushing with papaverine solution, and correction of systemic metabolic acidosis.
- To avoid later thrombosis, reconnect only normal intima visualized under the microscope. A vein graft may be necessary. Tourniquet-induced ischemia may be continued until the anastomosis is complete, although bolus injection of heparin is recommended to prevent thrombosis.
- Ideally, 2 veins should be repaired for each artery. No tension should be present on the vessels. Perform nerve repair next, with fascicular or bundle repair. A nerve graft may be necessary.
- Skin coverage is the final step. Skin grafts or flaps may be required.

Transfer

- The prevalence of severe associated injuries is 0.8%. Prior to considering transfer, ensure that the patient has no life-threatening conditions other than the amputation, if applicable. Transfer is indicated in the following cases:
 - Amputations of thumbs and/or multiple digits
 - Amputations in children
 - Amputations of individual digits distal to the superficialis insertion
 - Complete amputations that might benefit from acute microsurgical reconstruction (eg, revascularization, coverage of free flap)
 - Clean amputations at the palm, wrist, or forearm
- Contraindications to transfer include the following:
 - Significant associated injuries
 - Coexisting medical problems (eg, recent stroke or myocardial infarction) that prohibit surgery
 - Prolonged warm ischemia time (>12 hours), especially with limb amputations
- Avoid use of bulky dressings during transport because these can conceal bleeding. Control bleeding before applying the dressing or before cooling the distal extremity without perfusion.

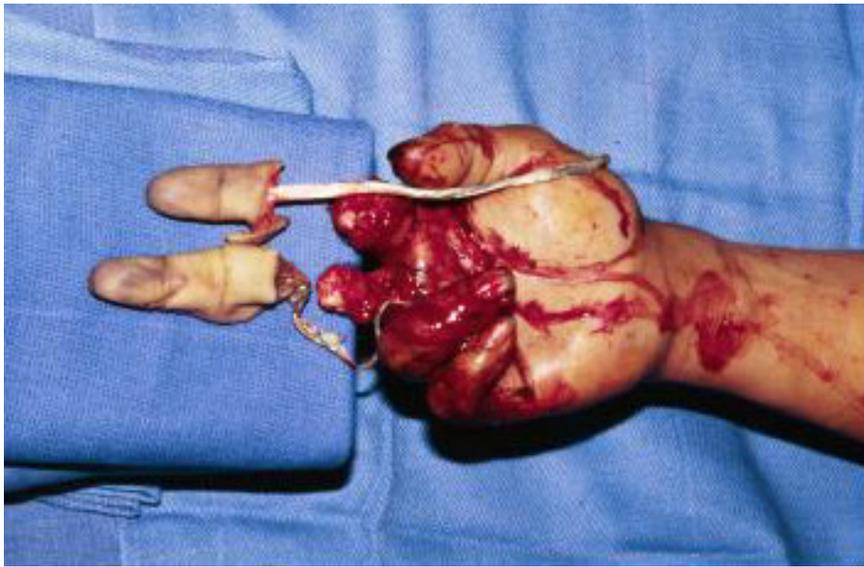
Complications

- Vessel thrombosis may lead to failed replantation.
- Infection may occur.
- Systemically, myonecrosis leading to rhabdomyolysis and renal insufficiency may occur if significant muscle mass that was transiently ischemic is replanted. These occur with forearm or lower leg replantations but not with finger replantations.
- Osteomyelitis may occur.
- Function may be limited after replantation.
- Cold intolerance of the replanted limb is a universal problem. Similarly, cold-induced vasospasm occurs in essentially all patients.
- Sensitivity to light touch and 2-point discrimination frequently is impaired, while limitations in the flexion of joints distal to the replantation vary.
- Cosmetic deformity may occur.

Prognosis

- Success rates as high as 90% have been reported for complete and incomplete amputations.
 - Multivariate analysis of factors that favor functional recovery after finger replantation or revascularization showed better recovery for patients younger than 40 years compared with older patients. Injuries caused by a sharp mechanism have a better prognosis than those caused by a crush mechanism; injuries caused by a crush mechanism have a better prognosis than those caused by avulsion; and injuries at the middle phalangeal level have a better prognosis than those at the proximal level.
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Pictures



Picture 1: Complete amputation of 2 digits

Picture type: Photo



Picture 2: Radiologic appearance of a hand with 2-digit amputation

Picture type: X-RAY



Picture 3: After 2-digit replantation

Picture type: Photo



Picture 4: Complete thumb amputation

Picture type: Photo



Picture 5: Radiologic appearance of a complete thumb amputation
Picture type: X-RAY



Picture 6: Complete thumb amputation
Picture type: Photo



Picture 7: After thumb replantation

Picture type: Photo



Picture 8: Surgical amputation of a left big toe

Picture type: Photo



Picture 9: Toe-to-thumb transfer

Picture type: Photo

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Rhabdomyolysis

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Introduction

Background

Rhabdomyolysis was first described in the victims of crush injury during the 1940-1941 London, England, bombing raids of World War II. It has many etiologies.

Pathophysiology

Rhabdomyolysis is the breakdown of muscle fibers with leakage of potentially toxic cellular contents into the systemic circulation. The final common pathway of rhabdomyolysis may be a disturbance in myocyte calcium homeostasis.

Clinical sequelae of rhabdomyolysis include the following:

- Hypovolemia (sequestration of plasma water within injured myocytes)
- Hyperkalemia (release of cellular potassium into the systemic circulation)
- Metabolic acidosis (release of cellular phosphate and sulfate)
- Acute renal failure (nephrotoxic effects of liberated myocyte components)
- Disseminated intravascular coagulation (DIC)

Frequency

- **In the US:** Rhabdomyolysis accounts for an estimated 8-15% of cases of acute renal failure.

Mortality/Morbidity

The overall mortality rate for patients with rhabdomyolysis is approximately 5%; however, the mortality rate of any single patient is dependent upon the underlying etiology.

Sex

Incidence is higher in males than in females, especially in the subgroups of trauma and inherited enzyme deficiencies.

Age

Rhabdomyolysis may occur in infants, toddlers, and adolescents who have inherited enzyme deficiencies of carbohydrate or lipid metabolism or who have inherited myopathies, such as Duchenne muscular dystrophy and malignant hyperthermia.

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Clinical

History

The clinical presentation is often subtle, underscoring the need for a high index of suspicion.

- In a study of 87 episodes of rhabdomyolysis in 1982, Gabow found that only 50% of patients initially complained of muscle pain. A minority of patients reported dark discoloration of the urine.
- In Gabow's series, 97% of patients reported at least 1 risk factor for rhabdomyolysis. Fifty-nine percent reported multiple risk factors.
 - Common risk factors included alcohol abuse (67%), recent soft tissue compression (39%), and seizure activity (24%).
 - Other causative factors included trauma (17%), drug abuse (15%), metabolic derangements (8%), hypothermia (4%), flulike illness (3%), sepsis (2%), and gangrene (1%).

Physical

- Focal or diffuse skeletal muscle swelling is rare. In Gabow's series, only 5% of the patients presented with muscle edema.
- Tense and tender muscle compartments suggest compartment syndrome; peripheral pulses that are within reference range do not rule out compartment syndrome because loss of distal pulses is a very late sign.

Causes

The etiologies may be subdivided into traumatic, exercise induced, toxicologic, environmental, metabolic, infectious, immunologic, and inherited classifications.

- Rhabdomyolysis may occur after traumatic events, including the following:
 - Significant blunt trauma
 - High voltage electrical injury
 - Extensive burns
 - Near drowning
 - Prolonged immobilization (eg, after excess alcohol or drug consumption, after an unwitnessed incapacitating stroke, following prolonged surgical procedures)
- Toxin-mediated rhabdomyolysis may result from substance abuse, including abuse of the following:
 - Ethanol
 - Methanol
 - Ethylene glycol
 - Isopropanol
 - Heroin
 - Methadone
 - Barbiturates
 - Cocaine
 - Amphetamine
 - Phencyclidine
 - 3, 4-methylenedioxymethamphetamine (MDMA, ecstasy)
 - Lysergic acid diethylamide (LSD)
- Rhabdomyolysis may be caused by other toxins, including the following:
 - Carbon monoxide
 - Toluene
 - Hemlock herbs from quail (Rhabdomyolysis after the consumption of quail is well-known in the Mediterranean region; it occurs as the result of intoxication by hemlock herbs that the quails consume.)
 - Snake, spider (eg, black widow spider), and massive bee envenomations
- Metabolic causes of rhabdomyolysis include the following:

- Hyponatremia or hypernatremia
- Hypokalemia
- Hypophosphatemia
- Hypothyroidism or hyperthyroidism
- Diabetic ketoacidosis
- Nonketotic hyperosmolar diabetic coma
- Bacterial infectious agents may cause rhabdomyolysis, including the following:
 - *Francisella tularensis*
 - *Streptococcus pneumoniae*
 - Group B streptococci
 - *Streptococcus pyogenes*
 - *Staphylococcus epidermidis*
 - *Escherichia coli*
 - *Borrelia burgdorferi*
 - *Clostridium perfringens*
 - *Clostridium tetani*
 - Viridans streptococci
 - *Plasmodium* species
 - *Rickettsia* species
 - *Salmonella* species
 - *Listeria* species
 - *Legionella* species
 - *Mycoplasma* species
 - *Vibrio* species
 - *Brucella* species
 - *Bacillus* species
 - *Leptospira* species
- Causative connective tissue diseases that can cause rhabdomyolysis include the following:
 - Polymyositis
 - Dermatomyositis
- Rhabdomyolysis also has been reported in patients with sickle cell anemia and has mistakenly been identified as a pain crisis

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Differentials

Anemia, Sickle Cell

Burns, Thermal

Toxicity, Theophylline

Toxicity, Toluene
Compartment Syndrome, Extremity
Diabetic Ketoacidosis
Disseminated Intravascular Coagulation
Electrical Injuries
HIV Infection and AIDS
Heat Exhaustion and Heat Stroke
Hypernatremia
Hyperosmolar Hyperglycemic Nonketotic Coma
Hypokalemia
Hyponatremia
Hypophosphatemia
Hypothermia
Hypothyroidism and Myxedema Coma
Myopathies
Neuroleptic Malignant Syndrome
Pediatrics, Inborn Errors of Metabolism
Plant Poisoning, Hemlock
Polymyositis
Renal Failure, Acute
Snake Envenomations, Rattle
Spider Envenomations, Widow
Toxicity, Alcohols
Toxicity, Amphetamine
Toxicity, Barbiturate
Toxicity, Carbon Monoxide
Toxicity, Cocaine
Toxicity, Cyclic Antidepressants
Toxicity, Ethylene Glycol
Toxicity, Hallucinogen
Toxicity, MDMA
Toxicity, Medication-Induced Dystonic Reactions
Toxicity, Methamphetamine
Toxicity, Neuroleptic Agents
Toxicity, Phencyclidine
Toxicity, Salicylate

Other Problems to be Considered

Serotonin syndrome

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Workup

Lab Studies

- Preliminary diagnosis of rhabdomyolysis requires a high index of suspicion. Definitive diagnosis is made by laboratory evaluation.
- The most useful measurement is for serum creatine kinase (CK). This assay is widely available and 100% sensitive.
 - Total CK elevation is a sensitive but nonspecific marker for rhabdomyolysis. Suspect early rhabdomyolysis in patients with serum CK levels in excess of 2-3 times the reference range and risk factors for rhabdomyolysis; initiate a full laboratory workup.
 - Patients with other disorders, such as acute myocardial infarction and acute stroke, may have high CK levels. CK levels have a wide range of distribution among patients with rhabdomyolysis (several hundred to hundreds of thousands of units per liter).
 - Serum CK levels peak within 24 hours and should decrease by approximately 40% per day after the initial insult. Persistent elevation suggests continuing muscle injury or development of a compartment syndrome.
- Aldolase, lactate dehydrogenase (LDH), and serum glutamic-oxaloacetic transaminase (SGOT) are nonspecific enzyme markers that are elevated in patients with rhabdomyolysis.
- Hyperkalemia, an immediate threat to life in the hours immediately after injury, occurs in 10-40% of cases.
 - Liberated potassium can cause life-threatening dysrhythmias and death.
 - Measure and closely monitor serum levels.
- Hyperphosphatemia does not require specific therapy.
- Hypocalcemia occurs early in the course of rhabdomyolysis. Supplemental calcium is not recommended.
- Increased purine metabolism causes hyperuricemia. Specific therapy with uricosuric agents or allopurinol is not indicated.
- Obtain the prothrombin time, activated partial thromboplastin time, and platelet count in all patients with rhabdomyolysis. Thromboplastin released from injured myocytes can cause DIC.

Imaging Studies

- Imaging studies generally play no role in the initial diagnosis of rhabdomyolysis.
- Magnetic resonance imaging (MRI) may be useful in distinguishing various etiologies of rhabdomyolysis.
 - One study suggests that bacterial myositis, focal myositis, and idiopathic rhabdomyolysis show a characteristic gadolinium enhancement on MRI. Abscesses were found only in bacterial myositis.
 - Polymyositis and dermatomyositis have a characteristic uniform distribution pattern with

emphasis on the quadriceps muscles.

Other Tests

- Obtain an electrocardiogram (ECG) early in the course of ED evaluation.
- ECG may reveal changes of acute hyperkalemia, including peaked T waves, prolongation of the PR and QRS intervals, and loss of the P wave or the sine wave.

Procedures

- Measure the compartment pressures in any patient with severe focal muscle tenderness and a firm muscle compartment.
- Perform a fasciotomy if compartment pressures are sustained in excess of 25-30 mm Hg.

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Treatment

Prehospital Care

Vigorous hydration with isotonic crystalloid is the cornerstone of therapy for rhabdomyolysis. Retrospective studies of patients with severe crush injuries resulting in rhabdomyolysis suggest that prognosis is better when prehospital personnel provide fluid resuscitation. Support of the intravascular volume increases the glomerular filtration rate (GFR) and oxygen delivery and dilutes myoglobin and other renal tubular toxins.

- Immediately obtain intravenous access with a large bore catheter.
- Administer isotonic crystalloid 500 cc/h and then titrate to maintain a urine output of 200-300 cc/h.
- Because injured myocytes can sequester large volumes of extracellular fluid, crystalloid requirements may be surprisingly large.

Emergency Department Care

Assess ABCs and support as needed. Treat any underlying conditions, such as trauma, infection, or toxins. General recommendations for the treatment of rhabdomyolysis include fluid resuscitation and prevention of end-organ complications.

- Patients with CK elevation in excess of 2-3 times the reference range, appropriate clinical history, and risk factors should be suspected of having rhabdomyolysis. Administer isotonic crystalloid 500 cc/h and titrate to maintain a urine output of 200-300 cc/h. Consider central venous pressures or Swan-Ganz catheterization in patients with cardiac or renal disease. These invasive studies can assist in the assessment of the intravascular volume.
- Acute renal failure develops in 30-40% of patients with rhabdomyolysis. Suggested mechanisms include precipitation of myoglobin and uric acid crystals within renal tubules, decreased glomerular perfusion, and the nephrotoxic effect of ferrihemate (formed upon dissociation of myoglobin in the acidic environment of the renal parenchyma). In a 1988 review, Ward suggested that predictors for the development of renal failure include peak CK more than 6000 IU/L, dehydration (hematocrit >50, serum sodium >150 mEq/L, orthostasis, pulmonary wedge pressure <5 mm Hg, urinary fractional excretion of sodium <1%), sepsis, hyperkalemia or hyperphosphatemia on admission, and the presence of hypoalbuminemia. Acute renal failure has occasionally developed in patients with peak CK as low as 2000 IU/L. To prevent renal failure, many authorities advocate urine alkalinization, mannitol, and loop diuretics.
 - Urinary alkalinization is recommended for patients with rhabdomyolysis and CK levels in excess of 1000 IU/L. A suggested regimen is 0.5 isotonic sodium chloride solution with one ampule of sodium bicarbonate administered at 100 cc/h and titrated to a urine pH higher than 7.
 - After establishing an adequate intravascular volume, mannitol may be administered to enhance renal perfusion.
 - Loop diuretics may be used to enhance urinary output in oliguric patients, despite adequate intravascular volume.
- Hypocalcemia is noted early in the course of rhabdomyolysis and generally is not of clinical significance. Calcium supplementation is not recommended.
- Compartment syndrome requires immediate orthopedic consultation for fasciotomy.
- DIC should be treated with fresh frozen plasma, cryoprecipitate, and platelet transfusions.
- Hyperuricemia and hyperphosphatemia rarely are of clinical significance and rarely require treatment.

Consultations

Indications for hemodialysis include hyperkalemia that is persistent despite therapy, severe acid-base disturbances, refractory pulmonary edema, and progressive renal failure.

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Medication

Medical therapy focuses on restoring adequate intravascular volume using isotonic crystalloid.

Adjunctive measures that may decrease the incidence of acute myoglobinuric renal failure include urinary alkalinization and osmotic and loop diuresis.

Alkalinizing Agents

Sodium bicarbonate is administered IV to alkalinize urine in patients with rhabdomyolysis. This may prevent toxicity caused by presence of myoglobin in acidic urine and crystallization of uric acid.

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| Drug Name | Sodium bicarbonate (Neut)- Useful in alkalization of urine to prevent acute myoglobinuric renal failure. Titrate dose to increase pH to >7. |
| Adult Dose | 1 ampule (44 mEq) of sodium bicarbonate is added to 1 L of 0.45 NS and infused at 100 cc/h IV |
| Pediatric Dose | 1.9 mEq/kg IV q1-2h prn |
| Contraindications | Alkalosis, hypernatremia, hypocalcemia, severe pulmonary edema, and unknown abdominal pain |
| Interactions | Urinary alkalinization, induced by increased sodium bicarbonate concentrations, may cause decreased levels of lithium, tetracyclines, chlorpropamide, methotrexate, and salicylates; Increases levels of amphetamines pseudoephedrine, flecainide, anorexiant, mecamylamine, ephedrine, quinidine, and quinine |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Only use to treat documented metabolic acidosis and hyperkalemia-induced cardiac arrest; can cause alkalosis, decreased plasma potassium, hypocalcemia, and hypernatremia; caution in electrolyte imbalances, such as in patients with CHF, cirrhosis, edema, corticosteroid use, or renal failure; when administering, avoid extravasation because can cause tissue necrosis |

Osmotic Diuretics

Increase osmolarity of glomerular filtrate and induce diuresis. Hinders tubular reabsorption of water, causing sodium and chloride excretion to increase.

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|-----------|---|
| Drug Name | Mannitol (Osmitol)- Alternative diuretic used when urine output is inadequate despite aggressive fluid therapy. Initially assess for adequate renal function in adults by administering a test dose of 200 mg/kg IV over 3-5 min. Should produce a urine flow of at least 30-50 mL/h over 2-3 h. In children, assess for adequate renal function by administering a test dose of 200 mg/kg IV over 3-5 min. It should produce a urine flow of at least 1 mL/h over 1-3 h. |
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| Adult Dose | 1.5-2 g/kg IV as 20% solution (7.5-10 mL/kg) or as 15% solution (10-13 mL/kg) over a period as short as 30 min |
| Pediatric Dose | 0.5-1 g/kg IV initial; followed by 0.25-0.5 g/kg IV q4-6h maintenance dose |
| Contraindications | Documented hypersensitivity; anuria, severe pulmonary congestion, progressive renal damage, severe dehydration, active intracranial bleeding, and progressive heart failure |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Carefully evaluate cardiovascular status before rapid administration of mannitol because a sudden increase in extracellular fluid may lead to fulminating CHF; avoid pseudoagglutination, when blood given simultaneously, add at least 20 mEq of sodium chloride to each liter of mannitol solution; do not give electrolyte-free mannitol solutions with blood |

Loop Diuretics

Elicit a loss of free water, increasing diuresis.

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| Drug Name | Furosemide (Lasix)- Increases water excretion by interfering with the chloride-binding cotransport system, resulting in inhibition of sodium and chloride reabsorption in the ascending loop of Henle and distal renal tubule. Individualize doses. Depending on the response, administer at increments of 20-40 mg q6-8h until desired diuresis occurs. When treating infants, titrate with 1-mg/kg/dose increments until a satisfactory effect is achieved. |
| Adult Dose | 20-40 mg IV q2h prn to maintain urine output; may increase dose by 20 mg q2h prn to desired response |
| Pediatric Dose | 1-2 mg/kg IV q6h; titrate to desired urine output; not to exceed 6 mg/kg/d |
| Contraindications | Documented hypersensitivity; hepatic coma, anuria, and state of severe electrolyte depletion |
| Interactions | Metformin decreases concentrations; interferes with hypoglycemic effect of antidiabetic agents and antagonizes muscle relaxing effect of tubocurarine; auditory toxicity appears to be increased with coadministration of aminoglycosides and furosemide; hearing loss of varying degrees may occur; anticoagulant activity of warfarin may be enhanced when taken concurrently with this medication; increased plasma lithium levels and toxicity are possible when taken concurrently with this medication |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

Precautions

Perform frequent serum electrolyte, carbon dioxide, glucose, creatinine, uric acid, calcium, and BUN determinations during first few months of therapy and periodically thereafter

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Follow-up

Further Inpatient Care

- Admit patients with rhabdomyolysis for continued volume support and urinary alkalinization. Obtain serial CK measurements to verify that values have peaked and are returning to reference range.
- Serial physical examinations and laboratory studies are indicated to monitor for compartment syndrome, hyperkalemia, acute oliguric or nonoliguric renal failure, and DIC.
- In patients with no apparent precipitating factors for rhabdomyolysis, consider inherited disorders of carbohydrate or lipid metabolism and myopathies.

Transfer

- Patients may be transferred to another facility after establishing intravenous access and addressing life- and limb-threatening conditions. Follow guidelines of the Consolidated Omnibus Budget Reconciliation Act (COBRA) and the Emergency Medical Treatment and Labor Act (EMTALA).

Complications

- Death from hyperkalemia or renal failure
- Compartment syndrome
- DIC
- Hepatic insufficiency
- Acute renal failure

Prognosis

- Prognosis is dependent upon the underlying etiology.

Patient Education

- Advise patients with rhabdomyolysis caused by hyperthermia and/or inordinate exertion to exercise in moderation with careful attention to hydration and external methods of cooling.
- Advise patients with rhabdomyolysis related to ethanol, recreational drugs, or prescription medications to discontinue use of the offending agent and refer them to a rehabilitation program.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to recognize rhabdomyolysis on initial presentation
 - Failure to institute early and aggressive volume replacement
 - Insufficient monitoring for serious complications, such as compartment syndrome and severe hyperkalemia
-

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Rotator Cuff Injuries

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Introduction

Background

Rotator cuff injuries are problems commonly encountered in athletic and nonathletic patients. Symptoms include pain, weakness, and decreased range of motion. Early diagnosis is important for identifying causes, implementing effective treatment, and preventing further injury.

Pathophysiology

Knowledge of the mechanical and normal anatomic structure allows for understanding of rotator cuff injuries. The rotator cuff muscles are the supraspinatus, infraspinatus, subscapularis, and teres minor.

The subscapularis is a humeral head depressor and, in certain positions, an internal rotator. The infraspinatus and teres minor are external rotators. These muscles work as a unit, rather than individually, to maintain the dynamic glenohumeral stability. All are innervated by subscapular and axillary nerves. The vascular supply largely is dependent upon the anterior humeral circumflex artery, which supplies the anterior cuff, and the posterior humeral circumflex and suprahumeral, which supply the posterior cuff.

Microscopically, all of the tendons of the rotator cuff fuse to form one continuous band, which is composed of a 5-layer structure. Because of this structure, none of the individual muscles have a higher incidence of tear, per se. However, the joint-side portion of the supraspinatus tendon is more susceptible to mechanical failure than the bursal side.

Most of the tears of the cuff are the end result of chronic degeneration, which makes them susceptible to rupture. The chronic deterioration of the cuff results from the coracoacromial arch, which is composed of the bony acromion, the coracoacromial ligament, and coracoid process. Because of its position above the rotator cuff, the coracoacromial arch forms the roof through which the supraspinatus tendon must pass (ie, supraspinatus outlet). Repetitive microtrauma and anatomical variations lead to most of the rotator cuff injuries.

Tendon degeneration is classified in 3 stages (classification of the impingement syndrome) based on the supraspinatus outlet.

- Stage I - Edema and hemorrhage, affecting persons younger than 25 years
- Stage II - Fibrosis and tendinitis, affecting persons aged 25-40 years
- Stage III - Tears of cuff, affecting persons older than 50 years

Frequency

- **In the US:** Precise incidence of symptomatic rotator cuff injuries is not known. Many individuals with full thickness cuff tears are not only asymptomatic, but they have minimal functional disability. The most accepted figure is 20-30%. Cadaver studies of elderly persons have estimated full thickness tears as high as 30%.

Mortality/Morbidity

An estimated 4% of cuff ruptures develop a cuff arthropathy. Various authors report a rate of success with conservative treatment ranging from 33-90%, with longer recovery time in older patients. Surgery results in improved function regardless of the patient's age.

Age

Rotator cuff injuries and tears usually do not occur in persons younger than 40 years (5-30%). The great majority is found in patients aged 55-85 years. Approximately 15% of patients with shoulder pain who are older than 70 years have rotator cuff injuries.

- Younger patients are more likely to have rotator cuff dysfunction because of overuse, subtle instability, and muscle imbalance.
- Older patients tend to have chronic shoulder pain and degeneration.

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Clinical

History

Assess any patient with shoulder pain with respect to the patient's age and occupation. Characterize pain according to its duration of onset, location, radiation, timing, and quality. In addition, investigate pain for its relationship to activities and sport.

- Pain is the most common symptom encountered with rotator cuff injury.
 - Pain usually is located anterolaterally and superiorly and referred to the level of the deltoid insertion with full thickness tears.
 - Pain is aggravated in activities where the arm must be in an overhead or in a forward-flexed position.
 - In an acute injury, pain suddenly is elicited after a fall, after lifting of a heavy object, or even after a trivial amount of force.
- The patient also may complain of clicking, catching, stiffness, and crepitus.

Physical

Approach the shoulder examination systematically in every patient with inspection, palpation, range of motion, strength testing, neurologic assessment, and performances of special shoulder tests. Also, include evaluation of the cervical spine and upper extremity.

- Inspect for scars, color, edema, deformities, muscle atrophy, and asymmetry.
- Palpate the bony and soft-tissue structures, noting any areas of tenderness.
 - The subdeltoid and subacromial bursae can be palpated anteriorly under the acromion, and laterally with the deltoid muscle and the arm in extension.
 - The supraspinatus is palpated anteriorly when the arm is externally rotated and flexed.
 - Hyperextension permits the palpation of the infraspinatus.
- Neer impingement test: An injection of 1% lidocaine into the subacromial bursae, using the lateral or posterior approach, creates signs of relief on forward flexion in patients with rotator cuff disease, distinguishing cuff disease from other sources of shoulder pain. However, rotator cuff tears are not distinguished from early stages of inflammation or fibrosis.

Causes

An emerging consensus suggests that the etiology of rotator cuff disease is multifactorial. Extrinsic factors exist, such as the morphology of the coracoacromial arch, tensile overload, repetitive use, and kinematics abnormalities. Intrinsic factors also exist, such as altered tendon vascular supply, microstructural collagen fiber abnormalities, and regional variations.

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Differentials

Cervical Strain
Dislocations, Shoulder
Myocardial Infarction
Myopathies

Other Problems to be Considered

Avascular necrosis of humerus

Acromioclavicular injury

Neurological injuries (C5-C6) caused by repetitive trauma

Septic arthritis

Spleen rupture

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Workup

Imaging Studies

- Routine radiographic evaluations are an essential component of shoulder evaluation in the ED. Perform a routine x-ray examination in every patient with suspected rotator cuff injury. Shoulder x-rays should include anteroposterior, axillary, and lateral views.
- A modified transscapular or suprascapular outlet view is useful for surgical purposes. Radiographic changes are as follows:

- Subacromial sclerosis (ie, "eyebrow" sign)
- Osteophyte formation
- Sclerosis and cystic changes in the greater tuberosity
- Reduction of the acromiohumeral distance (<7 mm).

(Only the last 2 points were found to have 78% sensitivity and 98% specificity.)

- Reserve advanced imaging modalities for suspected rotator tears with no improvement in symptoms, despite adequate therapy for 3-6 weeks.
 - Arthrography of glenohumeral joint has been used to diagnose rotator cuff disease. A complete tear is diagnosed when communication between the glenohumeral joint cavity and the bursae, either subacromial or subdeltoid, is evident. Partial tears are better evaluated with ultrasound or MRI.
 - Ultrasonography also is used to evaluate rotator cuff disease. The 4 criteria for rotator cuff pathology are nonvisualization of the cuff, localized absence or focal nonvisualization, discontinuity, and focal abnormal echogenicity. Sensitivity and specificity are operator dependent and have been reported to be greater than 90%.
 - A magnetic resonance imaging (MRI) can reveal a great spectrum of rotator cuff disease from degeneration to partial or complete tears. MRI also can reveal soft tissue injuries. As a postoperative imaging modality, it has proven to be invaluable.

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Treatment

Prehospital Care

Stabilization with a shoulder sling and an ice pack are sufficient for prehospital care.

Emergency Department Care

- Conservatively treat patients with chronic injuries that have progressed to a rotator cuff tear.
- The goals are to reduce inflammation, relieve stress on the rotator cuff, and correct any biomechanical dysfunction.
- Nonoperative therapy consists of rest and activity modification, shoulder sling, nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroid injections, and a basic shoulder-strengthening program.
- Various studies indicate a heterogeneous success rate ranging from 33-90%. Steroid injections

reduced pain, but no difference in improvement of function was observed.

- Schedule follow-up treatment as soon as possible; if the patient has not improved by a 6-week assessment, consider surgical therapy.
- Surgical therapy is indicated in patients younger than 60 years with a full-thickness tear demonstrated clinically or arthrographically, in patients who fail to improve after 6 weeks of rehabilitation, or in patients performing activity that requires shoulder use.
 - Emergent orthopedic evaluation is warranted in acute injuries or even severe extension of chronic rotator cuff injuries because they have poor prognosis if conservative modalities are used.
 - The success rate of surgical therapy is reported to be 77-86%.
 - Findings generally suggest that early treatment precipitates a better outcome than late treatment.
 - Many studies have concluded that the need for surgery should consider not only age but also type of tear, duration of symptoms, and the patient's ability to comply with the rehabilitation regimen.

Consultations

Consider an orthopedic consultation in primarily acute injuries or even severe extension of chronic rotator cuff injuries. An orthopedic consultation for possible surgical intervention is required under the following conditions:

- In patients younger than 60 years
- For full-thickness tear demonstrated clinically or arthrographically
- For failure to improve after 6 weeks of rehabilitation
- If the patient's employment requires shoulder use

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Medication

The goal of pharmacotherapy is to reduce pain and inflammation.

Analgesics

Pain control is essential to quality patient care, ensuring patient comfort, promoting pulmonary toilet, and enabling physical therapy regimens. Most analgesics have sedating properties, which are beneficial for patients who have sustained painful skin lesions.

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Ibuprin, Advil, Motrin)- Usually the DOC for the treatment of mild to moderate pain if no contraindications exist. Inhibits inflammatory reactions and pain by decreasing prostaglandin synthesis. |
| Adult Dose | 400 mg PO q4-6h, 600 mg PO q6h, or 800 mg PO q8h while symptoms persist; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 10-70 mg/kg/d PO divided tid/qid; begin at lower end of the dosing range and titrate upward; not to exceed 2.4 g/d >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease, recent GI bleeding or perforation, renal insufficiency, or high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Ketoprofen (Orudis, Actron, Oruvail)- For relief of mild to moderate pain and inflammation. Small dosages initially are indicated in small and elderly patients and in persons with renal or liver disease. Doses more than 75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe patients for response. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | 3 months to 14 years: 0.1-1 PO mg/kg q6-8h >14 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; GI disease |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |

| | |
|-------------|---|
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |
|-------------|---|

| | |
|-------------------|--|
| Drug Name | Acetaminophen (Tylenol, Aspirin-free Anacin, Acephen)- DOC for the treatment of pain in patients with documented hypersensitivity to aspirin or NSAIDs, persons with upper GI disease, or those taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg PO tid/qid; not to exceed 4 g/d; Alternately, 1000 mg PO tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg PO q4h; not to exceed 5 dose/d |
| Contraindications | Documented hypersensitivity; G-6-PD deficiency |
| Interactions | Rifampin can reduce analgesic effects; coadministration with barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholism following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; APAP is contained in many OTC products, and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose |

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Follow-up

Further Inpatient Care

- Admit to orthopedic service in preparation for the operating room (only required if surgery is the treatment of choice).

Further Outpatient Care

- Arrange outpatient follow-up care within 1-2 days to an orthopedic surgeon and rehabilitation services to continue conservative therapy.
- A follow-up reassessment examination 6 weeks after beginning conservative therapy is essential to determine if treatment is successful or if further surgical treatment is needed.

in/Out Patient Meds

- All NSAIDs are equally effective.

Deterrence/Prevention

- Instruct patient to limit activities to ensure rest of the affected shoulder.

Complications

- Failure of conservative treatment requires surgical intervention.
- Decreased range of motion may occur.

Prognosis

- An estimated 4% of cuff ruptures develop a cuff arthropathy.
- Various authors report the success rate of conservative treatment to be 33-90%, with longer recovery time required in older patients.
- Surgery results in better function regardless of the patient's age.

Patient Education

- Refer patients to a physical therapist in conservative treatment and postoperatively.

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Miscellaneous

Medical/Legal Pitfalls

- Proper follow-up and care ensure prevention of rotator cuff arthropathies and long-term sequelae.
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Shock, Hemorrhagic

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Synonyms, Key Words, and Related Terms

blood loss, hemorrhage, shocklike state

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Introduction

Background

Shock is a state in which adequate perfusion to sustain the physiologic needs of organ tissues is not present. Many conditions, including sepsis, blood loss, impaired autoregulation, and loss of autonomic tone, may produce shock or shocklike states.

Pathophysiology

In hemorrhagic shock, blood loss exceeds the body's ability to compensate and provide adequate tissue perfusion and oxygenation. This frequently is due to trauma, but it may be caused by spontaneous hemorrhage (eg, GI bleeding, childbirth), surgery, and other causes.

Most frequently, clinical hemorrhagic shock is caused by an acute bleeding episode with a discrete precipitating event. Less commonly, hemorrhagic shock may be seen in chronic conditions with subacute blood loss.

Physiologic compensation mechanisms for hemorrhage include initial peripheral and mesenteric vasoconstriction to shunt blood to the central circulation. This is then augmented by a progressive tachycardia. Invasive monitoring may reveal an increased cardiac index, increased oxygen delivery (ie, DO_2), and increased oxygen consumption (ie, VO_2) by tissues. Lactate levels, the acid-base status, and other markers also may provide useful indicators of physiologic status. Age, medications, and comorbid factors all may affect a patient's response to hemorrhagic shock.

Failure of compensatory mechanisms in hemorrhagic shock can lead to death. Without intervention, a classic trimodal distribution of deaths is seen in severe hemorrhagic shock. An initial peak of mortality occurs within minutes of hemorrhage due to immediate exsanguination. Another peak occurs after 1 to several hours due to progressive decompensation. A third peak occurs days to weeks later due to sepsis and organ failure.

Frequency

- **In the US:** Accidental injuries are the leading cause of death in individuals aged 1-44 years.

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Clinical

History

History taking should address the following:

- Specific details of the mechanism of trauma or other cause of hemorrhage are essential.
- Inquire about a history of bleeding disorders and surgery.
- Prehospital interventions, especially the administration of fluids administered, and changes in vital signs should be determined. Emergency medical technicians or paramedics should share this information.

Physical

Findings at physical examination may include the following:

- Head, ears, eyes, nose, and throat
 - Sources of hemorrhage usually are apparent.
 - The blood supply of the scalp is rich and can produce significant hemorrhage.
 - Intracranial hemorrhage usually is insufficient to produce shock, except possibly in very young individuals.
- Abdomen
 - Injuries to the liver or spleen are common causes of hemorrhagic shock.
 - Blood irritates to the peritoneal cavity; diffuse tenderness and peritonitis are common when blood is present. However, the patient with altered mental status or multiple concomitant injuries may not have the classic signs and symptoms at physical examination.
 - Progressive abdominal distention in hemorrhagic shock is highly suggestive of intraabdominal hemorrhage.
- Extremities
 - Hemorrhage from extremity injuries may be apparent, or tissues may obscure significant bleeding.
 - Femoral fractures may produce significant blood loss.

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Differentials

Abdominal Trauma, Blunt

Abdominal Trauma, Penetrating

Anemia, Acute

Anemia, Chronic

Aneurysms, Abdominal

Blast Injuries

Disseminated Intravascular Coagulation

Pneumothorax, Tension and Traumatic

Pregnancy, Ectopic

Pregnancy, Postpartum Hemorrhage

Pregnancy, Trauma

Shock, Cardiogenic

Shock, Hypovolemic

Shock, Septic

Spinal Cord Injuries

Other Problems to be Considered

Abortion complications

Cardiac tamponade

Knife wounds

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Workup

Lab Studies

- Laboratory studies are essential in management of many forms of hemorrhagic shock. Baseline levels are determined frequently, but these infrequently change the initial management after trauma. Serial evaluations of the following can help guide ongoing therapy.
 - CBC
 - Prothrombin time and/or activated partial thromboplastin time
 - Urine
 - ABGs (Levels reflect acid-base and perfusion status.)
- Typed and cross-match packed red blood cells should be obtained immediately.
- Fresh frozen plasma and platelets also may be required to correct coagulopathies that develop in severe hemorrhagic shock.

Imaging Studies

- Standard radiography
 - Cervical spine, chest, and pelvis radiographs are the standard screening images for severe trauma.
 - Other radiographs may be indicated for orthopedic injuries.
- Ultrasonography
 - Bedside abdominal ultrasonography can be useful for the rapid detection of free intra-abdominal fluid and, sometimes, specific parenchymal injury.
 - Thoracic ultrasonographic findings can immediately confirm hemothorax or pericardial tamponade.

Other Tests

- An ECG can be useful for detecting dysrhythmias and cardiac sequelae of shock.

Procedures

- Tube thoracostomy is necessary in hemothorax and hemothorax with or without pneumothorax.
- Central venous access facilitates fluid resuscitation and monitoring of central venous pressure and is necessary if peripheral intravenous access is inadequate or impossible to obtain.
- Diagnostic peritoneal lavage is used to detect intra-abdominal blood, fluid, and intestinal contents. It is sensitive but not specific for abdominal injury. It is not used to evaluate the retroperitoneum, which can hold significant hemorrhage, and does not identify the source of hemorrhage.

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Treatment

Prehospital Care

- The standard care consists of rapid assessment and expeditious transport to an appropriate center for evaluation and definitive care.
- Intravenous access and fluid resuscitation are standard. However, this practice has become controversial.
 - For many years, aggressive fluid administration has been advocated to normalize hypotension associated with severe hemorrhagic shock. Recent studies of urban patients with penetrating trauma have shown that mortality increases with these interventions; these findings call these practices into question.
 - Reversal of hypotension prior to the achievement of hemostasis may increase hemorrhage, dislodge partially formed clots, and dilute existing clotting factors. Findings from animal studies of uncontrolled hemorrhage support these postulates. These provocative results raise the possibility that moderate hypotension may be physiologically protective and should be permitted, if present, until hemorrhage is controlled.
 - These findings should not yet be clinically extrapolated to other settings or etiologies of hemorrhage. The ramifications of permissive hypotension in humans remain speculative, and safety limits have not been established yet.

Emergency Department Care

- Management of hemorrhagic shock should be directed toward optimizing perfusion of and oxygen delivery to vital organs.
- Diagnosis and treatment of the underlying hemorrhage must be performed rapidly and concurrently with management of shock.

- Supportive therapy, including oxygen administration, monitoring, and establishment of intravenous access (eg, 2 large-bore catheters in peripheral lines, central venous access) should be initiated.
 - Intravascular volume and oxygen-carrying capacity should be optimized.
 - In addition to crystalloids, some colloid solutions, hypertonic solutions, and oxygen-carrying solutions (eg, hemoglobin-based and perfluorocarbon emulsions) are used or being investigated for use in hemorrhagic shock.
 - Blood products may be required.
- Control of hemorrhage may be achieved in the ED, or control may require consultations and special interventions.

Consultations

Consult a general or specialized surgeon, gastroenterologist, obstetrician-gynecologist, radiologist, and others as required

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Medication

Achievement of hemostasis, fluid resuscitation, and use of blood products are the mainstays of treatment. Pressor agents may be useful in some settings (eg, spinal shock), but these agents should not be substitutes for adequate volume resuscitation.

Vasopressors

These agents augment both coronary and cerebral blood flow during the low-flow state associated with shock.

| | |
|----------------|---|
| Drug Name | Dopamine (Intropin)- Stimulates both adrenergic and dopaminergic receptors. Hemodynamic effect is dependent on the dose. Lower doses predominantly stimulate dopaminergic receptors that in turn produce renal and mesenteric vasodilation. Higher doses produce cardiac stimulation and renal vasodilation |
| Adult Dose | 1-5 mcg/kg/min IV; not to exceed 50 mcg/kg/min IV; after initiating therapy, increase dose by 1-4 mcg/kg/min IV q10-30min until optimal response is obtained; in more than 50% of patients, satisfactorily maintenance is achieved with doses <20 mcg/kg/min IV |
| Pediatric Dose | Administer as in adults |

| | |
|-------------------|--|
| Contraindications | Documented hypersensitivity; pheochromocytoma; ventricular fibrillation |
| Interactions | Phenytoin, alpha-adrenergic and beta-adrenergic blockers, general anesthesia, and MAOIs increase and prolong effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Closely monitor urine flow, cardiac output, pulmonary wedge pressure, and BP during infusion; prior to infusion, correct hypovolemia with whole blood or plasma, as indicated; monitoring of central venous pressure or left ventricular filling pressure may be helpful in detecting and treating hypovolemia |

| | |
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| Drug Name | Norepinephrine (Levophed) and epinephrine (Adrenalin)- Used in protracted hypotension following adequate fluid-volume replacement. Stimulates beta1-adrenergic and alpha-adrenergic receptors, which in turn increase cardiac muscle contractility and heart rate, as well as vasoconstriction; result is increased systemic BP and coronary blood-flow increases. |
| Adult Dose | 2 mcg/kg/min IV; titrate to effect (low normal BP, eg, 80-100 mm Hg systolic, which is sufficient to perfuse vital organs) |
| Pediatric Dose | 0.1 mcg/kg/min IV; titrate to effect |
| Contraindications | Documented hypersensitivity; peripheral or mesenteric vascular thrombosis because (ischemia may be increased; area of infarct may be extended) |
| Interactions | Atropine may enhance the pressor response by blocking reflex bradycardia |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Correct blood-volume depletion, if possible, before therapy; administer into a large vein (extravasation may cause severe tissue necrosis); caution in occlusive vascular disease |

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|-------------------|---|
| Drug Name | Vasopressin (Pitressin)- Has vasopressor and ADH activity. Increases water resorption at the distal renal tubular epithelium (ADH effect) and promotes smooth muscle contraction throughout the vascular bed of the renal tubular epithelium (vasopressor effects); however, vasoconstriction also is increased in splanchnic, portal, coronary, cerebral, peripheral, pulmonary, and intrahepatic vessels. |
| Adult Dose | 0.1-0.5 U/min IV, titrate as needed; after bleeding stops, continue at the same dose for 12 h and taper over 24-48 h |
| Pediatric Dose | Initial dose: 0.002-0.005 U/kg/min IV, titrate dose to a maximum 0.01 U/kg/min IV |
| Contraindications | Documented hypersensitivity; coronary artery disease |
| Interactions | Lithium, epinephrine, demeclocycline, heparin, and alcohol may decrease effects; chlorpropamide, urea, fludrocortisone, and carbamazepine may potentiate effects |

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| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Caution in cardiovascular disease, seizure disorders, nitrogen retention, asthma, or migraine headache; excessive doses may result in hyponatremia |

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Follow-up

Further Inpatient Care

- Admit the patient to an ICU, surgical ICU, or pediatric ICU.
- Patients with hemorrhagic shock should be admitted to an intensive care or monitored setting appropriate for the underlying condition and physiologic state.

Transfer

- In hospitals without facilities to provide definitive care, patients should be stabilized as much as possible and transferred to a facility with a higher level of care.

Complications

- Coagulopathies may occur in severe hemorrhage. Fluid resuscitation, while necessary, may exacerbate coagulopathies.
- Sepsis and multiple organ system failure occur days after acute hemorrhagic shock.
- Death is a possible complication.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to recognize occult hemorrhage
- Assumption that hypotension after trauma is due to head injury
- Failure to perform a rectal examination

- Failure to diagnose the cause(s) of hemorrhage
- Inadequate resuscitation (Therapy for hemorrhagic shock should be rapidly initiated and aggressively pursued.)
- Failure to make appropriate consultations in a timely fashion

Special Concerns

- Pregnancy: Optimization of perfusion in the mother is the treatment of choice for the fetus.
 - Pediatric: Compensatory mechanisms may be effective in children. Hypotension is a late finding and represents significant hemorrhage.
 - Geriatric: Medications and underlying diseases may modify responses to hemorrhage.
-

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Sternoclavicular Joint Injury

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Synonyms, Key Words, and Related Terms

SCJ injury, SJI

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Introduction

Background

A freely moveable synovial joint links the upper extremity to the torso, with the sternoclavicular joint (SCJ) participating in all movements of the upper extremity. Function resembles that of a ball-and-socket joint, allowing motion in nearly all the planes, including rotation. Ability to thrust the arm and shoulder forward requires sound function of the SCJ. Even though only about 50% of the medial end of the clavicle articulates with the manubrium, surprising strength and stability exists in this joint. On every side, structural supports, such as the dense fibrous capsule and ligaments, reinforce the joint, making dislocations uncommon events. Attachments include the interclavicular ligament, the anterior and posterior sternoclavicular ligaments, and the costoclavicular ligaments.

Pathophysiology

Only through the application of significant force do the ligaments supporting the SCJ become completely disrupted, enabling dislocation of the joint. Whether the SCJ subluxes or dislocates depends on the extent of the damage to the supporting ligaments and capsule. Simple sprains predominate among sternoclavicular joint injuries (SJIs) and are graded into 3 types. A first-degree injury constitutes an incomplete tear of the sternoclavicular and costoclavicular ligaments. With a second-degree lesion, the clavicle subluxes from its manubrial attachment, signifying a complete breach of the sternoclavicular ligament but only a partial tear of the costoclavicular ligament. With a third-degree wound, complete rupture of the sternoclavicular and costoclavicular ligaments permits the clavicle to completely dislocate from the manubrium.

A substantial direct or indirect force to the shoulder region can cause a traumatic dislocation of the SCJ. Anterior dislocations of the SCJ are much more common (by a 9:1 ratio), usually resulting from an indirect mechanism such as a blow to the anterior shoulder that rotates the shoulder backwards and transmits the stress to the joint. Traumatic contact driving the shoulder forward can cause posterior dislocations of the SCJ, but direct impact to the superior sternal or medial clavicle also can ensue.

Frequency

- **In the US:** Notwithstanding that the sternum and clavicle are incongruous and the SCJ is frequently in flux, the ligaments and capsule contribute enough stability to make this one of the least dislocated joints in the body. Row and Marble (1958) noted that out of 1603 shoulder girdle injuries, only 3% were sternoclavicular dislocations. Posterior dislocations are considerably less common than anterior dislocations. Only 1 of the 1603 injuries was a posterior dislocation.

Mortality/Morbidity

Problems and subsequent mortality and morbidity, other than cosmetic deformities, occur infrequently with anterior dislocations of the SCJ. However, posterior SCJ dislocation has a 25% complication rate. Complications from posterior displacement of the clavicle include pneumothorax, laceration of the superior vena cava, occlusion of the subclavian artery or vein, and disruption of the trachea. All of these complications can cause significant disability and even death.

Sex

Overall incidence is higher in males than in females. However, recurrent atraumatic anterior subluxation of the SCJ (usually associated with overall joint laxity) occurs more frequently in young girls.

Age

Incidence is increased in young adult males, since this population is engaged more often in the activities associated with SJI, such as motor vehicle crashes and sports.

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Clinical

History

- Determine the onset of pain and the mechanism of injury.
 - SCJ dislocations may follow direct trauma to the anteromedial aspect of the clavicle that drives it backward and causes a posterior dislocation.
 - More commonly, dislocations arise from an indirect force applied to the anterolateral or posterolateral shoulder that compresses the clavicle down toward the sternum. The direction the shoulder is driven determines the type of dislocation. When overwhelming compression propels the shoulder forward, the force directed towards the clavicle produces a posterior dislocation of the sternoclavicular joint. If the shoulder is pressed and rotated backward, the force directed down the clavicle produces an anterior dislocation of the sternoclavicular joint.
- Patients commonly complain of chest and shoulder pain exacerbated by arm movement or by assuming a supine position.
- Pain tends to be more severe with posterior dislocations.
- Additional symptoms may be caused by associated injuries or by compression of adjacent structures by a posterior SCJ dislocation.
 - Dyspnea
 - Dysphagia
 - Paresthesias

Physical

- Patients present with their head tilted towards the affected side and hold the affected arm across the trunk with the uninjured arm.
- Check vital signs, especially respirations.
 - Tachypnea, stridor, and other signs of respiratory distress (posterior dislocations) may be present.
 - Verify adequacy of circulation. Venous congestion of the head, neck, and/or affected arm may result from posterior dislocations.
- When viewed from the level of the patient's knees, anterior SCJ dislocations demonstrate a conspicuous asymmetry, with the medial aspect of the affected clavicle appearing prominent. Palpation reveals a medial protrusion.

- Findings tend to be subtler with posterior SCJ dislocations, and an evident defect may be present.
 - The corner of the sternum on the affected side may be palpated more readily than on the noninjured side.
 - Palpation often reveals exquisite tenderness medially.
 - Soft tissue swelling may obscure any defect and create the false impression of an anterior dislocation.

Causes

- Motor vehicle accidents are the most common mechanism producing sternoclavicular dislocation.
- Athletic injury
 - In a "pile-on" in football, the shoulder off the ground may be rolled backwards, causing an anterior dislocation, or rolled forwards, causing a posterior dislocation.
 - During a sporting event, an athlete lying on his or her back may be jumped on with the knee of the jumper landing directly on the medial end of the clavicle. A kick delivered to the front of the medial clavicle can produce dislocation.
- Dislocations of the sternoclavicular joint also may result from congenital, degenerative, and inflammatory processes.
- Ligamentous laxity, more common in young girls, is associated with recurrent atraumatic anterior dislocations of the sternoclavicular joint. This tends to be a self-limited condition.

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Differentials

Fractures, Clavicle
Fractures, Rib
Fractures, Scapular
Fractures, Sternal

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Workup

Imaging Studies

- Routine x-rays of the sternoclavicular joint are often difficult to interpret and may falsely appear

normal.

- A specialized view, known as the serendipity view and described by Rockwood (1975), may reveal the medial clavicle position. For this technique, the beam is tilted to 40° from vertical and directed cephalad through the manubrium of the supine patient. Normal clavicles should appear in the same horizontal plane, while anterior and posterior dislocations appear above and below the plane, respectively.
- In the Hobbs view, the patient sits at the x-ray table and leans forward so that the anterior chest is in contact with the film cassette and the flexed elbows straddle the cassette and support the patient. The x-ray beam is aimed directly down through the cervical spine, projecting the sternoclavicular joints onto the film cassette.
- Other imaging studies, such as angiography or esophagoscopy, may be indicated when mediastinal injuries from a posterior dislocation are suspected.
- MRI provides the same information as a CT scan while better documenting the soft tissue anatomy and associated mediastinal structures.

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Treatment

Prehospital Care

- Depending upon the mechanism of injury (eg, motor vehicle crash) and the close proximity of the sternum and clavicle to the vital structures of the neck and chest, patients with SCJ injuries may incur severe and life-threatening additional injuries.
- Foremost, address the ABCs during prehospital care, with rapid transport to an appropriate trauma care facility.
- For patients with seemingly isolated SJI, immobilization of the affected upper extremity with a sling stabilizes the joint and minimizes pain.

Emergency Department Care

Patients with posterior SCJ dislocations frequently (up to 25% of the time) sustain associated serious injuries that take treatment precedence over the dislocation.

- Sprains of the SCJ require only symptomatic treatment, ie, immobilization with a sling, ice for 24-48 hours, analgesics, and anti-inflammatory medications
- Acute anterior dislocations usually can be treated nonoperatively, but interposition of the joint capsule or the ligaments can make the joint irreducible. Additionally, maintaining reduced anterior dislocations often is difficult. Carry out closed reduction as follows:

- If time and circumstance permit, administer conscious sedation.
- Situate the patient in a supine position on the stretcher.
- Place a 3- to 4-inch-thick bolster (rolled sheet or sandbag) between the scapula and spine (helps separate the clavicle from the manubrium).
- Have an assistant abduct (to 90 degrees) and apply traction to the affected arm.
- For an anterior dislocation, press the medial clavicle posteriorly and inferiorly.
- To emergently relocate a posterior dislocation, pull the medial clavicle forward while an assistant maintains traction and abductive force on the affected limb.
 - In situations in which the clavicle cannot adequately be grasped by the fingers, employ a towel clip to grip the clavicle (after sterile preparation of the skin) and pull forward.
 - General anesthesia often is needed for reduction of posterior dislocations.
 - An alternative technique to prepare for reduction of a posterior SCJ dislocation (proposed by Buckerfield and Castle) suggests caudal traction accompanying adduction of the affected arm, along with downward pressure on both shoulders.
- Closed reduction may be unsuccessful or not attempted, depending on the age and activity level of the patient. In such patients, an immobilizing sling, analgesics, and anti-inflammatory agents may be used for symptomatic relief.

Consultations

- Consult an orthopedic surgeon for reduction and possible operative stabilization of posterior dislocations.
- Suspicion of tracheal disruption or mediastinal damage secondary to a posterior dislocation necessitates evaluation by a capable thoracic surgeon.

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Medication

The goal of therapy is to reduce inflammation and minimize severe pain. To achieve this goal, anti-inflammatory agents and analgesics are the drugs of choice (DOCs).

Analgesics

These agents commonly are used for the relief of mild to moderate pain. Pain control is essential to quality patient care. Analgesics ensure patient comfort, promote pulmonary toilet, and enable physical therapy regimens. Most analgesics have sedating properties that are beneficial for patients with injuries. Although the effects of NSAIDs in the treatment of pain tend to be patient specific, ibuprofen is usually

the DOC for the initial therapy. Other NSAIDs may be considered.

| | |
|-------------------|---|
| Drug Name | Ibuprofen (Motrin, Advil, Nuprin)- In the absence of contraindications, this is usually the DOC for treating mild to moderate pain. Inhibits inflammatory reactions and pain by decreasing prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h while symptoms persist; not to exceed 3.2 g/d |
| Pediatric Dose | Children 6 months to 12 years: 30-70 mg/kg/d PO divided tid/qid; start at the lower end of the dosing range and titrate upward to a maximum of 2.4 g/d >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease, recent GI bleeding or perforation, renal insufficiency, high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related side effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

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|-------------------|---|
| Drug Name | Naproxen (Aleve, Anaprox, Naprelan, Naprosyn)- For relief of mild to moderate pain. Inhibits inflammatory reactions and pain by decreasing the activity of the enzyme cyclooxygenase, which results in a decrease of prostaglandin synthesis. |
| Adult Dose | 500 mg PO, followed by 250 mg PO q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related side effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |

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|-------------|--|
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |
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|-------------------|---|
| Drug Name | Ketoprofen (Orudis, Oruvail, Actron)- For the relief of mild to moderate pain and inflammation. Administer small dosages initially to patients with a small body size, the elderly, and those with renal or liver disease. When administering this medication, doses >75 mg do not increase therapeutic effects. Administer high doses with caution and closely observe patients for response. |
| Adult Dose | 25-50 mg PO q6-8h prn; not to exceed 300 mg/d |
| Pediatric Dose | Children 3 months to 12 years: 0.1-1 mg/kg PO q6-8h >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; GI disease; cardiovascular disease; renal or hepatic impairment; patients receiving anticoagulants |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related side effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|---|
| Drug Name | Acetaminophen (Tylenol, Aspirin Free Anacin, Feverall)- DOC for pain in patients with documented hypersensitivity to aspirin or NSAIDs, with upper GI disease, or who are taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg PO q4h; not to exceed 5 doses in 24 h |
| Contraindications | Documented hypersensitivity; documented G-6-PD deficiency |
| Interactions | Rifampin can reduce analgesic effects of acetaminophen; coadministration with barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity |

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| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholism following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; acetaminophen is contained in many OTC products and combined use with these products may result in cumulative acetaminophen doses exceeding recommended maximum dose |

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| Drug Name | Propoxyphene and acetaminophen (Darvocet N-100)- Drug combination indicated for mild to moderate pain. |
| Adult Dose | 1-2 tabs q4h prn; not to exceed 600 mg/d |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | May increase the serum concentrations of MAOIs, tricyclic antidepressants, carbamazepine, phenobarbital, and warfarin |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates (substitution may result in acute opiate withdrawal symptoms); caution in severe renal or hepatic dysfunction |

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|-------------------|--|
| Drug Name | Acetaminophen with codeine (Tylenol with codeine)- Drug combination indicated for treating mild to moderate pain. |
| Adult Dose | 30-60 mg/dose PO based on codeine content q4-6h or 1-2 tab q4h; not to exceed 12 tab/d |
| Pediatric Dose | Recommended dose based on codeine: 0.5-1 mg/kg/dose PO Recommended based on acetaminophen: 10-15 mg/kg/dose PO q4h; not to exceed 2.6 g/24 h of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | Toxicity increases with CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates, since this substitution may result in acute opiate withdrawal symptoms; caution in severe renal or hepatic dysfunction |

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| Drug Name | Hydrocodone and acetaminophen (Lorcet, Vicodin)- Drug combination indicated for relieving moderate to severe pain. |
| Adult Dose | 1-2 tabs or caps PO q4-6h prn pain |

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| Pediatric Dose | Do not exceed the following doses of hydrocodone bitartrate: <2 years: 1.25 mg/dose PO 2-12 years: 5 mg/dose PO >12 years: 10 mg/dose PO |
| Contraindications | Documented hypersensitivity; high-altitude cerebral edema or elevated intracranial pressure |
| Interactions | Coadministration with phenothiazines may decrease analgesic effects; toxicity increases with CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tabs contain metabisulfite, which may cause hypersensitivity; caution in patients dependent on opiates since this substitution may result in acute opiate withdrawal symptoms; caution in severe renal or hepatic dysfunction |

| | |
|-------------------|--|
| Drug Name | Oxycodone and acetaminophen (Percocet)- Drug combination indicated for relieving moderate to severe pain. |
| Adult Dose | 1-2 tabs or caps PO q4-6h prn pain |
| Pediatric Dose | 0.05-0.15 mg/kg/dose PO; not to exceed 5 mg/dose of oxycodone q4-6h prn |
| Contraindications | Documented hypersensitivity |
| Interactions | Phenothiazines may decrease analgesic effects of this medication; toxicity increases with coadministration of either CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Duration of action may increase in elderly patients; be aware of total daily dose of acetaminophen patient is receiving; do not exceed 4 g/d of acetaminophen; higher doses may cause liver toxicity |

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Follow-up

Further Inpatient Care

- Inpatient admission may be necessary for patients with posterior SCJ dislocations or for patients in need for treatment of associated injuries.

Further Outpatient Care

- Reductions performed in the ED require stabilization of the affected shoulder with a soft figure-of-eight dressing, a commercial clavicular harness, or secure sling. Maintain immobilization for at least 4 weeks.
- To ensure adequate healing of sprains, arrange for a follow-up visit to the appropriate physician.
- For anterior/posterior dislocations, a follow-up visit with a qualified physician is indicated to determine the need for further treatment (eg, elective reduction, internal fixation) and to evaluate current functional capacity.

in/Out Patient Meds

- Analgesics and anti-inflammatory agents

Transfer

- Patients thought to have sustained additional major injuries, either because of the force of the mechanism of injury or documented presence of serious associated wounds (eg, pneumothorax, tracheal injury, venous compromise), may require triage to a trauma center.
- Issues of patient stability and transfer benefit need to be addressed based on the clinical setting and available resources.
- Patients with posterior SCJ dislocation and/or potential complications may benefit from transfer to a facility with thoracic and orthopedic consultation services.

Complications

- Approximately 25% of posterior SCJ dislocations are associated with tracheal, esophageal, or great vessel injury and may involve the following specific complications:
 - Tracheal rupture or erosion
 - Pneumothorax
 - Laceration of the superior vena cava
 - Occlusion of the subclavian artery and/or vein
 - Recurrent dislocation
 - Decreased range of motion
 - Residual swelling or deformity

Prognosis

- Most patients have adequate upper extremity function following sternoclavicular joint injuries.
- Individual prognosis depends on such factors as extent and type of joint damage, activity level, and concomitant medical illness of the patient.

Patient Education

- Patients with sprains initially should restrict activity involving the affected extremity.
- Anterior/posterior dislocations
 - Patients should restrict activity and follow up as instructed.
 - Patients with posterior dislocations who are discharged home should return for medical care if they exhibit symptoms of mediastinal injury.

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Miscellaneous

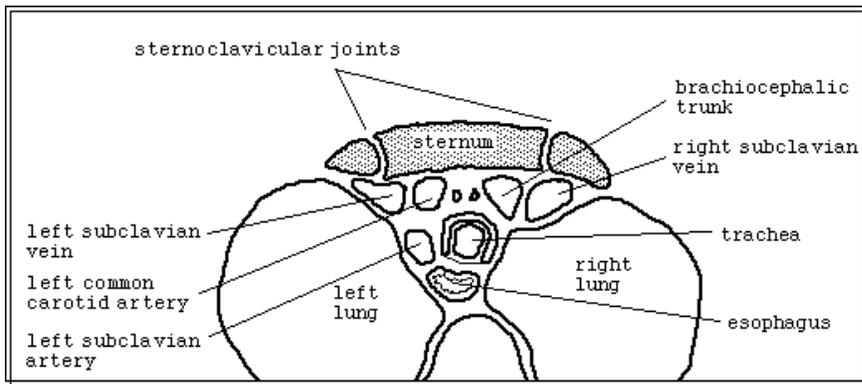
Medical/Legal Pitfalls

- Failure to diagnose posterior dislocation and its potential complications
- Failure to detect coexistent injuries (ie, additional injuries due to the mechanism of injury)
- Failure to consider life-threatening referred etiologies of shoulder and sternoclavicular pain, such as myocardial infarction and rupture of the spleen

Special Concerns

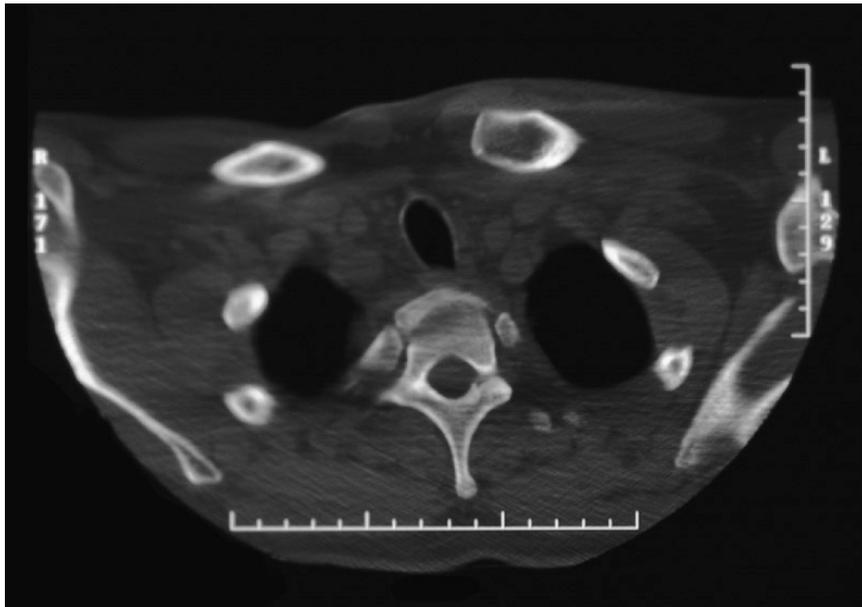
- Pregnancy
 - The need for radiographs outweighs the radiation risk to the fetus.
 - Use appropriate shielding.
 - Elderly patients
 - In older patients, anterior dislocations may transpire without any clear history of trauma. This most commonly manifests as a painless mass over the medial aspect of the clavicle.
 - In geriatric or less active younger patients, anterior dislocations may be left unreduced and still permit adequate range of motion.
-

Pictures



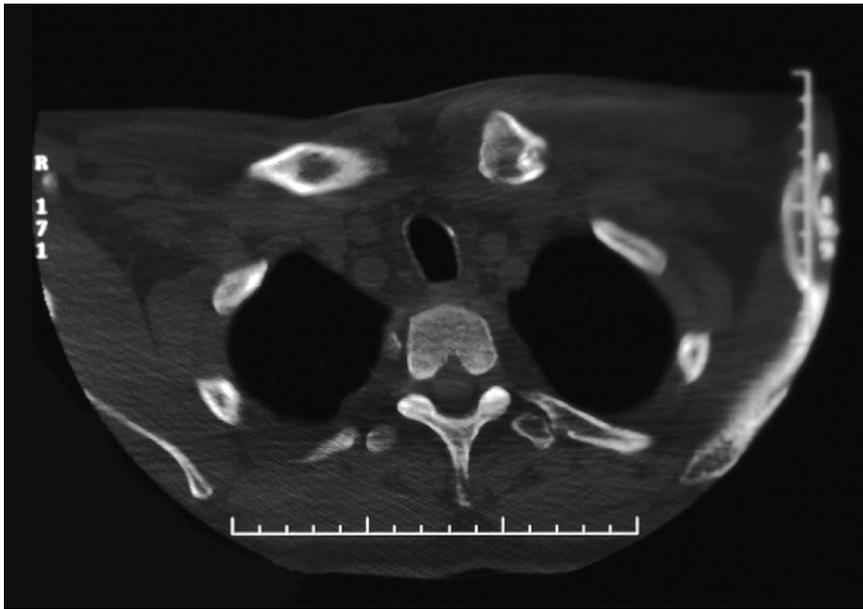
Picture 1: Superior mediastinal contents may be threatened in posterior dislocations of the sternoclavicular joint.

Picture type: Photo



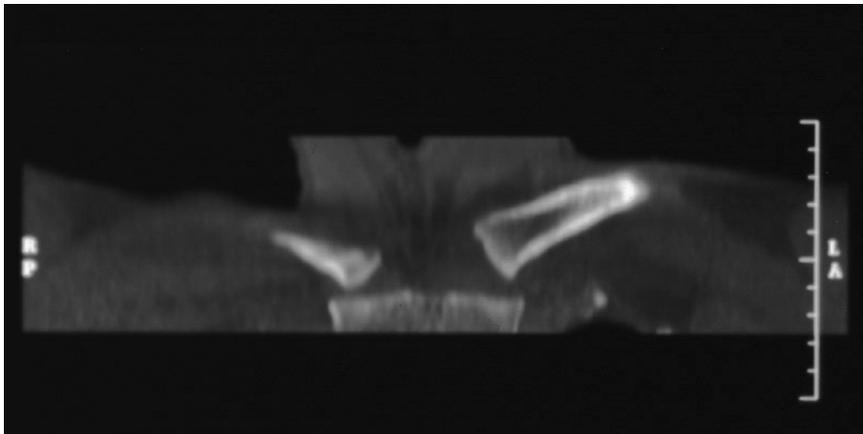
Picture 2: CT scan of left sternoclavicular dislocation demonstrates anterior and superior displacement of the clavicle from its normal articulation with the manubrium. The right sternoclavicular joint is normal.

Picture type: CT



Picture 3: CT scan of left sternoclavicular dislocation demonstrates anterior and superior displacement of the clavicle from its normal articulation with the manubrium. The right sternoclavicular joint is normal.

Picture type: CT



Picture 4: CT scan of left sternoclavicular dislocation demonstrates anterior and superior displacement of the clavicle from its normal articulation with the manubrium. The right sternoclavicular joint is normal.

Picture type: CT

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Subdural Hematoma

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Introduction

Background

An acute subdural hematoma (SDH) is a rapidly clotting blood collection below the inner layer of the dura but external to the brain and arachnoid membrane. Two further stages, subacute and chronic, may develop with untreated acute SDH. Each type has distinctly different clinical, pathological, and imaging characteristics.

Generally, the subacute phase begins 3-7 days after acute injury. (Surgical literature favors 3 days; radiological, 7).

The chronic phase begins about 2-3 weeks after acute injury.

Pathophysiology

Typically, low-pressure venous bleeding of bridging veins (between the cortex and venous sinuses) dissects the arachnoid away from the dura and layers out along the cerebral convexity. Cerebral injury results from direct pressure, increased intracranial pressure (ICP), or associated intraparenchymal insults.

In the subacute phase, the clotted blood liquifies. Occasionally, in the prone patient, the cellular elements

layer, which can appear on CT imaging as a hematocritlike effect.

In the chronic phase, cellular elements have disintegrated, and a collection of serous fluid remains in the subdural space. In rare cases, calcification develops.

Frequency

- **In the US:** Frequency is related directly to the incidence of blunt head trauma. An SDH is the most common type of intracranial mass lesion, occurring in about a third of those with severe head injuries (Glasgow Coma Scale [GCS] score <9).

Mortality/Morbidity

Acute SDH is associated with high mortality and morbidity rates.

- Simple SDH accounts for about half of all cases and implies that no parenchymal injury is present. Simple SDH is associated with a mortality rate of about 20%.
- Complicated SDH accounts for the remaining cases and implies that parenchymal injury (eg, contusion or laceration of a cerebral hemisphere) is present. Complicated SDH is associated with a mortality rate of about 50%.

Age

The majority of SDHs are associated with age factors related to the risk of blunt head trauma. Certain age factors are related to more unusual variants of this disease.

- SDH is more common in people older than 60 years. The elderly are predisposed to cerebral atrophy because they have less resilient bridging veins. Moreover, these veins can be damaged more easily in the elderly.
- Since the adhesions existing in the subdural space are absent at birth and develop with aging, bilateral SDHs are more common in infants.
- Interhemispheric SDHs often are associated with child abuse.

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Clinical

History

- Suspect acute SDH whenever the patient has experienced a mechanism of moderately severe to severe blunt head trauma.
- Patients generally lose consciousness, but this is not an absolute.
- Chronic SDH is more difficult to anticipate, and about half of such cases offer no history of head trauma. Patients often present with progressive symptoms such as unexplained headache, personality changes, signs of increased ICP, or hemiparesis/plegia.
- Any degree or type of coagulopathy should heighten suspicion of SDH.
- Hemophiliacs can develop SDH with a seemingly trivial head trauma. An aggressive approach to diagnosis and immediate correction of the factor deficiency to 100% activity is paramount.
- Alcoholics are prone to thrombocytopenia, prolonged bleeding times, and blunt head trauma.
 - Maintain a high level of suspicion in this population.
 - Promptly obtain a CT scan of the head when the degree of trauma is severe, focal neurologic signs are noted, or intoxication does not resolve as anticipated.
 - In alcoholics, more than any other cohort, acute or chronic SDHs can be due to the deadly combination of repetitive trauma and alcohol-associated coagulopathies.

Physical

- Physical examination of patients with head trauma should emphasize assessment of neurologic status using the GCS. Search for any focal neurologic deficits or signs of increased ICP.
- Signs of external trauma alert the physician to the expected location of coup or contrecoup injuries on CT scan.
- Any abnormality of mental status that cannot be explained completely by alcohol intoxication or the presence of another mind-altering substance should increase suspicion of SDH in the patient with blunt head trauma. Obtain an urgent CT scan.
- GCS score less than 15 after blunt head trauma, in a patient with no intoxicating substance use (or impaired mental status baseline), warrants consideration of an urgent CT scan.
- Presence of a focal neurologic sign following blunt head trauma is ominous and requires an emergent explanation.

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Differentials

Elder Abuse

Epidural Hematoma

Meningitis

Pediatrics, Child Abuse

Stroke, Hemorrhagic

Stroke, Ischemic
Subarachnoid Hemorrhage

Other Problems to be Considered

Dementia

Brain tumor

Subdural empyema

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Workup

Lab Studies

- Complete blood count
- Coagulation profile
- Electrolytes
- Type and screen/cross

Imaging Studies

- While MRI is superior for demonstrating the size of an acute SDH and its effect on the brain, noncontrast head CT scans are the primary means of making a diagnosis and suffice for immediate management purposes.
- Acute SDH typically appears on a noncontrast head CT scan as a hyperdense (white) crescentic mass along the inner table of the skull, most commonly over the cerebral convexity in the parietal region (see [Picture 1](#)). The second most common area is above the tentorium cerebelli.
 - Small SDHs may blend in with the adjacent skull and may be appreciated only by adjusting the CT scan window width to between those generally used to view brain and bone.
 - Some degree of midline shift should be present with moderate or large SDHs. Suspect a contralateral mass when midline shift is absent. If midline shift seems excessive, suspect underlying cerebral edema.
 - SDHs are relatively uncommon in the posterior fossa since the cerebellum undergoes little movement, which is protective of its bridging cortical veins. SDHs that do occur in that location are usually a result of parenchymal cerebellar injury.

- Interhemispheric SDH causes the falx cerebri to appear thickened and irregular and often is associated with child abuse.
- In the chronic phase, the lesion becomes hypodense and is easy to appreciate on a noncontrast head CT scan.

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Treatment

Emergency Department Care

- Consider endotracheal intubation when GCS score is less than 12 or other indications are present; this guarantees airway protection during the diagnostic workup.
- Obtain an immediate head CT scan in patients with head trauma who experienced clear loss of consciousness, are symptomatic, are disoriented/amnestic, or have any focal neurologic signs.
- Burr holes are a temporizing option when rapid demise is associated with severe head trauma, especially if a herniation syndrome is clinically evident.
 - Burr holes can guide surgical therapy when head CT imaging is unavailable.
 - Generally, because the lesion represents clotted blood, the burr hole is not curative, and emergent craniotomy is necessary.

Consultations

When a patient who experienced head trauma presents with a GCS score less than 12, consider immediate neurosurgical consultation while stabilizing and diagnostic maneuvers are in progress.

- Small, asymptomatic, acute SDHs may be managed by observation, serial examinations, and serial CT scanning.
- Patients with focal findings, neurologic worsening, significant midline displacement (>5.0 mm), or increased intracranial or posterior fossa pressure require operative intervention.
- The usual treatment for acute SDH is craniotomy and evacuation.
 - After making a large cranial flap, open the dura.
 - Remove the clot with suction, cup forceps, and/or irrigation. Identify and control bleeding sites.

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Follow-up

Transfer

- Detected SDHs require patient transfer to facilities offering neurosurgical evaluation and treatment.
- Transfer may be emergent, with appropriate stabilization measures taken and with appropriately skilled personnel accompanying the patient.

Complications

- Postoperative complications
 - Elevated ICP
 - Brain edema
 - New or recurrent bleeding/hematoma
 - Infection
 - Seizures

Prognosis

- Definitive prognosis often is not possible at the time of emergency department evaluation.
- Ultimate prognosis is related to the amount of associated direct brain damage and the damage resulting from the mass effect of the SDH.

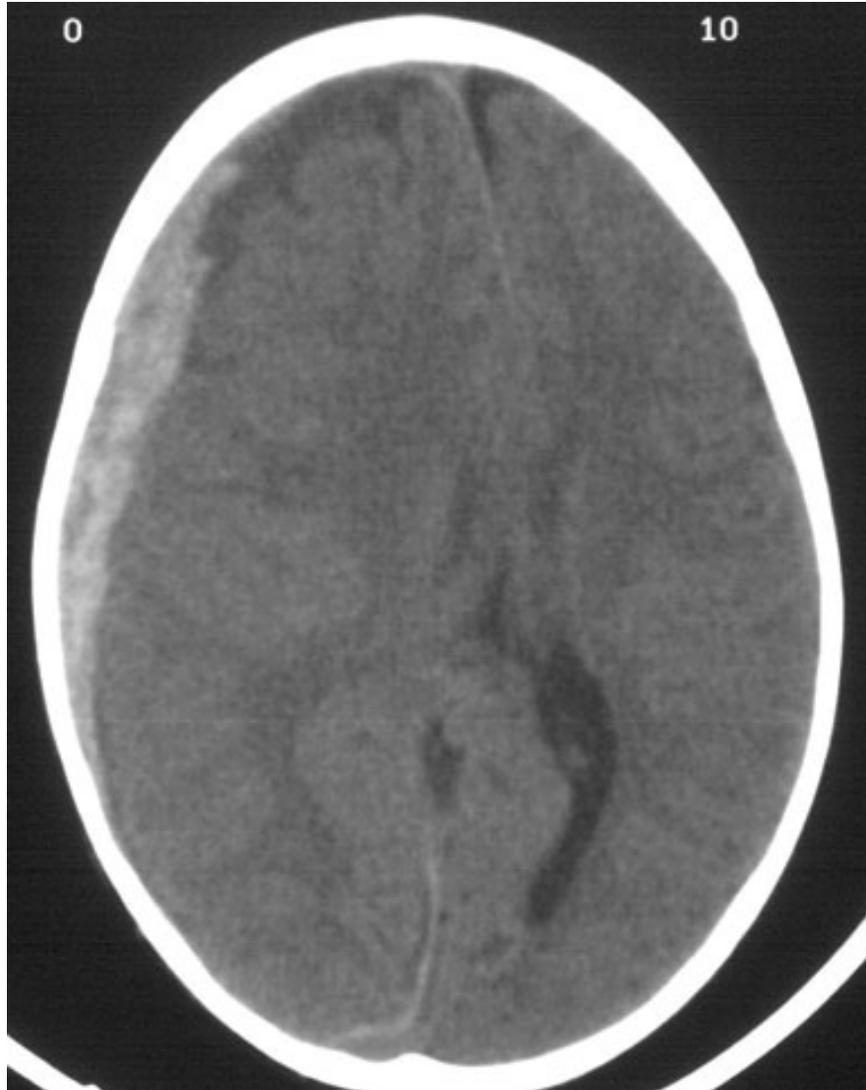
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Miscellaneous

Medical/Legal Pitfalls

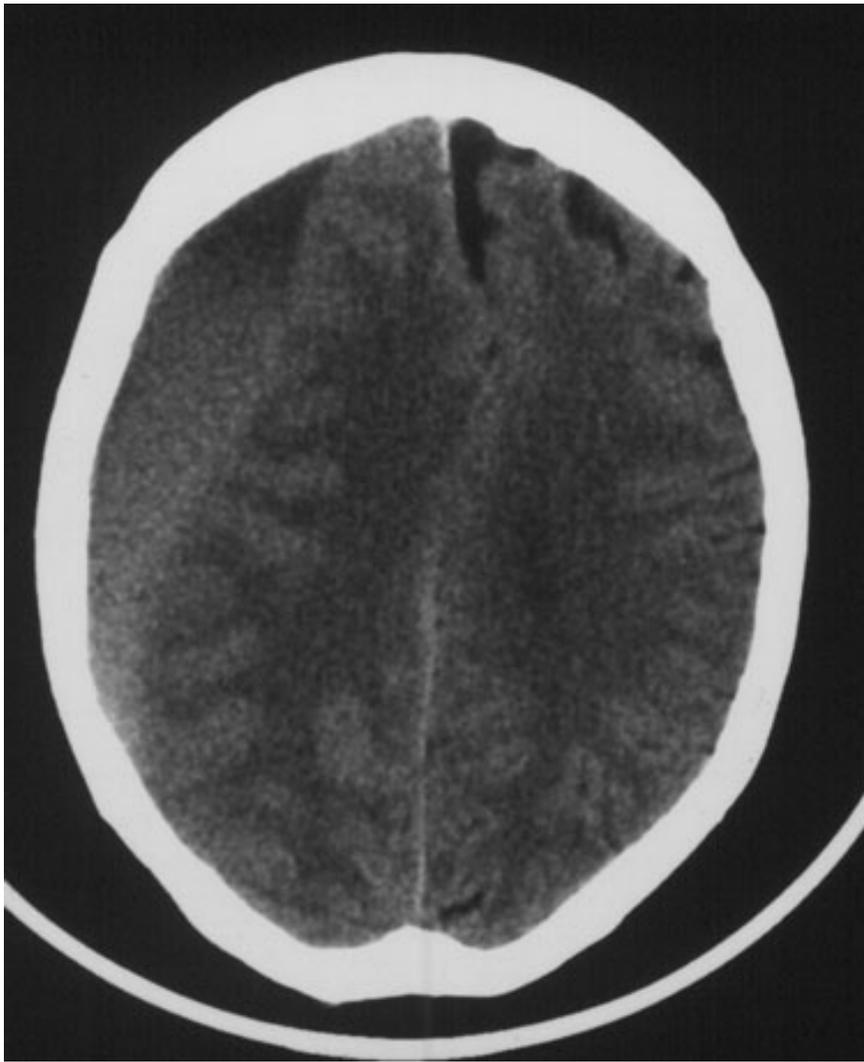
- Failure to identify a patient with SDH and delayed diagnosis of the hematoma are constant risks.
 - Consider neuroimaging for patients taking anticoagulants.
 - Alcoholics may have an associated coagulopathy placing them at high risk for SDH. Altered mental status may be from an SDH, not ethanol.
-

Pictures



Picture 1: Acute subdural hematoma: note the bright (white) image properties of the blood on this noncontrast cranial CT scan. Note also the midline shift. (CT scan courtesy of J. Stephen Huff, MD)

Picture type: CT



Picture 2: Subacute subdural hematoma: the crescent-shaped clot is less white than on CT scan of acute subdural hematoma (see Picture 1). In spite of the large clot volume, this patient was awake and ambulatory. (CT scan courtesy of J. Stephen Huff, MD)

Picture type: CT

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Toenails, Ingrown

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Synonyms, Key Words, and Related Terms

unguis incarnatus

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Introduction

Background

Ingrown toenails (unguis incarnatus) are a common toenail problem of uncertain etiology. Various causes include poorly fit (tight) footwear, infection, improperly trimmed toenails, trauma, and heredity. The great toe is the most commonly involved. The lateral side is involved more commonly than the medial side.

Pathophysiology

The underlying cause of this condition is a foreign body reaction. When the nail bed is compressed from the side, the edge of the nail then penetrates the cuticle. The presence of the keratinaceous material of the nail in the flesh of the toe sets up a foreign body reaction.

Frequency

- **In the US:** The occurrence of this common disorder is poorly measured, because many instances are not brought to the attention of the medical community.
- **Internationally:** The frequency is unknown.

Mortality/Morbidity

The principle morbid condition of this disorder is pain. However, it can be the initiating pathway for more serious disorders in certain patients at risk, especially those with diabetes or arterial insufficiency.

- Particular attention must be paid to high-risk patients. Referral to specialty clinics for follow-up (eg, surgeon, podiatrist) is recommended.
- No direct mortality for this disorder exists.

Age

This disorder is not found in the preambulatory stages. Rare in preteens, it is more common in teenagers, and its occurrence increases throughout life.

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Clinical

History

- Patients have a painful, swollen and tender toe.
- When infection is present, the patient may have local discharge.
- Important components of the history include a previous history of risk factors for diabetes and arterial insufficiency.

Physical

- The affected toe has all the classic signs of infection: edema, erythema, and warmth.
- Lymphangitis is rare.
- The affected side is readily apparent.
- Inspection for other contributing causes, particularly mycoses, is important.

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Differentials

Nailbed Injuries

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Workup

Imaging Studies

- Radiography should be considered when it is necessary to rule out osteomyelitis (rare) or in the setting of trauma to rule out toe fractures (common).

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Treatment

Emergency Department Care

- Once nails have started to grow in, which opens the basement membranes of the cuticle to bacterial invasion, action is needed to forestall progression. The edge of the nail must be elevated from the bed.
 - This elevation can be accomplished by simply rolling a cotton wisp from the lateral side of the nail (in the case of a lateral ingrowth) gently under the edge of the nail. (Forcing it in from the tip is much more painful.)
 - If the nail is too ingrown to do this without pain, try soaking the foot in warm water with an antibacterial agent. Soaking may soften the nail enough to allow elevation of the edge without much pain.
 - If soaking fails, perform a digital block (outlined below) before elevating the nail edge. The toe is exquisitely sensitive. The block may hurt more than the procedure, if it is not slowly performed with a small (30-gauge) needle and buffered lidocaine.
 - These conservative measures should be enacted as soon as possible and may be sufficient to render unnecessary the surgical treatment outlined below.

Consultations

Consultation is encouraged for those patients with risk factors (eg, those with diabetes or compromised circulation), related to either the disease or the procedure.

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Medication

Medications are needed for only those with complications. Antibiotics are not indicated unless lymphangitic spread is noted. Antifungal agents are needed for onychomycosis. Ibuprofen is used for pain.

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and enables physical therapy regimens. Most analgesics have sedating properties, which are beneficial for patients who have painful lesions.

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Advil, Motrin, Nuprin, and Genpril)- Usually the DOC for the treatment of mild to moderate pain, if no contraindications are present. Inhibits inflammatory reactions and pain by decreasing prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h while symptoms persist; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 30-70 mg/kg/d PO tid/qid; not to exceed 2.4 g/d >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease, recent GI bleeding or perforation, renal insufficiency, or high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |

| | |
|-------------|---|
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, decreased renal and hepatic function, anticoagulation abnormalities, or during anticoagulant therapy |
|-------------|---|

| | |
|-------------------|--|
| Drug Name | Acetaminophen (Tylenol, Aspirin Free Anacin)- DOC for pain in patients with documented hypersensitivity to aspirin or NSAIDs, those with upper GI disease, or those taking oral anticoagulants. |
| Adult Dose | 325-650 mg PO q4-6h or 1000 mg PO tid/qid; not to exceed 4 g/d |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose PO q4-6h prn; not to exceed 2.6 g/d >12 years: 325-650 mg PO q4h; not to exceed 5 doses in 24 h |
| Contraindications | Documented hypersensitivity; known G-6-PD deficiency |
| Interactions | Rifampin can reduce analgesic effects; coadministration with barbiturates, carbamazepine, hydantoins, and isoniazid may increase hepatotoxicity |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatotoxicity possible in chronic alcoholism following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; many OTC products contain acetaminophen, and combined use may result in cumulative acetaminophen doses exceeding recommended maximum dose |

| | |
|-------------------|--|
| Drug Name | Acetaminophen and Codeine (Tylenol with codeine)- Drug combination indicated for the treatment of mild to moderate pain. |
| Adult Dose | 30-60 mg/dose based on codeine content PO q4-6h or 1-2 tab PO q4h; not to exceed 12 tabs in 24 h |
| Pediatric Dose | 0.5-1 mg/kg/dose based on codeine PO q4-6h; 10-15 mg/kg/dose based on acetaminophen content PO; not to exceed 2.6 g/d of acetaminophen |
| Contraindications | Documented hypersensitivity |
| Interactions | Toxicity increases with CNS depressants or tricyclic antidepressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients dependent on opiates (substitution may result in acute opiate-withdrawal symptoms); caution in severe renal or hepatic dysfunction |

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Follow-up

Further Outpatient Care

- Follow-up for uncomplicated cases is needed only to reassure the patient.
- A lot of drainage (but little bleeding) may occur in the 2-3 days following removal. The toe looks better, the patient has less pain, and the redness decreases.
- Patients with risk factors require close follow-up, as noted in [Consultations](#).

Deterrence/Prevention

- If inciting factors are present, counseling about prevention is indicated.
- Preventive measures include the use of properly fitted footwear and correct trimming of nails.
 - Shoes should have a toe box large enough to fit the toes without pressure and to allow for normal spreading of the toes with walking.
 - Nails should be cut straight across with clean, sharp, preferably bulldog-type nail trimmers. Nails should not be cut shorter at the lateral edges.

Complications

- Complications are very rare, except in those predisposed because of underlying pathologic conditions.
- Complications include infection and loss of the nail.

Prognosis

- Generally, the prognosis is excellent.
- Recurrence and/or regrowth of the treated side occurs in 10-30% of cases.

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Miscellaneous

Medical/Legal Pitfalls

- Good communication regarding the risks and purposes of the cauterizing procedure is the best protection.
-

Pictures



Picture 1: Appearance of typical ingrown toenail.

Picture type: Photo



Picture 2: Cutting the nail.

Picture type: Photo



Picture 3: Cauterizing the matrix.

Picture type: Photo



Picture 4: Appearance of toenail at end of the cauterizing procedure.

Picture type: Photo

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Trauma, Lower Genitourinary

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Introduction

Background

Injuries to the lower genitourinary (GU) tract alone are not life threatening, but their association with other injuries necessitates an organized approach to diagnosis and management. Without such an approach, prolonged morbidity and disability may occur. As trauma is a multisystem disease, other injuries may take priority and interfere with full urologic assessment. Coordinated efforts between various services caring for the patient are crucial to ensure comprehensive care.

Initial evaluation of the injured patient suspected to have GU trauma should not differ from that of other patients. Follow the protocols of the Advanced Trauma Life Support program of the [American College of Surgeons](#).

Pathophysiology

The lower GU tract comprises the urinary bladder, urethra, and external genitalia. Bladder injuries mostly occur in blunt trauma. Eighty-five percent of these injuries occur with pelvic fractures; 15% occur with penetrating trauma and blunt mechanism without a pelvic fracture (ie, full bladder blowout).

Urethral injury is predominantly a male problem. In males, the urethra is divided into the proximal (posterior) segment and the distal (anterior) segment by the urogenital diaphragm. The anterior urethra is further divided into membranous (sphincteric) and prostatic segments. About 3 centimeters long, the anterior urethra extends from the bladder to the urogenital diaphragm.

Injuries to the proximal urethra are mostly secondary to pelvic fractures, while injuries to the distal urethra are caused by straddle-type (eg, bicycles, skateboards) or penetrating (often self-inflicted) injuries. Urethral injuries constitute only 10% of all GU injuries, but iatrogenic urethral injuries constitute a significant fraction of urethral injuries.

Penile injuries are secondary to injuries caused by penetration, blunt trauma, continence- or sexual pleasure-enhancing devices, and mutilation (self-inflicted or otherwise). Scrotal injuries follow the same pattern.

Frequency

- **In the US:** Of trauma patients, 3-10% have GU involvement, while 10-15% of trauma patients with abdominal injuries have GU involvement. Urethral injuries constitute 10% of all GU traumas; bladder injuries, 40%.

Mortality/Morbidity

Mortality from lower GU trauma is attributed to associated injuries, especially pelvic fractures.

Sex

Urethral trauma is mainly a male problem.

Age

Urethral trauma affects all age groups but seems to have a higher incidence in persons aged 15-25 years.

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Clinical

History

In blunt trauma, history is obtained regarding the time and mechanism of injury, eg, the position of the patient in a motor vehicle accident (MVA) and whether restraints were used. The speed of the vehicle and the manner in which the accident occurred provide information about forces applied to the victim.

In penetrating trauma, knowing the size of the stabbing weapon or the caliber of the gun and the distance from which it was discharged helps in assessment. Question paramedics as to the condition of the patient immediately after injury occurred and during transport to the care facility.

In patients with GU trauma, symptoms are nonspecific and may be masked by or attributed to other injuries.

- Bladder trauma
 - In the ED, question the patient about suprapubic abdominal pain and the ability to void after the injury.
 - If the patient cannot provide such information and gross hematuria is present, suspect bladder injury.
- In external genitalia trauma, a history of psychiatric problems, use of rings, and excessive sexual activity is pertinent in specific conditions. A history of sudden pain, loss of erection, and swelling is important.

Physical

Signs of lower GU injury are a small part of a massive conglomeration of signs related to associated injuries; therefore, always keep a high index of suspicion.

- Bladder trauma
 - Bruising or edema of the lower abdomen, perineum, or genitalia indicates bladder injury.
 - Always suspect urethral and bladder injuries in patients with pelvic fractures and inability to void.
 - Inability to retrieve all fluid used to irrigate the bladder through a Foley catheter indicates bladder injury.
- Penile trauma
 - Loss of skin
 - Edema
 - Angulation
 - Level of mutilation
 - Viability of mutilated segment

Causes

Bladder injuries are best classified as intraperitoneal and extraperitoneal. Extraperitoneal bladder injuries

account for 65-85% of bladder injuries and are associated with pelvic fractures, especially pubic bone fractures (95%). Intraperitoneal injuries account for 15-35% of bladder injuries and infrequently are associated with pelvic fractures. Blunt rupture of a distended bladder and penetrating injuries cause the remaining intraperitoneal injuries.

Blunt trauma is responsible for 60% of urethral injuries, and penetrating and iatrogenic etiologies cause 40%. Blunt injury in the anterior urethra usually is caused by a straddle-type mechanism compressing the urethra between a hard object and the symphysis pubis. In 70% of patients, penetrating trauma to the anterior urethra involves the perineum and bulbar urethra, and in 30%, the pendulous urethra is involved.

Posterior urethral injury in blunt trauma is secondary to pelvic fractures because of proximity to the bony pelvis. Missiles and knives can cause penetrating injury to the posterior urethra.

In gunshot wounds, look for associated injuries to the pelvis, bladder, rectum, and sphincter mechanism.

- Main causes of bladder injuries
 - MVAs
 - Motorcycle accidents (MCAs)
 - Bicycle accidents
 - Stabbings
 - Impalements
 - Gunfire
 - Iatrogenic

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Differentials

Trauma, Upper Genitourinary

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Workup

Lab Studies

- Complete blood count (CBC) to obtain a hematocrit and a platelet count

- Prothrombin time (PT) and activated partial thromboplastin time (aPTT) to check for coagulopathy
- Blood type and crossmatch
- Urinalysis to assess for gross hematuria

Imaging Studies

- Plain radiograph of the pelvis to assess presence and extent of bony injury
- Retrograde urethrogram
 - This is indicated prior to the insertion of a Foley catheter when urethral injury is suspected.
 - Urethrography is performed with water-soluble contrast material and preferably under fluoroscopy. If fluoroscopy is unavailable, multiple plain films are obtained with 10-cc injections of contrast material.
- CT scan of abdomen and pelvis
 - Specific in diagnosis of bladder injuries but carries low sensitivity
 - Useful for diagnosis of associated abdominal and pelvic injuries
- Simultaneous suprapubic cystography and retrograde urethrography
 - If the urethrogram is inconclusive and the patient still cannot void with a distended bladder, a suprapubic cystostomy catheter is inserted pending further investigation.
 - This is the procedure of choice about a week after the injury.

Procedures

- Bladder irrigation: Bladder rupture is indicated by inability to retrieve the total amount of the irrigant.

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Treatment

Prehospital Care

Advancement of prehospital care for the trauma patient is one of the biggest leaps forward in trauma care. Principles do not change with varying organ injuries.

- Paramedics quickly assess the patient and mechanism of injury, especially for patency of ABCs.
 - Establish an airway if needed and/or administer oxygen.
 - Establish 2 large-bore IVs.

- Take cervical spine precautions (eg, hard collar, board).
- Leave the scene as soon as possible and quickly transport the patient to the trauma center.

Emergency Department Care

- Administer oxygen and ventilatory support if needed.
- Resuscitate with crystalloids (lactated Ringer solution or isotonic sodium chloride solution) and blood if indicated (O-negative packed red blood cells).
- Treat life-threatening injuries (eg, tension pneumothorax, open pneumothorax, cardiac tamponade).
- Use diagnostic procedures as indicated (cystogram and retrograde urethrogram).

Consultations

- Trauma surgeon for associated intra-abdominal injuries
- Urologist for lower GU tract injury
- Orthopedic surgeon for management of frequently associated pelvic fractures
- Other specialists as injuries require

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Medication

Medications for patients with lower GU tract injuries relate to the management of patients as critically injured rather than management specific to the GU injury.

Antibiotics

Used for prophylaxis against infections of the GU tract. Empiric antimicrobial therapy must be comprehensive, covering all likely pathogens in the clinical setting.

| | |
|------------|---|
| Drug Name | Ampicillin and sulbactam (Unasyn)- Drug combination of beta-lactamase inhibitor with ampicillin. Covers skin, enteric flora, and anaerobes. Not ideal for nosocomial pathogens. |
| Adult Dose | 1.5 (1 g ampicillin + 0.5 g sulbactam) to 3 (2 g ampicillin + 1 g sulbactam) g IV/IM q6-8h; not to exceed 4 g/d sulbactam or 8 g/d ampicillin |

| | |
|-------------------|---|
| Pediatric Dose | 3 months to 12 years: 100-200 mg ampicillin/kg/d (150-300 mg Unasyn) IV divided q6h >12 years: Administer as in adults; not to exceed 4 g/d sulbactam or 8 g/d ampicillin |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid and disulfiram decrease renal excretion and increase antibiotic levels; allopurinol increases excretion; also may potentiate ampicillin rash and decrease effect of PO contraceptives |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Dose adjustment may be required in patients diagnosed with renal failure; mononucleosis increases incidence of rash with ampicillin-sulbactam therapy; carefully evaluate rash appearance to differentiate a nonallergic ampicillin rash from a hypersensitivity reaction |

| | |
|-------------------|--|
| Drug Name | Cefotetan (Cefotan)- Second-generation cephalosporin indicated for infections caused by susceptible gram-positive cocci and gram-negative rods. Dosage and route of administration depends on condition of patient, severity of infection, and susceptibility of causative organism. |
| Adult Dose | Loading dose: 2 g IV Maintenance dose: 1-2 g q12h IV/IM for 5-10 d |
| Pediatric Dose | 20-40 mg/kg/dose IV/IM q12h for 5-10 d |
| Contraindications | Documented hypersensitivity |
| Interactions | Alcoholic beverages consumed concurrently <72 h after taking cefotetan may produce acute alcohol intolerance (disulfiramlike reaction); hypoprothrombinemic effects of anticoagulants may be increased; monitor renal function in patients receiving potent diuretics (eg, loop diuretics); risk of nephrotoxicity may be increased; aminoglycoside nephrotoxicity may potentiate effects in the kidney when used concurrently; monitor renal function closely |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Reduce dosage by a half for patients with CrCl of 10-30 mL/min and by a quarter for patients with a CrCl of <10 mL/min; antibiotics (especially prolonged or repeated therapy) may result in bacterial or fungal overgrowth of nonsusceptible organisms, leading to a secondary infection; take appropriate measures if superinfection occurs |

Anticoagulants

Used prophylactically against deep venous thrombosis.

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|-------------------|---|
| Drug Name | Heparin (Hep-Lock)- Augments activity of antithrombin III and prevents conversion of fibrinogen to fibrin; does not actively lyse but can inhibit further thrombogenesis; prevents reaccumulation of a clot after spontaneous fibrinolysis. |
| Adult Dose | Loading dose: 40-170 U/kg IV Maintenance infusion: 18 U/kg/h IV Alternative dose: 50 U/kg/h, followed by continuous infusion of 15-25 U/kg/h IV; increase by 5 U/kg/h IV q4h prn using aPTT results |
| Pediatric Dose | Loading dose: 50 U/kg Maintenance infusion: 15-25 U/kg/h IV; increase by 2-4 U/kg/h q6-8h prn using aPTT results |
| Contraindications | Documented hypersensitivity; subacute bacterial endocarditis, active bleeding, or history of heparin-induced thrombocytopenia |
| Interactions | Digoxin, nicotine, tetracycline, and antihistamines may decrease effects; NSAIDs, aspirin, dextran, dipyridamole, and hydroxychloroquine may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Some preparations contain benzyl alcohol as a preservative; when used in large amounts, may be associated with fetal toxicity (gasping syndrome); use of preservative-free heparin is recommended in neonates; caution in shock or severe hypotension |

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| Drug Name | Enoxaparin (Lovenox)- Prevents DVT, which may lead to pulmonary embolism in patients undergoing surgery who are at risk for thromboembolic complications. Enhances inhibition of factor Xa and thrombin by increasing antithrombin III activity. In addition, preferentially increases inhibition of factor Xa. Average duration of treatment is 7-14 d. |
| Adult Dose | 30 mg SC q12h |
| Pediatric Dose | Not established Suggested dosing: <2 months: 0.75 mg/kg/dose SC bid 2 months to 18 years: 0.5 mg/kg/dose SC bid |
| Contraindications | Documented hypersensitivity; major bleeding, thrombocytopenia |
| Interactions | Platelet inhibitors or PO anticoagulants, such as dipyridamole, salicylates, aspirin, NSAIDs, sulfipyrazone, and ticlopidine, may increase risk of bleeding |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |

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| Precautions | If thromboembolic event occurs despite prophylaxis, discontinue and initiate appropriate therapy; reversible elevation of hepatic transaminases is occasionally observed; heparin-associated thrombocytopenia has been observed with LMWH; for significant bleeding complications, 1 mg of protamine sulphate reverses the effect of approximately 1 mg of enoxaparin |
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H2-Receptor Antagonists

Useful in prophylaxis against stress ulcerations; reversible competitive blockers of histamine at H2 receptors, particularly those in the gastric parietal cells, where they inhibit acid secretion. H2 antagonists are highly selective, do not affect H1 receptors, and are not anticholinergic agents.

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| Drug Name | Ranitidine hydrochloride (Zantac)- Competitively inhibits histamine at H2 receptor of the gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and reduced hydrogen concentrations. |
| Adult Dose | 150 mg PO bid; not to exceed 600 mg/d 50 mg/dose IV/IM q6-8h |
| Pediatric Dose | <12 years: Not established. >12 years: 1.25-2.5 mg/kg/dose PO q12h; not to exceed 300 mg/d 0.75-1.5 mg/kg/dose IV/IM q6-8h; not to exceed 400 mg/d |
| Contraindications | Documented hypersensitivity |
| Interactions | May decrease effects of ketoconazole and itraconazole; may alter serum levels of ferrous sulfate, diazepam, nondepolarizing muscle relaxants, and oxaprozin |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Caution in renal or liver impairment; if changes in renal function occur during therapy, consider adjusting dose or discontinuing treatment |

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|-------------------|---|
| Drug Name | Famotidine (Pepcid)- Competitively inhibits histamine at H2 receptor of gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and reduced hydrogen concentrations. |
| Adult Dose | 40 mg/d PO bid for 4-8 wk 20 mg IV bid |
| Pediatric Dose | Not established Suggested dose: 1-2 mg/kg/d PO/IV divided q6h; not to exceed 40 mg/dose |
| Contraindications | Documented hypersensitivity |
| Interactions | May decrease effects of ketoconazole and itraconazole |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |

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| Precautions | If changes in renal function occur during therapy, consider adjusting dose or discontinuing treatment |
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|-------------------|---|
| Drug Name | Nizatidine (Axid)- Competitively inhibits histamine at H2 receptor of the gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and reduced hydrogen concentrations. |
| Adult Dose | 300 mg PO hs or 150 mg bid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in renal or liver impairment; if changes in renal function occur during therapy, consider adjusting dose or discontinuing treatment |

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Follow-up

Further Inpatient Care

- Bladder contusion
 - Adequate drainage of the bladder should result in resolution within a few days.
 - Follow-up cystography is recommended to assess integrity of the bladder wall.
- Extraperitoneal rupture
 - Perform a cystogram after 7-10 days with adequate bladder drainage and broad-spectrum antibiotics.
 - Remove the catheter if extravasation has resolved. If the extravasation is persistent, surgical intervention is required.
 - Persistent severe hematuria and infection of the pelvic hematoma are contraindications to conservative therapy.
 - Surgical repair is performed by opening the dome of the bladder and repairing the laceration from within.
- Management of urethral injuries - Related to type of injury sustained, but basic principles apply
 - Drain the bladder with a suprapubic catheter percutaneously or open to prevent further extravasation.
 - Initial urethral repair is not recommended because of risk of hemorrhage, impotence, and infection of pelvic hematoma.

- Commence definitive management of urethral injuries after stabilizing the patient and attending to associated injuries, if present.
- Repair can be performed as immediate primary closure, delayed primary closure (10-14 d), or late primary closure (>3 mo).
- Blunt trauma
 - Radionuclide scan or ultrasonography can help assess the condition of the testes.
 - Surgical exploration and repair of ruptured testis reduces pain and duration of recovery.
 - With significant scrotal skin loss, the testes can be moved to an alternate location (ie, to the perineum or subcutaneously) and the skin debrided and closed. With time, the scrotum dilates and the testes can be returned.

Further Outpatient Care

- Further outpatient care in the patient with lower GU tract trauma mainly depends on the extent of associated injuries. The need for rehabilitation secondary to either orthopedic or neurologic injuries must be assessed on a patient-by-patient basis.
 - Arrange for follow-up care for delayed repair of urethral injuries.
 - Penile injuries require close follow-up care, especially if skin grafting was performed.
 - Perform follow-up hormonal studies and semen analysis on patients with scrotal or testicular injuries.

in/Out Patient Meds

- Use prophylaxis against infections of the GU tract, especially for penile injuries.

Transfer

- Assess capabilities of staff and center to handle the patient who has multiple injuries with lower GU trauma; the decision to transfer is based on that assessment.
- Treat all life-threatening injuries prior to transfer; stabilize and resuscitate the patient.
- The responsibility of the transfer, choice of transfer modality, and selection of accepting facility lies with the transferring physician.
- The receiving physician confirms the ability of the receiving institution to handle the patient's condition.
- An institutional transfer protocol facilitates the transfer process.
- Lower GU trauma patients benefit from transfer when the following conditions exist at the transferring center:
 - CT scan not available
 - No staff urologist
 - Multiple injuries that surpass hospital's resources
 - Unavailability of specialized care required by patient's injuries

Deterrence/Prevention

- Patients with urethral and penile injuries should refrain from sexual activity until the injury has healed.

Complications

- Bladder injuries
 - Urinomas
 - Fistulization (rectum, vagina, bowel, cutaneous)
 - Pelvic hematoma infection
 - Difficulties voiding
 - Distal ureteral obstruction
- Penile injury
 - Angulation
 - Painful erection
 - Impotence

Prognosis

- Prognosis for patients with lower GU tract injuries is related to their associated injuries.

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Miscellaneous

Special Concerns

- Pediatrics
 - Preferential use of suprapubic catheters in boys to avoid complications of prolonged indwelling catheters in small urethras
 - Cutaneous vesicostomy desirable in infants
-

Pictures



Picture 1: Retrograde urethrogram showing an irregularity of the urethra indicating injury secondary to a shotgun wound

Picture type: X-RAY

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Trauma, Peripheral Vascular Injuries

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Introduction

Background

Peripheral vascular injuries occur because of direct or indirect trauma and may result in loss of limb or function. Prompt diagnosis and intervention is crucial. The emergency physician is responsible for expedient recognition of injuries and rapid, appropriate consultation while stabilizing the patient.

Pathophysiology

Blunt trauma causes vascular injury by 2 mechanisms—tensile strain and shear strain. Vessel or intimal rupture is caused by excess longitudinal force from tensile strain, which exposes flowing blood to a large surface area rich in thrombogenic substances, resulting in local thrombosis. Shear strain is secondary to lateral forces acting on the vessel wall. It frequently is encountered with deceleration injuries and can result in partial or complete transection.

Penetrating and blunt trauma can cause direct or indirect injury. Examples of direct vascular injury include partial or complete transection, contusion, laceration, and arteriovenous (AV) fistula formation.

Indirect injuries can be more subtle in presentation and include vessel spasm, external compression, mural contusion, thrombosis, and aneurysm formation.

Peripheral vascular injuries to extremity tissues can be tolerated without ischemia when collateral vascular flow is present and adequate. This may not always be the situation, depending on the mechanism, location, and extent of injury and on the patient's baseline circulation to the involved extremity. In general, extremity tissues tolerate 4-6 hours of ischemia before irreversible injury occurs. Ischemia is less likely to occur in the presence of adequate collateral flow.

Frequency

- **In the US:** Peripheral vascular injuries comprise approximately 3% of major injuries. Most are caused by acts of violence or vehicular collisions.

Mortality/Morbidity

Mortality directly relates to other injuries and to the degree of hemorrhage.

- Isolated peripheral vascular trauma usually has a low mortality rate.
- Morbidity increases with prolonged ischemia and gangrene, which can result in amputation of the affected limb. False aneurysms and AV fistula formation may complicate this injury.
- Morbidity associated with arterial reconstruction includes postrepair edema, infection, arterial compression, and anastomotic thrombosis.

Sex

Penetrating peripheral vascular injuries secondary to gunshots or stab wounds are more common in males than in females.

Age

Traumatic injury disproportionately affects younger persons and is the leading cause of death in those aged 1-44 years.

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Clinical

History

- A detailed history usually is not required to determine the nature of penetrating vascular injury.
- Knowing the type of weapon used and the velocity or potential path of the missile may help in assessment.
- In blunt vascular injury, a detailed mechanism of injury often helps in appreciating subtle injuries.

Physical

- Penetrating vascular injuries usually are easy to diagnose.
- Profuse red blood suggests arterial injury, and a continuous flow of dark blood suggests venous injury. An apparently innocuous entrance wound may have a significant underlying pathology.
- Blunt vascular injuries usually are more obscure and severe and typically are surrounded by greater local injury.
- Skin that appears intact may obscure diagnosis.
- The following factors can obscure diagnosis:
 - Shock
 - Hypothermia
 - Vasospasm
 - External vascular compression

Causes

Most injuries are secondary to acts of violence involving gunshot or stab wounds. Vascular injuries usually are caused by penetrating trauma from knives, bullets, and glass. Motor vehicle accidents, heavy machinery-related injuries, and falls cause a small proportion of blunt vascular injuries secondary to decelerating or crushing forces.

Penetrating injuries cause damage to vascular structures by direct injury (eg, secondary to stab or low-velocity missile wounds) and/or high-velocity injury (eg, cavitation effect by passage of a high-velocity missile through tissue with transfer of large amounts of kinetic energy). Destructive power increases proportionately with missile velocity and mass. These types of injuries can cause severe damage, even in the absence of direct vascular trauma.

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Differentials

Abdominal Trauma, Blunt

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Workup

Lab Studies

- Laboratory studies do not help diagnose injury but may assist with management. Order the following studies in patients with vascular trauma:
 - Serial hemoglobin measurements
 - Typing and crossmatching of blood
 - BUN and creatinine as a measure of baseline renal function in anticipation of a contrast study
 - Baseline measurement of coagulation profile, especially if any associated medical conditions may effect coagulation (eg, alcoholism)

Imaging Studies

- Plain radiographs
 - Radiographs help diagnose fractures, foreign bodies, or missiles that may be responsible for neurovascular compromise.
 - Certain fractures more commonly are associated with blunt vascular injury.

Other Tests

- Color-flow duplex ultrasound
 - Images the vessel and measures blood flow and velocity
 - Noninvasive alternative to arteriography in monitoring occult injuries
 - Disadvantage - Operator-dependent test
 - Advantage - Noninvasive and can be performed serially at bedside
- Emergency center arteriography
 - Involves manual contrast injection followed by immediate radiography
 - Rapid, accurate, and does not require transportation of an unstable patient to angiography suite
 - Currently used in young children, as formal angiography is difficult to obtain in an uncooperative patient

Procedures

- Splint fractures and reduce dislocations.
- Unstable patients may require central venous access.
- Doppler limb pressure measurements, documenting decreased pulse pressure in the affected limb, help establish a diagnosis of vascular injury.

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Treatment

Prehospital Care

- Secure airway as needed.
- Fluid resuscitation requires vascular access.
- Control hemorrhage by external compression.
- Splint extremity as needed.

Emergency Department Care

- As with all traumas, maintain ABCs as the first priority.
- Conduct a more detailed secondary evaluation to assess for vascular injury.
- If penetrating injuries, particularly high-velocity injuries, are near major vascular structures, assume damage to those structures. Approximately 20% of such patients have occult arterial injuries.
- Frequently assess vascular status.
 - Doppler examination for pulses helps in patients with diminished pulses.
 - Measure blood pressure in an injured and uninjured extremity. A 10-mm Hg difference suggests vascular injury, as does an ankle-brachial index less than 1.0.
- Generally, extremity tissues tolerate 4-6 hours of ischemia. Carefully monitor popliteal artery injuries because of minimal collateral circulation present in the lower extremity.
- Patients presenting with shock often require central access; however, a large-bore antecubital line is best for fluid resuscitation.
- If using peripheral access, establish it in the uninjured extremity.
- Anatomic repositioning and splinting may help restore circulation in dislocations or fractures.
- Obvious vascular injury with evidence of ischemia indicates emergent surgical exploration once the patient is stabilized.

Consultations

Prompt consultation with the trauma team is routine at most major urban trauma centers. If isolated

peripheral vascular injury is present, consult the vascular surgeon as soon as the patient stabilizes to reduce ischemia time.

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Medication

The goal of therapy is to control pain and infections.

Analgesics

Pain control is essential to quality patient care. It ensures patient comfort, promotes pulmonary toilet, and aids physical therapy regimens. Most analgesics have sedating properties that benefit patients with traumatic injuries.

Use of pain medications in trauma victims is a difficult issue. Narcotics have several drawbacks, including exacerbating hypotension in hemorrhaging patients and mental status changes in patients with head injuries. These agents also may mask pain caused by subtle injuries. Nevertheless, in cases of isolated extremity trauma in stable patients, use pain medications. Use IV administration for more precise titration.

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| Drug Name | Fentanyl (Duragesic, Fentanyl Oralet)- Potent narcotic analgesic with much shorter half-life than morphine sulfate. DOC for conscious sedation analgesia. Ideal for short-term (30-60 min) analgesic action during anesthesia and immediate postoperative period. Excellent choice for short-term pain management and sedation; easy to titrate. Easily and quickly reversed by naloxone. After initial dose, do not titrate subsequent doses more frequently than q3-6h. When using transdermal dosage form, most patients are controlled with 72-h dosing intervals, although some patients require dosing 48-h dosing intervals. |
| Adult Dose | Emergency: 0.5-2 mcg/kg/dose IV/IM Analgesia: 0.5-1 mcg/kg/dose IM/IV q30-60min Transdermal: Apply a 25 mcg/h system q48-72h |
| Pediatric Dose | <2 years: 2-3 mcg/kg/dose IV/IM q30-60min 2-12 years: 1-2 mcg/kg/dose IV/IM q60min >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; hypotension or potentially compromised airway in which it would be difficult to establish rapid airway control |

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| Interactions | Phenothiazines may antagonize analgesic effects of opiate agonists; tricyclic antidepressants may potentiate adverse effects when coadministered |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hypotension, respiratory depression, constipation, nausea, emesis, and urinary retention; an idiosyncratic reaction (chest wall rigidity syndrome) may require neuromuscular blockade to increase ventilation |

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| Drug Name | Meperidine (Demerol)- Analgesic with multiple actions similar to those of morphine. May produce less constipation, smooth muscle spasm, and depression of cough reflex than similar analgesic doses of morphine. |
| Adult Dose | 50-150 mg PO/IM/SC q3-4h prn |
| Pediatric Dose | 1-1.8 mg/kg (0.5-0.8 mg/lb) PO/IM/SC q3-4h prn; not to exceed adult dose |
| Contraindications | Documented hypersensitivity; upper airway obstruction or significant respiratory depression; during labor when delivery of premature infant is anticipated; in patients taking MAOIs currently or within past 14 d |
| Interactions | Monitor for increased respiratory and CNS depression with coadministration of cimetidine; hydantoin may decrease effects; avoid with protease inhibitors |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution if administering narcotics to patients with head injuries, since may increase respiratory depression and CSF pressure; administer in such patients only if absolutely necessary; if dose is increased substantially above recommended levels due to tolerance, seizures may be aggravated or may occur even in individuals that do not have a history of convulsive disorders; closely observe patients for morphine-induced seizure activity if they have prior history of seizures; caution when using narcotic analgesics postoperatively and in patients with a history of pulmonary disease; may suppress the cough reflex |

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| Drug Name | Morphine sulfate (Duramorph, Astramorph, MS Contin)- DOC for narcotic analgesia due to reliable and predictable effects, safety profile, and ease of reversibility with naloxone; IV administration may be dosed in a number of ways and commonly is titrated until desired effect is obtained. |
| Adult Dose | Starting dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q2-4h Relatively hypovolemic patients: Start with 2 mg IV/IM/SC and reassess hemodynamic effects of dose |
| Pediatric Dose | Neonates: 0.05-0.2 mg/kg dose IV/IM/SC prn Children: 0.1-0.2 mg/kg dose IV/IM/SC q2-4h prn |

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| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects of opiate agonists; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects of morphine when coadministered |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

Antibiotics

Indicated in all open wounds. Empiric antimicrobial therapy must be comprehensive, covering all likely pathogens in the clinical setting.

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| Drug Name | Cefazolin (Ancef, Kefzol, Zolicef)- First-generation semisynthetic cephalosporin that arrests bacterial cell wall synthesis, inhibiting bacterial growth. Primarily active against skin flora, including <i>Staphylococcus aureus</i> . Typically used alone for skin and skin-structure coverage. IV and IM dosing regimens are similar. |
| Adult Dose | 250 mg to 2 g IV/IM q6-12h, depending on severity of infection; not to exceed 12 g/d |
| Pediatric Dose | 25-100 mg/kg/d IV/IM divided q6-8h depending on severity of infection; not to exceed 6 g/d |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid decreases renal clearance and prolongs effects; concurrent use with aminoglycosides may increase renal toxicity; administration may yield false-positive urine dip for glucose |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal impairment; prolonged use of antibiotics is associated with superinfections and promotion of nonsusceptible organisms; complications usually are reversible |

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| Drug Name | Clindamycin (Cleocin)- Lincosamide for treatment of serious skin and soft tissue staphylococcal infections. Also effective against aerobic and anaerobic streptococci (except enterococci). Inhibits bacterial growth, possibly by blocking dissociation of peptidyl t-RNA from ribosomes causing cessation of RNA-dependent protein synthesis. |
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| Adult Dose | 600-1200 mg/d IV/IM divided q6-8h |
| Pediatric Dose | 8-16 mg/kg/d IV/IM divided tid/qid |
| Contraindications | Documented hypersensitivity, regional enteritis, ulcerative colitis, hepatic impairment, and antibiotic-associated colitis |
| Interactions | Increases duration of neuromuscular blockade, induced by tubocurarine and pancuronium; erythromycin may antagonize effects; antidiarrheals may delay absorption |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Dose adjustment may be necessary in patients with severe hepatic dysfunction; no adjustment necessary in patients diagnosed with renal insufficiency; use has been associated with severe and possibly fatal colitis |

Tetanus Immunization

Indicated when 10 years have passed since last booster shot. If immunization status is unclear, tetanus immune globulin is required.

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| Drug Name | <p>Tetanus toxoid- Used to induce active immunity against tetanus in selected patients. The immunizing agents of choice for most adults and children >7 y are tetanus and diphtheria toxoids. Necessary to administer booster doses to maintain tetanus immunity throughout life.</p> <p>Pregnant patients should receive only tetanus toxoid, not a diphtheria antigen-containing product.</p> <p>In children and adults, may administer into deltoid or midlateral thigh muscles. In infants, preferred site of administration is the mid thigh laterally.</p> |
| Adult Dose | <p>Primary immunization: 0.5 mL IM; administer 2 injections 4-8 wk apart and a third dose 6-12 mo after second injection</p> <p>Booster dose: 0.5 mL IM q10y</p> |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; history of any type of neurologic symptoms or signs following administration of this product; FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are an important cause of provocative poliomyelitis |

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| Interactions | Patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization because of poor immune response; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent use of systemic chloramphenicol, since it may impair anamnestic response to tetanus toxoid; concurrent use of tetanus Ig may delay development of active immunity by several days; this interaction is clinically insignificant and does not preclude concurrent use |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Never use to treat actual tetanus infections or for immediate prophylaxis of unimmunized individuals; instead use tetanus antitoxin, preferably human tetanus Ig; diminished antibody response to active immunization may be seen in patients receiving immunosuppressive therapy; it is better to defer primary diphtheria immunization until immunosuppressive therapy is discontinued; routinely immunize symptomatic and asymptomatic persons who are infected with HIV |

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| Drug Name | Tetanus immune globulin (Hyper-Tet)- Used for passive immunization of patients with a wound that may be contaminated with tetanus spores. |
| Adult Dose | 250-500 U IM in extremity opposite to tetanus toxoid lesion |
| Pediatric Dose | Administer as in adults |
| Contraindications | Since antibodies in globulin preparation may interfere with immune response to vaccination, do not administer within 3 mo of live virus Ig administration; may be necessary to revaccinate patients who received Ig shortly after live virus vaccination |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Patients with isolated IgA deficiency may develop antibodies to IgA and may have anaphylactic reactions to subsequent administration of blood products that contain IgA; do not perform skin testing, since intradermal injection of concentrated gamma-globulin may cause a localized area of inflammation and can be misinterpreted as a positive allergic reaction rather than a localized chemical tissue irritation; medication mistakenly may be withheld from a nonallergic patient; true allergic responses to human gamma-globulin administered in the prescribed IM manner are extremely rare; do not mix with other medications as they are usually incompatible |

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Follow-up

Further Inpatient Care

- Most patients require surgical consultation and admission.

Transfer

- If angiography or surgical consultation is not available at the primary institution, transfer the patient as quickly as possible after stabilization.

Complications

- Associated nerve damage occurs in a large percentage of vascular injuries; 45% of those result in permanent deficits.

Prognosis

- Prognosis depends upon ischemic time and number and extent of associated injuries.
 - Extent of soft tissue injury correlates with probability of limb loss.
 - Infection also plays a major role in amputation rate.
 - Often, infection rate is high despite adequate antibiotic coverage.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to promptly diagnose peripheral vascular injury is a pitfall. The legal implications of delayed diagnosis resulting in limb amputation are self-explanatory; however, amputation sometimes is necessary in severe injury. In such instances, do not delay amputation, as this results in increased risk of sepsis and higher morbidity.
- Failure to provide thorough physical examination, prompt consultation, early IV antibiotics, and tetanus immunization if indicated also are pitfalls. The goals are stabilization of the patient and minimization of ischemic time.

Special Concerns

- Pediatrics

- A thorough neurovascular examination is more difficult in young children.
- Children have a higher risk of developmental abnormalities secondary to ischemia.
- Emergency center arteriography may be an alternative to aid rapid diagnosis in young patients.

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Trauma, Upper Genitourinary

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Introduction

Background

As trauma is a multisystem disease, multiple injuries may take priority and interfere with a full urologic assessment. Coordinated efforts between various services caring for the patient are crucial to ensure comprehensive care.

Initial evaluation of the injured patient with possible genitourinary (GU) trauma should not differ from that of other patients. Follow the protocols of the Advanced Trauma Life Support (ATLS) program of the [American College of Surgeons](#) to provide total patient care.

Pathophysiology

The kidneys, pelvocalyceal system, and ureters comprise the upper GU tract. Adult kidneys are well protected by the rib cage and vertebral column, but lateral forces can compress them between these structures, leading to injury. Sudden deceleration can cause avulsion injuries to the renal pedicle and pelvocalyceal system.

Only 6% of patients with GU trauma have ureteral injuries, probably due to its small size and mobility, and the protection provided by the psoas muscle posteriorly, the abdominal viscera anteriorly, and the vertebral column medially.

Frequency

- **In the US:** Three to 10% of trauma patients have GU involvement; 10-15% of trauma patients with abdominal injuries have GU involvement. Renal injuries constitute 45% of all GU injuries; ureteral injuries constitute 6%.

Mortality/Morbidity

Mortality from upper GU tract injuries is attributed primarily to associated injuries, and morbidity is 26%. Trauma is the leading cause of death in persons aged 1-40 years and is the third-ranked cause of mortality in all age groups.

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Clinical

History

- In blunt trauma, history is obtained regarding the time and mechanism of injury, position of the patient, speed of the vehicle, and use of restraints.
- In penetrating trauma, knowing the size of the stabbing weapon or the caliber of the gun and the distance from which it was discharged aids assessment.
- Question the paramedics as to the condition of the patient immediately after injury occurred and during transport to the hospital.
- In patients with GU trauma, symptoms are nonspecific and may be masked by or attributed to other injuries.
- Renal injuries are most commonly from motor vehicle accidents (MVAs).
 - Renal injuries occur in 3% of patients hospitalized with trauma and in 10% of patients with abdominal trauma.
 - Most renal injuries (80%) are minor and do not require surgical intervention.
 - Suspect renal injury when fractures of lower ribs and/or spinal processes are observed and/or when a history of sudden deceleration or significant lateral force on the patient exists.
 - Trajectory of the bullet or penetrating object helps indicate the possibility of renal injury.

Physical

- Flank ecchymosis or mass indicates a retroperitoneal process but is not specific to renal injuries and rarely occurs acutely.
 - The most important indicator of renal trauma is gross or microscopic hematuria.
 - The absence of hematuria, although rare, does not exclude renal injury because it is absent in 5% of patients.
- In suspected ureteral injury, physical examination is of minimal use except in diagnosis of associated injuries.
- Perform a rectal examination to help establish the presence or absence of a urethral injury prior to Foley catheter insertion. Look for a high-riding prostate, rectal tear, bony abnormality, or frank blood.

Causes

The most common cause of renal injury is blunt trauma, followed by penetrating trauma. MVAs and gunshot wounds account for 80% of renal injuries. Conversely, the etiology of ureteral trauma is mostly iatrogenic (82%). Other causes of ureteral trauma are penetrating trauma in 90% of patients (missile injury in 90%, stabbing injury in 10%); a blunt avulsing-type mechanism causes the remaining 10%.

- Blunt trauma
 - MVAs
 - Motorcycle accidents
 - Falls from high elevations
 - Bicycle accidents
 - Assaults with blunt weapons
- Iatrogenic

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Differentials

Abdominal Trauma, Blunt
Abdominal Trauma, Penetrating
Trauma, Lower Genitourinary

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Workup

Lab Studies

- Complete blood count (CBC) to obtain hematocrit level and platelet count
- Prothrombin time (PT) and activated partial thromboplastin time (aPTT) to check for coagulopathy; may be unnecessary in young, otherwise healthy patients
- BUN and serum creatinine: Elevation of BUN without elevation in creatinine indicates urine reabsorption.
- Urinalysis to diagnose hematuria
- Blood type and crossmatch

Imaging Studies

- Use of diagnostic imaging techniques is crucial in functional and anatomic assessment of the injured and uninjured kidney.
- Perform urologic imaging only when indicated, as persistent low yield occurs when imaging patients with microscopic hematuria, patients with no associated injuries, and patients who are hemodynamically stable.
- Indications for imaging
 - Gross hematuria
 - Microscopic hematuria with hemodynamic instability
 - Persistent microscopic hematuria
 - Hemodynamic instability with history of significant deceleration mechanism
- Intravenous pyelogram
 - IVP provides information about the function of both kidneys; when performed with a double dose, it is the preferred test in suspected ureteral injuries.
 - In renal injuries, CT scan is preferred because of lesser sensitivity and relatively poor anatomic details in IVP.
 - If CT scan is not readily available and renal imaging is required, IVP is a reasonable screening test.
 - CT scan or laparotomy should follow an abnormal IVP finding.
- Ultrasonography
 - Ultrasonography is inferior to CT scan in anatomic detail and sensitivity.
 - It may be helpful in follow-up care of renal injuries and in detection of urinomas.
- Retrograde ureterogram is useful in diagnosing ureteral injury, especially in missed injury. It is invasive and requires a cystoscopy suite.

Procedures

- Insert a Foley catheter only after urethral injury is excluded.

- Use suprapubic cystostomy when Foley catheter insertion is contraindicated. The procedure can be performed in a percutaneous or open manner. Indications for insertion are the same as those for Foley catheter (ie, urine output measurement, detection of hematuria).

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Treatment

Prehospital Care

Advancement of prehospital care for trauma patients is one of the biggest leaps forward in trauma care. Principles do not change with different organ injuries.

- Paramedics quickly assess the patient and mechanism of injury, with special attention to patency of ABCs.
 - Establish an airway if needed and/or administer oxygen.
 - Establish 2 large-bore IVs.
- Quickly transport the patient to the trauma center.

Emergency Department Care

Adherence to ATLS principles is necessary for proper care of the trauma patient.

- Administer oxygen and ventilatory support if needed.
- Resuscitate with crystalloids (lactated Ringer solution or isotonic sodium chloride solution) and blood if indicated (O-negative or type-specific blood if known).
- Treat life-threatening injuries (eg, tension pneumothorax, open pneumothorax, cardiac tamponade) in the ED.

Consultations

- Trauma or general surgeon for management of associated abdominal injuries
- Urologist for management of specific GU injuries
- Other specialists as injuries dictate

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Medication

Medications for patients with upper GU injuries relate to the management of patients as critically injured rather than management specific to the GU injury.

Antibiotics

Used in prophylaxis of infections of the injured GU tract. Empiric antimicrobial therapy must be comprehensive, covering all likely pathogens in the clinical setting.

| | |
|-------------------|---|
| Drug Name | Ampicillin and sulbactam (Unasyn)- Drug combination of beta-lactamase inhibitor with ampicillin. Covers skin, enteric flora, and anaerobes. Not ideal for nosocomial pathogens. |
| Adult Dose | 1.5 (1 g ampicillin + 0.5 g sulbactam) to 3 (2 g ampicillin + 1 g sulbactam) g IV/IM q6-8h; not to exceed 4 g/d sulbactam or 8 g/d ampicillin |
| Pediatric Dose | 3-12 years: 100-200 mg ampicillin/kg/d (150-300 mg Unasyn) IV divided q6h >12 years: Administer as in adults; not to exceed 4 g/d sulbactam or 8 g/d ampicillin |
| Contraindications | Documented hypersensitivity |
| Interactions | Probenecid and disulfiram decrease renal excretion and increase levels of the antibiotic; allopurinol increases ampicillin excretion; also may potentiate ampicillin rash and decrease effect of PO contraceptives |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Dose adjustment may be required in patients diagnosed with renal failure; mononucleosis increases incidence of rash with ampicillin-sulbactam therapy; carefully evaluate rash appearance to differentiate a nonallergic ampicillin rash from a hypersensitivity reaction |

| | |
|-------------------|--|
| Drug Name | Cefotetan (Cefotan)- Second-generation cephalosporin indicated for infections caused by susceptible gram-positive cocci and gram-negative rods. Dosage and route of administration depend on condition of patient, severity of infection, and susceptibility of causative organism. |
| Adult Dose | 1-2 g IV/IM q12h for 5-10 d |
| Pediatric Dose | 20-40 mg/kg/dose IV/IM q12h for 5-10 d |
| Contraindications | Documented hypersensitivity |

| | |
|--------------|--|
| Interactions | Alcoholic beverages consumed concurrently <72 h after taking cefotetan may produce acute alcohol intolerance (disulfiramlike reaction); may increase hypoprothrombinemic effects of anticoagulants; monitor renal function in patients receiving potent diuretics (eg, loop diuretics); risk of nephrotoxicity may be increased; aminoglycosides may potentiate cefotetan effects in the kidney when used concurrently; monitor renal function closely |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Reduce dosage by a half for patients with CrCl of 10-30 mL/min and by a quarter for patients with CrCl of <10 mL/min; antibiotics (especially prolonged or repeated therapy) may result in bacterial or fungal overgrowth of nonsusceptible organisms, leading to a secondary infection; take appropriate measures if superinfection occurs |

Anticoagulants

Agents used in prophylaxis of DVT. Any patient presenting with thromboembolic complications must undergo prompt anticoagulation therapy if he or she does not have a therapeutic INR of 2.5-3.5.

| | |
|-------------------|---|
| Drug Name | Heparin (Hep-Lock)- Augments activity of antithrombin III and prevents conversion of fibrinogen to fibrin; does not actively lyse but can inhibit further thrombogenesis; prevents reaccumulation of a clot after spontaneous fibrinolysis. |
| Adult Dose | Loading dose: 40-170 U/kg IV Maintenance infusion: 18 U/kg/h Alternative dose: 50 U/kg/h; followed by continuous infusion of 15-25 U/kg/h; increase dose by 5 U/kg/h q4h prn using PTT results |
| Pediatric Dose | Loading dose: 50 U/kg/h Maintenance infusion: 15-25 U/kg/h; increase by 2-4 U/kg/h q6-8h prn using PTT results |
| Contraindications | Documented hypersensitivity; subacute bacterial endocarditis, active bleeding, or history of heparin-induced thrombocytopenia |
| Interactions | Digoxin, nicotine, tetracycline, and antihistamines may decrease effects; NSAIDs, aspirin, dextran, dipyridamole, and hydroxychloroquine may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Some preparations contain benzyl alcohol as a preservative; when used in large amounts, may be associated with fetal toxicity (gassing syndrome); use of preservative-free heparin is recommended in neonates; caution in shock or severe hypotension |

| | |
|-------------------|--|
| Drug Name | Enoxaparin (Lovenox)- LMWH used in prevention of DVT, which may lead to pulmonary embolism in patients undergoing surgery who are at risk for thromboembolic complications; enhances inhibition of factor Xa and thrombin by increasing antithrombin III activity; slightly affects thrombin and clotting time and preferentially increases the inhibition of factor Xa. Average duration of treatment is 7-14 d |
| Adult Dose | 30 mg SC q12h |
| Pediatric Dose | Not established Suggested dosing: <2 months: 0.75 mg/kg/dose bid 2 months to 18 years: 0.5 mg/kg/dose bid |
| Contraindications | Documented hypersensitivity; major bleeding and thrombocytopenia |
| Interactions | Use LMWH with caution in patients receiving platelet inhibitors or PO anticoagulants (eg, aspirin, NSAIDs, dipyridamole, salicylates, sulfinpyrazone, ticlopidine) because of increased risk of bleeding |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | If thromboembolic event occurs despite prophylaxis, discontinue and initiate appropriate therapy; reversible elevation of hepatic transaminases is occasionally observed; heparin-associated thrombocytopenia has been observed with LMWH; for significant bleeding complications, 1 mg of protamine sulphate reverses the effect of approximately 1 mg of enoxaparin |

H2-Receptor Antagonists

Reversible competitive blockers of histamine at H₂ receptors, particularly those in the gastric parietal cells, where they inhibit acid secretion. H₂ antagonists are highly selective, do not affect H₁ receptors, and are not anticholinergic agents.

| | |
|-------------------|---|
| Drug Name | Ranitidine (Zantac)- Competitively inhibits histamine at H ₂ receptor of gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and reduced hydrogen concentrations. |
| Adult Dose | 150 mg PO bid; not to exceed 600 mg/d 50 mg/dose IV/IM q6-8h |
| Pediatric Dose | <12 years: Not established >12 years: 1.25-2.5 mg/kg/dose PO q12h; not to exceed 300 mg/d 0.75-1.5 mg/kg/dose IV/IM q6-8h; not to exceed 400 mg/d |
| Contraindications | Documented hypersensitivity |

| | |
|--------------|---|
| Interactions | May decrease effects of ketoconazole and itraconazole; may alter serum levels of ferrous sulfate, diazepam, nondepolarizing muscle relaxants, and oxaprozin |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Caution in renal or liver impairment; if changes in renal function occur during therapy, consider adjusting dose or discontinuing treatment |

| | |
|-------------------|---|
| Drug Name | Famotidine (Pepcid)- Competitively inhibits histamine at H2 receptor of gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and hydrogen concentrations. |
| Adult Dose | 300 mg PO hs or 150 mg bid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | None reported |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Caution in renal or liver impairment; if changes in renal function occur during therapy, consider adjusting dose or discontinuing treatment |

| | |
|-------------------|---|
| Drug Name | Nizatidine (Axid)- Competitively inhibits histamine at H2 receptor of gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and hydrogen concentrations. |
| Adult Dose | 300 mg PO hs or 150 mg bid |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in renal or liver impairment; if changes in renal function occur during therapy, consider adjusting dose or discontinuing treatment |

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Follow-up

Further Inpatient Care

- Management of renal injuries depends on the grade of injury and is linked to management of associated injuries. Grade of renal injury is best depicted in the scale developed by the American Association for the Surgery of Trauma and published in *J Trauma* in 1989 (see [Bibliography](#)).
 - Grade I - Renal contusion less than or equal to a small subcapsular hematoma
 - Grade II - Parenchymal laceration less than 1.0 cm deep without an expanding hematoma
 - Grade III - Parenchymal laceration more than 1.0 cm deep without urinary extravasation
 - Grade IV - Parenchymal laceration extending into the collecting system and/or segmented renal artery thrombosis
 - Grade V - Thrombosis of the main renal artery, avulsion of the pedicle, and shattered kidney with poor visualization
- In grade IV injuries, expectant treatment of the extravasation has a 60% success rate if ureteral outflow is not impeded.
 - Correct flow obstruction with stenting.
 - If urinary extravasation does not improve, perform percutaneous drainage.
 - Vascular injury indicates surgical intervention for repair, provided the warm ischemia time does not exceed 4 hours. Hemorrhage control also indicates surgical exploration.
- Management of acute ureteral injury primarily involves repair.
 - Perform debridement until a healthy bleeding ureter is reached.
 - Form a spatulated, stented, and tension-free repair.
 - The type of repair depends on the level of ureter injury and the length of ureter lost to injury and debridement.

Further Outpatient Care

- Further outpatient care depends upon the associated injuries and need for rehabilitation (eg, orthopedic or neurologic injuries).
- A follow-up CT scan is indicated in patients with renal injuries to assess the progress of healing; IVP is required as follow-up care for ureteral repairs.

in/Out Patient Meds

- Provide broad-spectrum antibiotics for prophylaxis against infection, especially in patients with urinary extravasation.

Transfer

- Assess capabilities of the staff and ED to handle patients who have multiple injuries with upper GU trauma; the decision to transfer is based on that assessment.
- Treat life-threatening injuries prior to transfer; stabilize and resuscitate the patient.
- The responsibility for transfer, choice of transfer modality, and selection of accepting facility lies with the transferring physician.

- The receiving physician confirms the ability of the receiving institution to handle the patient's condition.
- Institutional transfer protocol facilitates the process.
- Patients with upper GU trauma benefit from transfer when the following conditions exist at the transferring center:
 - CT scan is not available
 - No staff urologist
 - Multiple injuries that surpass hospital's resources
 - Unavailability of specialized care required by patient's injuries

Complications

- Renal trauma
 - Hemorrhage
 - Urinoma
 - Loss of function
 - Pseudoaneurysm formation
 - Arteriovenous fistula (rare)
 - Renal hypertension
 - Obstruction of the collecting system and renal artery aneurysm (pseudo)

Prognosis

- Prognosis in upper GU trauma depends on associated injuries.

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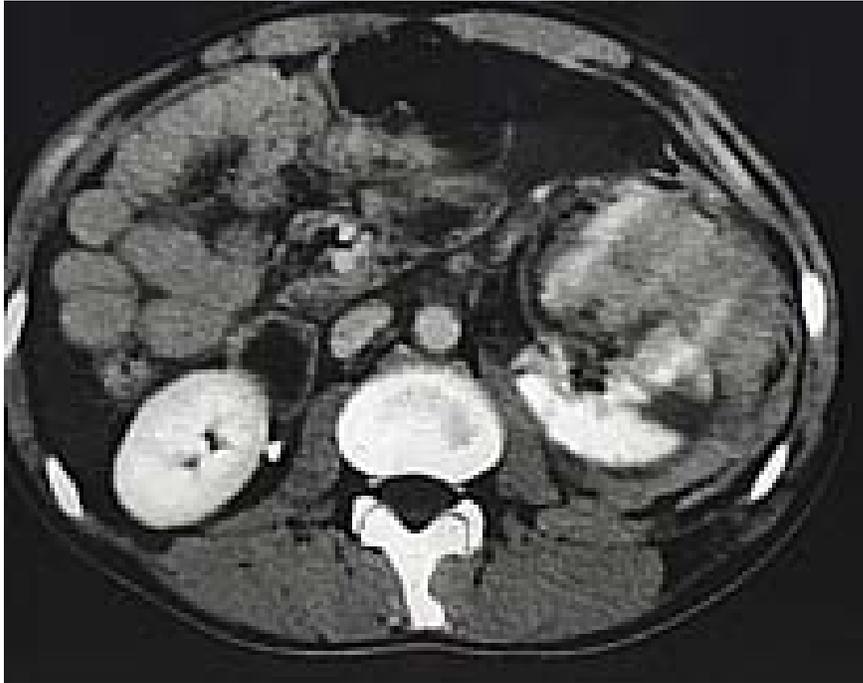
Miscellaneous

Special Concerns

- Pediatrics
 - The most frequently injured visceral organ is the kidney.
 - A higher incidence of preexisting renal disease or anomaly exists.
 - There is a higher incidence of hematuria on presentation and a lower incidence of rib fractures.
 - A dose of contrast material is 1 cc/kg.
 - CT scan is the preferred study.
 - Avoid arteriography.

- Avoid total nephrectomy when possible.
 - Ureteral trauma is extremely rare in children.
-

Pictures



Picture 1: CT scan of abdomen showing a lacerated left kidney with perirenal hematoma

Picture type: CT



Picture 2: Retrograde urethrogram showing a leak in the distal right ureter with an element of obstruction

Picture type: X-RAY



Picture 3: CT scan of abdomen and pelvis showing a urinoma

Picture type: CT

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CBRNE - Anthrax Infection

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Synonyms, Key Words, and Related Terms

anthrax, *Bacillus anthracis*, black bane, the fifth plague, wool-sorter's disease

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Introduction

Background

"Anthrax" means coal in Greek, and the disease is named after the appearance of its cutaneous form. Anthrax is described in the Old Testament, by the poet Virgil, and by the Egyptians. At the end of the 19th century, Robert Koch's experiments with anthrax led to the original theory of bacteria and disease. John Bell's work in inhalation anthrax led to wool disinfection processes and the term "wool sorter's disease."

Anthrax is caused by inhalation, skin exposure, or gastrointestinal (GI) absorption. Disease caused by inhalation is fatal, and symptoms usually begin days after exposure. This delay makes the initial

exposure to anthrax difficult to track.

An additional concern is use of anthrax as a biologic warfare agent. Recent reports reveal that Iraq produced 8500 L of anthrax for use during the Gulf War. During the last 2 years, the Centers for Disease Control and Prevention (CDC) have investigated several threats in the US including Indiana, Kentucky, Tennessee, and California. Most recently, an outbreak in Florida has been reported. Suspicion also should be raised if other potential biologic agents are diagnosed (ie, plague, smallpox, Ebola, Q fever).

Pathophysiology

Anthrax (*Bacillus anthracis*) is a large spore-forming gram-positive rod. Persistence of spores is aided by nitrogen and organic soil content, environmental pH greater than 6, and ambient temperature greater than 15°C. Drought or rainfall can trigger anthrax spore germination, while flies and vultures spread the spores.

B anthracis has a diameter of 1-1.5 µm and a length of 3-10 µm. It grows in culture as gray-white colonies that measure 4-5 mm and have characteristic comma-shaped protrusions. Anthrax is differentiated from other gram-positive rods on culture by lack of hemolysis and motility and by preferential growth on phenylethyl alcohol blood agar with characteristic gelatin hydrolysis and salicin fermentation.

Virulence depends on the bacterial capsule and the toxin complex. The capsule is a poly-D-glutamic acid that protects against leukocytic phagocytosis and lysis. Experiments by Sterne demonstrated that the capsule is vital for pathogenicity.

Anthrax toxins are composed of 3 entities: a protective antigen, a lethal factor, and an edema factor. The protective antigen is an 83-kd protein that binds to cell receptors within a target tissue. Once bound, a fragment is cleaved free to expose an additional binding site. This site can combine with edema factor to form edema toxin or with lethal factor to form lethal toxin. Edema toxin acts by converting adenosine triphosphate (ATP) to cyclic adenosine monophosphate (cAMP). Cellular cAMP levels are increased, leading to cellular edema within the target tissue. Lethal factor is not well understood, but recent work suggests that it may inhibit neutrophil phagocytosis, lyse macrophages, and cause release of tumor necrosis factor and interleukin-1.

Frequency

- **In the US:** During the last 20 years, the indigenous US incidence has been less than 1 case per year. From 1955-1994, US cases totaled 235, with 224 cases of cutaneous anthrax, 11 cases of inhalational anthrax, and 20 fatalities.
- **Internationally:** In 1958, approximately 100,000 cases of anthrax occurred worldwide. Exact figures do not exist because of reporting difficulties in Africa. Anthrax is endemic in Africa and

Asia despite vaccination programs. Sporadic outbreaks have occurred as a result of both agricultural and military disruptions. During the 1978 Rhodesian civil war, failure of veterinary vaccination programs led to a human epidemic, causing 6500 anthrax cases and 100 fatalities. A mishap at a military microbiology facility in the former Soviet Union in 1979 resulted in 66 deaths. Human anthrax often is associated with agricultural or industrial workers who come in contact with infected animal tissue.

Mortality/Morbidity

Anthrax is primarily zoonotic. Most anthrax disease is cutaneous (95%). The remaining cases of the disease are inhalational (5%) and GI (<1%).

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Clinical

History

Exposure comes from contact with animals or animal products (eg, hides in African export shops). Members of military forces may become exposed in biologic warfare situations.

Physical

Physical findings are nonspecific. The incubation period is 1-6 days following exposure. Chest radiography may reveal a widened mediastinum, due to hilar adenopathy, with pleural effusions.

- Cutaneous anthrax
 - In the most common cutaneous form of anthrax, spores inoculate a host through skin lacerations, abrasions, or biting flies. Incubation is 2-5 days.
 - The disease begins as a nondescript papule that becomes a 1-cm vesicle within 2 days. Occasionally, surrounding edema is severe and can lead to airway compromise if present in the neck.
 - The skin in infected areas may become edematous and necrotic but not purulent. Such skin lesions have been described as "malignant pustules" after their characteristic appearance, despite being neither malignant nor pustular. Lesions are painless but on occasion are slightly pruritic.
 - Spore germination occurs within macrophages at the site of inoculation. Anthrax bacilli are isolated easily from the vesicular lesions and can be observed on Gram stain.
 - The initial lesion ruptures after a week and progresses to a characteristic black eschar.

- Differential diagnoses of the skin lesion include tularemia, plague, cutaneous diphtheria, *Staphylococcus* infections, *Rickettsia* infections, and orf (a viral disease of livestock).
- Cutaneous anthrax usually remains localized, but without treatment, it disseminates systemically in up to one fifth of cases. Antibiotic therapy prevents dissemination but does not affect the natural history of the lesion. With treatment, mortality is approximately 1%.
- Gastrointestinal anthrax
 - GI anthrax is a rare form of infection. It occurs from eating infected, undercooked meat. Only 11 cases have been reported, all in underdeveloped countries.
 - Symptoms usually begin a few days after ingestion of the contaminated meat. Abdominal pain and fever occur first, followed by nausea, vomiting, and diarrhea.
 - In this illness, spores invade GI mucosa. As spores are transported to mesenteric lymph nodes, replication and bacteremia begin. Ascites and ileus follow as the lymphatic system becomes occluded with the large number of bacilli. Peritoneal fluid is turbid with the presence of leukocytes and red blood cells from hemorrhagic adenitis. Vascular stasis occurs, and the stomach and intestine become edematous.
 - In some cases, necrosis and ulceration at the site of infection produce GI hemorrhage. Shock occurs from interstitial and intraperitoneal volume losses.
 - Anthrax toxin further causes intrinsic renal failure independent of prerenal azotemia.
 - Death is rapid without antibiotic therapy and aggressive volume resuscitation. Mortality is 50%.
- Meningeal anthrax
 - Meningeal anthrax is usually the result of bacteremia from the cutaneous, GI, or inhalational form of the disease. It also has occurred without a primary focus.
 - The meninges are characteristically hemorrhagic and edematous.
 - Of the Soviet cases that underwent autopsy, 21 of 42 (50%) had meningeal involvement.
 - Mortality is near 100%.

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Differentials

Aneurysms, Abdominal

CBRNE - Plague

Coccidioidomycosis

Diphtheria

Dissection, Aortic

Gastroenteritis

Meningitis

Pleural Effusion

Pneumonia, Bacterial

Pneumonia, Mycoplasma
Pneumonia, Viral
Subarachnoid Hemorrhage
Superior Vena Cava Syndrome

Other Problems to be Considered

Ecthyma (*Pseudomonas aeruginosa* and *Staphylococcal* infections)

Glanders (*Pseudomonas pseudomallei*)

Histoplasmosis

Leprosy

Orf (*Rickettsia akari*)

Psittacosis

Rat bite fever (*Streptococcus moniliformis*, *Spirillum minus*)

Rickettsia

Tularemia

Typhoid

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Workup

Lab Studies

- The diagnostic assay is the serum enzyme-linked immunosorbent assay test (ELISA). Measurement of antibody titers can prove past infection or vaccination.
- Cerebral spinal fluid contains blood and leukocytosis in meningeal anthrax.
- Anthraxin skin test is available in the former Soviet Union.
- Polymerase chain reaction (PCR) may help in early diagnosis of disease but remains in development.

- A Gram stain is the easiest means of first identifying suggested cases. Anthrax appears as a large gram-positive rod.

Imaging Studies

- Chest radiography: Inhalational anthrax often does not appear on chest films as a typical pneumonia; therefore, pulmonary densities often are absent. A prominent mediastinum with pleural effusions may be present. The prominent mediastinum is due to hilar lymphadenopathy.

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Treatment

Prehospital Care

As with any potential epidemic biologic exposure, patients should be decontaminated in the field when possible, and paramedical health care workers should wear masks and gloves.

US Army Medical Research Institute of Infectious Diseases (USAMRIID) recommends barrier procedures (ie, gown, gloves, surgical mask) with secretion precautions.

Emergency Department Care

- Workup includes isolation; barrier protection; resuscitation; sampling of blood, wounds, and cerebral spinal fluid (when applicable); radiography; and rapid initiation of intravenous antibiotic therapy.
- If risk of exposure is considerable, initiate postexposure prophylaxis. This includes chemoprophylaxis and vaccination, if the vaccine is available.
- Patients can be placed in a normal hospital room with barrier nursing procedures (ie, gown, gloves, mask) and secretion precautions (ie, special handling of potentially infectious dressings, drainage, and excretions).

Consultations

Anthrax is a reportable disease; notify local health care authorities of suspected cases. In addition, consultation with an infectious disease specialist or the CDC may be warranted, although treatment of patients in whom anthrax is suspected is straightforward. If biologic terrorism is a threat, consider contacting the Federal Bureau of Investigations (FBI) through the local police department.

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Medication

First-line treatment for anthrax is penicillin, unless biologically mutant strains are suspected (as in germ warfare). For such situations, the CDC recommends ciprofloxacin. A wide variety of antibiotics provides adequate coverage. For patients with severe anthrax, corticosteroids are recommended in addition to antibiotics.

Measures to prevent anthrax infection include vaccination, decontamination, and prophylactic treatment. The CDC recommends chemoprophylaxis plus vaccination, if the vaccine is available. Prophylaxis for asymptomatic patients lasts 4-6 wk. Vaccination administered to people with high risk of exposure may not confer complete immunity and currently has a high rate of associated adverse effects.

Despite early treatment, those infected with inhalational, GI, or meningeal anthrax have a very poor prognosis. Although prophylaxis and vaccinations confer almost complete protection, adequately providing immunity to a potentially exposed community is extremely difficult.

A recent CDC recommendation indicates treatment until anthrax is excluded. Initial therapy is administration of an oral fluoroquinolone, including to pregnant women. Children should receive amoxicillin. Continue treatment for 4 wk (with 3 doses of vaccine) or for 8 wk if no vaccine is available. In general, continue antibiotics for 2 wk after symptoms resolve. Modify treatment in the presence of penicillin-resistant strains.

Antibiotics

Therapy must cover all likely pathogens in the context of this clinical setting.

| | |
|-------------------|--|
| Drug Name | Penicillin G (Pfizerpen), Penicillin V (Veetids)- DOC; interferes with synthesis of cell wall mucopeptide during active multiplication, resulting in bactericidal activity against susceptible microorganisms. Begin treating all patients with IV dosing. |
| Adult Dose | PCN G: 8-12 million U IV divided q4-6h PCN V: 200-500 mg PO q6h |
| Pediatric Dose | PCN V: 25-50 mg/kg/d PO divided bid/qid PCN G: 100,000-150,000 U/kg/d IV divided q4-6h |
| Contraindications | Documented hypersensitivity |

| | |
|--------------|---|
| Interactions | Probenecid can increase effects of penicillin; coadministration of tetracyclines can decrease effects of penicillin |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Caution in impaired renal function |

| | |
|-------------------|---|
| Drug Name | Ciprofloxacin (Cipro)- DOC when mutant strains are suspected (as in germ warfare). Indicated for inhalational anthrax host exposure. Inhibits bacterial DNA synthesis and, consequently, growth by inhibiting DNA-gyrase in susceptible organisms. Initiate treatment immediately following suspected or confirmed anthrax exposure. |
| Adult Dose | 500 mg PO q12h for 60 d; alternatively, 400 mg IV q12h for 60 d |
| Pediatric Dose | 15 mg/kg PO q12h for 60 d; not to exceed 500 mg/dose; alternatively, 10 mg/kg IV q12h for 60 d; not to exceed 400mg/dose |
| Contraindications | Documented hypersensitivity |
| Interactions | Antacids, iron salts, and zinc salts may reduce serum levels; administer antacids 6 h before or 2 h after taking fluoroquinolones; ciprofloxacin reduces therapeutic effects of phenytoin; probenecid may increase ciprofloxacin serum concentrations May increase toxicity of theophylline, caffeine, and cyclosporine; may increase effects of anticoagulants (monitor PT) |
| Pregnancy | C - Safety for use during pregnancy has not been established. However, it is recommended for anthrax inhalation post-exposure. |
| Precautions | In prolonged therapy, perform periodic evaluations of organ system functions (eg, renal, hepatic, hematopoietic); adjust dose in renal function impairment; superinfections may occur with prolonged or repeated antibiotic therapy |

| | |
|-------------------|---|
| Drug Name | Levofloxacin (Levaquin)- Inhibits growth of susceptible organisms by inhibiting DNA gyrase and promoting breakage of DNA strands. |
| Adult Dose | 500 mg PO q24h for 60 d |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Antacids, iron salts, and zinc salts may reduce serum levels; administer antacids 2-4 h before or after taking fluoroquinolones; cimetidine may interfere with metabolism of fluoroquinolones; levofloxacin reduces therapeutic effects of phenytoin; probenecid may increase levofloxacin serum concentrations May increase toxicity of theophylline, caffeine, cyclosporine, and digoxin (monitor digoxin levels); may increase effects of anticoagulants (monitor PT) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

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|-------------|---|
| Precautions | In prolonged therapy, perform periodic evaluations of organ system functions (eg, renal, hepatic, hematopoietic); adjust dose in renal function impairment; superinfections may occur with prolonged or repeated antibiotic therapy |
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|-------------------|---|
| Drug Name | Streptomycin sulfate- Aminoglycoside antibiotic recommended when less potentially hazardous therapeutic agents are ineffective or contraindicated. |
| Adult Dose | 30 mg/kg/d IM; not to exceed 2 g/d |
| Pediatric Dose | 25-30 mg/kg/d IM; not to exceed 1-1.5 g/d |
| Contraindications | Documented hypersensitivity, non-dialysis-dependent renal insufficiency |
| Interactions | Nephrotoxicity may be increased with aminoglycosides, cephalosporins, penicillins, amphotericin B, and loop diuretics |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Narrow therapeutic index; not intended for long-term therapy; caution in patients with renal failure not receiving dialysis; caution with myasthenia gravis, hypocalcemia, and conditions that depress neuromuscular transmission |

| | |
|-------------------|--|
| Drug Name | Tetracycline (Sumycin)- Treats susceptible bacterial infections caused by gram-positive and gram-negative organisms and infections caused by <i>Mycoplasma</i> , <i>Chlamydia</i> , and <i>Rickettsia</i> species. Inhibits bacterial protein synthesis by binding with 30S and, possibly, 50S ribosomal subunit(s) of susceptible bacteria. |
| Adult Dose | 500 mg PO/IV q6h |
| Pediatric Dose | <8 years: Not recommended >8 years: 10-20 mg/lb (25-50 mg/kg) PO/IV q6h |
| Contraindications | Documented hypersensitivity; severe hepatic dysfunction |
| Interactions | Bioavailability decreases with antacids containing aluminum, calcium, magnesium, iron, or bismuth subsalicylate; can decrease effects of oral contraceptives, causing breakthrough bleeding and increased risk of pregnancy; tetracyclines can increase hypoprothrombinemic effects of anticoagulants |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Photosensitivity may occur with prolonged exposure to sunlight or tanning equipment; reduce dose in renal impairment; consider drug serum level determinations in prolonged therapy; tetracycline use during tooth development (last one half of pregnancy through age 8 y) can cause permanent discoloration of teeth; Fanconilike syndrome may occur with outdated tetracyclines |

| | |
|-------------------|---|
| Drug Name | Doxycycline (Bio-Tab, Doryx, Vibramycin)- Inhibits protein synthesis and, thus, bacterial growth by binding with 30S and, possibly, 50S ribosomal subunits of susceptible bacteria. |
| Adult Dose | Loading dose: 100 mg PO/IV Maintenance dose: 50-100 mg PO/IV q12h |
| Pediatric Dose | <8 years: Not recommended <45 kg: 2.5 mg/kg q12h >45 kg: Administer as in adults |
| Contraindications | Documented hypersensitivity; severe hepatic dysfunction |
| Interactions | Bioavailability decreases with antacids containing aluminum, calcium, magnesium, iron, or bismuth subsalicylate; tetracyclines can increase hypoprothrombinemic effects of anticoagulants; tetracyclines can decrease effects of oral contraceptives, causing breakthrough bleeding and increased risk of pregnancy |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Bioavailability decreases with antacids containing aluminum, calcium, magnesium, iron, or bismuth subsalicylate; tetracyclines can increase hypoprothrombinemic effects of anticoagulants; tetracyclines can decrease effects of oral contraceptives, causing breakthrough bleeding and increased risk of pregnancy |

| | |
|-------------------|---|
| Drug Name | Chloramphenicol (Chloromycetin)- Binds to 50S bacterial-ribosomal subunits and inhibits bacterial growth by inhibiting protein synthesis. Effective against gram-negative and gram-positive bacteria. |
| Adult Dose | 50-100 mg/kg/d PO/IV divided q6h |
| Pediatric Dose | 50-75 mg/kg/d PO/IV divided q6h |
| Contraindications | Documented hypersensitivity |
| Interactions | Concurrently with barbiturates, chloramphenicol serum levels may decrease while barbiturate levels may increase, causing toxicity; manifestations of hypoglycemia may occur with sulfonyleureas; rifampin may reduce serum chloramphenicol levels, presumably through hepatic enzyme induction; may increase effects of anticoagulants; may increase serum hydantoin levels, possibly resulting in toxicity; chloramphenicol levels may be increased or decreased |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
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| Precautions | Use only for indicated infections or as prophylaxis for bacterial infections; serious and fatal blood dyscrasias (aplastic anemia, hypoplastic anemia, thrombocytopenia, granulocytopenia) can occur; evaluate baseline and perform periodic blood studies approximately every 2 d while in therapy; discontinue upon appearance of reticulocytopenia, leukopenia, thrombocytopenia, anemia, or findings attributable to chloramphenicol; adjust dose in liver or kidney dysfunction; caution in pregnancy at term or during labor because of potential toxic effects on fetus (gray syndrome) |
|-------------|--|

Corticosteroids

Agents used for severe edema, meningitis, or swelling in the head and neck region.

| | |
|-------------------|--|
| Drug Name | Dexamethasone (Decadron, Dexasone)- Used in various inflammatory diseases. May decrease inflammation by suppressing migration of polymorphonuclear leukocytes and reversing increased capillary permeability. |
| Adult Dose | 0.75-0.90 mg/kg/d PO/IV/IM divided q6h |
| Pediatric Dose | 0.25-0.5 mg/kg PO/IV/IM q6h |
| Contraindications | Documented hypersensitivity; active bacterial or fungal infection |
| Interactions | Effects decrease with coadministration of barbiturates, phenytoin, and rifampin; dexamethasone decreases effect of salicylates and vaccines used for immunization |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Increases risk of multiple complications, including severe infections; monitor adrenal insufficiency when tapering drug; abrupt discontinuation of glucocorticoids may cause adrenal crisis; hyperglycemia, edema, osteonecrosis, myopathy, peptic ulcer disease, hypokalemia, osteoporosis, euphoria, psychosis, myasthenia gravis, growth suppression, and infections are possible complications of glucocorticoid use |

| | |
|-------------------|---|
| Drug Name | Prednisone (Deltasone, Orasone, Meticorten)- Useful in inflammatory and allergic reactions. May decrease inflammation by reversing increased capillary permeability and suppressing PMN activity. |
| Adult Dose | 1-2 mg/kg or 5-60 mg PO qd |
| Pediatric Dose | 0.5-2 mg/kg/d PO |
| Contraindications | Documented hypersensitivity; in patients with viral, fungal, or tubercular skin infections |

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| Interactions | Coadministration with estrogens may decrease prednisone clearance; concurrent use with digoxin may cause digitalis toxicity secondary to hypokalemia; phenobarbital, phenytoin, and rifampin may increase metabolism of glucocorticoids (consider increasing maintenance dose); monitor for hypokalemia with coadministration of diuretics |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Abrupt discontinuation of glucocorticoids may cause adrenal crisis; hyperglycemia, edema, osteonecrosis, myopathy, peptic ulcer disease, hypokalemia, osteoporosis, euphoria, psychosis, myasthenia gravis, growth suppression, and infections may occur with glucocorticoid use |

Vaccination

The FDA approved a standard anthrax vaccine designated "anthrax vaccine adsorbed" (AVA). Sterile filtrate of cultures of an avirulent strain that elaborates protective antigen. No controlled trials are available. Efficacy in inhalation (biowarfare) anthrax is questionable. Although not endorsed by this site, the [Anthrax Vaccine Homepage](#) is a helpful web site with information on current research and controversy.

| | |
|-------------------|---|
| Drug Name | Anthrax vaccine adsorbed (AVA)- Vaccine used in high-risk situations. Along with prophylactic antibiotics, AVA can be administered in potential exposure. |
| Adult Dose | 0.5 mL SC at 2 and 4 wk and at 6, 12, and 18 mo |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | No controlled trials available demonstrating consistent efficacy |

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Follow-up

Prognosis

- Inhalational anthrax and its subsequent systemic infection have a mortality rate approaching 100%. If treatment is initiated in the incubation period of 1-6 days and before the manifestation of

symptoms, mortality can decrease to 1%.

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Miscellaneous

Medical/Legal Pitfalls

- Vaccine: Better protection, more extensive testing, more rigorous Food and Drug Administration (FDA) approval, reduction of adverse effects, and a simpler dosing schedule are needed. No human studies are available that document efficacy of available vaccines.
-

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CBRNE - Arsenicals, Arsine

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Synonyms, Key Words, and Related Terms

AsH₃, arsine gas, arseniuretted hydrogen, arsenous hydride, arsenic trihydride, hydrogen arsenide

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Introduction

Background

Arsine, the most toxic form of arsenic, has some properties that may make it useful as a chemical warfare (CW) agent; it is a colorless, odorless, nonirritating gas that is 2.5 times denser than air. At concentrations above 0.5 ppm, a garliclike odor may be noted, but arsine is toxic at much lower concentrations.

Although it has been investigated as a CW agent, arsine has no recorded battlefield use. During and prior

to World War II, the British studied this agent and rejected its use in the field. They concluded it was more than 10 times less toxic than phosgene (CG). In addition, it is difficult to manufacture and is highly flammable. So although it was determined that arsine was not a useful battlefield CW agent, it may be useful as a small-scale weapon of assassination or terror.

In contrast, several arsine-derived organoarsenic compounds have been developed and used as CW agents, including lewisite (beta-chlorovinylchloroarsine), adamsite (diphenylaminearsine), Clark I (diphenylchlorarsine), and Clark II (diphenylcyanoarsine).

Possible sources of occupational exposure are many and include the semiconductor industry during microchip production and other industries in which workers are involved in galvanizing, soldering, etching, and lead plating. It also can be produced inadvertently by mixing arsenic-containing insecticides and acids.

Pathophysiology

Inhaled arsine gas is distributed rapidly and causes massive red blood cell hemolysis that potentially can lead to global cellular hypoxia. The mechanisms of hemolysis are not elucidated fully, but studies have revealed the following: arsine causes a nonspecific disruption of ion gradients, leading to cell membrane instability; cell membrane sulfhydryl groups are probable targets of arsine toxicity; and hemoglobin is an important subcellular target of arsine toxicity.

Arsine poisoning can lead to acute renal tubular necrosis and ultimately to oliguric/anuric renal failure by the following mechanisms: direct toxic effect of arsine on renal tissue; heme-pigment nephropathy; and renal hypoxia secondary to massive hemolysis and decreased oxygen carrying capacity of the blood.

Mortality/Morbidity

Most reported deaths are believed to be secondary to acute renal failure. Prior to the advent of hemodialysis, 100% of arsine-induced renal failure cases were fatal. More recently, mortality rates for patients with acute arsine toxicity are no higher than 25%.

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Clinical

History

Most patients report little or no discomfort at the time of exposure. Although a garliclike odor may be

noted with higher ambient arsine concentrations, serious toxicity may result from clinically nondetectable exposures. Following exposure, a dose-dependent latent period ensues, lasting up to 24 hours.

The classic triad of symptoms in sublethal arsine exposures includes abdominal pain, hematuria, and jaundice. Symptoms by organ system are as follows:

- General - Fever, chills, shivering, thirst, malaise
- Neurologic - Headache, confusion, dizziness, paresthesias
- Pulmonary - Dyspnea
- Gastrointestinal - Nausea, vomiting, abdominal pain, anorexia, jaundice
- Genitourinary - Red or dark-colored urine, flank pain, decreased urine output
- Muscle - Weakness, cramping

Physical

Physical signs and their severity depend on the concentration of arsine gas and the length of the patient's exposure to it.

- Vital signs - Hyperthermia, tachypnea, tachycardia, hypotension
- Head, ears, eyes, nose, and throat (HEENT) - Discoloration of conjunctivae (red, orange, brown, or brassy; reportedly distinct from hyperbilirubinemia), scleral icterus, garlic odor to breath (possible)
- Pulmonary - Crackles (from acute respiratory distress syndrome [ARDS] in severe exposures)
- Gastrointestinal - Abdominal tenderness, hepatomegaly
- Genitourinary - Costovertebral angle tenderness, colored urine (red, brown, or green from hemoglobinuria and/or methemoglobinuria)
- Extremities - Possible paresthesias and Mees lines with chronic arsenic toxicity from arsine exposure

Causes

- Arsine gas is used in the semiconductor industry when depositing arsenic on microchips. Exposures also may occur from producing, cleaning, or reclaiming gallium arsenide wafers.
- Arsine is released during the (usually accidental) production of hydrogen in an acid medium in contact with arsenic-contaminated metals.
- Arsine potentially may be used as a CW agent.
 - Suspect arsine exposure if multiple victims present in a delayed fashion with hematuria, abdominal or flank pain, and jaundice.
 - Severe acute arsine exposures result in sudden death.

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Differentials

Anemia, Acute
CBRNE - Chemical Warfare Mass Casualty Management
CBRNE - Cyanides, Hydrogen
CBRNE - Evaluation Of A Chemical Warfare Victim
Cholecystitis and Biliary Colic
Cholelithiasis
Hemolytic Uremic Syndrome
Hepatitis
Hyperkalemia
Leptospirosis
Malaria
Methemoglobinemia
Renal Calculi
Renal Failure, Acute
Rhabdomyolysis
Smoke Inhalation
Toxicity, Arsenic
Urinary Tract Infection, Female
Urinary Tract Infection, Male

Other Problems to be Considered

Cold agglutinin disease

Paroxysmal nocturnal hemoglobinuria

Pyrogalllic acid toxicity

Stibine gas toxicity

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Workup

Lab Studies

- Complete blood count
 - Hemolytic anemia - Coombs test, negative; may be severe and rapidly developing, with pink serum resulting from free hemoglobin
 - Elevated white blood cell count - May be seen early
- Urinalysis
 - Hemoglobinuria (possible methemoglobinuria)
 - Proteinuria (with possible tubular casts)
- Arsenic levels
 - Blood and urine arsenic levels are elevated acutely, but these findings are not necessarily helpful in treatment decisions.
 - A 24-hour urine arsenic may help in monitoring chronic, low-level arsine exposures.

Imaging Studies

- No routine imaging studies are indicated.
- Chest radiography is indicated to detect ARDS in patients with pulmonary symptoms.

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Treatment

Prehospital Care

- Properly protected personnel should remove the victim from continued exposure to arsine.
- Provide high-flow oxygen.

Emergency Department Care

- Exchange transfusion is the treatment of choice for patients with severe hemolysis.
 - Supports oxygen carrying capacity of the blood
 - Removes free hemoglobin
 - Removes arsine and arsenic dihydride residues
- Chelating agents (eg, 2,3-dimercaptopropanol, British antilewisite [BAL]) have not been shown to be of benefit in acute arsine toxicity.

Consultations

- Alert the blood bank regarding the possibility of exchange transfusion.
- Initiate nephrology consultation to start hemodialysis in patients with acute renal failure.

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Medication

Diuresis with IV mannitol and urinary alkalinization with sodium bicarbonate may be of benefit prior to the onset of renal failure.

Diuretics

Promote urine flow.

| | |
|-------------------|--|
| Drug Name | Mannitol (Osmitol, Resectisol)- Increases osmotic pressure of glomerular filtrate, inducing an osmotic gradient that inhibits tubular resorption of water and electrolytes, resulting in increased urinary output. |
| Adult Dose | Usual loading dose is 0.5-1 g/kg body weight IV, followed by 0.25-0.5 g/kg q4-6h to maintain diuresis |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity, anuria, severe pulmonary congestion, progressive renal damage, severe dehydration, active intracranial bleeding, and progressive heart failure |
| Interactions | May enhance renal excretion of lithium |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Carefully evaluate cardiovascular status before rapid administration of mannitol, since a sudden increase in extracellular fluid may lead to fulminating CHF; avoid pseudoagglutination; when blood is given simultaneously, add at least 20 mEq of sodium chloride to each L of mannitol solution; do not give electrolyte-free mannitol solutions with blood |

Urinary Alkalinization Agents

Decreases risk of heme pigment-induced renal injury from arsine-related hemolysis.

| | |
|-------------------|--|
| Drug Name | Sodium bicarbonate (Neut)- Dosing of sodium bicarbonate to induce urinary alkalization not standardized; urinary alkalization may prevent heme-pigment nephropathy by decreasing hemoglobin crystallization in renal tubules and/or by decreasing iron uptake by tubular epithelium. |
| Adult Dose | 1 mEq/kg IV as bolus, followed by continuous IV maintenance infusion of sodium bicarbonate-containing fluids; examples of infusate include D5W with 3 ampules of sodium bicarbonate (132-150 mEq) per L or D5-1/2NS with 2 ampules of sodium bicarbonate (88-100 mEq) per L |
| Pediatric Dose | Administer as in adults |
| Contraindications | Alkalosis, hypernatremia, hypocalcemia, severe pulmonary edema, and unknown abdominal pain |
| Interactions | Urinary alkalization induced by increased sodium bicarbonate concentrations may cause decreased levels of lithium, tetracyclines, chlorpropamide, methotrexate, and salicylates; increases levels of amphetamines, pseudoephedrine, flecainide, anorexiant, mecamlamine, ephedrine, quinidine, and quinine |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Establish urinary flow with intravascular volume repletion prior to alkalizing urine; administering bicarbonate may induce hypokalemia and other electrolyte disorders |

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Follow-up

Further Inpatient Care

- Monitor renal function; initiate hemodialysis as necessary for acute renal failure.
- Monitor hemoglobin levels; perform transfusions to maintain oxygen carrying capacity of the blood.

Further Outpatient Care

- Monitor patient for signs of chronic arsenic toxicity.

in/Out Patient Meds

- Chelating agents (eg, BAL) may be used to treat chronic arsenic toxicity.

- Chronic arsenic toxicity from arsine exposure is treated no differently than exposure from other sources.
- See [Toxicity, Arsenic](#) for more information.

Deterrence/Prevention

- Train workers in high-risk industries to avoid toxic arsine exposures.
- Screen workers in the same environment as those persons already exposed to acute arsine poison.

Complications

- Hemolytic anemia
- Renal failure
- Hyperkalemia
- Death
 - Overwhelming exposures cause rapid death from massive hemolysis.
 - In those who survive acute exposures, most deaths occur from renal failure.

Prognosis

- Patients who reach medical attention should survive with modern, supportive medical care.
- Historically, patients who developed renal failure had 100% mortality. More recent (but still dated) studies report a mortality rate from arsine poisoning of approximately 25%.

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Miscellaneous

Medical/Legal Pitfalls

- A multiple casualty arsine exposure incident may overwhelm hospital resources to perform exchange transfusions.
 - Monitor arsine-exposed victims serially until the possibility of chronic arsenic toxicity has been ruled out.
-

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CBRNE - Biological Warfare Agents

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Historic Aspects of Biological Warfare Agents

Biological weapons include any organism or toxin found in nature that can be used to incapacitate, kill, or otherwise impede an adversary. Biological weapons are characterized by low visibility, high potency, substantial accessibility, and relatively easy delivery.

The potential spectrum of bioterrorism ranges from hoaxes and use of nonmass casualty agents by individuals or small groups to state-sponsored terrorism that employs classic biological warfare (BW) agents and can produce mass casualties. Such scenarios would present serious challenges for patient treatment and for prophylaxis of exposed persons. Environmental contamination could pose continuing threats.

The use of biological agents is not a new concept, and history is replete with examples of biological weapon use. Prior to the 20th century, biological warfare took on 3 main forms: (1) deliberate poisoning of food and water with infectious material, (2) use of microorganisms or toxins in some form of weapon system, and (3) use of

biologically inoculated fabrics.

Attempts to use BW date back to antiquity. Scythian archers infected their arrows by dipping them in decomposing bodies or in blood mixed with manure as far back as 400 BC. Persian, Greek, and Roman literature from 300 BC quote examples of the use of animal cadavers to contaminate wells and other sources of water. In 190 BC, at the Battle of Eurymedon, Hannibal won a naval victory over King Eumenes II of Pergamon by firing earthen vessels full of venomous snakes into the enemy ships.

In the 12th century AD, during the battle of Tortona, Barbarossa used the bodies of dead soldiers to poison wells. In the 14th century AD during the siege of Kaffa, the attacking Tartar force hurled the corpses of those who died of plague into the city to attempt to inflict a plague epidemic upon the enemy. This was repeated in 1710 when the Russians besieging Swedish forces at Reval in Estonia catapulted plague cadavers.

In the 18th century AD during the French and Indian War,

British forces in North America gave blankets from smallpox patients to the Native Americans to create a transmission of the disease to the immunologically naïve tribes. In 1863, a confederate surgeon was arrested and charged with attempting to import yellow fever-infected clothes into the northern parts of the US during the Civil War.

Biological warfare became more sophisticated against both animals and humans during the 1900s. During World War I, the Germans developed anthrax, glanders, cholera, and a wheat fungus for use as biological weapons. They allegedly spread plague in St Petersburg, infected mules with glanders in Mesopotamia, and attempted to do the same with the horses of the French Cavalry.

In 1925, the Geneva Protocol was signed by 108 nations, including the 5 permanent members of the UN Security Council. This was the first multilateral agreement that extended prohibition of chemical agents to biological agents. No method for verification of compliance was addressed.

During

World War II, the Japanese operated a secret BW research facility in Manchuria and carried out human experiments on Chinese prisoners. They exposed more than 3000 victims to plague, anthrax, syphilis, and other agents. Victims were observed for development of disease, and autopsies were performed.

In 1942, the US formed the War Research Service. Anthrax and botulinum toxin initially were investigated for use as weapons, and sufficient quantities of botulinum toxin and anthrax cattle cakes were stockpiled by June 1944 to allow limited retaliation if the Germans first used biological agents. The British tested anthrax bombs on Gruinard Island off the northwest coast of Scotland in 1942 and 1943 and then prepared and stockpiled anthrax-laced cattle cakes.

The US continued research on various offensive biological weapons during the 1950s and 1960s. From 1951-1954, simulants (*Bacillus globigii*, *Serratia marcescens*) were released off both coasts of the US to demonstrate the

vulnerability of American cities to biological agent attacks. This vulnerability was tested again in 1966 when the simulant *B globigii* was released in the New York subway system.

In 1957, the British government decided to end its offensive BW capabilities and destroy its weapon stockpiles.

The US terminated its offensive biological weapons program in 1969 for microorganisms and in 1970 for toxins. The US is a signatory nation of the Biological Toxin Weapons Convention of 1972. This convention addressed the prohibition of the development, production, stockpiling, and destruction of bacteriologic and toxin weapons. Signatories to this agreement are required to submit information annually to the United Nations concerning facilities where biological defense research is being conducted, scientific conferences that are held at specified facilities, exchanges of scientists or information, and disease outbreaks. American stockpiles of biological weapons were destroyed completely by

1973.

During the Vietnam War, Vietcong guerrillas used punji stakes dipped in feces to increase the morbidity from wounding by these stakes.

The Soviet Union (USSR) continued to develop biological weapons from 1950-1980. In the 1970s, the USSR and its allies were suspected of having used "yellow rain" (trichothecene mycotoxins) during campaigns in Laos, Cambodia, and Afghanistan. In 1979, an accidental release of anthrax from a weapons facility in Sverdlovsk, USSR, killed at least 66 people. The Russians denied this accident until 1992.

Since the 1980s, terrorist organizations have become users of biological agents. The most frequent bioterrorism episodes have involved contamination of food and water. In September and October of 1984, 751 persons were infected with *Salmonella typhimurium* after an intentional contamination of restaurant salad bars in Oregon by followers of the Bhagwan Shree Rajneesh.

In 1985, Iraq began an offensive biological weapons program producing

anthrax, botulinum toxin, and aflatoxin. During Operation Desert Shield, the coalition of allied forces faced the threat of chemical and biological agents. Following the Persian Gulf War, Iraq disclosed that it had bombs, Scud missiles, 122-mm rockets, and artillery shells armed with botulinum toxin, anthrax, and aflatoxin. They also had spray tanks fitted to aircraft that could distribute 2000 L over a target.

Currently, 17 countries are suspected of having an offensive BW program. In 1992, 20 people were administered chemoprophylaxis after a Virginia man sprayed his roommates with a substance that he

claimed was anthrax. In 1994, a Japanese sect of the Aum Shinrikyo cult attempted an aerosolized release of anthrax from the tops of buildings in Tokyo. In 1995, 2 members of a Minnesota militia group were convicted of possession of ricin, which they had produced themselves for use in retaliation against local government officials. In 1996, an Ohio man was able to obtain bubonic plague cultures through the mail.

In 1997, the Defense Against Weapons of Mass Destruction Act directed the Department of Defense to establish a domestic preparedness program to improve the ability of local, state, and federal agencies to respond to biological incidents. During 1998 and 1999, multiple hoaxes occurred involving the threatened release of anthrax in the US that resulted in decontamination and antibiotic prophylaxis for the intended victims. Nearly 6000 persons across the US have been affected by these threats. To date, none of these cases involved actual anthrax bacteria. According to a study by the Centers for Disease Control and Prevention (CDC), an intentional release of anthrax by a bioterrorist in a major US city would result in an economic impact of \$477.8 million to \$26.2 billion per 100,000 persons exposed.

The threat that biological agents will be used on both military forces and civilian populations is now more likely than at any point in all of history.

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Delivery, Dissemination, and Detection of Biological Warfare Agents

Biological agents are easy to acquire, synthesize, and use. The small amount of agents necessary to kill hundreds of thousands of people in a metropolitan area make the concealment, transportation, and dissemination of biological agents relatively easy. In addition, BW agents are difficult to detect or protect against; they are invisible, odorless, tasteless, and their dispersal can be performed silently.

Dissemination of BW agents may occur by aerosol sprays, explosives (artillery, missiles, detonated bombs), or food or water contamination. Variables that can alter the effectiveness of a delivery system include particle size of the agent, stability of the agent under desiccating conditions, UV light, wind speed, wind direction, and atmospheric stability.

The use of an explosive device to deliver and disseminate biological agents is not very effective, since such agents tend to be inactivated by the blast. Contamination of municipal water supplies requires an unrealistically large amount of

agent and introduction into the water after it passes through a regional treatment facility.

To be an effective biological weapon, airborne pathogens must be dispersed as fine particles less than 5 μm in size. Infection with an aerosolized agent usually requires deep inspiration of an infectious dose. Advanced weapons systems (eg, warheads, missiles) are not required for the aerosolized delivery of biological agents. Low-technology aerosolization methods including agricultural crop-dusters; aerosol generators on small boats, trucks, or cars; backpack sprayers; and even purse-size perfume atomizers suffice. Aerosolized dispersal of biological agents is the mode most likely to be used by terrorists and military groups.

Detection of biological agents involves either finding the agent in the environment or medical diagnosis of the agent's effect on human or animal victims. Early detection of a biological agent in the environment allows for early specific treatment and time

during which prophylaxis would be effective. Unfortunately, currently no reliable detection systems exist for BW agents. The US Department of Defense has placed a high priority on research and development of a detector system. Methods are being developed and tested to detect a biological aerosol cloud using an airborne pulsed laser system to scan the lower altitudes upwind from a possible target area. A detection system mounted on a vehicle also is being developed. This system will analyze air samples to provide a plot of particle sizes, detect and classify bacterial cells, and measure DNA content, ATP content, and identify agents using immunoassays.

A BW agent attack is likely to be covert. Thus, detection of such an attack requires recognition of the clinical syndromes associated with various BW agents. Physicians must be able to identify early victims and recognize patterns of disease. This requires integrated epidemiologic surveillance systems performing real-time monitoring with information shared at many levels of the health care system (eg, ED to ED or ED to public health officials). Preliminary criteria for suspicious outbreaks of disease that could provide indications of a possible biological weapons event include the following:

- Disease (or strain) not endemic
- Unusual antibiotic resistance patterns
- Atypical clinical presentation
- Case distribution geographically and/or temporally inconsistent (eg, compressed time course)
- Other inconstant elements (eg, number of cases, mortality and morbidity rates, deviations from disease occurrence baseline)

Indications of possible BW agent attack include the following:

- Disease entity that is unusual or that does not occur naturally in a given geographical area or combinations of unusual disease entities in the same patient population

- Multiple disease entities in the same patients, indicating that mixed agents have been used in the attack
- Large numbers of both military and civilian casualties when such populations inhabit the same area
- Data suggesting a massive point-source outbreak
- Apparent aerosol route of infection
- High morbidity and mortality relative to the number of personnel at risk
- Illness limited to fairly localized or circumscribed geographic areas
- Low attack rates in personnel who work in areas with filtered air supplies or closed ventilation systems
- Sentinel dead animals of multiple species
- Absence of a competent natural vector in the area of outbreak (for a biological agent that is vector-borne in nature)

PROTECTIVE MEASURES

Protective measures can be taken against BW agents. These should be implemented early (if warning is received) or later (once suspicion of BW agent use is made). Currently, available masks such as the military gas mask or high-efficiency particulate air (HEPA) filter masks used for tuberculosis (TB) exposure filter out most BW particles delivered by aerosol. Multilayered HEPA masks can filter 99.9% of 1-5 μm particles, but face-seal leaks may reduce the efficacy by as much as 10-20%. Individual face-fit testing is required to correct seal leak problems.

Most aerosolized biological agents do not penetrate unbroken skin, and few organisms adhere to skin or clothing. After an aerosol attack, simple removal of clothing eliminates a great majority of surface contamination. Thorough showering with soap and water removes 99.99% of the few organisms left on the victim's skin after disrobing. The use of sodium hypochlorite is not

recommended over soap and water.

The use of special suits by health care providers is not necessary. Normal clothing provides a reasonable degree of protection against dermal exposure. Latex gloves and universal precautions provide sufficient protection when treating most infected patients. Place patients in a private negative-pressure room and practice proper sanitation with universal precautions. Proper disposal of corpses is essential. In the case of anthrax spores, this should be performed by incineration.

Of the potential BW agents, only plague, smallpox, and viral hemorrhagic fevers are spread readily person to person by aerosol and require more than standard infection control precautions (gown, mask with eye shield, gloves). Regardless, place all potential victims of BW agents in isolation. Medical personnel caring for these patients should wear a HEPA mask in addition to standard precautions pending the results of a more complete evaluation.

Broad-spectrum intravenous antibiotic

coverage is recommended initially for victims when a BW agent is suspected. Institute this even prior to the identification of the specific BW agent. Vaccinations currently are available for anthrax, botulinum toxin, tularemia, plague, Q fever, and smallpox. The widespread immunization of nonmilitary personnel has not been recommended by any governmental agency. Immune protection against ricin and staphylococcal toxins may be feasible in the near future.

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Bacterial Agents

ANTHRAX

INTRODUCTION

Bacillus anthracis is a large, aerobic, gram-positive, spore forming, nonmotile bacillus. The bacterium ordinarily produces a zoonotic disease in domesticated and wild animals such as goats, sheep, cattle, horses, and swine. Humans become infected by contact with infected animals or contaminated animal products. Infection occurs predominantly through the cutaneous route and only rarely via the respiratory or gastrointestinal (GI) route.

Anthrax occurs worldwide. The organism exists in the soil as a spore. The form of the organism in infected animals is the bacillus. Sporulation occurs only when the organism in the carcass is exposed to air.

The true incidence of human anthrax is unknown. Reporting of illness has been unreliable. In 1958, an estimated 20,000-100,000 cases occurred worldwide. In the US, the annual incidence of human anthrax has declined steadily from approximately 127 cases in the early years of this century to approximately 1

per year for the past 10 years.

PATHOPHYSIOLOGY - ANTHRAX

B anthracis possesses 3 known virulence factors, an antiphagocytic capsule and 2 protein exotoxins (lethal and edema toxin). The role of the capsule in pathogenesis was demonstrated in the early 1900s when anthrax strains, lacking a capsule, were demonstrated to be virulent. In more recent years, the genes encoding synthesis of the capsule were found to be encoded on a 110-kilobase plasmid. The capsule is composed of a polymer of poly-D-glutamic acid, which confers resistance to phagocytosis and may contribute to the resistance of anthrax to lysis by serum cationic proteins.

The anthrax toxins, like many bacterial and plant toxins, possess the following 2 components: a cell binding B-domain and an active A-domain. The A-domain confers enzymatic activity and toxicity. Edema toxin, which consists of the same protective antigen together with a third protein, edema factor, causes edema when injected into the skin of

experimental animals.

Infection begins when the spores are inoculated through skin or mucosa. It is believed that spores are ingested locally by tissue macrophages. Subsequently, spores germinate within macrophages to the vegetative bacilli, which produce capsules and toxins. Bacteria proliferate at these tissue sites and produce the edema and lethal toxins that impair host leukocyte function and lead to the following distinctive and pathologic findings: edema, hemorrhage, tissue necrosis, and a relative lack of leukocytes. In inhalation anthrax, the spores are ingested by alveolar macrophages, which transport them to the regional tracheobronchial lymph nodes, where germination occurs.

Once in the tracheobronchial lymph nodes, the local production of toxins by extracellular bacilli gives rise to the characteristic pathologic picture of massive hemorrhagic, edematous, and necrotizing lymphadenitis and mediastinitis. The bacillus then can spread to the blood, leading to septicemia and frequently

causing hemorrhagic meningitis. Death results from respiratory failure, overwhelming bacteremia, septic shock, and meningitis.

CLINICAL FEATURES - ANTHRAX

Cutaneous: More than 95% of cases of anthrax are cutaneous. After inoculation, the incubation period is 1-5 days. The disease first appears as a small papule that progresses over 1-2 days to a vesicle containing serosanguinous fluid with many organisms and a paucity of leukocytes. This often has been referred to as a malignant pustule; however, this is a misnomer because no pustular lesions are found in anthrax patients. The vesicle ruptures, leaving a necrotic ulcer. The lesion usually is painless, and varying degrees of edema may be present around it. The edema occasionally may be massive, encompassing the entire face or limb, and is described as malignant edema. Patients generally experience fever, malaise, and headache, which may be severe in those with extensive edema. Local lymphadenitis also may be present. The ulcerbase develops a characteristic 1-5 cm black eschar. (The black appearance of the eschar gives anthrax its name [Greek *anthrakos* = coal].) After a period of 2-3 weeks, the eschar separates, often

leaving a scar. Septicemia is rare. Mortality should be less than 1% with adequate treatment.

Inhalation: Also known as woolsorter's disease, inhalation anthrax has a typical incubation period of 1-6 days, but a latent period as long as 60 days has been described. Initial manifestations are nonspecific and include headache, malaise, fatigue, myalgia, and fever. Associated nonproductive cough and mild chest discomfort may occur. These symptoms usually persist for 2-3 days, and in some patients a short period of improvement may occur. This is followed by the sudden onset of increasing respiratory distress with dyspnea, stridor, cyanosis, increased chest pain, and diaphoresis. Associated edema of the chest and neck may be present. Chest x-ray usually shows the characteristic widening of the mediastinum and often, pleural effusion. Pneumonia is an uncommon finding. The onset of respiratory distress is followed by the rapid onset of shock and death within 24-36 hours. Mortality is nearly 100% despite appropriate treatment. Inhalation anthrax is the most likely form of disease to follow military

or terrorist attack. Such an attack likely will involve the aerosolized delivery of anthrax spores.

Oropharyngeal and gastrointestinal: These result from the ingestion of infected meat that has not been cooked sufficiently. After an incubation period of 2-5 days, patients with oropharyngeal disease present with severe sore throat or a local oral or tonsillar ulcer, usually associated with fever, toxicity, and swelling of the neck due to cervical or submandibular lymphadenitis or edema. Dysphagia and respiratory distress also may be present. GI anthrax begins with nonspecific symptoms of nausea, vomiting, and fever. These are followed in most patients by severe abdominal pain. The presenting sign may be an acute abdomen, which may be associated with hematemesis, massive ascites, and diarrhea. Mortality in both forms may be as high as 50%, especially in the GI form.

Meningitis: This may occur following bacteremia as a complication of any of the other clinical forms. Meningitis also may occur, rarely, without any of the other clinical forms of the disease. It often is hemorrhagic and

almost invariably fatal.

DIAGNOSIS - ANTHRAX

The most critical aspect in making a diagnosis is a high index of suspicion associated with a compatible history of exposure. Consider cutaneous anthrax following the development of a painless, pruritic papule, vesicle, or ulcer. This area often is associated with surrounding edema that develops into a black eschar. With extensive or massive edema, such a lesion is almost pathognomonic. Gram stain or culture of the lesion confirms the diagnosis. The differential diagnosis should include tularemia and staphylococcal or streptococcal species.

The diagnosis of inhalation anthrax is extremely difficult, but suspect the disease with a history of exposure to a *B anthracis*-containing aerosol. Early symptoms are entirely nonspecific. The development of respiratory distress in association with radiographic evidence of a widened mediastinum due to

hemorrhagic mediastinitis and the presence of hemorrhagic pleural effusions or hemorrhagic meningitis should suggest the diagnosis. Sputum Gram stain and culture usually are not helpful, since pneumonia is an uncommon feature of illness. Gram stain of peripheral blood may be positive for gram-positive bacilli and should be performed.

GI anthrax also is exceedingly difficult to diagnose because of the rarity of the disease and nonspecific symptoms. Diagnosis usually is confirmed only with a history of ingesting contaminated meat in the setting of an outbreak. Once again, cultures generally are not helpful in making the diagnosis.

Meningitis from anthrax is clinically indistinguishable from meningitis due to other etiologies. A distinguishing feature is that the spinal fluid is hemorrhagic in as many as 50% of patients. The diagnosis can be confirmed by identifying the organism in the spinal fluid by microscopy, culture, or both.

Serology can be used to make a retrospective diagnosis. Antibody develops in 68-93% of reported cases of cutaneous anthrax and 67-94% of reported cases of oropharyngeal anthrax. A positive skin test to anthracin also has been used to make a retrospective diagnosis of anthrax.

The most useful microbiologic test is the standard blood culture, which is almost always positive in patients with

systemic illness. Blood cultures should show growth in 6-24 hours. If the laboratory has been alerted to the possibility of anthrax, biochemical testing and review of colonial morphology should provide a preliminary diagnosis 12-24 hours later. However, if the laboratory has not been alerted to the possibility of anthrax, *B anthracis* may not be identified correctly.

New rapid diagnostic tests for *B anthracis* and its proteins include polymerase chain reaction (PCR), enzyme-linked immunoassay (ELISA), and direct fluorescent antibody (DFA) testing. Currently, these tests are only available at national reference laboratories.

TREATMENT - ANTHRAX

A number of possible therapeutic strategies have yet to be fully explored experimentally or submitted for approval to the Food and Drug Administration (FDA). The recommendations provided do not represent uses currently approved by the FDA but are a consensus based on best available information of recent studies.

Given the fulminant course of inhalation anthrax, early antibiotic administration is essential to maximize patient survival. Given the difficulty in achieving timely microbiologic diagnosis of anthrax, all persons with fever or evidence of systemic disease in an area where anthrax cases are occurring should be treated empirically for anthrax until the disease is excluded.

No clinical studies exist of the treatment of inhalation anthrax in humans. Most naturally occurring strains of anthrax are sensitive to penicillin, and penicillin historically has been the preferred therapy for the treatment of anthrax. Penicillin and doxycycline are FDA-approved antibiotics for anthrax. Doxycycline is the preferred option from the tetracycline class of antibiotics because of its proven efficacy in monkey studies. Experts currently recommend initiation of ciprofloxacin or other fluoroquinolones in adults with presumed inhalation anthrax infection. Following a terrorist attack, assume resistance to

penicillin and tetracycline class antibiotics until laboratory testing demonstrates otherwise.

In a contained casualty setting (a situation in which a modest number of patients require therapy), initiate intravenous antibiotics for symptomatic patients. In adults, ciprofloxacin 400 mg IV q12h is recommended. Traditionally, ciprofloxacin and other fluoroquinolones are not recommended for use in children younger than 16-18 years because of a link to permanent arthropathy in adolescent animals and transient arthropathy in a small number of children.

Balancing these small risks against the real risk of death and resistant strains of *B anthracis*, experts recommend that ciprofloxacin be given to a pediatric population for initial therapy or postexposure prophylaxis following anthrax attack. In children, ciprofloxacin at 20-30 mg/kg/d IV in 2 daily doses (not to exceed 10 g/d) is recommended. If antibiotic susceptibility testing allows, substitute intravenous penicillin for the fluoroquinolones. For adults and children older than 12 years, penicillin G at 4 million U IV q4h is recommended for 60 days. Doxycycline at 100 mg IV q12h for 60 days is an acceptable alternative for adults. For children younger than 12 years, penicillin G is dosed 50,000 U/kg IV q6h for 60 days.

In experimental models, antibiotic therapy during anthrax infection has prevented development of an immune response. This suggests that even if the antibiotic-treated patient survives anthrax infection, risk of recurrence remains for at least 60 days. Oral therapy should replace intravenous therapy as soon as a patient's clinical condition improves.

Historically, the treatment of cutaneous anthrax has been oral penicillin. Recent recommendations suggest that oral fluoroquinolones or tetracycline antibiotics, as well as amoxicillin, are suitable alternatives if antibiotic susceptibility is proven. Although previous guidelines have suggested treating cutaneous anthrax with 7-10 days of therapy, recent recommendations suggest treatment for 60 days in the setting of bioterrorism, given the presumed exposure to the primary aerosol. Treatment of cutaneous anthrax generally prevents progression to systemic disease, although it does not prevent the formation and evolution of the eschar.

Other

antibiotics effective against *B anthracis* in vitro include chloramphenicol, erythromycin, clindamycin, extended spectrum penicillins, macrolides, aminoglycosides, vancomycin, cefazolin, and other first-generation cephalosporins.

In pregnant women, experts recommend that ciprofloxacin be given for therapy and postexposure prophylaxis following anthrax attack. Substitute intravenous penicillin for the fluoroquinolones if microbiologic testing confirms penicillin susceptibility.

PREVENTION/PROPHYLAXIS-ANTHRAX

No FDA-approved chemoprophylactic regimens are available following exposure to an anthrax aerosol. For postexposure prophylaxis, experts recommend the same oral regimen as that recommended for treatment of mass casualties. For adults, administer ciprofloxacin 500 mg PO bid for 60 days. Ciprofloxacin may be changed to amoxicillin at 500 mg PO tid or doxycycline 100 mg PO bid for 60 days if microbiologic testing confirms such antibiotic susceptibility. In children,

administer ciprofloxacin at 20-30 mg/kg/d PO taken twice daily (not to exceed 1 g/d) for 60 days. If the strain is susceptible to penicillins and patient weight is greater than 20 kg, amoxicillin may be given at 500 mg PO tid. For a child who weighs less than 20 kg, amoxicillin is administered at 40 mg/kg/d divided tid for 60 days.

A licensed vaccine, an aluminum hydroxide-absorbed preparation, is derived from culture fluid supernatant taken from an attenuated strain. The vaccination series consists of 6 subcutaneous doses at 0, 2, and 4 weeks, then at 6, 12, and 18 months, followed by annual boosters. Insufficient data are available regarding efficacy against inhalation anthrax in humans, although studies in rhesus monkeys indicate that it is protective. If information indicates that a BW attack is imminent or may have occurred, prophylaxis of unimmunized individuals with ciprofloxacin (500 mg PO bid) or doxycycline (100 mg PO bid) is recommended. Initiate the vaccination series for unimmunized

individuals. Should an anthrax attack be confirmed, continue chemoprophylaxis for at least 4 weeks and until all those exposed receive 3 doses of vaccine (at 0, 2, and 4 wk).

PLAGUE

INTRODUCTION

Plague is a zoonotic infection caused by *Yersinia pestis*, a gram-negative coccobacillus, which has been the cause of 3 great human pandemics in the Common Era, in the 6th, 14th, and 20th centuries. Throughout history, the oriental rat flea (*Xenopsylla cheopis*) has been largely responsible for spreading bubonic plague. After the flea ingests a blood meal on a bacteremic animal, bacilli can multiply and essentially block the flea's foregut with a fibrinoid mass of bacteria. When an infected flea with a blocked foregut attempts to feed again, it regurgitates clotted blood and bacteria into the victim's blood stream and so passes the infection onto the next victim, whether rat or human. As many as 24,000 organisms may be inoculated into the host.

Although the largest outbreaks of plague have been associated with *X cheopis*, all fleas should be

considered dangerous in plague-endemic areas. The most important vector in the US is *Diamesus montanus*, the most common flea of rock squirrels and California ground squirrels. The black rat, *Rattus rattus*, has been most responsible worldwide for the persistence and spread of plague in urban epidemics.

Plague is characterized by the abrupt onset of high fevers, painful lymphadenopathy, and bacteremia. Septicemic plague sometimes can ensue from untreated bubonic plague or, de novo, after a flea bite. Patients with the bubonic form of the disease may develop secondary pneumonic plague. This complication can lead to human-to-human spread by the respiratory route and cause primary pneumonic plague. Pneumonic plague is the most severe form of disease and, untreated, has a mortality rate approaching 100%.

Mortality from endemic plague continues at low rates throughout the world despite the availability of effective antibiotics. People continue to die of plague, not because the bacilli have become resistant but, most often, because physicians do not include plague in their differential

diagnosis, and treatment is delayed.

PATHOPHYSIOLOGY - PLAGUE

Y pestis is a gram-negative, nonacid-fast, nonmotile, nonsporulating coccobacillus. Its bipolar appearance is best appreciated when Wright-Giemsa, Wayson, or Gram stains are used. *Y pestis* grows optimally at 28°C. Biochemically, the plague bacillus produces no hemolysins; is positive for catalase; and is negative for hydrogen sulfide, oxidase, and urease.

The known virulence factors of *Y pestis* are encoded on the chromosomes of its 3 plasmids. The pH6 antigen, a protein located on the surface of the bacterium, is necessary for complete virulence. It is induced in vivo at sites of inflammation and cellular necrosis and within phagocytic cells. The low calcium response (LCR) plasmid, which is homologous in *Y pestis* and the other 2 *Yersinia* pathogens, *Yersinia pseudotuberculosis* and *Yersinia enterocolitica*, codes for several secreted proteins and is also necessary for

virulence.

As few as 1-10 organisms of *Y pestis* are sufficient to infect rodents and primates via the oral, intradermal, subcutaneous, or intravenous routes. After being introduced into the mammalian host by a flea, the organism is thought to be susceptible initially to phagocytosis and killing by neutrophils. However, some of the bacteria may grow and proliferate within tissue macrophages. Within the human host, several environmental signals (temperature of 37°C, contact with eukaryotic cells, location within mononuclear cells, pH) are thought to induce the synthesis and activity of a multitude of factors that contribute to virulence. Bacteria become resistant to phagocytosis and proliferate unimpeded extracellularly.

During the incubation phase, the bacilli most commonly spread to regional lymph nodes, where supportive lymphadenitis develops, producing the characteristic bubo. Dissemination from the local site is thought to be related to the action of both plasminogen activator and Yop M. Infection progresses if untreated; septicemia develops, and the infection spreads to other organs. The endotoxin probably contributes to the development of septic shock, which is similar to the shock states observed with other causes of gram-negative sepsis.

Tissues most commonly infected include the spleen, liver, lungs, skin, and mucous membranes. Late infection of the meninges also occurs, especially if suboptimal antibiotic therapy has been administered.

Primary pneumonic plague, the most severe form of the disease, arises from inhalation of an infectious aerosol. Primary pneumonic plague is more rapidly fatal than the secondary form, because the inhaled droplets already contain phagocytosis-resistant bacilli, which have arisen from their growth in the vertebrate host.

Primary septicemia plague can arise from direct inoculation of bacilli into the bloodstream, bypassing initial multiplication in the lymph nodes.

CLINICAL FEATURES - PLAGUE

In the US, most patients (85-90%) with human plague present clinically with the bubonic form, 10-15% with the primary septicemia form, and 1% with the pneumonic form. Secondary septicemic plague occurs in 23% of patients who present with bubonic plague, and secondary pneumonic

plague occurs in 9%. If *Y pestis* were used as a BW agent, it most likely would be inhaled as an infectious aerosol and result in primary pneumonic plague (epidemic pneumonia). If fleas were used as carriers of disease, bubonic or septicemic plague would result.

Bubonic plague: Buboes manifest after a 1- to 8-day incubation period. Their appearance is associated with the onset of sudden fever, chills, and headache, which often are followed by nausea and vomiting several hours later. Presenting symptoms include severe malaise (75%), headache (20-85%), vomiting (25-49%), chills (40%), altered mentation (26-38%), cough (25%), abdominal pain (19%), and chest pain (13%). Buboes occur in the groin (90% femoral, more frequent femoral than inguinal), axillary, or cervical regions, depending on the site of inoculation, 6-8 hours after the onset of symptoms. Buboes become visible within 24 hours and are characterized by severe pain. Untreated, septicemia develops in 2-6 days. Approximately 5-15% of

bubonic plague patients develop secondary pneumonic plague and thus the ability to spread illness from person to person by respiratory droplets.

Septicemia plague: Septicemia plague may occur primarily or secondarily as a result of hematogenous dissemination of bubonic plague. Presenting signs and symptoms of primary septicemic plague are

essentially the same as those for any gram-negative septicemia and include fever, chills, nausea, vomiting, and diarrhea; later, purpura, disseminated intravascular coagulation (DIC), and acrocyanosis and necrosis occur.

Pneumonic plague: Pneumonic plague may occur primarily from inhalation of aerosols or secondarily from hematogenous dissemination. Patients typically have a productive cough with blood-tinged sputum within 24 hours of symptom onset. The findings on chest x-ray are variable, but bilateral alveolar infiltrates appear to be the most common findings in pneumonic plague.

Plague meningitis: This is observed in 6-7% of

patients. The condition manifests itself most often in children after 9-14 days of ineffective treatment. Symptoms are similar to those of other forms of acute bacterial meningitis.

DIAGNOSIS - PLAGUE

The diagnosis of bubonic plague should be made readily on clinical grounds if a patient presents with a painful bubo, fever, prostration, and history of exposure to rodents or fleas in an endemic area. However, if the patient presents in a nonendemic area or without a bubo, then the diagnosis can be difficult to make. When a bubo is present, the differential diagnosis should include tularemia, cat scratch disease, lymphogranuloma venereum, chancroid, TB, streptococcal adenitis, and scrub typhus.

The differential diagnosis of septicemic plague also includes meningococemia, gram-negative sepsis, and rickettsioses. A presentation of systemic toxicity, a productive cough, and bloody sputum suggests a large differential diagnosis. However, demonstration of gram-negative coccobacilli

in the sputum readily should suggest the correct diagnosis, because *Y pestis* is perhaps the only gram-negative bacterium that can cause extensive, fulminant pneumonia with bloody sputum in an otherwise healthy, immunocompetent host. In addition, *Y pestis* has unique bipolar, safety-pin morphology.

In patients with lymphadenopathy, perform a bubo aspiration. Air-dry the aspirate on a slide for Gram, Wright-Giemsa, or Wayson stain. If available, obtain a DFA stain of the aspirate for the presence of *Y pestis* capsular antigen. A positive DFA is more specific for *Y pestis* than the other stains listed.

Perform cultures of blood, bubo aspirate, sputum, and cerebrospinal fluid (CSF). Tiny 1- to 3-mm beaten copper colonies appear on blood agar in 48 hours. It is important to remember that colonies may be negative at 24 hours.

Complete blood counts (CBCs) often reveal leukocytosis with a left shift. Platelet counts may be normal or low, and activated partial thromboplastin

times (aPTTs) may be increased. When DIC is present, fibrin degradation products are elevated. Because

of liver involvement, alanine aminotransferase, aspartate aminotransferase, and bilirubin levels may be increased.

Most naturally occurring strains of *Y pestis* produce an F1-antigen in vivo, which can be detected in serum samples by immunoassay. Because fractional antigen and antibody do not occur early in the infection, perform titers for both on several sequential blood specimens. A four-fold rise in antibody titer in patient serum is retrospectively diagnostic.

TREATMENT - PLAGUE

Isolate patients with plague for the first 48 hours after treatment initiation. If pneumonic plague is present, continue isolation for 4 days.

Since 1948, streptomycin has been the treatment of choice for bubonic, septicemic, and pneumonic plague. Administer it in a dose of 30 mg/kg/d IM divided bid. In patients with meningitis or hemodynamic instability, add intravenous

chloramphenicol (50-75 mg/kg/d) divided qid. Gentamicin has had much less clinical usage but can be used as an alternative to streptomycin. Continue treatment for a minimum of 10 days or 3-4 days after clinical recovery. In patients with very mild bubonic plague who are not septic, tetracycline can be used orally at a dose of 2 g/d divided qid for 10 days. Doxycycline, ofloxacin, and ceftriaxone have been demonstrated to be effective in animal models.

In pregnant women, use streptomycin or gentamicin unless chloramphenicol specifically is indicated. Streptomycin is also the treatment of choice for newborns.

If treated with antibiotics, buboes typically recede in 10-14 days and do not require drainage. Patients are unlikely to survive primary pneumonic plague if antibiotic therapy is not initiated within 18 hours of symptom onset. Without treatment, mortality is 60% for bubonic plague and 100% for the pneumonic and septicemic forms.

PREVENTION/PROPHYLAXIS - PLAGUE

All

plague control measures must include insecticide use, public education, and reduction of rodent populations with chemicals such as cholecalciferol. Fleas always must be targeted before rodents, because killing rodents may release massive amounts of infected fleas.

Treat contacts of patients with pneumonic plague and individuals who have been exposed to aerosols with tetracycline 15-30 mg/kg/d divided qid for 6 days. If tetracycline is not available, doxycycline 100 mg bid is an effective alternative. Pregnant women and children younger than 8 years should receive

trimethoprim/sulfamethoxazole (40 mg sulfa/kg/d) divided bid for 6 days.

Contacts of patients with bubonic plague do not require prophylactic therapy. However, administer prophylaxis to people who were in the same environment and potentially exposed to the same source of infection. In addition, previously vaccinated individuals should receive prophylactic antibiotics if they have been exposed to a plague aerosol.

Only individuals

at high risk for plague should be immunized with a licensed, killed, whole cell vaccine. Vaccinate military troops and personnel working in endemic areas, lab personnel working with *Y pestis*, and people who reside in enzootic or epidemic areas. While epidemiologic evidence supports the efficacy of the current vaccine against bubonic plague, its efficacy against aerosolized *Y pestis* is believed to be poor.

CHOLERA

INTRODUCTION

Cholera is an acute and potentially severe GI disease caused by *Vibrio cholerae*. *V cholerae* is a short, curved, motile, gram-negative, nonsporulating rod. Two serogroups (01, 0139) have been associated with cholera in humans. The 01 serotype exists as 2 biotypes, classical and El Tor. The organisms are strongly anaerobic, preferring alkaline and high-salt environments. They do not invade the intestinal mucosa but rather adhere to it. Cholera is the prototype toxigenic diarrhea, which is secretory in nature.

This agent has been investigated in the past as a biological weapon. Cholera does not spread easily from human to human; therefore, it appears that major drinking water supplies would have to be contaminated heavily for this agent to be effective as a biological weapon. The rate of symptomatic-to-asymptomatic cases during exposures is 1:400.

PATHOPHYSIOLOGY- CHOLERA

All strains of *V cholerae* elaborate the same

enterotoxin, a protein molecule with a molecular weight of 84,000 daltons. The entire clinical syndrome is caused by the action of the toxin on the intestinal epithelial cell. Cholera toxin causes active secretion of chloride and blocks sodium absorption in the small intestine, with the colon relatively insensitive to the toxin. The large volume of fluid produced in the upper intestine overwhelms the capacity of the lower intestine to absorb. The diarrhea is classically thin, grayish brown, and mucoid and may reach a rate of 1 L/h.

Transmission is made through direct or indirect fecal contamination of water or foods and by heavily soiled hands or utensils. All populations are susceptible, while natural resistance to infection varies.

Drying easily kills the organism. It is not viable in pure water but survives up to 24 hours in sewage and as long as 6 weeks in certain types of relatively impure water containing organic matter. It can withstand freezing for 3-4 days. It is killed readily by dry

heat at 117°C, steam and boiling, short-term exposure to ordinary disinfectants, and chlorination of water.

CLINICAL FEATURES - CHOLERA

Infection generally occurs within a week of exposure and is classically of abrupt onset following a brief nonspecific prodrome. Fever is rare. The syndrome is characterized by sudden onset of nausea and vomiting and profuse diarrhea with a classic rice water appearance. If untreated, the disease generally lasts 1-7 days. The clinical manifestations of cholera are related to the profound fluid and electrolyte depletion that occurs. Acute treatment consists of rapid, aggressive fluid resuscitation with isotonic solutions and potassium.

Children may experience seizures caused by hypoglycemia and hypernatremia and may have potassium depletion severe enough to cause an arrhythmia. The rapid loss of body fluids often leads to toxemia and frequent cardiovascular collapse. Mortality can range as high as 50% in untreated cases.

DIAGNOSIS-

CHOLERA

The incubation period ranges from 12-72 hours and depends on the dose of ingested organisms. Onset of illness usually is sudden. Initially, the disease presents with intestinal cramping and painless diarrhea. Vomiting, malaise, and headache often accompany the diarrhea, especially early in the illness.

On microscopic examination of the stool, few or no red cells, white cells, and almost no protein are found. The absence of inflammatory cells and erythrocytes reflects the noninvasive character of *V cholerae* infection of the intestinal lumen. The organism can be identified in liquid stool or enrichment broths by darkfield or phase contrast microscopy and by identifying darting motile *Vibrio* species. Bacteriologic diagnosis is not necessary for treatment, as it can be diagnosed clinically.

TREATMENT - CHOLERA

Treatment depends on replacement of fluids and electrolyte losses. This is best accomplished using oral rehydration therapy, but

intravenous fluid replacement is occasionally necessary for persistent vomiting or high rates of stool loss (10 mL/kg/h). Antibiotics shorten the duration of diarrhea and reduce fluid losses. Tetracycline (500 mg

q6h for 3 d) or doxycycline (300 mg once or 100 mg bid for 3 d) is an acceptable alternative. However, due to resistance, ciprofloxacin (500 mg q6h for 3 d) or erythromycin (40 mg/kg/d divided qid for 3 d) also has been accepted.

PREVENTION/PROPHYLAXIS - CHOLERA

A licensed, killed vaccine is available for use in those considered to be at risk for exposure. The vaccine is protective for only approximately 50% of those immunized, and protection lasts for no more than 6 months. The vaccination schedule is an initial dose followed by another dose 4 weeks later, with booster doses every 6 months.

An inactivated oral vaccine (WC/rBs) is safe and provides rapid short-term protection. WC/rBs requires 2 doses and has approximately 85% efficacy lasting 2-3 years for both El Tor and classic biotypes.

TULAREMIA

INTRODUCTION

Tularemia is a zoonosis caused by the gram-negative, facultative intracellular bacterium *Francisella tularensis*. The disease is characterized by fever, localized skin or mucous membrane ulceration, regional lymphadenopathy, and occasionally pneumonia. GW McCay discovered the disease in Tulare County, California, in 1911. The first confirmed case of human disease was reported in 1914. Edward Francis, who described transmission by deer flies via infected blood, coined the term tularemia in 1921. *F tularensis* has been considered an important BW agent because of its high infectivity after aerosolization.

F tularensis is a nonmotile, obligate aerobic, gram-negative coccobacillus with 2 subspecies. *F tularensis* subsp *tularensis* is the most common in the US. *F tularensis* subsp *paleoartica* is more common outside the US. The subspecies are indistinguishable serologically, although

they may be distinguished by 16S ribosomal ribonucleic acid (rRNA) analysis. A capsule has been reported to contribute to virulence. No known toxins are produced.

The principal reservoir in North America is the tick. In North America, the rabbit is the most common vertebrate associated with transmission of tularemia. In other areas of the world, tularemia is maintained in water rats and other aquatic animals.

PATHOPHYSIOLOGY - TULAREMIA

F tularensis usually is introduced into the host through breaks in the skin or through the mucous membranes of the eye, respiratory tract, or GI tract. Ten virulent organisms injected subcutaneously and 10-50 organisms given by aerosol can cause infection in humans. After inoculation, *F tularensis* is ingested by and multiplies within macrophages. The host defense against *F tularensis* is mediated by a T

cell-independent mechanism, which appears early after infection (<3d), and a T cell-dependent mechanism, which appears later(>3 d) after infection. The role of humoral-mediated immunity and neutrophils in the host defense against *F tularensis* remains unclear.

CLINICAL FEATURES - TULAREMIA

Tularemia can be divided into the ulceroglandular (75% of patients) and typhoidal (25% of patients) forms based on clinical findings. Patients with ulceroglandular tularemia have lesions of the skin or mucous membranes, lymph nodes greater than 1 cm in diameter, or both. Patients with typhoidal tularemia present with lymph nodes less than 1 cm in diameter and without skin or mucous membrane lesions.

After an incubation period of 3-6 days, patients with the ulceroglandular form of the disease develop a constellation of symptoms consisting of fever (85%), chills (57%), headache (45%), cough (38%), and myalgia (31%). Patients also may complain of chest pain, vomiting, arthralgia, sore throat, abdominal pain, diarrhea, dyspnea, back pain, or neck stiffness.

A cutaneous chancrelike ulcer occurs in approximately 60% of patients and is the most common sign of tularemia. Ulcers are generally single lesions with heaped up borders 0.4-3 cm in diameter. Lesions associated with infection acquired from mammalian vectors usually are located on the upper extremities, whereas lesions associated with infection from arthropod vectors usually are located on the lower extremities.

Enlarged lymph nodes are seen in approximately 85% of patients and may be the initial or the only sign of infection. Although enlarged lymph nodes usually occur as single lesions, they may appear in groups. The appearance of enlarged lymph nodes in upper or lower extremities and the correlation with the vector is the same as for ulcerative lesions. Enlarged lymph nodes may become fluctuant, drain spontaneously, or persist for as long as 3 years. When fluctuant, they may be confused with buboes of bubonic plague. A minority of patients with typhoidal disease develop a morbilliform eruption.

Pharyngitis may occur in up to 25% of patients with tularemia. On occasion, patients with pharyngitis also may develop a retropharyngeal abscess or suppuration of regional lymph nodes. Pharyngeal ulcers may be found in patients with aerosol-induced disease.

The lower respiratory tract is involved in 47-94% of patients. Approximately 30% of patients with ulceroglandular and 80% of patients with typhoidal tularemia have pneumonia. Patients present with productive or nonproductive cough and less commonly with pleuritic chest pain, shortness of breath, or hemoptysis. Fifty percent of patients have radiographic evidence of pneumonia, and 1% or fewer have hilar adenopathy. Pleural effusions are seen in 15% of patients with pneumonia.

DIAGNOSIS - TULAREMIA

Tularemia can be diagnosed by recovery of *F tularensis* in culture. Although difficult to culture, it can be recovered from blood, ulcers, sputum, conjunctival exudate, pharyngeal exudates, and gastric washings. On

media containing cysteine, *F tularensis* appears as small, smooth, opaque colonies after 24-48 hours of incubation at 37°C. Identification of the organism is made on the basis of its growth characteristics and bacterial agglutination or fluorescent stain using antiserum specific for *F tularensis*.

Most diagnoses of tularemia are made serologically using bacterial agglutination or ELISA. The serologic response may be blunted by the use of antibiotics and may not appear for more than 2 weeks.

Patients usually do not have abnormalities in the hemoglobin, hematocrit, or platelet count. The peripheral white blood cell count usually is elevated only mildly and often shows a lymphocytosis late in the disease. Patients may have microscopic pyuria, which may lead to erroneous diagnosis of urinary tract infection. Some patients show mild elevations in lactic dehydrogenase, serum transaminases, and alkaline phosphatase. CSF is usually normal.

TREATMENT -

TULAREMIA

Patients with tularemia who do not receive appropriate antibiotic therapy may have a prolonged illness characterized by malaise, weakness, and weight loss. With appropriate therapy, tularemia has a mortality of only 1-2.5%.

Streptomycin (30 mg/kg/d IM divided bid for 10-14 d) is the drug of choice for tularemia. Gentamicin (3-5 mg/kg/d parenterally for 10-14 d) is also effective. Tetracycline and chloramphenicol are effective as well but have been associated with significant relapse rates. Although laboratory-related infections with this organism are common, human-to-human spread is unusual, and respiratory isolation is not required.

PROPHYLAXIS/PREVENTION - TULAREMIA

Antibiotic prophylaxis after exposure to tularemia is difficult, because the ideal drug, streptomycin, must be administered parenterally. Tetracycline is effective after exposure to an aerosol of tularemia if administered within 24 hours of the exposure at an oral dose of 2 g/d for 14

days.

A live attenuated vaccine has been developed and used in humans since 1940. In the 1960s, a further purified derivative was introduced and called live vaccine strain (LVS). Extensive studies have demonstrated that the LVS vaccine protected humans against an aerosol challenge with virulent *F tularensis*. Evidence indicates that immunization with the LVS vaccine prevents the typhoidal and

ameliorates the ulceroglandular forms of tularemia.

BRUCELLOSIS

INTRODUCTION

Brucellosis is a zoonotic infection of domesticated and wild animals caused by an organism of the genus *Brucella*. The organism infects mainly cattle, sheep, goats, and other ruminants, causing abortion, fetal death, and genital infection. Humans, who usually are infected incidentally by contact with infected animals, may develop numerous symptoms in addition to the usual ones of fever, malaise, and muscle pain. The disease often becomes chronic and may relapse, even with appropriate treatment. The ease of transmission by aerosol suggests that *Brucella* species may be useful as a BW agent.

PATHOPHYSIOLOGY - BRUCELLOSIS

Brucella species are small, nonmotile, nonsporulating, aerobic, gram-negative coccobacilli that may represent a single species. However, they are classified into 6 species. Each species has a characteristic predilection to infect certain animal species. Only *Brucella melitensis*, *Brucella suis*,

Brucella abortus, and *Brucella canis* cause disease in man. Infection of humans with *Brucella ovis* and *Brucella neotomae* has not been described.

Animals may transmit *Brucella* organisms during septic abortion, at the time of slaughter, and in their milk. Brucellosis is rarely, if ever, transmitted from human to human. *Brucella* species can enter mammalian hosts through skin abrasions or cuts, the conjunctiva, the respiratory tract, and the GI tract. Organisms are ingested rapidly by polymorphonuclear leukocytes, which generally fail to kill them. Organisms also are phagocytized by macrophages, which traffic to lymphoid tissue and eventually localize in lymph nodes, liver, spleen, joints, kidneys, and bone marrow.

Brucellosis also can replicate extracellularly in host tissue. The host cellular response may range from abscess formation to granuloma formation with caseous necrosis.

CLINICAL FEATURES - BRUCELLOSIS

Clinical manifestations of brucellosis are diverse, and the course of the disease varies. Patients may present with an acute, systemic, febrile illness; an insidious chronic infection; or a localized inflammatory process. The disease may be abrupt or insidious in onset, with an incubation period of 3 days to several weeks. Patients usually have nonspecific symptoms such as fever, sweats, fatigue, anorexia, and muscle or joint aches. Neuropsychiatric symptoms such as depression, headache, and irritability occur frequently. In addition, focal infection of bones, joints, or the genitourinary tract may cause local pain. Cough and pleuritic chest pain also may be noted.

Symptoms often last 3-6 months and occasionally for longer than a year. Brucellosis usually does not cause leukocytosis, and patients may be neutropenic. *B melitensis* tends to cause more severe, systemic illness than the other *Brucella* species. *B suis* is more likely to cause localized, suppurative disease.

Infection with *B melitensis* leads to bone or joint disease in approximately 30% of patients. Sacroiliitis develops in 6-15%, particularly in young adults. Arthritis of large joints occurs with about the same frequency as sacroiliitis. In contrast to septic arthritis caused by pyogenic organisms, joint inflammation observed with *B melitensis* is mild, and erythema of overlying skin is uncommon. Synovial fluid is exudative, with cell counts in the low thousands, predominantly mononuclear. In both sacroiliitis and peripheral joint infections, destruction of bone is unusual. Organisms can be cultured from fluid in approximately 20% of patients. Spondylitis tends to affect middle-aged or elderly patients, causing back (usually lumbar) pain, local tenderness, and occasionally radicular symptoms.

Radiographic findings, similar to those of tuberculous infection, include disk space narrowing and epiphysitis. Paravertebral abscesses occur rarely. In contrast to frequent infection of the axial

skeleton, osteomyelitis of long bones is rare.

Infection of the genitourinary tract, an important target in ruminant animals, also may lead to signs and symptoms of disease in humans. Pyelonephritis, cystitis, and, in males, epididymo-orchitis may occur. Both diseases may mimic their tuberculous counterparts with sterile pyuria on routine bacteriologic cultures.

Lung infections also have been described. Although up to 25% of patients may complain of respiratory symptoms (mostly cough, dyspnea, or pleuritic pain), chest radiographic examinations usually are normal. Diffuse or focal infiltrates, pleural effusions, abscesses, and granulomas may be seen.

Hepatitis and, rarely, liver abscess also occur. Mild elevations of serum lactate dehydrogenase and alkaline phosphatase are common. Biopsy may show well-formed granulomas or nonspecific hepatitis with collections of mononuclear cells.

Other sites of infection include the heart, central nervous system (CNS), and skin. *Brucella*

endocarditis, a rare but feared complication, accounts for 80% of deaths from brucellosis. CNS infection usually manifests itself as chronic meningoencephalitis, but subarachnoid hemorrhage and myelitis also occur. Cases of skin abscess also have been reported.

DIAGNOSIS - BRUCELOSIS

A thorough history eliciting details of appropriate exposures (animals, animal products, environmental exposures) is the most important diagnostic tool. Strongly consider brucellosis in the differential diagnosis when military troops exposed to a biological attack have febrile illnesses. PCR and antibody-

based antigen detection systems may demonstrate the presence of organisms in environmental samples collected from attack areas.

When the disease is considered, diagnosis usually is made by serology. The tube agglutination test remains the criterion standard. This test reflects the presence of anti-O-polysaccharide antibody. Most patients already have high titers at the time of clinical

presentation. Serum testing always should include dilution to at least 1:320. The tube agglutination test does not detect antibodies to *B canis*, because this organism does not have O-polysaccharide on its surface.

In addition to serologic testing, pursue diagnosis by microbiologic cultures of blood or body fluid samples. Hold cultures for at least 2 months. The reported frequency of isolation from blood varies widely, from less than 10% to 90%. *B melitensis* is said to be cultured more readily than *B abortus*. Culture of bone marrow may increase the yield.

TREATMENT - BRUCELLOSIS

Therapy with a single drug has resulted in a high relapse rate, so use combined antibiotic regimens whenever possible. A 6-week regimen of doxycycline 200 mg/d PO with the addition of streptomycin 1 g/d IM for the first 2 weeks is effective in most adults with most forms of brucellosis. Patients with spondylitis may require longer treatment. A 6-week oral regimen with both rifampin

900 mg/d and doxycycline 200 mg/d is effective. Several studies have demonstrated that treatment with a combination of streptomycin and doxycycline may result in less frequent relapse than treatment with the combination of rifampin and doxycycline.

Endocarditis likely is best treated with a combination of rifampin, streptomycin, and doxycycline for 6 weeks. Replace infected valves early. CNS disease responds to a combination of rifampin and trimethoprim/sulfamethoxazole but may need prolonged therapy. The latter combination is also effective for children younger than 8 years. Rifampin is recommended for pregnant women.

PREVENTION/PROPHYLAXIS - BRUCELLOSIS

Animal handlers should wear appropriate protective clothing when working with infected animals. Meat should be well cooked and milk should be pasteurized. Laboratory workers should culture the organism only with appropriate Biosafety level 2 or 3.

In the event of a biological attack, the standard gas mask should protect

personnel adequately from airborne *Brucella* species. No commercially available vaccine exists for humans.

Q FEVER

INTRODUCTION

Q fever is a zoonotic disease caused by *Coxiella burnetii*, a rickettsialike organism of low virulence but remarkable infectivity. A single organism may initiate infection. In addition, despite the fact that *C burnetii* is unable to grow or replicate outside host cells, a sporelike form of the organism is extremely resistant to heat, pressure, and many antiseptic compounds. This allows *C burnetii* to persist in the environment for long periods under harsh conditions. In contrast to this high degree of inherent resilience and transmissibility, the acute clinical disease associated with Q fever is usually a benign, although temporarily incapacitating, illness in humans. Even without treatment, most patients recover.

The primary reservoir for natural human infection is livestock, particularly parturient females, and the distribution is worldwide. Humans who work in animal husbandry, especially those who assist during parturition, are at risk for

acquiring Q fever.

The potential of *C burnetii* as a BW agent is related directly to its infectivity. It has been estimated that 50 kg of dried *C burnetii* would produce casualties at a rate equal to that of similar amounts of anthrax or tularemia organisms.

The causative agent of Q fever was designated *Coxiella burnetii* to recognize the outstanding contribution of both Harold Cox and MacFarlane Burnet in the isolation and characterization of the pathogen. The disease now has been identified in at least 51 countries and on 5 continents.

PATHOPHYSIOLOGY - Q FEVER

The genus *Coxiella* has only 1 species. *C burnetii* is extremely infectious. Under experimental conditions, a single organism is capable of producing infection and disease in humans.

The host range of *C burnetii* is diverse and includes a large number of mammalian species and arthropods. Among these, man is the only host identified that experiences an illness as a result

of infection. A number of different strains of *C burnetii* have been identified worldwide, and different clinical manifestations and complications may be associated with the various strains.

Humans have been infected most commonly by contact with domestic livestock, particularly goats, cattle, and sheep. The risk of infection is increased substantially if humans are exposed to these animals at parturition. During gestation, the proliferation of *C burnetii* in the placenta facilitates aerosolization of large numbers of the pathogen during parturition. Survival of the organism on inanimate surfaces, such as

straw, hay, or clothing, allows for transmission to individuals who are not in direct contact with infected animals.

Human infection with *C burnetii* is usually the result of inhalation of infected aerosols. Following this, host cells phagocytize the organisms. After phagocytosis by host cells, dissemination of the pathogen occurs as a result of circulation of organism free in

the plasma, on the surface of the cells, and carried by circulatory macrophages.

Little host reaction occurs at the initial portal of entry, either in the lung following inhalation of aerosol or in the skin following a tick bite. Q fever develops without formation of a primary infectious focus in the area of the tick bite, and the organism does not infect the vascular endothelium, as do other rickettsial pathogens. The presence of a lipopolysaccharide on the cell surface of *C burnetii* protects the pathogen from host microbicidal activity.

CLINICAL FEATURES - Q FEVER

Humans are the only hosts that commonly develop an illness as a result of the infection. Incubation varies from 10-40 days. The duration of the incubation period is correlated inversely with the magnitude of the inoculum. A higher inoculum also increases the severity of the disease. Q fever in humans may be manifested by asymptomatic seroconversion, acute illness, or chronic disease. The frequency of chronic

disease (usually endocarditis) is probably less than 1% of the total infected population.

No characteristic illness is described for acute Q fever, and manifestations may vary considerably between locations where the disease is acquired. The onset of symptomatic Q fever may be abrupt or insidious. Fever, chills, and headache are the most common signs and symptoms. Diaphoresis, malaise, myalgias, fatigue, and anorexia are also common. Arthralgias are relatively uncommon. Cough often occurs later in the illness. Chest pain occurs in a minority of patients. Although nonspecific, evanescent skin eruptions have been reported. No characteristic rash results.

Most patients appear mildly to moderately ill. The temperature tends to fluctuate, with peaks at 39-40°C, and is biphasic in approximately 25% of patients. The fever generally lasts less than 13 days but has been reported to last longer in older adults.

Encephalopathic symptoms, headache, hallucinations (visual, auditory), expressive

dysphasia, facial pain resembling trigeminal neuralgia, diplopia, and dysarthria have been reported.

Physical findings in acute Q fever are as nonspecific as the clinical symptomatology. Rales are probably the most commonly observed physical finding; evidence of pleural effusion and consolidation also may be noted but not in most infections.

Reports of abnormalities on chest radiographic examination vary with locale, but abnormalities probably are seen 50-60% of the time. The most common abnormality reported is a unilateral, homogenous infiltrate involving 1 or 2 lobes. Rounded opacities and hilar adenopathy are not uncommon. Consider the diagnosis of Q fever when these abnormalities are observed in the setting of acute pneumonia.

Patients with acute Q fever may present with a clinical picture of acute hepatitis with elevations of aminotransferases that are 2- to 3-fold higher than the upper limit of normal. The total bilirubin can be expected to be elevated in 10-15% of patients with acute

Q fever. The white blood cell count is usually normal. The erythrocyte sedimentation rate is elevated in 33% of patients. Mild anemia or thrombocytopenia also may be observed.

Chronic infection with *C burnetii* usually is manifested by infective endocarditis, which also is the most severe complication of Q fever. In addition, hepatitis, infected vascular prostheses, aneurysms, osteomyelitis, pulmonary infection, cutaneous infection, and an asymptomatic form have been reported.

In Q fever endocarditis, fever has been recorded in 85% of patients, along with other systemic symptoms (eg, chills, headache, myalgias, weight loss). Other frequently reported clinical features of Q fever endocarditis include heart failure, splenomegaly, hepatomegaly, clubbing, and cutaneous signs. Routine blood cultures in Q fever endocarditis are negative, and Q fever should be considered when culture-negative endocarditis is encountered. The diagnosis of infective endocarditis secondary to Q fever is confirmed by serologic testing.

DIAGNOSIS- Q FEVER

Diagnosis of Q fever usually is accomplished by serologic testing; the most common methods are complement fixation, indirect fluorescent antibody, and ELISA. Significant antibody titers usually are not identifiable until 2-3 weeks into the illness.

Of the methods currently used for the diagnosis of Q fever, ELISA is the most sensitive and easiest to perform. This assay can establish a diagnosis of Q fever from a single serum specimen with a sensitivity of 80-84% in early convalescence and 100% in intermediate and late convalescence.

TREATMENT OF Q FEVER

Tetracycline has been the mainstay of therapy since the 1950s. When initiated within the first few days of the illness, treatment significantly shortens its course. Macrolide antibiotics, such as erythromycin and azithromycin, are also effective.

When chronic Q fever infection is manifested by infective endocarditis, the mortality is 24% even when patients receive appropriate treatment. At least 2 years of therapy are required, usually with a tetracycline

combined with rifampin or aquinolone, although trimethoprim-sulfamethoxazole also has been used.

PREVENTION/PROPHYLAXIS - Q FEVER

Although an effective vaccine (Q-vax) is licensed in Australia, all Q fever vaccines used in the US are investigational. Q fever can be prevented by immunization.

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Viral Agents

SMALLPOX

INTRODUCTION

Variola, the causative agent of smallpox, is the most notorious of the poxviruses (family Poxviridae). Smallpox was an important cause of morbidity and mortality in the developing world until recent times. In 1980, the World Health Organization (WHO) declared endemic smallpox eradicated, with the last occurrence in Somalia in 1977.

Variola represents a significant threat as a BW agent. Variola is highly infectious and is associated with high mortality and secondary spread. Currently, the majority of the US population has no immunity, little vaccine is readily available, and no effective treatment exists for the disease. Currently, 2 WHO-approved and inspected repositories remain: the CDC in the US and Vector Laboratories in Russia; however, clandestine stockpiles may exist.

PATHOPHYSIOLOGY - SMALLPOX

Variola virus is highly infectious by aerosol, environmentally stable, and can retain infectivity for long periods. Infection through

contaminated fomites is infrequent. After exposure to aerosolized virus, the virus multiplies locally in the respiratory tract. After an incubation period of 7-17 days, variola is spread hematogenously (primary viremia) to regional lymph nodes, where additional replication occurs. Subsequently, variola is spread hematogenously (secondary viremia) to small dermal blood vessels, where skin inflammatory changes (pox) occur. Two types of smallpox generally are recognized. Variola major, the most severe form, has a fatality rate of 30% in unvaccinated individuals and 3% in those previously vaccinated. Variola minor, a more mild form of smallpox, produces lethality in only 1% of unvaccinated individuals.

CLINICAL MANIFESTATIONS - SMALLPOX

After a 7- to 17-day incubation period, symptoms begin acutely with high fever, headache, rigors, malaise, myalgias, vomiting, and abdominal and back pain. During the initial phase, 15% of patients develop delirium, and 10% of light-skinned patients may

develop a fleeting erythematous exanthem. After 2-3 days, an exanthem develops on the face, hands, and forearms and extends gradually to the trunk and lower extremities. The lesions progress synchronously from macules to papules to vesicles to pustules that often are umbilicated, such as in molluscum contagiosum. Centrifugal distribution of the rash is an important diagnostic feature, with a greater number of lesions on the face and extremities compared to the trunk. Patients are most infectious on days 3-6 after the onset of fever. Virus is shed from oropharyngeal and respiratory secretions.

The above described manifestations are known as variola major. In variola minor (ie, alastrim), cutaneous lesions are similar but smaller and fewer in number. Patients are not as ill as those who have variola major are. Small numbers (3%) of patients develop hemorrhagic lesions, and these patients typically die of disease before papules develop. Flat smallpox with macular, soft, velvety lesions develop in 4% of patients and forebodes a poor prognosis. Modified smallpox occurs in those who have been vaccinated and develop a mild prodrome with rapid development of lesions and crusting by day 7. Frequently patients with modified disease form no pustules.

DIAGNOSIS - SMALLPOX

The most difficult aspect of diagnosing smallpox is the current lack of familiarity with the disease for most physicians. Other viral exanthems, such as chicken pox, erythema multiforme with bullae, or allergic contact dermatitis, can look similar. Smallpox is distinguished from chicken pox by the centrifugal distribution of its rash and the presence of lesions at the same stage of development everywhere on the body. The failure to recognize mild cases of smallpox in persons with partial immunity permits rapid person-to-person transmission. Exposed people may shed virus from the oropharynx without ever manifesting disease.

The usual method of diagnosis is demonstration of characteristic virions on electron microscopy of vesicular scrapings. Gispem modified silver stain is rapid but relatively insensitive. When microscopy is unavailable, the gel diffusion test, in which vesicular fluid antigen from a pus lesion is incubated with vaccine hyperimmune serum,

may be used. However, none of the above tests differentiate smallpox from monkeypox or cowpox. PCR techniques have been developed and may provide for more accurate diagnosis in the near future.

TREATMENT - SMALLPOX

It is critical for medical personnel to recognize a vesicular exanthem in possible terrorist areas or warfare theaters as possible smallpox. Immediate reporting of all possible cases must be made to public health

authorities and to the chain of command. Strict quarantine with respiratory isolation for 17 days is applied to all people in direct contact with the index case or cases. All personnel exposed to either weaponized variola or clinical cases must be vaccinated immediately. Immediate vaccination is effective at ameliorating or preventing illness if accomplished within a few days of exposure. Give vaccinia immune globulin (VIG) to patients who cannot receive the vaccine. Treatment of smallpox is mainly supportive. The antiviral agent, cidofovir, is effective in vitro

and may be involved in treatment of symptomatic illness.

PREVENTION/PROPHYLAXIS - SMALLPOX

Smallpox vaccine (attenuated vaccinia virus) is administered by intradermal inoculation with a bifurcated needle. The permanent scar results from a process known as scarification. A vesicle usually appears 5-7 days after inoculation; scabbing over and healing of the site occur over the next 1-2 weeks. Common adverse effects include low-grade fever and axillary lymphadenopathy. The most frequent complication is inadvertent inoculation to other skin or mucous membrane sites or to other people.

The absolute contraindication to vaccination is significant impairment of systemic immunity. Other relative contraindications include immunosuppression, HIV, pregnancy, and history or evidence of eczema. Evidence exists that VIG is of value in postexposure prophylaxis when administered within several days following smallpox exposure and concurrently with vaccination. VIG can be obtained from the CDC

and is administered at a dose of 0.6 mL/kg IM.

MONKEYPOX

The monkeypox virus is a naturally occurring relative of variola, which is formed in Africa. The first case of human monkeypox was identified in 1970, with subsequently confirmed cases totaling less than 400. Some concern exists that monkey pox may be weaponized; however, human monkeypox is less virulent than smallpox. Monkeypox has a case-fatality rate of 11% in humans not vaccinated against smallpox. However, pneumonia due to monkeypox has about a 50% mortality rate. The secondary attack rate is only 9%, far lower than the rates of 25-40% observed in smallpox.

The clinical picture of monkeypox is clinically indistinguishable from smallpox with the exception of enlarged cervical and inguinal lymph nodes. The virus is transmitted by respiratory aerosol or direct contact with an infected individual or fomites. Immunization to vaccinia virus provides protection to 85% of individuals exposed to monkeypox. The treatment

for monkeypox remains supportive.

VIRAL ENCEPHALITIDES

INTRODUCTION

The viral encephalitides, Venezuelan equine encephalitis (VEE) virus, western equine encephalitis (WEE) virus, and eastern equine encephalitis (EEE) virus, are members of the Alphavirus genus and regularly are associated with encephalitis. These viruses first were recovered from horses during the 1930s. VEE was isolated in the Guajira peninsula of Venezuela in 1930, WEE in the San Joaquin Valley of California in 1930, and EEE in Virginia and New Jersey in 1933.

Although natural infections with these viruses occur following bites from mosquitos, the viruses also are highly infectious by aerosol. Alphaviruses replicate readily to very high titers and are relatively stable. They once were used as model systems by which to study different aspects of viral replication genesis and vector relationships. These characteristics and the familiarity of the virus lend it to weaponization. The intentional release as a small particle aerosol may be expected to infect a high

percentage of individuals within an area of a least 10,000 km.

PATHOPHYSIOLOGY - VIRAL ENCEPHALITIDES

After exposure to these viruses, the tissues of the CNS and reticuloendothelial and/or lymphoid systems most commonly are affected in both humans and animals. A systemic viral febrile syndrome characterizes most infections. The severity of response is highly dependent upon host and viral factors, including the species and immune response of the host, route of infection, and strain and dose of the virus. VEE virus has the capacity to produce large human epidemics. Outcomes are significantly worse for young and elderly patients, with case fatalities ranging from 4-35%. WEE and EEE typically produce less fulminant and widespread disease but are associated with case-fatality rates of 50-75% in those with severe illness.

CLINICAL MANIFESTATIONS - VIRAL ENCEPHALITIDES

After an incubation period of 2-6 days, patients with VEE develop fevers, chills, headache,

malaise, myalgias, sore throat, and photophobia. CNS manifestations range from mild confusion and lethargy to seizures, paralysis, and coma. For those that survive, CNS recovery usually is complete.

The incubation period for EEE varies from 5-15 days. Adults may exhibit a viral prodrome of up to 11 days before the onset of CNS manifestations. Signs and symptoms include fever, chills, vomiting, muscle rigidity, lethargy, paresis, excess salivation, and impaired respiratory regulation. Children frequently develop facial and periorbital edema. CNS effects range from mild confusion to seizures and paralysis. Up to 30% of survivors of severe disease have permanent neurologic sequelae, which include seizures and various degrees of dementia.

WEE has an incubation period of 5-10 days. Most patients are asymptomatic or have a nonspecific febrile

illness/aseptic meningitis picture. Manifestations include fever, nausea, vomiting, malaise, headache, nuchal rigidity, and lethargy. The severity of CNS

involvement is inversely proportional to age. Up to 90% of patients younger than 1 year exhibit seizure activity. Typically, adults recover completely. Children have a higher incidence of persistent neurologic sequelae, which ranges from 50% in newborns to 1% in those older than 1 year.

DIAGNOSIS - VIRAL ENCEPHALITIDES

Laboratory evaluation may reveal leukopenia with all 3 viruses. CSF analysis demonstrates from 10 to thousands of white blood cells per microliter with a lymphocyte pleocytosis. Specific diagnosis depends on virus isolation, serology, or both. Virus may be collected from the nasopharynx for 3 days after the onset of symptoms.

TREATMENT - VIRAL ENCEPHALITIDES

No specific treatment is available for the viral encephalitides. Supportive care may include aggressive airway management and antipyretic and anticonvulsant drug administration.

PREVENTION/PROPHYLAXIS - VIRAL ENCEPHALITIDES

TC-83 is a live attenuated vaccine for

VEE. It is administered as 0.5-mL subcutaneous injection for those at high risk, such as laboratory field personnel. Approximately 20% of those who receive the vaccine fail to make a minimum neutralizing antibody response. An additional 25% of those vaccinated develop high fever, chills, and malaise that require bed rest. D-84 is a vaccination developed for those with incomplete inactivation from the TC-83 vaccine. It is an inactivated vaccine, which produces only mild tenderness at the injection site. It also is a 0.5-mL subcutaneous injection, administered at 2- and 4-week intervals until a satisfactory antibody response is measured.

The EEE vaccine is inactivated and administered in a 0.5-mL subcutaneous injection on days 0 and 28. Minimal side effects are noted, and no long-term problems have occurred. Boosters are required to maintain neutralizing titers. Like the EEE vaccine, the VEE vaccine is inactivated, produces no adverse effects, and requires boosters. The vaccine is administered on days 0,

7, and 28.

VIRAL HEMORRHAGIC FEVERS

INTRODUCTION

Viral hemorrhagic fevers are caused by 4 families of viruses, which include the Arenaviridae (Lassa, Argentine, Bolivian, Brazilian, Venezuelan hemorrhagic fevers), Bunyaviridae (Rift Valley, Crimean-Congo, Hantaan), Filoviridae (Marburg, Ebola 4 Fs), and Flaviviridae (Yellow, Dengue, Kyasanur Forest, Omsk HFs). The best known of the viral hemorrhagic fever agents is Ebola virus. First recognized in Zaire in 1976, the virus has been linked to 3 outbreaks in Africa and is associated with a 53-92% mortality rate. A related virus was discovered in Reston, VA, in 1989 in association with an outbreak of illness among cynomolgus monkeys imported from the Philippines. No human cases occurred with this latter outbreak.

These viruses each are characterized by an acute generalized illness that includes malaise, prostration, increased vascular permeability, and abnormalities of circulatory regulation. All agents are highly infectious via the aerosol route, and most are

stable as respiratory aerosols. Thus, they possess characteristics ideal for use by terrorists.

PATHOPHYSIOLOGY - VIRAL HEMORRHAGIC FEVERS

The agents that produce viral hemorrhagic fever are all simple RNA viruses with lipid envelopes. They are stable at a neutral pH and able to survive in blood for long periods, which leads to their infectivity of patients around domestic animal slaughters. These viruses are linked to the ecology of their vector, whether rodent or arthropod, which helps in searching for a diagnosis.

The specific viral hemorrhagic fever syndrome that develops in patients depends on numerous factors such as viral virulence and strain characteristic, routes of exposure, dose, and host factors.

CLINICAL MANIFESTATIONS - VIRAL HEMORRHAGIC FEVERS

All viral hemorrhagic fevers primarily target vascular beds. They produce microvascular damage and enhance vascular permeability. Clinical manifestations include fever, myalgia, prostration,

conjunctival injection, mild hypotension to severe shock, and mucosal and petechial hemorrhages. Neurologic, hematopoietic, hepatic, and pulmonary involvement can be found with more severe disease.

Among the arenaviruses, Lassa fever is not associated with hemorrhagic or neurologic manifestations, but deafness is common. It causes the most severe capillary leak syndrome among hemorrhagic fever viruses and causes massive edema. Argentine and Bolivian hemorrhagic fevers have much more pronounced hemorrhagic and neurologic manifestations.

Rift Valley fever virus is primarily hepatotropic; a small portion of patients demonstrates hemorrhagic signs. Crimean-Congo virus commonly is associated with severe DIC and the most severe hemorrhaging among the viral hemorrhagic fevers. Hantaan virus is associated with pulmonary and renal failure. A sunburn flush on the head, neck, and upper back is somewhat characteristic.

Limited clinical data are available on Marburg and Ebola hemorrhagic fevers. A

nonpruritic, centripetal, pinhead-sized papular erythematous exanthem (visible primarily in fair-skinned patients) and DIC are prominent manifestations of illness.

Of the flaviviruses, yellow fever virus is primarily hepatatrophic. Black vomit caused by hematemesis is common, and patients usually develop clinical jaundice and die from hepatorenal syndrome.

DIAGNOSIS - VIRAL HEMORRHAGIC FEVERS

A high index of suspicion and detailed travel history are the most important steps in making the diagnosis of viral hemorrhagic fever. These agents are linked tightly with their natural geographic area and ecology of the reservoir species and vectors. Patients often recall exposures to rodents (Arenavirus, Hantavirus), mosquitoes (Rift Valley fever virus, yellow and dengue fever viruses), or even slaughtered horses (Rift Valley fever virus, Crimean-Congo virus). Suspect viral hemorrhagic fever in patients with fever, diffuse mucosal and dermal bleeding, and known travel to an endemic area

for the illness.

Laboratory tests may be helpful. Leukopenia and thrombocytopenia are common, except in Lassa fever and Hantavirus. Proteinuria and hematuria are both common, but their absence rules out Hantavirus and Argentine and Bolivian hemorrhagic fevers.

Definitive diagnosis requires specific virologic diagnosis. ELISA can detect early immunoglobulin antibody responses during the acute illness; however, results may take 3-10 days. Reverse transcriptase PCR also may be used to identify a specific RNA virus. Testing can be conducted at the CDC in Atlanta or the US Army Medical Research Institute of Infectious Disease in Frederick, Maryland.

TREATMENT - VIRAL HEMORRHAGIC FEVERS

Treatment for a viral hemorrhagic fever is largely supportive. Patients benefit from rapid nontraumatic hospitalization to prevent damage to the capillary bed. Air transport is contraindicated. Sedative and pain-relieving medications are helpful, but aspirin and other antiplatelet agents should be avoided.

Avoid intravenous lines and catheters unless absolutely necessary. Secondary infections should be sought and aggressively treated.

Immunosuppressive agents such as steroids are contraindicated. The treatment for bleeding is controversial. Generally, mild bleeding should not be treated, whereas severe hemorrhage requires appropriate replacement therapy. Fluid infusions are typically safe for severe volume depletion except in the setting of pulmonary edema.

Specific treatment with ribavirin has been used and currently is being investigated as therapy for Lassa fever, Hantavirus, Crimean-Congo, and Rift Valley Fever. The dosage

is 130 mg/kg IV followed by 15 mg/kg q6h for 4 days, then 7.5 mg/kg q8h for 6 days. Treatment is most effective if begun within 7 days. Ribavirin has poor activity against the filoviruses and flaviviruses.

PREVENTION/PROPHYLAXIS - VIRAL HEMORRHAGIC FEVERS

The only established and licensed virus-specific vaccine against any of the VHF viruses is the yellow fever vaccine. It is mandatory for those traveling into endemic areas of Africa and South America. Current trials are underway for further vaccines and antibody therapies.

[Top](#)

Biological Toxins

STAPHYLOCOCCAL ENTEROTOXIN B

INTRODUCTION

Staphylococcal enterotoxin B (SEB) is one of the best studied and therefore best understood toxins. Staphylococcal enterotoxin is one of the most common causes of food poisoning. Nausea, vomiting, and diarrhea normally occur following ingestion of contaminated foodstuffs. The toxin causes a markedly different clinical syndrome when exposure is through a nonenteric route. In a BW or terrorist situation, the toxin is likely to be acquired through inhalation of an SEB aerosol. SEB is stable as an aerosol, and the inhaled dose necessary to incapacitate individuals is small (0.004 mcg/kg). Within 24 hours of inhalation of SEB toxin, exposed individuals are likely to be incapacitated by systemic illness created by the toxin.

PATHOPHYSIOLOGY - STAPHYLOCOCCAL ENTEROTOXIN B

SEB is an extracellular product produced by coagulase-positive staphylococci. The toxin is heat stable and generally forms in the presence of overgrowth of staphylococcal organisms, as occurs with poorly handled food. The effects of the enterotoxin are mediated by its interactions with the host's own immune system. The toxin binds directly to the major histocompatibility complex and subsequently stimulates large numbers of T lymphocytes. These activated T lymphocytes then stimulate the release of various cytokines, which are thought to mediate the toxic effects of SEB.

CLINICAL MANIFESTATIONS - STAPHYLOCOCCAL ENTEROTOXIN B

After exposure, signs and symptoms begin in 2-12 hours. Mild-to-moderate exposure to SEB produces nonspecific systemic illness that is characterized by fever, chills, headache, nausea, vomiting, dyspnea, chest pain, myalgias, and a nonproductive cough. Severe exposures can lead to a toxic shocklike picture and even death.

In mild-to-moderate cases, the physical examination is typically unremarkable. In severe instances, rales are common from pulmonary edema. Depending on the severity of exposure, duration of illness varies from 3-10 days.

DIAGNOSIS - STAPHYLOCOCCAL ENTEROTOXIN B

Diagnosis of SEB intoxication can be difficult and is made primarily by clinical and epidemiologic methods. Laboratory studies may show a nonspecific neutrophilic leukocytosis and an elevated erythrocyte sedimentation rate. In severe exposures with significant pulmonary symptoms, a chest radiograph may show interstitial

edema.

Enterotoxin is a stable protein that can be collected from serum if performed quickly. However, by the time symptoms are noted, the detection of toxin is unlikely. SEB accumulates in urine and may be detected for several hours following exposure. If the source of infection is from an inhalation injury, the toxin may be isolated from nasal swabs for up to 12-24 hours.

TREATMENT - STAPHYLOCOCCAL ENTEROTOXIN B

Supportive care is the mainstay of treatment. Close attention to oxygenation and hydration are important. Patients with severe SEB may need ventilator support and diuretics. Most patients are expected to do well after the initial phase, but the time to full recovery may be prolonged.

PREVENTION/PROPHYLAXIS - STAPHYLOCOCCAL ENTEROTOXIN B

No approved human vaccine exists for SEB, although human trials are ongoing. Passive immunotherapy agents have demonstrated some promise when given within 4 hours of exposure, but such therapy is still investigational.

RICIN

INTRODUCTION

Ricin, a plant protein toxin derived from the beans of the castor plant, is one of the most toxic and easily

produced of the plant toxins. Although the lethal toxicity of ricin is approximately 1000-fold less than botulinum toxin, the worldwide ready availability of castor beans and the ease with which toxin can be produced give it significant potential as a biological weapon.

Since ancient times, more than 750 cases of ricin intoxication have been described. Ricin may have been used in the highly publicized assassination of Bulgarian exile Georgi Markov in London in 1978. He was attacked with a device that implanted a ricin-containing pellet into his thigh.

PATHOPHYSIOLOGY - RICIN

The toxicity of ricin varies greatly with the route of administration. Ricin is extremely toxic to cells and acts by inhibiting protein synthesis. Inhalation exposure causes primarily pulmonary symptoms, ingestion causes GI symptoms, and intramuscular

exposure results in a localized reaction.

CLINICAL MANIFESTATIONS - RICIN

Following inhalation exposure of ricin, toxicity is characterized by the sudden onset of nasal and throat congestion, nausea and vomiting, itching of the eyes, urticaria, and tightness in the chest. If exposure is significant, pulmonary manifestations occur after 12-24 hours and include airway lesions, alveolar flooding, and severe respiratory distress. In animal studies, death occurs 36-48 hours after severe exposure.

Ingestion of ricin is generally less toxic because of its poor absorption and enzymatic degradation in the digestive tract. Out of 751 ingestions recorded, only 14 resulted in a fatality. Clinical manifestations occur rapidly and are characterized by nausea, vomiting, abdominal pain and cramping, diarrhea, fever and chills, hematochezia, and eventually, shock and vascular collapse. Autopsy findings have revealed significant hepatic, splenic, and renal necrosis.

At low doses, intramuscular exposures produce flulike symptoms, myalgias, nausea, vomiting, and localized pain and swelling at the injection site. Severe intoxication results in local lymphoid necrosis and GI hemorrhage, as well as diffuse hepatic, splenic, and renal necrosis.

DIAGNOSIS - RICIN

The diagnosis of ricin poisoning is made on the basis of clinical and epidemiologic factors. In a BW or terrorist situation, exposure is likely to occur by inhalation of a toxin aerosol. Thus, consider ricin poisoning when patients experience upper airway and pulmonary symptoms in the setting of a known or suspected mass casualty incident.

Patients may have neutrophilic leukocytosis, hypoxemia, and bilateral infiltrates on chest radiograph.

Confirmation of ricin exposure can be made by ELISA analysis of a swab sample from nasal mucosa. Ricin can be identified for up to 24 hours after exposure.

TREATMENT - RICIN

Treatment is supportive. Determine specific treatment largely by the route of exposure and clinical manifestations. Inhalation injury may require treatment of pulmonary edema, with respiratory support as indicated. Early following ingestion, patients should undergo GI decontamination with gastric lavage and the administration of activated charcoal. Intravenous crystalloid infusion and pressor support may be necessary for patients with hypotension.

PREVENTION/PROPHYLAXIS - RICIN

Currently, no vaccine is available for ricin exposure. Investigational vaccines have proven effective in animals. Some chemotherapeutic agents are being studied as well.

BOTULINUM

TOXIN

INTRODUCTION

The anaerobic, spore-forming, gram-positive bacillus, *Clostridium botulinum*, produces botulinum toxins. Botulinum toxins are the most lethal toxins known, with an estimated lethal dose to 50% of the exposed population (LD50) of 0.001 mcg/kg in humans. Since botulinum toxin is so lethal and easy to manufacture and weaponize, it represents a credible threat as a BW agent. When used as a BW or terrorist agent, exposure is likely to occur following inhalation of aerosolized toxin or ingestion of food contaminated with the preformed toxin or microbial spores. Recently, Iraq admitted to active research on the offensive use of botulinum toxins and to weaponizing and deploying more than 100 munitions with botulinum toxin in 1995.

PATHOPHYSIOLOGY - BOTULINUM TOXIN

All 7 subtypes (A-G) of botulinum toxin act by similar mechanisms. The toxin produces similar effects whether ingested or inhaled. The time course and severity of illness vary with

route of exposure and dose received. Symptom onset is slower after inhalation exposure.

Botulinum toxins bind to the presynaptic nerve terminal at the neuromuscular junction and cholinergic autonomic sites. This prevents the presynaptic release of acetylcholine and blocks neurotransmission. Interruption of neurotransmission produces muscular weakness and paralysis.

CLINICAL MANIFESTATIONS - BOTULINUM TOXIN

Symptom onset may occur hours to several days after exposure. Initial signs and symptoms include blurred vision, mydriasis, ptosis, dysphagia, dysarthria, dysphonia, and muscle weakness. After 24-48 hours, neuromuscular manifestations progress to symmetric descending paralysis and respiratory failure. Varying degrees of muscular weakness may occur. Patients may become cyanotic or exhibit narcosis from carbon dioxide retention secondary to respiratory failure. Postural hypotension may occur from autonomic insufficiency. Deep tendon reflexes may be depressed or absent on

physical examination. Cranial nerve palsies often are present.

DIAGNOSIS - BOTULINUM TOXIN

The occurrence of multiple related cases of descending and progressive bulbar and skeletal paralysis in afebrile patients should suggest the diagnosis of botulinum toxicity. Laboratory tests, including CSF studies, generally are not helpful. Oral exposure can be detected by analyzing serum or gastric contents with a mouse neutralization assay. Intoxication by inhalation can be diagnosed by ELISA identification from nasal swabs up to 24 hours after exposure.

TREATMENT - BOTULINUM TOXIN

The most serious complication of toxicity is respiratory failure. With supportive care and ventilatory assistance, fatalities should be less than 5%. For confirmed exposures, a trivalent equine antitoxin is available from the CDC. This antitoxin has all of the disadvantages of horse serum products, including the risks for anaphylaxis and serum sickness. Skin testing is performed by

injecting 0.1 mL of a 1:10 dilution of antitoxin intradermally and monitoring the patient for 20 minutes. After a negative skin test, administer the antitoxin at a dose of 10 mL IV over 20 minutes, which is repeated until improvement ceases. With a positive skin test, administer 0.01-0.1 mL of antitoxin subcutaneously, gradually increasing the dose every 20 minutes until 2 mL can be sustained without reaction.

PREVENTION/PROPHYLAXIS - BOTULINUM TOXIN

A toxoid for *C botulinum*, which has been used in volunteers and occupationally at-risk workers, is available through the CDC. It was used to immunize US military troops in the Persian Gulf War. The current schedule for immunization is at 0, 2, and 12 weeks with an annual booster. Currently, no indication exists for prophylactic use of the antitoxin except under specialized circumstances.

MYCOTOXINS

INTRODUCTION

The trichothecene mycotoxins are highly toxic compounds produced by certain species of filamentous fungi (*Fusarium*, *Myrotecium*, *Cephalosporium*, *Trichoderma*, *Verticimonosporium*, *Stachybotrys* species). These mycotoxins (eg, T2, nivalenol) cause multiple organ effects, which include emesis, diarrhea, weight loss, nervous disorders, cardiovascular alterations, immunosuppression, hemostatic derangements, skin toxicity, and bone marrow damage.

Because of their antipersonnel properties, ease of large-scale production, and amenability to dispersal by various methods (dusts, droplets, aerosols, smoke, rockets, artillery mines, portable sprays), mycotoxins have an excellent potential for weaponization.

Strong evidence suggests that trichothecenes ("yellow rain") have been used as a BW agent in Southwest Asia and Afghanistan. From 1974-1981, numerous attacks resulted in a minimum of 6310 deaths in Laos, 981 deaths in Cambodia, and 3042 deaths in Afghanistan.

PATHOPHYSIOLOGY - MYCOTOXIN

These toxins are nonvolatile, low molecular weight compounds that are highly soluble in acetone, ethyl acetate, chloroform, ethanol, methanol, and propylene glycol. The trichothecenes vaporize when heated in organic solvents. Extraction of the mycotoxin from fungal cultures yields a yellow-brown liquid that evaporates into a yellow crystalline product (thus, the "yellow rain" appearance). These toxins require a 3-5% sodium hydroxide solution and heating at 900Â°F for 10 minutes or 500Â°F for 30 minutes for complete inactivation.

The trichothecene mycotoxins are cytotoxic to most eukaryotic cells by way of inhibiting protein synthesis and electron transport. Rapid absorption from the gut or

pulmonary mucosa can produce initial symptoms in 5 minutes and maximal effects by 60 minutes. Peak tissue levels occur 1-4 hours following exposure. These toxins are absorbed slowly through intact skin.

CLINICAL MANIFESTATIONS - MYCOTOXIN

After exposure to the mycotoxins, early symptoms begin within minutes. Cutaneous manifestations include burning, tender erythema, edema, and blistering with progression to dermal necrosis and sloughing of large skin areas in lethal cases. Respiratory exposure results in nasal itching, pain, sneezing, epistaxis, rhinorrhea, dyspnea, wheezing, cough, and blood-tinged saliva and sputum.

GI toxicity consists of anorexia, nausea and vomiting, abdominal cramping, and watery and/or bloody diarrhea. Following entry into the eyes, pain, tearing, redness, and blurred vision occurs. Systemic toxicity may occur and includes weakness, prostration, dizziness, ataxia, tachycardia, hyperthermia or hypothermia, diffuse bleeding, and hypotension. Death may

occur within minutes to days depending on the dose and route of exposure.

DIAGNOSIS - MYCOTOXIN

Diagnosis of an attack of trichothecene mycotoxin depends on clinical observations and identification of the toxin from biological and environmental samples. Many patients presenting with the above symptoms and reporting a yellow rain or smoke attack lend support to the diagnosis. Initial laboratory studies are nonspecific. Elevations of serum creatinine, potassium, and phosphorus may occur, as well as abnormalities of coagulation parameters. An initial rise in absolute neutrophils can be seen. Leukopenia, thrombocytopenia, and anemia may occur 2-4 weeks following initial exposure.

Currently, a rapid identification kit for any of the trichothecene mycotoxins does not exist. Gas-liquid chromatography has been used in the past with great success. However, chromatographic methods lack great sensitivity, and presently alternative methods of detection are under investigation (eg, radioimmunoassay, ELISA, mass-spectrometry).

TREATMENT - MYCOTOXIN

Treatment is supportive. The immediate use of protective clothing and mask during a mycotoxin aerosol attack should prevent illness. If a soldier is unprotected during an attack, the outer clothing should be removed within 4-6 hours and decontaminated with 5% sodium hydroxide for 6-10 hours. The skin should be washed with copious amounts of soap and uncontaminated water. The eyes, if exposed, should be irrigated with copious amounts of normal saline or sterile water. A skin decontamination kit (M291) currently is fielded to all US military personnel and is efficacious against most chemical warfare agents, including the mycotoxins.

No specific therapy exists for a trichothecene exposure. After appropriate skin decontamination, give victims of inhalation and oral exposures superactivated charcoal orally. Activated charcoal binds mycotoxins. Treat severe respiratory distress with endotracheal intubation and mechanical ventilation as needed. Early use of systemic steroids increases survival time by decreasing the primary injury and shocklike state that follows significant poisoning.

PREVENTION/PROPHYLAXIS - MYCOTOXIN

No vaccine exists for trichothecene mycotoxin exposure. Currently, 2 topical skin protectants as well as vaccines are in advanced development but have not been approved yet for use in humans.

Pictures



Picture 1: Al Hakam Single-Cell Protein Plant. Iraq's major facility for the production of biological warfare agents. Under the watchful eye of the United Nations Special Commission, this plant was destroyed by Iraqi workers in May and June 1996. (Photo courtesy of David Franz, DVM, PhD)

Picture type: Photo



Picture 2: The "eight ball" one million liter test sphere at Fort Detrick, Maryland. This aerobiology chamber was used before 1969 when the US was performing offensive biological warfare research. Animals were tethered within the sphere while aerosolized agents were aerosolized.

Picture type: Photo



Picture 3: The "eight ball" one million liter test sphere at Fort Detrick, Maryland. This aerobiology chamber was used before 1969 when the US was performing offensive biological warfare research. Animals were tethered within the sphere while aerosolized agents were aerosolized.

Picture type: Photo



Picture 4: Anthrax pilot plant used to produce billions of anthrax spores at Fort Detrick, Maryland, before the US unilaterally ended offensive biological warfare research in 1969. Spores were sent to other military sites where they were placed in weapons. The building is entirely free of anthrax spores and on the National Register of Historic Places. Tours frequently take visitors through this old production facility.

Picture type: Photo



Picture 5: Entrance to Fort Detrick with headquarters building in background.

Picture type: Photo



Picture 6: Front of main building at the United States Army Medical Research Institute of Infectious Diseases (USAMRIID) on the grounds of Fort Detrick, Maryland.

Picture type: Photo



Picture 7: A suppurative bubo of the femoral lymph node (a), the most common site of erythematous, tender, swollen nodes in a plague victim. The next most common lymph node regions involved are the inguinal, axillary (b), and the cervical areas. The child in (b) has an erythematous, eroded, crusting necrotic ulcer at the presumed primary inoculation site on the left upper quadrant. This type of lesion is uncommonly found in patients with plague. Bubo location is primarily a function of the region of the body in which an infected flea inoculates plague bacilli. (Photos courtesy of Jack Poland, PhD, Centers for Disease Control and Prevention, Fort Collins, CO)

Picture type: Photo



Picture 8: A suppurative bubo of the femoral lymph node (a), the most common site of erythematous, tender, swollen nodes in a plague victim. The next most common lymph node regions involved are the inguinal, axillary (b), and the cervical areas. The child in (b) has an erythematous, eroded, crusting necrotic ulcer at the presumed primary inoculation site on the left upper quadrant. This type of lesion is uncommonly found in patients with plague. Bubo location is primarily a function of the region of the body in which an infected flea inoculates plague bacilli. (Photos courtesy of Jack Poland, PhD, Centers for Disease Control and Prevention, Fort Collins, CO)

Picture type: Photo



Picture 9: Small femoral bubo and presumed inoculation site (inferior thigh) in a patient with tularemia. This gram-negative bacterial infection (*Francisella tularensis*) may closely mimic bubonic plague and is successfully treated with the same antibiotics. (Photo courtesy of Dermatology Service, Fitzsimons Army Medical Center, Aurora, CO)

Picture type: Photo



Picture 10: Vesicles and erosions on the back of hairless guinea pigs at (a) 1 day, (b) 2 days, (c) 7 days, and (d) 14 days after application of (bottom to top) 25 ng, 50 ng, 100 ng, or 200 ng of T-2 mycotoxin in 2mL of methanol. (Reprinted from Wannamacher RW Jr, Wiener SL. Trichithecene mycotoxins. In: Zajtchuk R, Bellamy RF, eds. *Medical Aspects of Chemical and Biological Warfare- Textbook of Military Medicine*. Washington, DC: US Department of the Army, Office of the Surgeon General, and Borden Institute; 1997: 666.) Government publication, no copyright on photos

Picture type: Photo



Picture 11: Adult with variola major with hundreds of pustular lesions distributed centrifugally. (Fitzsimons Army Medical Center Slide File)

Picture type: Photo



Picture 12: Boy with monkeypox in Democratic Republic of the Congo in 1996. Note the (a) centrifugal distribution and (b) synchronicity of lesions as was typical for smallpox. (Courtesy of William Clemm)

Picture type: Photo



Picture 13: Boy with monkeypox in Democratic Republic of the Congo in 1996. Note the (a) centrifugal distribution and (b) synchronicity of lesions as was typical for smallpox. (Courtesy of William Clemm)

Picture type: Photo



Picture 14: New World Arenavirus - Machupo. Oral mucosal hyperemia and hemorrhage in a patient with Bolivian Hemorrhagic Fever caused by Machupo virus (Photo courtesy of C.J. Peters, MD).

Picture type: Photo



Picture 15: Bunyavirus infection - CCHF Virus. Ecchymoses encompassing left upper extremity one week after onset of CCHF. Ecchymoses often are accompanied by hemorrhage in other locations: epistaxis, puncture sites, hematemesis, melena, and hematuria. (Reprinted from Jahrling PB. Viral Hemorrhagic Fevers. Chapter 29 In: Sidell Fr, Takafuji ET, Franz DR, eds. Medical Aspects of Chemical and Biological Warfare. In: Zajtchuk R, Bellamy RF, eds. Textbook of Military Medicine. Washington, DC: US Department of the Army, Office of the Surgeon General, and Borden Institute; 1997: 595.) (Photo courtesy of Robert Swaneopel, PhD, DTVM, MRCVS, National Institute of Virology, Sandringham, South Africa.) Government publication, no permission needed.

Picture type: Photo



Picture 16: Bunyavirus Infection - Hantaan Virus. Patient with Korean Hemorrhagic Fever caused by Hantaan virus demonstrating typical sunburn flush of cheeks, chin, and base of neck. (Photo courtesy of John Huggins, PhD)

Picture type: Photo



Picture 17: Ulceroglandular tularemia demonstrating inoculation site on cheek and cervical lymphadenopathy (Fitzsimons Army Medical Center Dermatology Slide File)

Picture type: Photo



Picture 18: Typical heaped up ulcer of tularemia. (Reprinted from Evans ME, Friedlander AM. Tularemia. Chapter 24 In: Sidell FR, Takafuji ET, Franz DR, eds. Medical Aspects of Chemical and Biological Warfare. In: Zajtchuck R, Bellamy RF, eds. Textbook of Military Medicine. Washington, DC: US Department of the Army, Office of the Surgeon General, and Borden Institute; 1997:505.) Government publication, no permission needed.

Picture type: Photo

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CBRNE - Botulism

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Introduction

Background

Botulism is a paralytic disease caused by the neurotoxins of *Clostridium botulinum* and in rare cases, *Clostridium butyricum* and *Clostridium baratii*. These gram-positive, spore-forming anaerobes can be found in soil samples and marine sediments throughout the world. With a lethal dose to humans of less than 1 mcg, botulinum toxins are among the most poisonous substances known and pose a great threat as an agent of biological warfare.

Classification

Three forms of botulism are recognized classically, grouped according to the mode of acquisition. The first form, food-borne botulism, follows the ingestion of preformed toxin in foods that have not been canned or preserved properly.

Wound botulism, caused by systemic spread of toxin produced by organisms inhabiting wounds, is associated with trauma, surgery, subcutaneous heroin injection, and sinusitis from intranasal cocaine abuse.

Infant botulism results from intestinal colonization of organisms in infants younger than 1 year. In this age group, normal intestinal flora may not have developed to the degree that prevents colonization by

these organisms in healthy adults.

More recently, a fourth form, adult intestinal colonization botulism, has been identified. Similar in pathogenesis to infant botulism, this form occurs in older children and adults in the presence of colitis, with a recent history of bowel surgery, or in association with other conditions that may create local or widespread disruption in the normal intestinal flora.

Differences in antigenicity among the toxins produced by different strains of botulism-causing organisms allow for separation of the organisms into 7 distinct types (A-G). Types A, B, and E are the toxins most often responsible for disease in humans, while types C and D only cause disease in other animals (eg, nonhuman mammals, birds, fish). In rare instances, a single strain of organism may produce 2 toxins.

As alluded to earlier, clostridia other than *C botulinum* have been associated with a handful of cases of botulism. These include reports of food-borne and infant botulism associated with type E toxin produced by *C butyricum*. Adult and infant intestinal colonization botulism, associated with type F toxin-producing *C baratii*, has been documented.

In addition, strains of *C botulinum* have been classified into 4 groups based on their phenotypic characteristics and DNA homology.

- Group I organisms are proteolytic and produce toxins A, B, or F.
- Group II is nonproteolytic and can make toxins B, E, or F.
- Group III organisms produce toxins C or D.
- Group IV organisms, now identified as *Clostridium argentinense*, produce toxin type G, which has not been shown to cause neuromuscular illness but has been associated with sudden death in Switzerland.

Pathophysiology

Epidemiology

Food-borne botulism, the first form of the disease to be identified, is responsible for 963 annual cases worldwide. While European cases most commonly are associated with type B contamination of home-processed meats, Alaskan, Canadian, and Japanese outbreaks often involve type E toxin in preserved seafood. Chinese cases involve type A toxin in home-processed bean products.

Most cases in the continental US are associated with home-canned vegetable products such as asparagus, green beans, and peppers. Of the average 30 food-borne US cases (from 14 different outbreaks) per year, 60% are type A, 18% type B, and 22% type E. Alaska, California, Michigan, Washington, New Mexico,

Illinois, Oregon, and Colorado are states with the highest incidence rates of food-borne botulism.

The toxin type most often responsible for food-borne illness corresponds well with the geographic distribution of the toxigenic species. Types A, B, and E are most common in the western US, the eastern US and Great Lakes area, and Alaska, respectively. Toxin type A produces a more severe illness than type B, which in turn is more severe than type E.

By far, home-processed foods are responsible for most (94%) outbreaks in the continental US. In fact, of the 6% of outbreaks caused by mass-produced commercial foods, most cases were attributed to consumer mishandling of commercial products.

Infant botulism occurs most commonly in those aged 1 week to 11 months, with peak susceptibility occurring at 2-4 months. In the 16 years following its identification in 1976, 1134 cases of infant botulism have been recorded in the US. With approximately 60 cases of infant botulism reported each year, it is now the most frequently occurring form of botulism. The disease is most common in the western part of the US. One half of all annual cases are reported in California, where the frequency of the toxin responsible is distributed equally between types A and B.

While the toxin types of food-borne botulism seem to reflect the distribution of toxigenic strains in the environment, the frequency of type B toxin in infantile botulism is disproportionately high. Although the case-fatality ratio for infant botulism in the US is less than 2%, the disease may be responsible for up to 5% of sudden infant death syndrome cases in California.

Although the ingestion of honey has been identified as a strong risk factor for the disease, it is found in fewer than 20% of case histories (and only 5% of case histories in California in recent years).

Other risk factors that have been reported include infants with higher birth weights and mothers who were older and better educated than the general population. Another reported risk factor was a decreased frequency of bowel movements (<1/d) for at least 2 months. Breastfeeding was associated with older age at onset of illness in type B cases.

Through 1992, only 1-3 cases of wound botulism were reported in the US each year. Two thirds of these cases were type A and almost one third were type B. One half of all cases were reported from California. In recent years, the number of reported cases of wound botulism has risen dramatically, with 11 cases in California in 1994 and 19 cases confirmed by the state's Department of Health Services during the first 11 months of 1995. All but 1 of 40 cases reported in California, at this writing, involved drug abusers, many with subcutaneous injection or skin-popping of heroin.

Seven cases of adult colonization botulism have been reported in the literature. In some of these cases, *C botulinum* organisms, but no preformed toxin, could be found in foods the patients had ingested. These cases were associated with a prolonged latent period of up to 47 days postingestion before onset of symptoms. In one study, 2 of 4 patients had surgical alterations of the gastrointestinal tract that may have

promoted colonization. Jejunioileal bypass, surgery of the small intestine, and Crohn disease are among other reported factors predisposing adult patients for intestinal colonization.

Pathogenesis

C botulinum is distributed widely throughout the environment and can be found in soil, freshwater and saltwater sediments, household dust, and on the surfaces of many foods. The toxins produced are cytoplasmic proteins (mass = 150 kDa) released as cells lyse. While the spores survive 2 hours at 100°C (but die rapidly at 120°C), the exotoxin is heat labile. It becomes inactivated after 1 minute at 85°C or 5 minutes at 80°C.

Although the mode of entry of toxin may differ between the different forms of diseases, once the toxin enters the bloodstream, it acts in a similar manner to produce the clinical symptoms. The toxin binds to receptors on presynaptic terminals of cholinergic synapses, is internalized into vesicles, and then is translocated to the cytosol. In the cytosol, the toxin mediates the proteolysis of components of the calcium-induced exocytosis apparatus to interfere with acetylcholine release. Blockade of neurotransmitter release at the terminal is permanent, and recovery only occurs when the axon sprouts a new terminal to replace the toxin-damaged one.

The effects of the toxin are limited to blockade of peripheral cholinergic nerve terminals, including those at neuromuscular junctions, postganglionic parasympathetic nerve endings, and peripheral ganglia. This blockade produces a characteristic bilateral descending paralysis of the muscles innervated by cranial, spinal, and cholinergic autonomic nerves but no impairment of adrenergic or sensory nerves.

Mortality/Morbidity

- For food-borne botulism, patients with an early onset of clinical symptoms (within 36 h of toxin ingestion), index patients in food-borne cases, and patients older than 60 years generally have a longer clinical course than their counterparts.
- Deaths occurring within the first 2 weeks of botulism are most often due to failure to recognize the severity of disease or from pulmonary or systemic infection. The average duration of respiratory support for those requiring mechanical ventilation is 6-8 weeks but may be as long as 7 months.
- For food-borne botulism, severity of disease seems to be associated with toxin type. Intubation is required for 67% of patients with type A botulism, 52% of patients with type B, and 39% of patients with type E. In addition to being more likely to need ventilatory support, patients with type A botulism tend to see a physician earlier and have a longer course of illness than those with type B botulism. The case-fatality rate for type A botulism is 10%, twice
- The overall case-fatality rate for the 1976-84 period was 7.5%, while the case-fatality rate for patients older than 60 years was 30%.
- Infant botulism progresses for 1-2 weeks and stabilizes for 2-3 weeks before recovery begins. The average length of a hospital stay for infants is approximately 1 month, although excretion of toxin

and organisms may continue for more than 3 months following discharge. The infant case-fatality rate (1.3%) is low compared to food-borne illness, but a 5% relapse rate is associated with infant botulism after apparent resolution of clinical symptoms.

- The case-fatality rate of wound botulism is 10%, and survivors experience significant morbidity requiring prolonged medical care.
- The occurrence of an episode of botulism does not necessarily confer immunity toward subsequent episodes. Immunization in the form of a pentavalent toxoid is available, but it is used only for those in high-risk areas such as laboratory workers and certain military personnel.

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Clinical

History

- Although laboratory confirmation is necessary for a definitive diagnosis, clinical presentation, patient history, and physical examination (particularly neurologic exam) can be used as strong indicators for the presence of botulism.
- Place special attention on eliciting a complete patient history, including the following:
 - History of foods eaten
 - History of drug abuse
 - Recent surgery or trauma
 - Gastrointestinal problems

Physical

- Food-borne botulism
 - The CDC suggests attention to the following cardinal features:
 - Patient is afebrile unless another infection is present.
 - Patient demonstrates symmetric neurologic symptomatology.
 - Patient remains responsive.
 - Patient has a normal or slow heart rate in the absence of hypotension.
 - Signs typically are not accompanied by sensory deficits, with the exception of blurred vision.
 - Respiratory difficulty arises from airway obstruction and diaphragmatic weakness. Diplopia, dysarthria, dry mouth, and generalized weakness are among the most common presenting symptoms. Other symptoms that have been associated with botulism include ptosis, dysphagia, sore throat, dysphonia, nystagmus, ataxia, paresthesias, paralytic ileus, severe constipation, urinary retention, and orthostatic hypotension.

- Pupils are dilated or unreactive (ophthalmoplegia) in 50% of patients. Unless secondary complications such as respiratory failure develop, patients are alert and mental function is unimpaired.
- Sensory deficits only have been reported in isolated cases. Neurologic symptoms may appear from 6 hours to 10 days after ingestion of toxin, with a median incubation period of 1 day.
- Nausea, vomiting, and diarrhea often precede or accompany neurologic manifestations; constipation typically follows after neurologic signs have appeared.
- Wound botulism
 - Patients often present with much of the same symptomatology that is observed in the food-borne form, including acute blurred vision, dysphagia, dysarthria, generalized weakness (with or without absence of deep tendon reflexes), and pupillary abnormalities. Gastrointestinal manifestations are absent.
 - The *Clostridium*-infected wound may appear benign unless infected by other bacteria, in which case a fever also may be present.
 - The average incubation period is 10 days.

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Differentials

Diphtheria
Encephalitis
Guillain-Barré Syndrome
Hypermagnesemia
Lambert-Eaton Myasthenic Syndrome
Myasthenia Gravis

Other Problems to be Considered

Congenital neuropathy or myopathy

Mushroom (muscarine) poisoning

Poliomyelitis

Tick paralysis

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Workup

Lab Studies

- Laboratory confirmation
 - Before treatment with antitoxin, obtain 10-15 mL of serum, 25-50 g of feces, and possibly 25-50 mL of fluid from gastric aspiration. Collect and refrigerate similar quantities of suspected food samples for testing.
 - Label each specimen container with the patient's name, specimen type, date of collection, and medications being received and send it to a state health department-approved reference laboratory in insulated cold packs.
 - Confirmation of the organism and/or toxin and toxin typing is obtained in almost 75% of cases. Early cases are more likely to be diagnosed by toxin assay, whereas later ones are more likely to have a positive culture. Laboratory confirmation of toxin presence is via a mouse bioassay, and identification of the toxin type is performed by a mouse toxin neutralization test.
- Infant botulism
 - In patients whom infant botulism is suspected, stools and enema fluids (with minimal water added to limit dilution of toxin) are the specimens of choice, as serum is only rarely toxin positive.
 - One also may wish to culture possible sources of clostridia, such as honey or house dust.
- Adult colonization botulism: Organisms may be detected in stool and toxin in serum for up to 119 days following the onset of symptoms.
- New methods of detection: In vitro methods of detection, including polymerase chain reaction-based detection of toxin genes, have been reported, but these methods still are being evaluated and are not widely available at present.

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Treatment

Emergency Department Care

- Food-borne botulism
 - Monitor asymptomatic individuals who have eaten food suspected of being contaminated for the appearance of neurologic signs and symptoms. Therapy is not recommended unless

definitive evidence of food contamination is available.

- Enemas and cathartics may be used (if no ileus is present) to purge the gut of toxin. If ingestion occurred within the past few hours, emetics or gastric lavage may aid in the removal of toxin.
- Wound botulism
 - Wound botulism requires thorough debridement of the wound site, even if it appears to be healing well.
 - Follow this by injection of 3% hydrogen peroxide to produce aerobic conditions. Hydrogen peroxide itself is not innocuous to tissues, and some have advocated using hyperbaric oxygen therapy if available.
 - Antitoxin may be injected directly into the wound site.
 - Urinary retention may require use of a catheter.

Consultations

- Pulmonology for respiratory sequelae
- Surgery for wound care
- Infectious disease specialist for management issues

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Medication

The goals of pharmacotherapy are to reduce morbidity and prevent complications. Medication commonly used in the treatment of botulism is described below. In addition to that described, guanethidine and 4-aminopyridine have been used for the treatment of botulinum paralysis but have not been shown to be effective.

The use of local antibiotics such as penicillin G or metronidazole may be helpful in eradicating *C botulinum* in wound botulism. Antibiotic use is not recommended for infant botulism because cell death and lysis may result in the release of more toxin. Aminoglycoside antibiotics and tetracyclines, in particular, may increase the degree of neuromuscular blockade by impairing neuronal calcium entry.

Antitoxin Therapy

Therapy consists of approximately 10,000 IU of antibodies against toxin types A, B, and E to neutralize serum toxin concentrations.

| | |
|-------------------|--|
| Drug Name | <p>Trivalent equine botulism antitoxin- CDC recommends administration of 1 vial of antitoxin for adult patients with botulism as soon as diagnosis is made, without waiting for laboratory confirmation; before administration of antitoxin, perform skin testing for sensitivity to serum or antitoxin; 1 vial of trivalent botulism antitoxin administered IV results in serum levels of type A, B, and E antibodies; capable of neutralizing serum toxin concentrations in substantial excess of those reported for botulism patients; administration of 1 vial of antitoxin IV recommended and need not be repeated (circulating antitoxins have a half-life of 5-8 d).</p> <p>Antitoxin packages, which include instructions for skin or conjunctival testing for hypersensitivity to horse serum and a regimen for desensitization, are available through the CDC (404-639-2206 or 404-639-3753 workdays, 404-639-2888 weekends and evenings); antitoxin packages also may be obtained through state health departments.</p> <p>Some sources report that antitoxin may be re-administered q4h for severe cases; others state that 2 vials (approximately 20 mL) of antitoxin are as effective as 4. Lower doses are associated with fewer adverse effects.</p> <p>Although evidence supporting use is limited, has been shown to lower fatality rates and shorten illness for type A food-borne botulism; particularly true when administered in the first 24 h after the onset of illness.</p> <p>Antitoxin neutralizes toxin not yet bound to nerve terminals and has circulating half-life of 5-8 d; patients who do not receive antitoxin treatment show free toxin in serum for up to 28 d.</p> <p>For infant botulism, IV immune globulin trials in California were completed in early 1997; trials demonstrated safety and efficacy of human-derived botulinum antitoxin and a reduced mean hospital stay from 5.5 wk to 2.5 wk. BIG is now available from California Department of Health Services (24-h telephone: 510-540-2646).</p> |
| Adult Dose | 1 vial (10 mL) IV once |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Adverse reactions include serum sickness (3.6%), urticaria (2.6%), and anaphylaxis (1.9%) |

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Follow-up

Further Inpatient Care

- Ventilatory support
- Surgical debridement of wounds
- Antitoxin or immune globulin, if indicated
- Pediatric nutritional support: Intravenous feeding (hyperalimentation) is discouraged because of its potential for secondary infection and because of the success with nasogastric or nasojejunal tube feeding.

Deterrence/Prevention

- Inform the public about the hazards of improperly preserved or canned foods.
- Inform expectant mothers not to administer honey to infants.

Complications

- Wound infection
- Respiratory distress

Prognosis

- Prognosis is generally good with early detection and intensive therapy.

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CBRNE - Brucellosis

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Synonyms, Key Words, and Related Terms

Malta fever, Crimean fever, undulant fever

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Introduction

Background

Brucellosis is a zoonotic infection transmitted from animals to humans by ingestion of infected food products, direct contact with an infected animal, or inhalation of aerosols. This last method of transmission is remarkably efficient given the relatively low concentration of organisms (as few as 10-100 bacteria) needed to establish infection in humans and has brought renewed attention to this old disease. First officially diagnosed as an infection in British soldiers, brucellosis now is touted as a potential biological warfare agent. However, its relatively long and variable incubation period (1-8 wk),

as well as the fact that many infections are asymptomatic, has made it a less desirable agent for weaponization.

Descriptions of the disease date back to the days of Hippocrates, although the organism was not isolated until 1887, when British Army physician David Bruce isolated the organism that bears his name from the spleens of 5 patients with fatal cases on Malta. The disease gets its names from both its course (undulant fever) and location (Malta fever, Crimean fever).

In the ensuing years, different species of *Brucella* were identified and named primarily either for the source animal or features of infection. Currently, of the 6 main species of *Brucella*, 4 have moderate-to-significant human pathogenicity: *Brucella suis* (from pigs; high pathogenicity); *Brucella melitensis* (from sheep; highest pathogenicity); *Brucella abortus* (from cattle; moderate pathogenicity); and *Brucella canis* (from dogs; moderate pathogenicity). Given the ease of aerosol transmission of *Brucella* species, researchers attempted to develop it into a biological weapon beginning in 1942, and in 1954 it became the first agent weaponized by the old US offensive biological weapons program. Field testing on animals soon followed. By 1955, the US was producing *B suis*-filled cluster bombs for the US Air Force at the Pine Bluff Arsenal in Arkansas. Of note, *B melitensis* actually produces more severe disease in humans.

Development of brucellae as a weapon was halted in 1967, and President Nixon later banned development of all biological weapons on November 25, 1969. Although the *Brucella* munitions never were used against human targets, the research performed resulted in concern that *Brucella* species someday may be used as a weapon against either military or civilian objectives.

Pathophysiology

Brucellae are aerobic gram-negative coccobacilli that produce urease and catalyze nitrite to nitrate. They have a lipopolysaccharide coat that is much less pyrogenic than other gram-negative organisms, which accounts for the rare presence of high fever in brucellosis. Brucellae can gain entry into humans through breaks in the skin, mucous membranes, conjunctiva, and respiratory and GI tracts. Sexual transmission is not documented convincingly. Ingestion usually occurs by way of unpasteurized milk, as meat products often have a low bacterial load. Percutaneous needlestick exposure, conjunctival exposure through eye splash, and inhalation are the most common routes in the US.

Both polymorphonuclear leukocytes and macrophages ingest brucellae, but the organism can prevent fusion of phagosome and lysosome. Brucellae are transported into the lymphatic system and may replicate there locally; they also may replicate in the kidney, liver, spleen, breast tissue, or joints, causing both localized and systemic infection. Granulomas may accompany extracellular replication of the bacteria, especially in the liver and spleen. *B abortus* can replicate in fetal tissue, causing abortion, although this usually is observed in cattle. The primary method of control is cell-mediated immunity rather than antibodies, although some immunity to reinfection is provided by serum immunoglobulins. Initially, immunoglobulin M (IgM) levels rise, followed by immunoglobulin G (IgG) titers. IgM may remain in the serum in low levels for several months, whereas IgG eventually declines. Persistently

elevated IgG titers or second rises in IgG usually indicate chronic or relapsed infection.

Frequency

- **In the US:** In the US, frequency is related to the number of infected animals. Infected animals are rare in the US, and pasteurization of milk has eliminated that potential reservoir, thus infection generally occurs via occupational exposure (cattlemen, veterinarians, slaughterhouse workers). The incidence is approximately 200 per year or 0.04 per 100,000. Patients in the US primarily are found in Texas, California, Virginia, and Florida.
- **Internationally:** Frequency of brucellosis varies across nations but obviously is higher in more agrarian societies and in places where handling of animal products and dairy products is less stringent. The highest incidence is observed in the Middle East, Mediterranean region, China, India, Peru, and Mexico.

Mortality/Morbidity

Mortality from brucellosis is rare and usually is secondary to endocarditis (which occurs in approximately 2% of patients). Because of the predilection to affect joints and the vague symptoms and chronic nature of the disease, symptoms can result in relatively long-term disability. However, nearly all patients respond to appropriate antibiotic therapy, with fewer than 10% relapsing. This potential for long-lasting infection that can disable workers in either military or civilian circles makes *Brucella* species an appealing choice for a biological weapon.

Race

Since exposures tend to be primarily occupational, no race predilection exists in the US.

Sex

Exposures are occupational and demonstrate no specific gender preference.

Age

Generally no specific age predilection exists because of limited chance for exposure, although brucellosis is unusual in very young or elderly patients.

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Clinical

History

History is the most helpful component in diagnosing brucellosis.

- Unless exposure is due to a weaponized attack, almost every case either directly or indirectly involves exposure to an affected animal.
- Elicit an occupational history (eg, farmer, veterinarian) that is suggestive of exposure to a source animal.
- Suspected biological attack should heighten awareness of potential infection.
- As brucellae typically take 1-8 weeks to incubate, include in the history any possible exposures in the preceding few months.
- Obtain exposure to potentially contaminated foodstuffs or travel to an area where the disease is endemic.
- Symptoms of brucellosis are protean and nonspecific. Somatic complaints (weakness, fatigue, malaise, body aches, depression, anorexia) may often predominate.
 - Onset may be an abrupt acute febrile illness, chronic infection, or localized infection.
 - When case reviews were performed, certain symptoms were noted to be more prevalent. Fever was observed in 90-95% of patients, malaise in 80-95%, myalgias in 40-70%, sweats in 40-90%, and arthralgias in 20-40%. Except for fever and malaise, most symptoms were observed in half or fewer than half of patients.
 - Neuropsychiatric complaints may include depression, headache, and irritability. In patients with advanced cases where meningoencephalitis is present, these complaints may include changes in mental status, coma, neurologic deficit, nuchal rigidity, or seizures.
 - Arthralgias may be diffuse or localized, with a predilection to bone ends and the sacroiliac joint. Although uncommon, acute monoarticular arthritis may be part of the presentation.
 - In respiratory infection, nonproductive cough and pleuritic chest pain predominate.
 - Patients with prolonged cases often experience weight loss, fatigue, and anorexia.
 - A significant percentage of patients may have GI complaints, primarily dyspepsia, although abdominal pain from hepatic abscesses may occur. Suspect hepatic abscesses in patients with signs of systemic toxicity and persistently elevated liver enzymes. The abscess can serve as a source of bacteremic seeding.

Physical

Physical examination findings in brucellosis, like history, often are nonspecific.

- Focal infection of bones, joints, or the genitourinary system may present with localized abnormal physical findings in the affected areas. Arthritis, joint effusions, urethritis, or, in patients with severe cases, costovertebral angle tenderness may be observed.

- Some patients may present with hepatosplenomegaly, 10-30% with hepatomegaly and 10-70% with splenomegaly. Right upper quadrant pain and jaundice may indicate hepatic abscess.
- In chronic infection (>3-6 mo), weight loss may be apparent.
- Infection of the nervous system may present with focal findings (abscesses) or nuchal rigidity (leptomeningitis). Of note, nuchal rigidity was present in fewer than half of patients with brucella leptomeningitis. Typical focal findings may steer toward an abscess. Global depression of cognition may occur.
- Dermal manifestations may include cutaneous ulcerations, petechiae, purpura, and erythema nodosum.
- Endocarditis may present with murmurs, and mycotic aneurysms of ventricles, brain, and aorta have been observed.
- Although pulmonary complaints are frequently present, physical findings of this organ system are almost always normal.
- Generally, physical examination findings are normal, and diagnosis is made from history and serology.

Causes

Brucellosis is caused by exposure to the pathogen via the routes discussed above. Occupational exposures tend to be isolated. A large-scale outbreak of the infection should raise suspicion that a biological weapon has been released, most likely via an infectious aerosol.

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Differentials

Abortion, Septic

Back Pain, Mechanical

Brain Abscess

Bronchitis

CBRNE - Biological Warfare Agents

Depression and Suicide

Endocarditis

Epididymitis

Gastroenteritis

Lumbar (Intervertebral) Disk Disorders

Meningitis

Osteomyelitis

Pneumonia, Bacterial

Pneumonia, Mycoplasma

Pneumonia, Viral
Subarachnoid Hemorrhage
Tuberculosis
Urinary Tract Infection, Female
Urinary Tract Infection, Male

Other Problems to be Considered

Typhus

Sacroiliitis

Erythema nodosum

Vasculitis

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Workup

Lab Studies

- Blood cultures
 - Blood cultures are positive in 10-90% of patients but are not particularly helpful in initial diagnosis of the disease.
 - Keep them for 2 months and reculture them onto solid media every week.
 - Because of the ease of aerosol transmission, handle any potential *Brucella* specimens under a biohazard hood.
- Cerebrospinal fluid cultures
 - Obtain cerebrospinal fluid (CSF) cultures for suggested meningitis.
 - CSF demonstrates lymphocytic pleocytosis, elevated protein, and normal-to-low glucose.
 - CSF cultures are positive for brucellosis less than 50% of the time, but antibody testing of the fluid yields a diagnosis.
- Urinalysis and/or urine cultures
 - Urinalysis and/or culture and sensitivity may be sent in the presence of symptoms of urinary tract infection. It most likely demonstrates a sterile pyuria, similar to tuberculosis.
 - Send urine cultures, since the organism grows from the urine if the genitourinary tract is infected.

Imaging Studies

- Chest x-ray: Obtain a chest x-ray if respiratory symptoms are present or if a source of infection is not apparent. Chest x-ray in brucellosis usually is normal.
- Cranial CT scan: Obtain a cranial CT scan for altered mental status or focal neurologic deficits. While often normal, it may reveal evidence of acute or chronic brucella leptomeningitis, subarachnoid hemorrhage, or cerebral abscess.
- Echocardiography
 - Echocardiography is used to evaluate for possible endocarditis. The primary site of vegetation is the aortic valve, with the sinus of Valsalva most commonly affected, followed by the mitral valve.
 - Mycotic aneurysms of the aorta or carotids may be observed on duplex arteriography.

Procedures

- Arthrocentesis: Perform arthrocentesis for suggested septic arthritis. The joint aspirate demonstrates an exudative fluid with low cell counts and mononuclear predominance. Patients with brucellosis rarely present with acute monoarticular arthritis.
- Bone marrow biopsy: Although not an ED procedure, bone marrow biopsy may be required to establish a diagnosis in certain patients.
- Liver biopsy: While not an ED procedure, percutaneous biopsy may be needed in the patient with liver granulomas to obtain a specimen for diagnosis.

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Treatment

Prehospital Care

Prehospital care for brucellosis is supportive.

- As the symptoms generally are vague and presentation rarely life threatening, emergency medical service (EMS) care should focus on stabilization, as needed, and transport.
- If a proximate bioterrorist attack is known or strongly suggested at the time of patient contact, appropriately decontaminate the patient. In the event of a covert undiscovered attack, patients may become symptomatic well after the time that decontamination is necessary.
- As in the care of any patient with a potentially transmissible disease, use appropriate precautions

(eg, gloves, mask, gown).

- If the patient presents as part of a known, immediately proximate bioterrorism incident, EMS providers should notify the hospital to undertake appropriate decontamination and isolation measures.

Emergency Department Care

Given the nonspecific patient complaints, a diagnosis of brucellosis is unlikely in the ED. With an appropriate history, an astute clinician may suspect it.

- Respiratory isolation usually is not necessary, as long as close contact with the respiratory tract is not made. Wear masks for intubation, suctioning, or other maneuvers that may expose the caregiver to a large concentration of aerosolized particles.
- The appropriate antibiotic therapy for brucellosis is combination therapy with doxycycline and rifampin or streptomycin. If brucellosis is strongly suggested, consult a specialist to determine the proper antibiotic regimen.
- Provide supportive care for any specific symptoms and obtain appropriate tests targeted to affected organ systems as determined by history and physical.

Consultations

- The primary specialist to consult is an infectious disease specialist. Determine proper serologic tests, cultures, further diagnostic evaluations, and the correct antibiotic therapy in conjunction with the infectious disease specialist.
- Depending on the degree of damage to individual organ systems, contact the appropriate specialist (eg, cardiology for endocarditis).

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Medication

The appropriate antibiotic therapy for brucellosis has been studied to some degree. Doxycycline (100 mg PO bid for 6 wk) is the most appropriate monotherapy in simple infection; however, relapse rates approach 40% for monotherapy treatment. Rifampin (600-900 mg/d) usually is added to doxycycline for a full 6-week course. In patients with spondylitis or sacroiliitis, doxycycline plus streptomycin (1 g/d IM for 3 wk) was found to be more effective than the doxycycline/rifampin combination. Streptomycin currently is favored over rifampin for combination therapy of any significant infection. In pediatric patients older than 8 years, doxycycline (5 mg/kg/d for 3 wk) plus gentamicin (5 mg/kg/d IM for the first 5 d) was the recommended therapy. For children younger than 8 years, trimethoprim/sulfamethoxazole

(TMP-SMZ) for 3 weeks and a 5-day course of gentamicin were most effective. TMP-SMZ also was effective in treating pregnant women, either as a single agent or in combination with rifampin or gentamicin.

Fluoroquinolones have a high relapse rate when used as monotherapy. No uniform recommendation exists for treatment of meningitis or endocarditis; however, TMP-SMZ plus rifampin remains the preferred combination. In endocarditis, early replacement of the infected valve is recommended, along with medical therapy. Corticosteroids are recommended in CNS infection, but data supporting their utility are lacking. Also prescribe symptomatic treatment for pain and fever.

Antibiotics

Indicated to abolish infection. Therapy must cover all likely pathogens in the context of the clinical setting.

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|-------------------|---|
| Drug Name | Doxycycline (Doryx, Vibramycin, Bio-Tab)- Several different controlled and retrospective trials have established efficacy as treatment for brucellosis. Because of concerns regarding treatment failures, combination therapy with rifampin or an aminoglycoside now is recommended, although it remains approved for use as monotherapy. |
| Adult Dose | 200 mg/d, usually divided into 100 mg PO bid; may be administered IV if needed; duration is 3-6 wk |
| Pediatric Dose | 5 mg/kg/d PO for 3 wk |
| Contraindications | Documented hypersensitivity |
| Interactions | None reported |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | May cause photosensitivity; can cause nausea and erosive esophagitis, especially if taken hs; may deposit in teeth, although less than with tetracycline; safe to use in renal failure |

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| Drug Name | Rifampin (Rifadin, Rimactane)- Used in combination therapy with doxycycline, TMP-SMZ, or gentamicin for treatment of brucellosis. |
| Adult Dose | 600-900 mg PO/IV qd |
| Pediatric Dose | 10-20 mg/kg PO/IV qd, not to exceed 600 mg |
| Contraindications | Documented hypersensitivity; preexisting liver disease |

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| Interactions | Multiple drug-drug interactions; notably, decreases serum levels of most antiretrovirals; decreases effectiveness of beta-blockers; decreases effectiveness of oral contraceptives; decreases phenytoin levels; decreases effectiveness of anticoagulants and sulfonyleureas; increases conversion of INH into its hepatotoxic metabolites; levels increase with concurrent use of antiretrovirals and TMP-SMZ; also decreases levels of methadone, precipitating withdrawal |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Monitor liver enzymes before starting therapy and repeat if symptoms of potential hepatotoxicity develop; causes brownish discoloration of body fluids; stains contact lenses; may cause drug-induced lupus; if taken irregularly or restarted after an interval of no medication, may cause "flu syndrome" with fever, chills, myalgias, and dyspnea |

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| Drug Name | Sulfamethoxazole and trimethoprim (Bactrim, Septra)- Used as adjunctive therapy with gentamicin in treating infection in children <8 y; used as monotherapy or combined with rifampin or gentamicin to treat infection in pregnant females. Inhibits bacterial growth by inhibiting synthesis of dihydrofolic acid. |
| Adult Dose | 1 double strength tab PO bid (160/800) 8-10 mg/kg IV divided q6, 8, or 12h |
| Pediatric Dose | 5 mL/10 kg PO bid; 5 mL: 40/200 |
| Contraindications | Documented hypersensitivity; relatively contraindicated in asthmatics, as sensitivity to the sulfa molecule may cause bronchospasm; relatively contraindicated in thrombocytopenic patients, as thrombocytopenia may worsen |
| Interactions | Competes with creatinine for tubular reabsorption and thus may increase serum creatinine; hyperkalemia observed in 20% of patients; may cause thrombocytopenia and aseptic meningitis; frequently causes GI disturbances; occasionally may cause severe reactions in form of Stevens-Johnson syndrome or TEN; increases levels of phenytoin, rifampin, and loperamide; increases activity of warfarin; enhances bone marrow suppression when administered with methotrexate; decreases effectiveness of oral contraceptives |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in sulfa-allergic patients or in concurrent use with rifampin |

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| Drug Name | Gentamicin (Garamycin, Gentacidin)- Aminoglycosides have been used for several years to treat brucellosis; studies to date have shown gentamicin to be the preferred aminoglycoside to treat infection as combined therapy with either TMP-SMZ or doxycycline in children. Adult dose is either once daily dosing or a multiple daily dose. |
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| Adult Dose | Once daily dose: 5.1 mg/kg IV/IM qd Multiple daily dose: 2 mg/kg loading dose, followed by 1.7 mg/kg IV/IM q8h; continue for 5 d |
| Pediatric Dose | 5 mg/kg IM for 5 d, in combination with either doxycycline or TMP-SMZ |
| Contraindications | Documented hypersensitivity; avoid if possible in patients with impaired renal function or sensorineural deafness because of known nephrotoxicity and ototoxicity; once daily dosing is associated with decreased risk of nephrotoxicity |
| Interactions | Increases nephrotoxicity of contrast agents, cyclosporin, <i>cis</i> -platinum, NSAIDs, amphotericin B, and vancomycin; increases ototoxicity of loop diuretics and noise; potentiates neuromuscular blocking agents |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in patients with renal failure or if IV contrast is planned; check levels at minimum q3d and adjust dose based on level and calculated creatinine clearance |

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| Drug Name | Streptomycin- Has been used for several years to treat brucellosis; used in combination with doxycycline, especially for spondylitis or sacroiliitis; augments bacteriocidal action of other agents used to treat brucellosis. |
| Adult Dose | 15 mg/kg, not to exceed 1 g/d IM qd for 3 wk |
| Pediatric Dose | 20-40 mg/kg IM qd, not to exceed 1 g qd |
| Contraindications | Documented hypersensitivity; if possible avoid in patients with preexisting renal disease or vestibular disease because of ototoxicity and nephrotoxicity |
| Interactions | Increases nephrotoxicity of contrast agents, cyclosporin, <i>cis</i> -platinum, NSAIDs, amphotericin B, and vancomycin; increases ototoxicity of loop diuretics and noise; potentiates neuromuscular blocking agents |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Caution in renal failure and preexisting vestibulocochlear disease; adjust dose based on creatinine clearance ratio; determine BUN and creatinine prior to starting therapy; perform weekly audiograms for treatment duration |

Corticosteroids

Indicated to reduce inflammation and improve neurologic outcome in patients with neurobrucellosis.

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| Drug Name | Dexamethasone (Decadron, AK-Dex)- Use of corticosteroids is reserved for symptomatic brucella meningitis. Although generally recommended, scientific evidence supporting their use is lacking. No consensus exists on optimal dosing, frequency, or duration of therapy. |
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| Adult Dose | 0.15 mg/kg IV q8h |
| Pediatric Dose | 0.6 mg/kg/d divided into q6h doses for 2 d prior to starting antibiotics |
| Contraindications | Documented hypersensitivity |
| Interactions | Barbiturates, carbamazepine, phenytoin, rifampin, and isoniazid may reduce effectiveness; estrogens enhance effect |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Prolonged use may cause mood changes, seizures, hyperglycemia, GI bleeding, and HPA axis suppression; long-term use is rare |

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Follow-up

Further Inpatient Care

- Starting the appropriate antibiotic therapy is the mainstay of care.
- Depending on what other systems are involved, more specialized care may be needed.
- Use appropriate precautions (eg, mask, gloves, eye protection) for respiratory procedures or handling body fluids.
- Handle specimens from the patient in the lab under biosafety level III conditions.
- Tests and other procedures are discussed in [Lab Studies](#).

Further Outpatient Care

- Outpatient care consists of completing the course of antibiotics, treating any exposed patients, and avoiding contact with the initial source of infection.
- Arrange outpatient follow-up care with the infectious disease specialist and any other necessary specialist.
- Strongly emphasize the need to complete the full 6-week course of antibiotics, as failure to do so increases the risk of relapse.

in/Out Patient Meds

- Administer antibiotic and corticosteroid therapy as outlined in the [Medication](#) section. Also administer any additional drugs needed for symptomatic treatment (eg, antipyretics, analgesics). Additional medication is based on the patient's presenting symptoms.

Transfer

- Transfer to another facility depends on the needs of the patient. As most patients do not require highly specialized interventions, the need to transfer should not be frequent.
- Personnel involved in the transfer should maintain respiratory and contact precautions, and the vehicle should be decontaminated after transport as needed.

Deterrence/Prevention

- Avoiding the source of infection prevents reinfection. Better handling of infected animals or animal products is paramount.
- As no human vaccine is available, immunization is not an option for humans, but immunization of animals reduces the pool of vectors and thereby reduces human infection.
- As the brucellosis vaccine is attenuated for animals but not humans, accidental percutaneous exposure to the vaccine may cause disease. Labs should handle all specimens under biosafety level III conditions.
- Military personnel should take appropriate precautions if use of biological weapons by an unfriendly source is anticipated.

Complications

- Complications are rare in the patient who is treated appropriately.
- The primary complication is the need for valve replacement in the patient with endocarditis.
- Residual musculoskeletal complaints may be present in the patient with long-term infection and sacroiliitis.
- Relapse of infection may occur in 10% of patients.

Prognosis

- Most patients with brucellosis recover completely without lasting sequelae, provided they receive appropriate antibiotic treatment. The relapse rate is approximately 10%, even with treatment. Prognosis generally is excellent.

Patient Education

- Center patient education on the need for strict compliance with the antibiotic regimen and the need to avoid potential sources of infection. This primarily involves avoidance of infected animals or stricter precautions (eg, gloves, mask) when dealing with a potentially infected animal.
- Advise farmers and ranchers to immunize their cattle against the disease as needed.
- Laboratory workers should maintain the appropriate level of containment.
- Travelers to regions where the disease is endemic need to take precautions against infection (eg,

avoid potentially contaminated dairy products).

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Miscellaneous

Medical/Legal Pitfalls

- As brucellosis presents in such a nonspecific manner, the diagnosis can be difficult. The primary pitfall is failure to consider possible *Brucella* infection in a patient with history that suggests a possible source of infection (eg, farmer, traveler to an endemic region, veterinarian).
- In a patient with endocarditis or meningitis and history suggestive of possible exposure, failure to treat for brucellosis is a potential downfall.
- *Brucella* species have not yet been implicated in any major bioterrorism incident; however, were they used in such a way, patients may not present until several weeks later. Detailed history and knowledge of such potential exposures in the recent past is essential.

Special Concerns

- Pregnancy: *B abortus* is associated strongly with miscarriage in cattle. Whether this increases the risk of spontaneous abortion in humans more than other severe bacterial infection is unknown. Therefore, consider any pregnant female with brucellosis to carry an increased risk of spontaneous abortion. *Brucella* species may be transmitted across the placenta.
- Pediatric: Pediatric brucellosis is more common than originally suspected; however, given the relative infrequency of infection in the US, it is still rare. The presentation is similar in neonates, children, and adults.
- Zoonosis: As brucellosis is a zoonotic infection, immunization of at-risk animals reduces the number of infected animals and therefore the reservoir of infection.

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CBRNE - Chemical Decontamination

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Synonyms, Key Words, and Related Terms

chemical warfare agents, hazardous materials, decon

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Introduction

Emergency departments (EDs) and emergency medical services (EMS) are responsible for managing potential chemical disasters, whether they result from industrial accidents or terrorist activities. In recognition of this responsibility, the Joint Commission on Accreditation of Healthcare Organizations (JCAHO) and the Occupational Safety and Health Administration (OSHA) require EDs to prepare for hazardous material incidents.

In treating patients with chemical exposures, decontamination is of primary importance provided the patient does not require immediate life-saving interventions. Any plan must include contingencies for contamination sources within the hospital and for ED evacuation. The determination of a workable hazardous materials plan requires careful thought and often professional input from medical toxicologists, hazardous materials teams, and industrial hygiene and safety officers. Using a patient decontamination plan implemented without specific adaptation to the hospital and without practice can result in undesirable outcomes.

Legal requirements apply to hospital-based decontamination. All EDs incorporated in an emergency response plan for hazardous materials incidents must meet OSHA requirements ([29 CFR 1910.120\(q\)](#)) for both staff training and response to hazardous materials, because they likely will be presented with a chemically exposed patient who has not been decontaminated at the scene. Under these regulations, emergency medical personnel who may decontaminate victims exposed to a hazardous substance should be trained at a minimum to the first-responder operational level. For response to an unknown hazard, OSHA regulations require Level B protection, which includes a positive-pressure self-contained breathing apparatus and splash-protective chemical-resistant clothing.

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Purpose of Chemical Decontamination

Chemical decontamination has 2 primary goals. Firstly, decontamination helps prevent further harm to the patient from the chemical exposure. Methods of patient decontamination include chemical dilution and chemical inactivation. Secondly, decontamination helps protect healthcare providers and maintains the viability of the ED as a treatment center. Mismanagement may result in illness in healthcare providers and contamination of the ED; severe ED contamination may necessitate departmental closure, which is potentially catastrophic in a mass casualty incident.

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Recognizing a Chemical Contamination

Before chemical decontamination can occur, chemical contamination must be recognized. The most important tool for assessing a patient for chemical exposure is a careful history. Continue to consider chemical exposures in the differential diagnosis for any mass casualty incident in which multiple ill persons with similar clinical complaints (point-source exposure) seek treatment at about the same time or in persons who are exposed to common ventilation systems or unusual patterns of death or illness.

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Personal Protective Equipment

Personal protective equipment (PPE) is the clothing and respiratory gear designed to protect the health care provider while he or she is caring for the contaminated patient (see [CBRNE - Personal Protective Equipment](#)). The minimum protective equipment required by OSHA regulations for healthcare providers caring for patients contaminated with an unknown substance include chemical-resistant suits that guard against splash exposures and positive-pressure full-faced respirators. Using this equipment requires specialized training; therefore, train appropriate personnel in the use of this equipment before they need to use it.

PPE is divided into 3 levels. Level A PPE is required in the area of chemical release if dangerous exposure levels potentially are present. A Level A suit is fully encapsulated and chemically resistant to both liquid and vapor exposures. Since this suit is fully encapsulated, it requires self-contained breathing apparatus. The level of protection typically provided to those involved in the decontamination procedure is a Level B suit. This suit requires a full-faced positive-pressure respirator, is chemically resistant, and provides protection against splash exposures. Use Level C protection when the chemical hazard is known, the concentration is at nontoxic levels, and ambient air oxygen levels are at or above 21% of atmospheric levels. Level C protection involves chemical-resistant clothing and air-purifying respirators to filter airborne contaminants.

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Patient Decontamination

ED staff has the following 3 primary goals in treating a patient who has been exposed to a hazardous material and may be contaminated or who has not undergone adequate decontamination before arrival at the hospital: (1) isolate the chemical contamination; (2) appropriately decontaminate and treat the patient(s) while protecting hospital staff, other patients, and visitors; and (3) reestablish normal service as quickly as possible.

- Healthcare providers caring for the patient should put on the appropriate PPE prior to coming into contact with contaminated patients. In most instances, this is Level B PPE.
- Ideally, decontamination occurs outside the hospital by EMS providers. If this does not occur, prepare a decontamination area for the patient. If possible, the ideal location is outdoors.

- If indoor decontamination is necessary, a decontamination room is the next ideal location. Indoor decontamination only should occur in cases in which a controlled indoor environment may be maintained safely.
 - Control volatilization of the chemical to prevent displacement of ambient room oxygen, prevent combustion, and to prevent levels of the chemical from reaching air concentrations deemed immediately dangerous to life or health for that specific hazard. In order to monitor this hazard effectively, the hospital requires testing equipment capable of identifying the chemical, its ambient air concentration, and ambient room oxygen concentrations.
 - If such a room is not available, try to isolate the patient in a single large room after removing nonessential and nondisposable equipment. Ideally, this room should be away from other patient care areas. Maintain ventilation to the area in which the patient is located, but be wary of further contaminating the hospital with recycled ventilation.
 - Establish a secure zone with yellow tape and permit only appropriately protected individuals to enter as needed. Include in the secure zone any area the patient may contaminate while entering the ED.
-
- Upon arrival of the patient, determine whether the patient requires any immediate life-saving interventions. If these are required, stabilize the patient before or during decontamination.
 - Having the patient perform as much of the decontamination as possible is preferable to decrease the amount of cross-contamination.
 - Remove the patient's clothes and jewelry and place them in plastic bags.
 - Wash the patient from head to toe with soap and water. Avoid vigorous scrubbing to prevent skin breakdown.
 - Decontaminate open wounds by irrigation with saline or water for an additional 5-10 minutes.
 - Try to avoid contaminating unexposed skin on the patient. Use surgical drapes if necessary.
 - Flush exposed areas with soap and water for 10-15 minutes with gentle sponging.
 - Irrigate exposed eyes with saline for 10-15 minutes, except in alkali exposures, which require 30-60 minutes of irrigation.
 - Clean under fingernails with a scrub brush.
 - Ideally, collect runoff water in steel drums if possible.

Special considerations for the chemical warfare patient

The best universal liquid decontamination agent for chemical warfare agents (CWAs) is 0.5% hypochlorite solution. It is prepared easily by diluting household bleach to one-tenth strength (ie, 9 parts water or saline to 1 part bleach). Hypochlorite solution works through physical removal and oxidation and/or hydrolysis of the agent; water does this at a much slower rate. Hypochlorite solutions are for use on the skin and soft-tissue injuries, including open lacerations. Do not use it in penetrating abdominal wounds (leads to development of peritoneal adhesions), in the eye (leads to corneal opacities), in open chest wounds, or in open brain or spinal cord injuries (effects unknown). Irrigate these areas with copious amounts of sterile saline solution. After using hypochlorite solution on either the skin or soft-tissue wounds, subsequently irrigate these areas with sterile saline solution.

The military also has access to a universal dry decontaminant known as M291 resin, which is available as pads packaged in small individual packets. M291 resin is a dry black carbonaceous material that decontaminates by absorption and physical removal of the CWA from the victim. M291 resin is used for spot decontamination of skin exposed to CWAs.

Organization of the military treatment area in chemical warfare

A full discussion of the military medical team response to a chemical warfare attack is beyond the scope of this article. Please refer to Medical NBC Website for access to the *Textbook of Military Medicine* for a complete discussion of this area (<http://www.nbc-med.org>). A basic understanding of the structure of the military's medical treatment facility is important for civilian health care providers, since they most likely will be working with the military in the event of a chemical warfare incident. The military medical treatment facility is divided into dirty and clean sides. The demarcation of the sides is known as the hotline. The concept of the hotline is to keep all contaminated equipment, personnel, and casualties out of the clean side until decontamination is completed.

The dirty side consists of a triage station, emergency treatment station, and a decontamination area. The triage station is the single entry point into the medical treatment facility. If the patient has an emergent medical condition that requires immediate medical intervention before decontamination, the patient is sent from triage to the emergency treatment station. The emergency treatment station is equipped to handle contaminated patients with emergent medical issues and stabilize them for either decontamination at the medical treatment facility or dirty evacuation to another facility for a higher level of care. The decontamination area is divided into ambulatory and nonambulatory patient decontamination areas.

The clean side consists of part of the decontamination area and the clean treatment area. The hotline extends through the decontamination area. Patients are decontaminated on the dirty side and are brought to the hotline nude except for their PPE mask. These patients are transferred across the line to a team on the clean side of decontamination area. The clean side decontamination team then brings patients into the clean treatment area.

The clean treatment area is located 30-60 meters upwind of the dirty side. The clean side decontamination team removes the patient's mask prior to transferring the patient to the clean

treatment area. In the clean treatment area, the patient can be treated definitively or transferred to another facility if needed.

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Supportive Care

Saving lives always depends on ensuring the ABCs: adequate airway, ventilation, and circulation. Greater contamination or exposure more likely results in victims who require early intubation and ventilation. Conversely, adequate ventilation may be impossible because of the intense muscarinic effects of certain nerve gas exposures (copious airway secretions, bronchoconstriction). In this situation, administer atropine before initiating other measures. In some patients, large quantities of atropine may be required, rapidly depleting hospital supplies. Administering succinylcholine to assist intubation is relatively contraindicated, since nerve agents prolong the drug's paralytic effects.

Benzodiazepines are the mainstays in seizure treatment. Liberal doses are required; titrate to effect. Termination of seizure activity may reflect onset of flaccid paralysis from the nerve agent rather than adequacy of antiseizure therapy. A bedside electroencephalograph (EEG) may be required to assess ongoing seizure activity.

Animal data suggest that routine administration of diazepam reduces the incidence of seizures and decreases severity of pathologic brain injury following nerve agent exposure.

CWAs are a diverse group of extremely hazardous materials. Emergency physicians must be familiar with the pathophysiology and various clinical presentations produced by CWAs and the principles and practices of appropriate medical management. Since deployment of CWAs also places emergency care providers at serious risk of exposure, emergency physicians must be familiar with the different levels of PPE, appropriate use, and decontamination procedures.

CWAs, as potential weapons of mass destruction with the capability of causing a catastrophic medical disaster, easily may overwhelm any healthcare system. Since civilian victims exposed to CWAs are likely to flee to the nearest hospital, emergency physicians provide the first line of treatment and must prepare their EDs for the treatment of persons exposed to CWAs.

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Identifying the Chemical and Obtaining Expert Advice

Ideally, a hazardous materials team at the scene will be able to provide assistance regarding the specifics of the exposure and the potential treatment. A local poison control center also may be able to provide assistance. The Chemical Manufacturers Association provides 24-hour assistance in the specifics of treating a particular chemical exposure; it can be reached at (800) 424-9300. The Domestic Preparedness Chem/Bio Helpline can be reached at (410) 436-4484. Online information is available at [Centers for Disease Control and Prevention](#).

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Deciding to Evacuate the Emergency Department

Evacuation of the ED rarely is indicated. In most situations, isolation of the contamination is all that is required.

Consider evacuation of the ED in the following situations:

- Toxic material spills in the ED.
- Nearby hazardous materials are threatening the hospital.
- The patient is contaminated with a volatile toxic or flammable chemical and is decontaminated insufficiently prior to entering the ED.

If symptoms start to occur outside of the isolation area or the situation requires urgent decision making without time to identify the contaminant, consider evacuation. Odor does not predict toxicity reliably.

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CBRNE - Chemical Detection Equipment

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Introduction

Chemical detection equipment (CDE) is an essential component of hazardous material (HAZMAT) emergency response. This equipment should detect the harmful agent, correctly identify the agent, and define the area of exposure. Rapid detection is essential so that responders and military targets can recognize a threat and don protective gear (ideally in <9 s). It also is important to know the extent of contamination. During several documented chemical attacks, first responder casualties have been vast enough to delay the rescue. During the Tokyo subway sarin attack in 1995, 9% of emergency medical services (EMS) providers suffered the affects of acute exposure. Effective CDE may help prevent these occurrences.

Several different technologies are used today to detect chemical agents (CAs). CAs are defined as chemicals intended to kill or seriously injure human beings. CDE usually detects the most common CAs: nerve agents, blister agents, and arsenical vesicants. A large variety of equipment is available that is capable of identifying liquid droplets of CAs on surfaces and in vapors. Laboratory-based equipment can detect agents in water. The main challenges with these technologies are ensuring an appropriate sample for analysis and filtering out nonhazardous environmental chemicals that may be present. This article focuses on the technologies and devices that may be used by first responder teams in the field.

Laboratory detection techniques are beyond the scope of this discussion.

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Enzymatic Detection Techniques

Chemical detection paper is a very sensitive technique for detecting CAs. It is one of the least sophisticated and thus least expensive methods of detection. It is used to detect liquids and aerosols and is a common means for defining a contaminated area.

Chemical detection paper is composed of 2 dyes soluble in CAs and a pH indicator integrated into cellulose fibers. When exposed to CAs, it can change color according to the type of agent. If an aerosolized droplet encounters the paper, the diameter and density of the spot can be used to determine the droplet size of the agent and the degree of contamination.

Chemical detection paper lacks specificity and is prone to error because it reacts with contaminants such as brake fluid, antifreeze, and insect repellent, resulting in false-positive readings. False readings are especially undesirable in civilian situations because they may lead to mass panic. Therefore, always use chemical detection paper with another modality for accuracy of detection.

M8/M9 chemical detection paper

M8 and M9 CA detection papers, commonly used by the military, are available commercially to HAZMAT response teams. M8 paper is packaged in 25 perforated sheets, 2.5 in X 4 in, and is blotted on liquids that arouse suspicion. It identifies CAs by changing colors within 30 seconds of exposure: dark green for vesicants, yellow for nerve agents, and red for blister agents.

M9 paper has adhesive backing that allows it to be attached to clothing and equipment. M9 paper detects the same agents as M8 paper but does not change color to enable identification. M9 paper tends to react faster than M8 paper and can be attached to vehicles that are entering areas filled with vapor to determine contamination. Vehicles thus equipped are limited to a speed of 30 km/h.

M256A1 chemical agent detection kit

The M256 CA detector kit originally was released in 1978 and was modified in 1987 to the M256A1, which is sensitive to lower concentrations of nerve agents. It was used extensively during the Gulf War but also is available commercially. It is another common component of CDE provided to civilian response teams. This portable kit detects nerve gas, mustard gas, and cyanide and usually is used to define areas of contamination. The M256A1 contains a package of M8 paper, detailed instructions, and a vapor sampler (12 enzymatic tickets that contain laboratory filter paper for detecting CA vapors). The

vapor sampler employs wet chemistry technology, in which ampoules containing different substrates are crushed so that the liquids interact with strips of filter paper, chromatographic media, and glass fiber filter. These substrates then are exposed to the vapor under suspicion. The reaction causes a color change, alerting the user to the presence of a CA. The reactions typically take 15 minutes to occur.

The M256A1 can detect nerve gas concentrations of 0.005 mg/m³, hydrogen cyanide concentrations of 11mg/m³, and mustard gas concentrations of 0.02 mg/m³. This is one of the military's most sensitive devices for detecting CAs and detects all agents at levels below those that can kill or injure people. It is prone to false-positive results, similar to other enzymatic detection techniques, but has not been demonstrated to produce false-negative results in real situations.

Colorimetric tubes

Colorimetric tubes such as those available from Draeger and RAE systems use enzymatic techniques to identify CAs. A hand pump is used to draw a sample into a specific tube, and the concentration of the substance is read from the tube. This is another simple and inexpensive way of detecting and identifying a CA. It is used extensively in civilian response units for this reason, but it has some disadvantages. Available are 160 substance-specific reagent tubes identifying different agents. For each agent, a different tube must be used. Efficient use of this system demands knowledge of which CA is likely to be present in a given environment. If a tube for vesicants is used to sample the air and the CA is a nerve agent, the tube reports a false-negative result. A tube for each possible CA must be used for thorough detection.

Draegar has made this process simpler by offering a chip measurement system analyzer (CMS). The analyzer integrates an optical system for analyzing the color reaction, a flow controller, a pump system, and 10 capillaries, each capable of detecting an agent. As long as the proper chip is inserted, 10 agents can be detected and measured accurately within 20 seconds using this device. Draegar offers this device as part of an emergency response kit available to the public.

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Ion Mobility Spectroscopy

Ion mobility spectroscopy (IMS) is used in many handheld and stand-alone detection devices that can be used to scan equipment, surfaces, and people for contamination. This technology involves drawing a gaseous sample into a reaction chamber using an air pump. The air molecules then are ionized, most commonly using radioactive beta emitters such as nickel-63 or americium-241. The ionized particles then are passed through a weak electrical field toward an ion detector. Contaminants are identified according to the time it takes to traverse the distance to the detector. This time is proportional to the mass of the molecule. The pattern is compared to a sample of clean air; if the pattern is markedly different and

unique to certain types of agents, the alarm sounds. These systems are capable of detecting and distinguishing between nerve gas, mustard gas, and vesicants. Its sensitivity ranges from 0.03 mg/m³ for nerve gases such as sarin to 0.1 mg/m³ for mustard gas.

IMS has certain advantages. It is less sensitive to contaminants, because it relies on a clean air sample for calibration. Thus, if an area has a certain baseline nonhazardous environmental vapor present, it is not detected.

Improved Chemical Agent Monitor and other handheld devices

IMS is the cornerstone of many devices used today. The Finnish M86 and M90 are handheld devices that use IMS, as is the Improved Chemical Agent Monitor (ICAM). The ICAM was used extensively in the Gulf War, even attached to certain vehicles. It is a handheld device that continuously displays the concentration of nerve agents or mustard agents. The ICAM is prone to erroneous detection in enclosed spaces and areas of strong vapor concentration (eg, heavy smoke). It also can become saturated, requiring recalibration. Versions of the ICAM are available for commercial purchase and are used by many medical response teams (see [Picture 1](#), [Picture 2](#)).

The APD 2000, manufactured by Environmental Technologies Group (ETG), is another common device that uses IMS and is sold commercially to HAZMAT response teams for domestic preparedness. This handheld device can be powered by batteries and can detect mace and pepper spray as well as nerve agents, blister agents, and hazardous compounds (see [Picture 3](#), [Picture 4](#)).

Stand-alone detectors - M8A1, Automatic Chemical Agent Alarm, and Fixed Site/Remote Chemical Agent Detector

Many stand-alone detectors also use IMS technology. The military employs the M8A1 detector that consists of a stand-alone detector, which continuously monitors the environment for hazardous vapors and aerosols, and up to 5 alarms that can be dispersed throughout an area. The M8A1 detects nerve agents and blister agents when the concentration is 0.1 mg/m³ or greater and alarms within 1-2 minutes. M8A1 is an ideal device for protection from off-target attacks, in which a vapor is released upwind from the targets. However, it is less effective for on-target attacks, in which the CA is released in large amounts within seconds. In this situation, the alarm sounds after the personnel have been exposed. This system was used during the Gulf War and has been upgraded to the Automatic Chemical Agent Alarm (ACAA) system. The ACAA is slightly larger and has a communications interface that is useful in combat.

ETG provides a commercial version of an IMS stand-alone detector called the Fixed Site/Remote Chemical Agent Detector. This system detects and identifies nerve and blister agents and offers superior reliability from interferences. The alarm information can be transmitted via radio, satellite, or hardwiring. This system can be useful if placed in hospital wards or at victim collection sites to detect contamination (see [Picture 5](#)).

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Infrared Radiation Detection Techniques

Infrared radiation (IR) is employed in several CA detectors, including long-range detectors and point detectors. IR can be used to excite molecules, and each agent has a unique infrared pattern referred to as a fingerprint. Several different detection techniques use IR, including photoacoustic infrared spectroscopy, filter-based infrared spectroscopy, forward-looking infrared spectroscopy (FLIR), and Fourier transform spectroscopy.

Photoacoustic infrared spectroscopy

This highly selective technique is used to identify CA vapors. It usually is used in point detector devices. Modulated IR is passed through the sample. Since the CA absorbs the radiation, the temperature increases, and per Boyle law, the gas expands. The pattern of expansion or contraction depends on the modulation of the IR, which in photoacoustic spectroscopy is an audible signal. A microphone detects the modulation and alarms when it is similar to a recognizable agent.

This technique's selectivity is based on the number of wavelengths transmitted through the sample. As more wavelengths are passed, the chance of contaminants causing false alarms decreases. These devices are sensitive to environmental variables such as external vibration. Like IMS, if these devices are calibrated in the operating environment, detection should be accurate.

Filter-based infrared spectroscopy

This technology also is based on comparing the amount of energy absorbed by the sample, using several different wavelengths of infrared light. A series of filters is used to direct the beam through a predetermined path. Concentrations of each vapor component are used to compile trends and identify the vapor.

Differential absorption light detection and ranging

This infrared technology is used mainly to track CA clouds that already have been identified. Two pulses of laser are transmitted into the distance, and the reflected IR is detected. One pulse is a frequency that is known to be absorbed by the CA; the other is not known to be absorbed. The difference in the intensity of the return signal is used to determine the concentration of the cloud, while the time of return is used to determine the distance from the observers. This technique also is subject to environmental noise but has been used effectively to track CAs.

Passive infrared detection

FLIR and Fourier transform infrared (FTIR) are techniques by which IR emitted from CA vapor is detected simply. These technologies commonly are used in stand-alone detection devices that simply alarm when a CA cloud is detected. Both of these methodologies depend on the collection of infrared information; however, the processing is different.

M21 Remote Sensing Chemical Agent Alarm

The military uses the M21 Remote Sensing Chemical Agent Alarm (RSCAAL) based on passive infrared detection. It is the first fielded standoff chemical detection device. This system can detect a vapor cloud from 5 km with an 87% detection rate. The M21 RSCAAL continuously monitors a background and notes the change in spectral information if a vapor cloud obstructs the background. It automatically scans along a 60° angle, allowing the operator to monitor horizontal movement. The M21 can be set up in 10 minutes and is unaffected by low light conditions. However, the M21 is limited in that it must be stationary and can be obstructed by snow and rain.

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Other Detection Technologies

Photo ionization detection

Gas vapors can be ionized using ultraviolet light. Photo ionization detection depends on exposing the suspect vapor to ultraviolet energy powerful enough to ionize agent molecules. Specific ranges of ultraviolet light ionize molecules in certain CAs. An ion detector then registers the amount of ionized molecules. Thus, these detectors can determine the concentration and identity of the agent. Handheld detectors produced by RAE systems and Photovac are examples of detectors that use this technology.

Flame photometry

Another technology employed is flame photometry. In this technique, a flame of hydrogen is used to burn a sample of air. The color of the flame is analyzed by a photometer for concentrations of sulfur and phosphorous (key components in nerve gas and mustard). Flame photometry is highly sensitive yet is prone to false-positive results by detecting other gases that contain significant concentrations of sulfur or phosphorus but are nonhazardous. Certain analysis algorithms can be employed to make these detectors less prone to error. If gas chromatography is integrated with flame photometry, the detectors are more accurate. Gas chromatography is a technique used in labs to separate mixtures of compounds. It involves using a carrier gas to separate a volatilized liquid or vapor based on its passage through a column. As each solute exits the column according to its properties and the temperature of the column, a detector

records an electrical signal plotted over time.

Miniature automatic continuous agent monitoring system

The miniature automatic continuous agent monitoring system (MINICAMS) is a system based on combining gas chromatography with flame photometry. A sample vapor is drawn into the machine and exposed to a heated preconcentrator loop. As each component exudes from the column, it is exposed to flame photometry. This system enables more specific detection. A typical cycle lasts 3-5 minutes, enabling continuous monitoring of the environment.

Surface acoustic wave detection

Surface acoustic wave (SAW) chemical detectors rely on chemically selective coated piezoelectric crystals that absorb target gases. The absorption causes a change in the resonant frequency of the crystal that is measured by a microcomputer. These detectors are able to identify and measure many CAs simultaneously. These devices are produced inexpensively, making them a popular choice among civilian response units. The SAW MINICAD mk II is a portable SAW array detector that is lightweight, battery operated, and available commercially. It is used remotely to define areas of decontamination but also can be used for active detection.

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Factors in Selecting Chemical Detection Equipment

A market survey of commercially available detection equipment in May 2000 identified 148 detection devices available to the military and first responders. With many different detectors and technologies available, the National Institute of Justice recommends examining 16 factors when choosing a detection device. The table below lists these factors. The most sensitive detectors tend to be most susceptible to false-positive alarms. Thus, for most practical applications, multiple detectors are needed to verify the findings of the initial detector.

Factors to be Examined When Choosing a Detection Device

| | |
|----------------------------|--|
| Sensitivity | Lowest concentration of CA that results in positive response; ideally, lower than levels necessary for injury to personnel |
| Resistance to interference | Factors such as smoke, moisture, or other chemicals that prevent the device from accurately providing a response |
| Response time | Time to collect, analyze, and provide feedback |

| | |
|-------------------------|---|
| Start-up time | Time to assemble and deploy the device |
| Detection status | Vapor, liquid, and/or aerosols |
| Alarm capability | Audible, visual, or both |
| Portability | Ease of transport, which encompasses weight and dimensions |
| Power capabilities | Battery versus alternating current |
| Battery needs | Quantity and type of batteries |
| Operational environment | Extremes of conditions under which the device operates |
| Durability | Amount of abuse the device withstands |
| Procurement costs | Cost per device needed |
| Operator skill level | Skill involved in using the device |
| Training requirements | Number of hours and type of educational background required for operation |

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Chemical Detection Equipment in Civilian Response to Terrorism

CDE technology has advanced primarily as a result of military necessity. More recently, the need for civilian preparedness for terrorist attacks with CA has been recognized. Civilian response is different from military response in many ways, and the choice of CDE must take this into account.

Key differences include the following:

- Civilian responders tend to be less experienced in chemical attacks.
- Civilian responders have less information concerning the origin and type of attack and may not recognize that it is a CA attack initially.
- Civilian responders have more stringent budget restraints and thus must use cost-effective equipment.
- Civilian responders have less latitude in incorrectly identifying a CA.
- Civilian responders are deployed primarily to provide medical care, leaving detection as a secondary goal.

In the civilian setting, EMS or other medical providers are the first to arrive (see [EMS and Terrorism](#)). Most EMS providers do not carry CDE to detect CAs and thus initially must recognize the potential threat in order to notify specialized HAZMAT response teams. These teams exist in many cities and are at a minimum equipped with pH paper and combustible gas indicators. This equipment is inadequate in identifying most CAs. Other teams now are equipped with colorimetric tubes (see [Enzymatic Detection Techniques](#)). Colorimetric tubes are much less expensive than more technical devices, such as the ICAM, and can be distributed generally.

Major cities in the US have a Metropolitan Medical Strike Response System (MMRS) organized by the Public Health Service. These are highly specialized, fully equipped, deployable teams to combat civilian threats from weapons of mass destruction. They are primarily medical providers who provide EMS services, decontamination, detection, and treatment. The first such team was organized in 1995 in Washington, DC, and a second was organized for the 1996 Olympics in Atlanta. They are now present in 27 major cities.

MMRS teams are often better equipped to respond to CA attacks than HAZMAT response teams. Even so, wide variability exists in the type of detection devices used. A recent study by the National Guard recognized that no standards regulate the detection devices among different civilian emergency response units. MMRS teams can employ any of the devices and technologies described above. They commonly use inexpensive CDE such as SAW detectors and enzymatic techniques such as M9 paper and the M256 kit. Some teams also use IMS devices such as the APD2000 and a modified ICAM for domestic preparedness.

Pictures



Picture 1: Improved Chemical Agent Monitors (ICAMs) available for domestic use (Image courtesy of Environmental Technologies Group, Inc)

Picture type: Photo



Picture 2: A soldier using an Improved Chemical Agent Monitor (ICAM) (Image courtesy of Environmental Technologies Group, Inc)

Picture type: Photo



Picture 3: APD2000, used by hazardous materials (HAZMAT) and Metropolitan Medical Strike Response System (MMRS) teams for domestic preparedness (Image courtesy of Environmental Technologies Group, Inc)

Picture type: Photo



Picture 4: APD2000 is being used to scan a first responder (Image courtesy of Environmental Technologies Group, Inc).

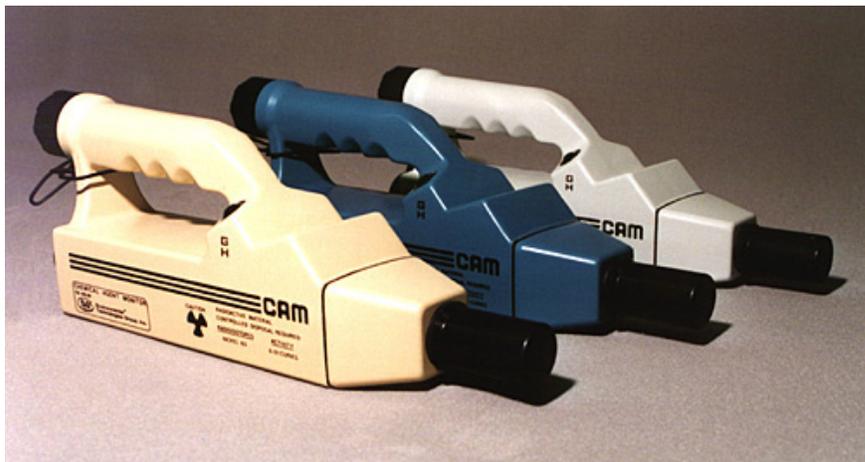
Picture type: Photo



Picture 5: Fixed Site/Remote Chemical Agent Detector using ion mobility spectroscopy (IMS; image courtesy of Environmental Technologies Group, Inc)

Picture type: Photo

Pictures



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Introduction

Emergency physicians must be able to care for victims of chemical weapon agents (CWAs). This chapter reviews the physical properties and general clinical effects of CWAs. It also describes the medical management of victims of CWAs, including the use of personal protective equipment (PPE), victim decontamination, provision of supportive care, and provision of specific antidotal therapy. To illustrate these principles, the properties, clinical effects, and medical management of nerve agents and mustards are reviewed briefly.

Risk of exposure to chemical warfare agents

Injury from CWAs may result from industrial accident, military stockpiling, war, or terrorist attack.

Industrial accidents are a significant potential source of exposure to the agents used in chemical weapons. Chemicals such as phosgene, cyanide, anhydrous ammonia, and chlorine are used widely and frequently are transported by industry. The accidental release of a methylisocyanate cloud (composed of phosgene and isocyanate) was implicated in the Bhopal disaster in 1984.

CWAs first were used in 1915, when the German military released 168 tons of chlorine gas at Ypres, Belgium, killing an estimated 5000 Allied troops. Two years later, the same battlefields saw the first

deployment of sulfur mustard. Sulfur mustard was the major cause of chemical casualties in World War I. CWAs have been used in at least 12 conflicts since, including the first Persian Gulf War (Iraq-Iran War). The Iraqi military also used chemical weapons against the Iraqi Kurds during the second Persian Gulf War.

Civilians also have been exposed inadvertently to chemical weapons many years after weapon deployment during war. Approximately 50,000 tons of mustard shells were disposed of in the Baltic Sea following World War I. Since then, numerous fishermen have been burned accidentally while hauling leaking shells aboard boats. Leaking mustard shells also have injured collectors of military memorabilia and children playing in old battlefields.

Although a number of international treaties have banned the development, production, and stockpiling of CWAs, these agents reportedly still are being produced or stockpiled in several countries.

Within the last decade, terrorists deployed chemical weapons against civilian populations for the first time in history. The release of sarin in Matsumoto, Japan, in June 1994 by the extremist Aum Shinrikyo cult left 7 dead and 280 injured. The following year, in March 1995, the Aum Shinrikyo cult released sarin vapor in the Tokyo subway system during morning rush hour, leaving 12 dead and sending more than 5000 casualties to local hospitals.

Several characteristics of CWAs lend themselves to terrorist use. Chemical substrates used in CWAs are widely available, and recipes for CWA production may be found on the Internet. CWAs are transported easily and may be delivered by a variety of routes. Chemical agents often are difficult to protect against and quickly incapacitate the intended targets. Most civilian medical communities are inadequately prepared to deal with a chemical terrorist attack.

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General Considerations

Types of chemical weapon agents

CWAs comprise a diverse group of hazardous substances. Major categories of CWAs include the following:

- Nerve agents (eg, sarin, soman, cyclosarin, tabun, VX)
- Vesicating or blistering agents (eg, mustards, lewisite)
- Choking agents or lung toxicants (eg, chlorine, phosgene, diphosgene)

- Cyanides
- Incapacitating agents (eg, anticholinergic compounds)
- Lacrimating or riot control agents (eg, pepper gas, cyanide, CS)
- Vomiting agents (eg, adamsite)

Physical properties

CWAs generally are stored and transported as liquids and deployed as either liquid aerosols or vapors. Victims usually are exposed to agents via one or more of 3 routes: skin (liquid and high vapor concentrations), eyes (liquid or vapor), and respiratory tract (vapor inhalation).

CWAs are characterized by 2 inversely related physical properties: volatility (ie, tendency of liquids to vaporize, which directly increases with temperature) and persistence (ie, tendency of liquids to remain in a liquid state).

In general, volatile liquids pose the dual risk of dermal and inhalation exposure, while persistent liquids are more likely to be absorbed across the skin. The effects of vapors largely are influenced by ambient wind conditions; even a slight breeze can blow nerve agent vapor away from its intended target. Effects of vapor are enhanced markedly when deployed within an enclosed space.

Clinical effects

Depending on the agent and the type and amount (concentration) of exposure, CWA effects may be immediate or delayed. Large inhalation exposures to nerve agents or mustards are likely to be lethal immediately. Small dermal exposures to nerve agents and mustards are particularly insidious and generally require expectant observation for variable periods because of possible delayed effects. Specific clinical effects of CWAs are as varied as the agents.

Medical management

To appropriately manage CWA exposures, emergency care personnel are required to protect themselves by performing the following:

- Using PPE
- Decontaminating patients immediately
- Providing supportive care

- Providing specific antidotes when indicated

Personal protective equipment

The primary responsibility of those who treat victims of CWAs is to protect themselves by wearing adequate PPE. First responders are at serious risk from the chemically contaminated environment (hot zone), either from direct contact with persistent liquid or from inhaling vapor. First responders and emergency care providers also are at risk from handling skin and clothing of victims contaminated with liquid CWAs (secondary skin and inhalation exposure). Conversely, providing care to those exposed only to vapor CWAs poses little risk to emergency care providers outside the hot zone. Unless a clear history of only vapor exposure is obtained, emergency medical personnel should assume that liquid contamination is present and wear PPE.

Standard protective garments are inadequate for most CWAs. Double layers of latex gloves are useless against liquid nerve and blister agents, and surgical masks and air-purifying respirators are inadequate against nerve agent vapors.

Levels of personal protective equipment

US regulatory agencies mandate the use of appropriate levels of PPE.

- Level A PPE is required for first responders and others working inside the hot zone, where vapor concentrations may be immediately dangerous to life and health. These suits are fully encapsulated, resistant to liquid and vapor chemical penetration, and include a self-contained breathing apparatus. Level A suits are also cumbersome, hot, and very difficult to wear for more than 30 minutes.
- Level B PPE is required for hospital personnel involved in decontamination of unknown hazardous materials. These suits provide adequate protection against liquid and vapor chemicals when accompanied by a self-contained breathing apparatus or supplied air respirator.
- Level C PPE is used when chemical agents have been identified and are amenable to removal by an air-purifying respirator. This suit also provides some protection against penetration by chemical liquids and vapors.

Decontamination

- Decontamination is the physical process of removing residual chemicals from persons, equipment, and the environment. Residual hazardous chemicals on those who have been exposed directly are a source of ongoing exposure to those persons and pose a risk of secondary exposure to first responders and emergency care personnel. Immediate decontamination is a major treatment priority for those with CWA exposure.

- Initial decontamination involves removal from the contaminated environment, removal of all contaminated clothes and jewelry, and copious irrigation with water.
- Rinse exposed persons with a 0.5% hypochlorite solution, which chemically neutralizes most CWAs (eg, nerve agents, mustards). A 0.5% hypochlorite solution conveniently is prepared by mixing 1 part 5% hypochlorite (household bleach) with 9 parts water.
- Avoid hot water and vigorous scrubbing, as they may increase chemical absorption.
- Vapor exposure alone does not require decontamination. Fully decontaminate patients with unclear exposure histories.
- Ideally, decontaminate as close as possible to the site of exposure to minimize duration of exposure and prevent further spread. Hospitals receiving contaminated persons should establish an area outside the emergency department in which to perform decontamination before people and equipment are allowed in. Portable decontamination equipment with showers and run-off water collection systems are commercially available. All hospitals should have the capacity to safely decontaminate at least one person.

Supportive and specific therapy

Supportive therapy for victims of CWAs generally follows the universally accepted algorithm of first ensuring the adequacy of airway, breathing, and circulation, with one important exception. Severe nerve agent poisoning may require immediate administration of parenteral atropine. Many CWAs only can be treated supportively. Specific, well-established antidotes are available only for nerve agent and cyanide exposures. Since no laboratory tests are available to rapidly confirm exposure to CWAs, treatment is based on clinical criteria.

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Nerve Agents - Properties and Clinical Effects

Mechanism of Action

The 5 nerve agents, tabun (GA), sarin (GB), soman (GD), cyclosarin (GF), and VX, have chemical structures similar to the common organophosphate pesticide malathion. Like organophosphate insecticides, these agents phosphorylate and inactivate acetylcholinesterase (AChE). Acetylcholine accumulates at nerve terminals, initially stimulating and then paralyzing cholinergic neurotransmission throughout the body.

Inhibition of AChE may not account for all of the toxic effects of nerve agents. These agents also are known to bind directly to nicotinic receptors and cardiac muscarinic receptors. They also antagonize gamma-aminobutyric acid (GABA) neurotransmission and stimulate glutamate *N*-methyl-d-aspartate (NMDA) receptors. These latter actions may partly mediate nerve agent-induced seizures and CNS neuropathology.

Physical Properties

Under temperate conditions, all nerve agents are volatile liquids. The most volatile agent, sarin, evaporates at approximately the same rate as water. The least volatile agent, VX, has the consistency of motor oil. This persistence and higher lipophilicity make VX 100-150 times more toxic than sarin when victims sustain dermal exposure. A 10-mg dose applied to the skin is lethal to 50% of unprotected individuals.

All nerve agents rapidly penetrate skin and clothing. Nerve agent vapors are heavier than air and tend to sink into low places (eg, trenches, basements).

Clinical Effects

Nerve agents produce muscarinic, nicotinic, and direct CNS toxicity with a wide variety of effects on the respiratory tract, cardiovascular system, CNS, gastrointestinal (GI) tract, muscles, and eyes. Onset and severity of clinical effects vary widely, since numerous variables determine predominant effects. Agent identity, dose (determined by concentration and duration of exposure), and type of exposure primarily determine nerve agent toxicity. Toxic effects result from dermal exposure to liquid and ocular and inhalation exposure to vapor.

Liquid exposure

Liquid agents easily penetrate skin and clothing. Onset of symptoms occurs from 30 minutes to 18 hours following dermal exposure.

Minimal liquid exposure (eg, a small droplet on the skin) may cause local sweating and muscle fasciculation, followed by nausea, vomiting, diarrhea, and generalized weakness. Even with decontamination, signs and symptoms may persist for hours.

In contrast, persons with severe liquid exposures may be briefly asymptomatic (1-30 min) but rapidly may suffer abrupt loss of consciousness, convulsions, generalized muscular fasciculation, flaccid paralysis, copious secretions (nose, mouth, lungs), bronchoconstriction, apnea, and death.

Vapor exposure

Vapor inhalation produces clinical toxicity within seconds to several minutes. Effects may be local or systemic. Exposure to even a small amount of vapor usually results in at least one of the following categories of complaints: (1) ocular (miosis, blurred vision, eye pain, conjunctival injection), (2) nasal (rhinorrhea), or (3) pulmonary (bronchoconstriction, bronchorrhea, dyspnea).

Exposure to a vapor concentration of 3.0 mg/m^3 for 1 minute causes miosis and rhinorrhea. Inhalation of a high concentration of vapor results in loss of consciousness after only one breath, convulsions, respiratory arrest, and death. For example, breathing 10 mg/m^3 of sarin vapor for only 10 minutes (100 mg/m^3 for 1 min) causes death in approximately one half of exposed individuals. Severe vapor exposures also are characterized by generalized fasciculations, hypersecretions (mouth, lungs), and intense bronchoconstriction with respiratory compromise.

Respiratory tract

Nerve agents act on the upper respiratory tract to produce profuse watery nasal discharge, hypersalivation, and weakness of the tongue and pharynx muscles. Laryngeal muscles are paralyzed, resulting in stridor. In the lower respiratory tract, nerve agents produce copious bronchial secretions and intense bronchoconstriction. If untreated, the combination of hypersecretion, bronchoconstriction, respiratory muscle paralysis, and CNS depression rapidly progresses to respiratory failure and death. Nerve agents depress the central respiratory drive directly. Thus, early death following large vapor exposure likely results from primary respiratory arrest, not from neuromuscular blockade, bronchorrhea, or bronchoconstriction.

Cardiovascular system

The cardiovascular effects of nerve agents vary and depend on the balance between their nicotinic receptor-potentiating effects at autonomic ganglia and their muscarinic receptor-potentiating effects at parasympathetic postganglionic fibers that innervate the heart.

Sinus tachydysrhythmias with or without hypertension (sympathetic tone predomination) or bradydysrhythmias with or without variable atrioventricular blockade and hypotension (parasympathetic tone predomination) may occur.

Superimposed hypoxia may produce tachycardia or precipitate ventricular tachydysrhythmias.

Nerve agent-induced prolonged QT and torsades de pointes have been described in animals.

In victims of the Tokyo sarin gas attack, sinus tachycardia and hypertension were common cardiovascular abnormalities, while sinus bradycardia was uncommon.

Central nervous system

Nerve agents produce a variety of neurologic signs and symptoms by acting on cholinergic receptors throughout the CNS. The most important clinical signs of neurotoxicity are a rapidly decreasing level of consciousness (sometimes within seconds of exposure) and generalized seizures. Symptoms such as headache, vertigo, paresthesias, anxiety, insomnia, depression, and emotional lability also have been reported.

Musculoskeletal system

Nerve agents initially stimulate and then paralyze neurotransmission at the neuromuscular junction. With minimal exposure, exposed persons may complain of vague weakness or difficulty walking. More significant exposures resemble the clinical effects that result from succinylcholine, with initial fasciculations followed by flaccid paralysis and apnea.

Ocular

Nerve agent liquid or vapor readily penetrates the conjunctiva and exerts direct muscarinic parasympathetic effects. This results in constriction of the iris (miosis, blurred and dim vision, headache), constriction of the ciliary muscle (pain, nausea, vomiting), and stimulation of the lacrimal glands (tearing, redness). Although miosis is the most consistent clinical finding after vapor exposure to nerve agents (occurred in 99% of persons exposed in Tokyo sarin attack), it may be absent or delayed in dermal exposure. Duration of miosis varies according to the extent of ocular exposure (up to 45 d).

Laboratory Tests

Routine toxicology testing does not identify nerve agents in serum or urine. Measurements of red blood cell (RBC) or plasma cholinesterase activity have been used as an index of the severity of nerve agent toxicity, but this approach is not always reliable. The reference range of RBC cholinesterase activity may vary widely, and mild exposures may be difficult to interpret without baseline measurement. In addition, RBC cholinesterase activity may not correlate with the severity of signs and symptoms following vapor exposure.

In the Tokyo subway sarin attack, 27% of patients with clinical manifestations of moderate poisoning had plasma cholinesterase activity in the normal range. Moreover, different organophosphates variably inhibit RBC and plasma cholinesterase. For example, in mild-to-moderate exposures to sarin or VX, RBC cholinesterase activity is decreased to a much greater extent than plasma cholinesterase activity.

Since plasma cholinesterase is produced by the liver, its activity also may be depressed in certain conditions (eg, liver disease, pregnancy, infections) or with certain drugs (eg, oral contraceptives). Conversely, a 20-25% reduction in RBC cholinesterase activity tends to correlate with severe clinical toxicity and, despite the exception noted above, activity of both enzymes approaches zero in most severely poisoned victims. Nevertheless, treatment decisions should be clinically based. Never withhold treatment from a symptomatic patient while awaiting laboratory confirmation. Conversely, decreased

cholinesterase activity in the absence of clinical signs of toxicity is not an indication for treatment.

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Nerve Agents - Medical Management

Personal Protective Equipment

First responders are at serious risk of exposure within the contaminated environment (hot zone), either from direct contact with persistent liquid or from inhaling residual vapors. First responders and subsequent emergency care providers outside the hot zone are at risk from handling persons contaminated with liquid nerve agent (through both dermal and inhalation exposure).

Conversely, victims exposed to nerve agent vapor pose little risk to emergency care providers outside the hot zone; residual agent is not present and off-gassing does not occur from the lungs. In the Tokyo sarin attack, approximately 90% of exposed persons reported to medical facilities by private or public transportation without notable contamination of others. Additionally, secondary injury to hospital staff was minimal and did not necessitate specific treatment.

Unless a clear history of vapor exposure only is obtained, emergency medical personnel should assume that liquid contamination is present and wear PPE. For most nerve agent exposures, first responders require level A PPE inside the hot zone, and hospital personnel involved in decontamination should wear level B PPE.

Decontamination

Decontamination should proceed as described in ["General Considerations."](#) Decontaminate with a triple wash, including initial irrigation with tepid water followed by 0.5% hypochlorite solution or soap and water (alkaline solutions also neutralize nerve agent) and repeated thorough water rinsing. In survivors, the amount of residual liquid contaminant is likely to be small, because victims with larger exposures probably will die before they reach the hospital. Other than removing clothing and jewelry, decontamination is unnecessary for victims of vapor nerve agent exposure.

Supportive Care

Saving lives always depends on ensuring adequate airway, ventilation, and circulation. The larger the exposure, the more likely victims require early intubation and ventilation. Conversely, adequate ventilation may be impossible due to the intense muscarinic effects of nerve gas exposure (copious airway secretions, bronchoconstriction). In this situation, administer atropine before initiating other

measures. The use of succinylcholine to assist intubation is not recommended, since nerve agents prolong the drug's paralytic effects.

Treat seizures with adequate oxygenation and liberal doses of benzodiazepines titrated to effect. Termination of seizure activity may reflect onset of flaccid paralysis from the nerve agent rather than adequacy of antiseizure therapy. A bedside electroencephalograph (EEG) may be required to assess ongoing seizure activity.

Animal data suggest that routine administration of diazepam reduces incidence of seizures and decreases severity of pathologic brain injury following nerve agent exposure.

Specific Therapy

Treatment of victims with nerve gas toxicity is broadly similar to the treatment of those poisoned by organophosphate insecticides.

Atropine sulfate

Symptomatic patients require immediate treatment with atropine. Atropine blocks muscarinic effects of nerve agents (eg, bronchorrhea, bronchoconstriction), improving ventilation by drying secretions and decreasing airway resistance. Atropine also blocks other muscarinic effects, such as nausea, vomiting, abdominal cramping, bradycardia, and diaphoresis. Atropine does not have nicotinic effects and thus does not reverse toxicity at autonomic ganglia and neuromuscular junctions. Atropine does not prevent or reverse paralysis.

Atropine therapy is guided by clinical signs and symptoms. Titrate dosing to the desired clinical effect. The goals of atropine therapy are to dry secretions and eliminate bronchoconstriction.

Administer more atropine if assisted ventilation remains difficult or secretions persist. Heart rate and pupil size are poor clinical indicators of adequate atropinization. Presence of tachycardia should not dissuade the clinician from initiating or continuing atropine therapy. Miosis may be absent or delayed in dermal exposures and is not reversed by systemic atropine.

- Adults with mild-to-moderate symptoms: 2 mg of atropine IV/IM/ET q2-5min
- Adults with severe symptoms: 5 mg IV/IM/ET
- Children: 0.02 mg/kg (0.1 mg minimum) IV/IM/ET
- Children with severe symptoms: 0.05 mg/kg IV/IM/ET

Up to 20 mg of atropine may be required the first day, unlike with organophosphate insecticide poisoning, where as much as 3000 mg of atropine may be required over 1 day. In the Tokyo sarin attack, only 19% of poisoned patients required more than 2 mg of atropine. Severely poisoned patients required 1.5-15 mg of atropine.

Oxime therapy

Oximes are nucleophilic substances that bind to the phosphate moiety of the nerve agent more avidly than AChE to reactivate the nerve agent-inhibited enzyme. Reactivation is impossible once dealkylation or "aging" of phosphorylated AChE occurs. Once aging occurs, new AChE must be synthesized. The rate of aging varies among nerve agents. Aging occurs within 2 minutes after soman exposure, 5-8 hours after sarin exposure, and more than 40 hours after tabun and VX exposure.

Pralidoxime chloride (2-PAM) is the only conventional oxime available for clinical use in the US. Administer pralidoxime to symptomatic patients as early as possible, ideally concurrent with adequate doses of atropine. Pralidoxime has its greatest effect at the neuromuscular junction.

- Adult dose: 1-2 g IV
- Pediatric dose: 15-25 mg/kg IV over 30 min

Administer pralidoxime slowly IV to minimize adverse effects such as hypertension, headache, blurred vision, epigastric pain, nausea, and vomiting. When IV access cannot be established, 2-PAM also may be given IM (1 mg with 3 mL sterile saline).

With adequate decontamination and appropriate initial therapy, serious signs and symptoms of nerve agent toxicity rarely last more than a couple of hours. In the unusual event that toxicity persists or worsens clinically, administer repeat doses of 2-PAM at hourly intervals. In the Tokyo sarin attack, severely poisoned patients required 1-36 g. Since 2-PAM is excreted in the urine, lower repeat doses for patients with renal failure and maintain adequate hydration. If hypertension increases during pralidoxime administration, IV phentolamine may help (adults: 5 mg IV; children: 1 mg IV).

Mark I kit

Mark I kit was designed for military self-administration in the field. It consists of two spring-loaded IM Autoinjectors containing 2 mg of atropine and 600 mg of pralidoxime, respectively. These antidote kits are not yet available for civilian use.

Hemodialysis

Japanese physicians reported successful use of hemodialysis and hemoperfusion in one severely intoxicated victim of the Tokyo Subway sarin attack who remained unresponsive to pharmacotherapy.

Disposition

Peak toxic effects occur within minutes to hours and resolve within 1 day. Observe asymptomatic patients exposed to nerve agent liquid for a minimum of 18 hours, since delayed onset of signs and symptoms have been reported (up to 18 h postexposure). Admit symptomatic patients with liquid exposure and monitor them for at least 1 day.

Cholinesterase levels alone should not guide disposition. Some sources recommend observation of asymptomatic patients exposed to nerve gas vapor for 1 hour. In reality, patients who present after inhaling nerve agent vapor have experienced peak effects long before arriving at the hospital, and no further absorption or worsening is expected. When patients experience no signs or symptoms other than eye findings, they may be discharged. Unlike with organophosphate insecticides, nerve agents have not been associated with delayed neuropathy.

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Mustards - Properties and Clinical Effects

Mechanism of action

Sulfur mustard (2,2,-dichloroethyl sulfide) has been used as a chemical weapon since World War I. Nitrogen mustard, a derivative of sulfur mustard, was one of the first chemotherapy agents but never has been used in warfare. These vesicating agents cause blistering of exposed surfaces. Both mustard agents rapidly penetrate cells and generate a highly toxic intermediate episulfonium ion, which irreversibly alkylates DNA, RNA, and protein. This disrupts cell function and causes cell death. The chemical reaction is both temperature dependent and facilitated by the presence of water, which explains why warm, moist tissues are affected more severely. Actively reproducing cells, such as epithelial and hematopoietic cells, are most vulnerable to alkylation. Mustards also produce cytotoxicity by binding to and depleting cellular glutathione, a free radical scavenger. Glutathione depletion leads to the inactivation of sulfhydryl-containing enzymes, loss of calcium

homeostasis, lipid peroxidation, cellular membrane breakdown, and cell death.

Physical Properties

Mustards are oily liquids with odors of mustard, onion, garlic, or horseradish. Highly soluble in oils, fats, and organic solvents, mustards quickly penetrate skin and most materials, including rubber and most textiles. Sulfur mustard is considered a persistent agent with low volatility at cool temperatures but becomes a major vapor hazard at high ambient temperatures. Exposure to mustard vapor, not mustard

liquid, is the primary medical concern. More than 80% of mustard casualties in World War I were caused by exposure to mustard vapor. Mustard vapor is 3 times more toxic than a similar concentration of cyanide gas; however, mustard liquid is also quite toxic. Skin exposure to as little as 1-1.5 tsp of liquid (7 g) is lethal to 50% of adults.

Clinical Effects

Mustards injure the skin, eyes, respiratory tract, GI mucosa, and hematopoietic system. The pattern of toxicity depends partly on whether the person is exposed to liquid or vapor. Liquid exposure primarily damages the skin, producing an initial erythema followed by blistering similar to a partial-thickness burn. Vapor exposure preferentially damages the upper respiratory tract (skin usually is not affected). Mustards penetrate cells and alkylate intracellular components in less than 2 minutes, yet signs and symptoms usually are delayed 4-6 hours (range, 1-24 h). The latent period is shorter with high-concentration exposures, such as those occurring at increased ambient temperature and humidity.

Skin

Chemical burns secondary to mustard often appear deceptively superficial on initial presentation. Earliest symptoms are pruritus, burning, and stinging pain over exposed areas. Moist, thinner skin is affected more severely. Affected areas appear erythematous and edematous. If contamination is more extensive, superficial bullae occur within 24 hours of exposure. Most burns are partial thickness, but full-thickness burns with deep bullae and ulcers may result from exposure to higher concentrations. Severe exposures clinically and histologically may resemble scalded skin syndrome or toxic epidermal necrolysis. Blister fluid does not contain active mustard and is not toxic.

Eyes

Eyes are especially sensitive to the effects of mustard. Ocular symptoms begin 4-8 hours postexposure. Earliest symptoms include burning pain, foreign body sensation, photophobia, tearing, and visual blurring. Clinical manifestations include eyelid edema, conjunctival injection and edema, chemosis, iritis, corneal abrasions, edema and ulceration, and decreased visual acuity. Permanent corneal scarring and blindness may occur with severe exposures.

Respiratory tract

Mustards primarily damage upper airway mucosa. Inhalation of mustard vapor produces a direct inflammatory effect on the respiratory tract, with damage occurring in a progressive downward pattern. The lower respiratory tract and lung parenchyma rarely are affected. Following a variable latent period of 2-24 hours, injury is characterized by hemorrhagic inflammation and airway erosion.

Upper respiratory tract is affected first, evidenced by sinusitis, sinus congestion, sore throat, and hoarseness. Lower respiratory tract symptoms include cough, dyspnea, and respiratory distress. Direct

necrotic effect of mustard on airway mucosa produces epithelial sloughing and pseudomembrane formation, causing small and large airway obstruction.

In severe cases, late pulmonary sequelae include bronchopneumonia and bronchial obstruction. Pulmonary edema rarely occurs, because mustard rarely affects the lung parenchyma and alveoli. Patients with extensive mucosal involvement may suffer fatal respiratory compromise as late as several days after exposure.

Gastrointestinal tract

Mustard damages rapidly proliferating cells of the intestinal mucosa. GI involvement results in abdominal pain, nausea, vomiting, diarrhea, and weight loss.

Hematopoietic system

Mustards cause unpredictable bone marrow suppression, as leukocyte precursors begin dying 3-5 days after exposure. A leukopenic nadir usually occurs in 3-14 days, depending on the severity of exposure. Anemia and thrombocytopenia are late findings. Complete bone marrow aplasia has been reported.

Laboratory Tests

Diagnosis of mustard exposure is clinical. No laboratory tests identify or characterize acute exposure.

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Mustards - Medical Management

Personal protective equipment

Liquid mustard contamination poses a dermal contact risk for emergency care personnel. Specialized protective military garments containing a charcoal layer to absorb penetrating sulfur mustard provide protection for up to 6 hours. These protective garments (chemical protective overgarment, battle dress overgarment, mission-oriented protective posture) are not available outside the military. Level A PPE provides the best protection for civilian first responders, and hospital-based emergency care personnel involved in subsequent decontamination should wear level A PPE.

Decontamination

Decontamination within 2 minutes of exposure is the most important intervention for patients with dermal exposure, since mustard rapidly becomes fixed to tissues, and its effects are irreversible. The classic description is an initial lack of signs and symptoms, which does not lessen the urgency to decontaminate

patients as soon as possible.

Remove clothing immediately and wash the underlying skin with soap and water. Ocular exposure requires immediate copious irrigation with saline or water. Next, decontaminate the skin with 0.5% hypochlorite solution or with alkaline soap and water, which chemically inactivates sulfur mustard. Because mustard is relatively insoluble in water, water alone has limited value as a decontaminant. Decontamination after the first few minutes of exposure does not prevent subsequent damage but at least protects emergency care personnel from further contact exposure.

Supportive care

Treatment of mustard exposure proceeds according to symptoms. Since the effects of mustards typically are delayed, persons with complaints immediately after exposure may have an additional injury. Patients with signs of upper airway obstruction require endotracheal intubation or the creation of a surgical airway. Also consider endotracheal intubation for persons with severe exposures. Use the largest endotracheal tube that can pass through, since sloughing epithelium may obstruct smaller tubes. Have patients inhale moist air. Mucolytics also are recommended for those with respiratory complaints.

Avoid overhydration, since fluid losses generally are less than with thermal burns. Monitor fluid and electrolyte status and replace losses accordingly. Mustard-induced burns are especially painful, warranting the liberal use of narcotic analgesia. Adequate burn care is essential, since skin lesions heal slowly and are prone to infection. Severe burns may require debridement, irrigation, and topical antibiotics, such as silver sulfadiazine. Address tetanus toxoid immunity.

Severe ocular burns require ophthalmologic consultation. Eye care typically includes daily irrigation, topical antibiotic solutions, topical corticosteroids, and mydriatics. Treat minor corneal injuries similarly to corneal abrasions. Apply petroleum jelly to prevent eyelid margins from sticking together. More severe corneal injuries may take as long as 2-3 months to heal. Permanent visual defects are rare.

Specific therapy

Although no antidotes currently are available to treat mustard toxicity, several agents are under investigation, including antioxidants (vitamin E), anti-inflammatory drugs (corticosteroids), mustard scavengers (glutathione, *N*-acetylcysteine), and nitric oxide synthase inhibitors (*L*-nitroarginine methyl ester).

Administer granulocyte colony-stimulating factor to patients with bone marrow suppression following mustard exposure.

Disposition

Patients with significant respiratory tract burns usually require ICU admission and aggressive pulmonary

care. Admit patients with significant dermal burns to a burn unit for aggressive wound management, analgesia, and supportive care. Arrange to monitor blood cell counts for 2 weeks following significant exposures. For 12 hours prior to discharge, observe patients who are initially asymptomatic following mustard exposure.

Most patients recover completely. Only a small fraction have chronic ocular or pulmonary damage. Approximately 2% of those exposed to sulfur mustard in World War I died, mostly due to burns, respiratory tract damage, and bone marrow suppression. Sulfur mustard is a known carcinogen, yet a single exposure causes only minimal risk.

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Conclusion

CWAs comprise a diverse group of extremely hazardous materials. Emergency physicians should be familiar with the pathophysiology and various clinical presentations produced by CWAs as well as the principles and practices of appropriate medical management. Since deployment of CWAs also places emergency care providers at serious risk of exposure, emergency physicians must be familiar with PPE and decontamination.

As potential weapons of mass destruction, CWAs are capable of causing a catastrophic medical disaster, which would overwhelm any healthcare system. Since civilian victims exposed to CWAs are likely to flee to the nearest hospital, emergency physicians play a key role in preparing emergency departments for the treatment of persons exposed to CWAs.

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CBRNE - Chemical Warfare Mass Casualty Management

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Specific Procedures for a Chemical Weapon Response

Specific measures to handle an incident involving a chemical weapon of mass destruction (WMD) should be integrated into existing hospital emergency preparedness plans. Although some of these measures are common, the demands that may result from a chemical WMD crisis require expanding existing hospital emergency preparedness plans, including the protection of employees and the physical plant.

First, establish a chain of command to supervise the hospital staff during a mass casualty event to provide an orderly response when great numbers of casualties arrive. This chain of command may be delineated in the health care facility incident command system (ICS; see <http://www.emsa.ca.gov/dms2/heics3.htm>) or in the emergency operations plan. The designated leader primarily is responsible for coordinating the activities of the departments and hospital staff in accordance with the emergency preparedness plan. In

addition, the leader is responsible for coordinating activities with outside agencies.

One strategy to promote such planning is to have department or clinic chiefs prepare a sequence of objectives for their staff to follow during a chemical incident. Include housekeeping, maintenance, administrative, pharmacy, and security staffs. To support the hospital plan, each department or unit must develop a standard operating procedure (SOP) that defines how each of their tasks will be accomplished. The unit's SOP must be simple but all-inclusive; each task must be defined. Cue cards or checklists to address specific tasks or the sequence of tasks can be prepared as appendices to the SOP. The cue cards, distributed at the outset of an event, prevent essential tasks from being overlooked. The SOPs must be consolidated and reviewed closely to identify shortcomings and to ensure a coordinated response. Staff members must practice frequently to maintain their competency.

A chemically hazardous environment requires additional safeguards to protect hospital workers and patients. Designate a safety officer. This officer monitors work and/or rest cycles of employees working in personal protective equipment (PPE), ensures contamination control, and identifies safety hazards that occur during the hospital response. In addition, the safety officer ensures that patients are decontaminated thoroughly before entering the hospital; therefore, the safety officer is the last line of defense for clinical personnel and the facility. Even with the urgent needs of many victims of a chemical attack, the hospital, staff, and patients must not be compromised by chemical contamination.

Establish a plan to handle decontaminated and contaminated victims of a chemical WMD incident. Ideally, hazardous materials (HAZMAT) teams decontaminate people at the incident site before evacuating them to a medical facility. However, planners should anticipate that less-injured patients will self-transport to the nearest medical facilities and may contaminate these facilities. The plan must include ways of diverting these people to a holding and/or treatment area that does not compromise the health care facility. The requirement to remove all chemical contamination from patients delays their entry into the health care system but should not delay lifesaving treatment by staff in appropriate PPE.

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Integration of Hospital Plans Into Community Disaster Plans

Detection, diagnosis, and mitigation of illness and injury caused by biological and chemical terrorism constitute a complex process that involves numerous partners and activities. A large-scale chemical event is not solely a medical issue. Many local and community resources will be called upon to assist in resolving the situation. Implementation of a plan requires collaboration with state and local public health agencies and groups, including the following:

- Public health organizations

- Poison control centers
- Medical research centers
- Health care providers and their professional networks
- Professional societies
- Medical examiners
- Emergency response units and first responder organizations
- Safety and medical equipment manufacturers
- Federal agencies
- Federal Bureau of Investigation (FBI)
- Local law enforcement

Establish an early notification system with emergency response units and first responder organizations to notify hospitals when a chemical WMD incident occurs. Early notification of an event helps a hospital prepare for mass casualties. Preparing a medical facility to receive casualties who may arrive without preliminary decontamination requires considerable effort. Early warning permits initiation of specific procedures and assembly of appropriate staff to receive casualties.

Prepare formal agreements with vendors (ie, hospital suppliers, pharmacies), health departments, emergency medical services (EMS), fire and police departments, other local hospitals, laboratories, city officials (transportation and emergency planners), the American Red Cross, and ambulance services to provide the best possible response. Review these agreements annually and practice procedures frequently to maintain proficiency.

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Communications

Establish a communication plan. An accurate and effective communications network is essential to a coordinated and efficient incident response. Communication equipment and expertise are likely to be among the weakest links in any response to a terrorist incident. During conventional incidents involving

only a few casualties, information frequently is inadequate, incorrect, or nonexistent. Backup systems with built-in redundancy inside the hospital are necessary to ensure that communication equipment is available at critical moments. Consider continuous open channels to maintain a flow of information. Communication plans should identify departments, units, or positions rather than individuals because of varying work shifts and rotation of personnel in key positions. Designated channels should be available for the FBI and the local law enforcement agency.

Method of communication

Select an effective method of communication. Mobile telephone systems may be overwhelmed and become ineffective as the first responders, members of the ICS and health care system, and citizens attempt to keep abreast of the situation. Handheld radios within the hospital are effective and frequently are the communication method of choice. The radios' short range is effective within the medical facility but does not interfere with similar devices a short distance away. Care must be taken with portable communication equipment to avoid interference with patient monitoring devices and other electronic equipment within the hospital. Coordinate communication plans with outside agencies early in the planning phase to avoid conflicts with local regulations and outside requirements. Tailor the methods of communication for each hospital's needs; they also must meet the requirements of the surrounding community. Anticipate communication needs that may occur during a crisis and plan for those needs.

Hospitals should coordinate and practice communication systems to be prepared during a chaotic situation. Anticipate having to shift personnel and move supplies to meet rapidly changing demands. Moving patients within the health care system requires effective communication between the receiving area, clinics, wards, radiology, and expedient patient holding areas so that patients are tracked effectively. Maintain a written log to follow patients to ensure that they reach the appropriate area for definitive care. Members of the American Red Cross or another volunteer group may be able to operate a patient locator system and permit hospital staff to be assigned to more critical or complex positions.

Establish procedures to receive calls from family members and friends to keep telephone lines free for operational purposes. Hospitals must be prepared to help families and friends when they inquire about patients' whereabouts and medical status. Finding and identifying patients is a major undertaking. Patient confidentiality must be maintained during this process.

Information dissemination

Remember that controlling sensitive or secure information may be a priority among law enforcement officials. Police and the ICS supervisor will be more willing to communicate with health care facilities if outsiders cannot access the communication system. The ICS is the model tool for command, control, and coordination of a response and provides a means to coordinate the efforts of individual agencies as they work toward the common goal of stabilizing the incident and protecting life, property, and the environment.

Implement an emergency communication plan that ensures rapid dissemination of health information to the public during actual, threatened, or suspected acts of chemical terrorism. Announcements to the news media must be open, reliable, and current. During a terrorist event, all public communication should be through the designated community or regional spokesperson, even though large medical centers may have communication procedures and a media spokesperson. Gaining public cooperation requires that consistent information from all sources is related by one spokesperson. Effective communication with the public through the news media is essential to limit the terrorist's ability to induce public panic and disrupt daily life. Gaining and retaining public confidence and cooperation is extremely important during a WMD incident.

Address what information to release and by which method. Prepare sample public health advisories and develop repositories of information on anticipated chemical warfare agents and industrial chemicals for use in a crisis. Specific information is available on traditional chemical warfare agents and toxic industrial chemicals at one or more of the web sites identified in this article. Provide the community spokesperson with a fact sheet prepared specifically for the public. In some instances, the hospital spokesperson may be called upon to comment on the situation. Reliable information from a known and respected community resource can dismiss rumors and inaccurate predictions from self-proclaimed experts. To achieve a more rapid response to warnings, provide the public with enough information to allow everyone to understand the instructions from local officials.

In the hospital's emergency preparedness plans, include plans for communicating with the media. The designated spokesperson or security person should assist the reporters and camerapersons but not allow them to interfere with hospital activities. During decontamination and treatment, consider the privacy of patients, and prohibit filming of patient areas. Staff members who come into contact with media personnel should refer the reporters to the appropriate hospital spokesperson. The designated spokesperson responds to the media according to hospital policy after coordinating with the regional or community spokesperson.

Improving the plan

Prepare a formal critique of the event response as an important step in improving the emergency plan. Afford the entire staff the opportunity to present comments through the chain of command. Presenting these comments during the first 24 hours after the event is resolved is critical to capture the lessons learned.

Personnel considerations

Develop a recall roster adapted to local needs. Prioritize and update the sequence of notification of personnel (see [Table](#)). Clinics or support departments may have their own recall roster in addition to maintaining one for key hospital staff. Because actual terrorist incidents may be unannounced, the treatment facility may have little time to prepare for receiving casualties. Having as many key staff members present as possible when casualties arrive greatly facilitates response. Although the presence of

clinical staff members is generally the first priority, support staff members also are necessary. Security, logistics, pharmacy, housekeeping, and patient decontamination personnel are other essential staff in a chemical incident scenario.

Recall Roster

| Name/Organization | Office/Beeper/Home/Cell Phone |
|------------------------------------|-------------------------------|
| Poison control center | ^ |
| Director ED | ^ |
| Physicians | ^ |
| Nurses | ^ |
| Security | ^ |
| Nursing supervisor | ^ |
| Decontamination team | ^ |
| Public relations person | ^ |
| Housekeeping administrator on-call | ^ |
| Others as determined locally | ^ |

Those individuals identified as essential in the event of a mass casualty incident must be aware that they are required to respond with little or no notice. Family considerations and support require advance planning and preparation. Many of today's workers are single parents, and in many families both spouses are employed. Essential personnel need to make prior arrangements for family support, child care, and pet care in case the operation continues for hours or up to days. Planning strategies should be developed that account for medication prophylaxis of essential personnel and their families.

Prepare a format to provide critical information to the local poison control center, public health department, and other agencies. Distribute the format to key personnel in the hospital and those agencies. Provide the following information as it becomes available:

- Number and types of casualties
- Substances involved
- Estimated time of arrival at the hospital
- Time of incident
- Incident site

- Method of contamination (vapor or liquid)
- Necessary decontamination (and extent)
- Hazards to health care providers
- Role of the health care facility in the incident
- Updated information as it becomes available

Enlisting the support of health care providers who normally work outside the hospital is important during an influx of casualties. Physicians and nurses in family practice clinics and urgent care centers may be available to augment the hospital staff. Have a list readily available of community health care providers who are trained and prepared to assist in emergencies. These providers are essential during sustained operations. Consider credentials and hospital privileges for these physicians and nurses. Address liability and reimbursement for services. Volunteers may be available to help with specific tasks. Identify in advance people from outside the normal hospital staff who can help in a severe situation and use these people when appropriate. These individuals require an orientation to the disaster plan to be most effective.

The hospital incident commander must know what staff is available at all times; therefore, maintain a roster of when staff members arrive and depart. When they report to work, have key and essential personnel pass through the hospital entry control point. The cue cards discussed earlier can be handed out as staff members enter the facility. This plan allows the hospital incident commander to identify shortfalls and tasks that need to be reassigned.

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Casualty Management

A health care facility must develop an area, preferably outside the hospital, for decontamination, and personnel must be trained in patient decontamination. This special area is crucial for the protection of the facility. After the Tokyo sarin attack, local hospitals were overwhelmed immediately with chemical agent casualties. Victims who arrived at the hospitals early were self-transported. No effort was made to decontaminate them; as a result, several health care providers were exposed to sarin vapor that off-gassed from clothing.

Protection of personnel

The first rule for personnel assigned to decontamination must be that they protect themselves.

(Information concerning PPE is presented in [CBRNE - Personal Protective Equipment](#)). Failure to use appropriate procedures or protective equipment places the individual, health care workers, and the medical treatment facility at risk. During a mass casualty incident, many people require medical care; neither health care personnel nor health care facilities can be compromised.

Decontamination area

The ambulance garage or parking area may be used as a decontamination area. Since the ambulance area frequently is located near the ED entrance, casualties may be sent to the ED after they are clean. The area also may provide adequate storage for decontamination equipment and supplies. The garage can be heated for patient protection or cooled to reduce the heat load of PPE worn by the workers; however, sites with overhead covers only can provide protection from radiant heat. A water source generally used for washing vehicles can be used to decontaminate casualties. Fire fighters can be consulted on how to modify the existing plumbing so that it is conducive to washing both ambulatory and nonambulatory casualties.

Consider how to move ambulatory patients and patients on litters. In addition, plan for and prepare sites for the separation of males and females for undressing, washing, and dressing; a heating source for water; and drainage and capture of potentially contaminated water. An effective decontamination site can be erected with limited expenditures by using available structures and equipment. Litters are now available with open mesh fabric that permits effective decontamination, which is not possible with a standard gurney. Conveyor roller units also can make handling nonambulatory patients easier as they are moved through the decontamination lane.

Personnel designated to decontaminate patients require initial training and periodic practice. Specific procedures that may be suitable to civilian needs are detailed in the *Guidelines for Mass Casualty Decontamination During a Terrorist Chemical Agent Incident* (<http://dp.sbcom.army.mil>) prepared by the US Army Soldier and Biological Chemical Command.

A decontamination site has 3 areas, as follows:

- Hot zone: Incoming traffic, personnel, and casualties potentially contaminate this area.
- Warm zone: Decontamination takes place in this area.
- Cold zone: In this clean area, casualties are triaged again and then moved into the health care facility.

Managing a chemical casualty is a continuum from triage to discharge from the hospital. Only the management principles required to get a chemical casualty safely into a medical treatment facility are discussed.

Triage

The first task is to triage casualties before decontamination and as they enter the hospital. The purpose of triage is to sort the injured by priority and determine the best use of available resources (eg, personnel, equipment, medications, ambulances, hospital beds). The emphasis is on saving as many people as possible. Triage must occur at each site due to changes in patient status. In a chemical incident, victims are triaged for 3 purposes at the incident site: decontamination, treatment, and evacuation. Triage at the hospital primarily is for decontamination and treatment. Evacuation to other medical facilities may be necessary to provide specialty care or to distribute the patients throughout the regional health care system.

Triage terminology and methods to identify the different categories vary and are a matter of preference. To avoid confusion or delays in care, standardize terminology and methods throughout the region. Four triage categories for treatment are described as follows:

- Immediate: Casualties need lifesaving measures performed without delay if they are to survive.
- Delayed: Casualties can wait for definitive treatment without causing additional harm.
- Expectant: Casualties will not survive or will require extensive resources and time if they are to be saved.
- Minor: Casualties are generally ambulatory and are injured only slightly.

Triaging for decontamination is necessary to move the casualties quickly and safely into the health care system. Divide casualties into ambulatory and nonambulatory groups. Ambulatory casualties most often are classified as minor or delayed. However, observe people triaged as minor or delayed for worsening signs and symptoms. Immediately decontaminate casualties who were closest to the point of chemical release, those with liquid contamination, and those who have severe signs or symptoms of chemical injury or severe conventional injuries. Victims may be classified as expectant when they have serious signs and symptoms after initial therapy or are unresponsive to antidotes.

Move deceased casualties to a site that is not observed readily by the public or other casualties. Keep the deceased at this site until law enforcement officer(s) have acquired any available evidence and living casualties have been moved into the health care facility. Deceased casualties require a thorough decontamination before they are moved to the morgue or they are ready for release.

Effective triage requires the presence of a triage officer who is trained to identify the type of casualties that will be sorted. For instance, a person contaminated with a liquid nerve agent who arrives as an ambulatory casualty may deteriorate rapidly once the agent is absorbed. Triage officers are in a key position, and the individual must be capable of making quick and frequently difficult decisions. Also, triage officers must be very familiar with the medical staff and the hospital's capabilities and limitations.

Admission procedures

Following a chemical event with many casualties, normal patient admission procedures are not effective. A system that initially captures essential information is sufficient. Additional patient information can be captured when time permits. Ambulatory patients and many nonambulatory casualties are able to provide appropriate information. However, identify unconscious casualties before their personal belongings are removed. A plastic driver's license with a photo may be decontaminated and used to accompany the patient. Photo identification is particularly useful for unconscious and deceased casualties. Additionally, a number can be assigned to each patient; the number then can be placed on the patient and on two bags. The first bag contains clothing; the second holds the patient's valuables. Clear bags facilitate identification at a later date. A meticulous, practical method of cataloging belongings helps ensure proper return and possibly helps in forensic investigations.

A secondary triage following decontamination further identifies casualties who require immediate attention and those who can wait for treatment. Move casualties who can wait for treatment to a clean holding area. A nurse, paramedic, or emergency medical technician assigned to the clean holding area can observe these individuals and provide reassurance if necessary. Additional clean holding areas can be established in dining rooms, physical therapy departments, and meeting rooms.

Emergency treatment

Emergency treatment for immediate casualties depends on the chemical agent(s) used and on the availability of definitive treatment measures. For many toxic agents, no antidotes are available, and treatment is supportive. Administer antidotes judiciously to ensure that they are not wasted on minor casualties who arrive early or on those with no chance of survival. The requirement for ventilators may exceed availability. Spreading the serious casualties among community hospitals or consolidating ventilators to one or two major medical treatment facilities are local options.

Take measures to protect hospital personnel and maintain the integrity of the health care facility. Have in place emergency procedures for exposure of staff members. If the decontamination procedure fails and staff members become contaminated, decontaminate and treat them. Until the cause of the contamination is determined, do not use the treatment facility or the contaminated area of the facility. If decontamination procedures fail, correct them immediately. Place exhaust fans to ventilate contaminated air. Keep doors between clean and contaminated areas closed, except when moving a casualty into a treatment area. Do not let chemical vapor contamination into an area with unprotected health care providers and casualties.

Human remains may provide essential evidence and are important to law enforcement personnel. Unconventional methods must be used to hold the bodies until they can be decontaminated and released. Refrigerated vans may be used to store the remains temporarily. Morticians and funeral directors may need to be given specific directions for handling the remains. Bodies exposed to classic chemical warfare agents can be decontaminated and released to funeral homes. Bodies contaminated by other chemicals require individual evaluation for safety in handling. The US Public Health Service has developed Disaster Mortuary Operational Response Teams to provide victim identification and mortuary services. State authorities may request assistance from these teams.

An important part of the medical response to a chemical terrorism event is dealing with the psychological reactions among health care workers, first responders, and the public. The death of family members, coworkers, and particularly children is traumatic. Plan to provide critical incident stress management support early in a chemical event.

Transportation issues

Transportation and evacuation of patients are major tasks during a WMD event. Have a community-wide plan, enforced by the local police department, to identify and mark areas of contamination so that hazardous areas can be avoided and traffic does not become congested. Health care providers must be able to identify themselves and use the routes identified by the police to travel to the hospitals. Although many of the victims will arrive early at the medical facilities by various modes of transportation, the more severely injured may be among the last to arrive. Contact the US Environmental Protection Agency for specific guidance on contamination identification and control.

Plan for the movement of equipment, supplies, and pharmaceuticals to support the influx of casualties. Access to critical areas must remain open, and traffic must not be permitted to become congested. A circular movement of traffic in the critical areas facilitates unloading casualties and rapid departure of vehicles. To ensure that vehicles follow the plan, assign a security guard wearing PPE to direct traffic flow. Security personnel in PPE must have training and medical clearance, required by the Occupational Safety and Health Administration (OSHA), when wearing respirators and protective clothing. During warm weather, security personnel must be rotated frequently to avoid becoming heat casualties.

A sufficient number of ambulances will not be available to transport hundreds of casualties in a short time. Consider city bus systems or school buses as alternatives. Methods of moving large numbers of victims may vary for different communities. As a general rule, do not mix resources used to move contaminated or potentially contaminated casualties with those used for moving uncontaminated patients.

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Logistics and Facility Management

When the number of patients exceeds the treatment capability of a medical treatment facility, resource management becomes crucial. Planning for the availability of essential supplies, equipment, and PPE at critical times requires close coordination between clinical and support personnel.

Maintain a list of the location and quantity of ventilators and establish agreements between vendors and hospitals prior to an incident. These agreements must be reviewed and agreed upon by all health care facilities in the community and not limited solely to agreements between one hospital and a supplier. Develop a respiratory therapy mutual aid plan among multiple hospitals and include ventilator vendors.

Other important equipment requires the same coordination and written agreements.

List vital pharmaceuticals and supplies and prepare them for rapid distribution. An integral part of an emergency plan includes listing specific antidotes and supporting pharmaceuticals for the most likely emergency situations. Such lists permit pharmacists to prepare preplanned packages to send to the emergency treatment area and save valuable time in meeting the treatment demands of many casualties. Awareness of the stockpile of antidotes maintained by the Metropolitan Medical Response Systems (MMRS), the National Medical Response Teams (NMRT), and the National Pharmaceutical Stockpile (NPS) of the Centers for Disease Control and Prevention (CDC) as well as the antidotes' expected time of arrival may alleviate the need for large quantities of antidotes to be stockpiled locally. A regional pharmaceutical mutual aid plan can be developed among hospitals and can include pharmaceutical suppliers.

Blueprints of the hospital and diagrams of the immediate area surrounding the hospital are useful in planning for a WMD event. Blueprints help determine the most appropriate routes for patient flow and vehicle movement. The identification of an area for decontamination with ready access to an emergency treatment area may be much more apparent when looking at a diagram. A large dining facility or waiting area may be prepared quickly to receive casualties. Warehouses may provide alternate facilities for initial triage, decontamination, and emergency treatment or serve as a holding area for minor casualties. Alternatives to the usual ways of conducting business and areas allowing for overflow of patients are important components of a comprehensive plan.

Alternate or back-up utilities may be required if the chemical incident site includes the local electrical plant or if electrical power is disrupted. Water systems also may be affected. A medical treatment facility must be self-sufficient for 24 hours following a terrorist event or natural disaster.

The influx of both patients and staff requires food service support. If the chemical event continues for hours or days, food service personnel need to provide food and water to meet the increased demands. Additional housekeeping service to remove excess trash, linens, and expendable items is required. Maintenance workers are needed to keep equipment functioning in treatment areas. Identify areas for the temporary storage of potentially contaminated clothing and other personal belongings that must be identified and stored until final disposition is determined.

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Security and Safety Plans

The normal hospital security plan requires modification to satisfy requirements of the emergency situation and to conform to community plans. To avoid the risk of the health care facility becoming contaminated and unusable, it must be secured and access limited. At the hospital staff entry point, guards check to ensure that people arriving are listed on the hospital disaster response roster, log those people in, and note

their time of arrival. The same procedures apply to personnel leaving the hospital. Identification badges (ID) can be issued at the entry control point. The ID may be issued to employees before an event, but invariably, some will not have their ID when they arrive. Badges can be color coded to identify a particular unit or service.

Security is necessary at the decontamination point. Guards need to wear PPE; rotate them frequently in warm weather. Crowd control, casualty flow, and traffic control are critical at the health care facility. Vehicles must not linger in the area, or others will be slowed or denied access. Crowds and media must be kept at a safe distance so that they are not harmed and do not impede the emergency process. Develop a plan to address any breach of security.

A terrorist may plant more than one device. Additional devices may not be the same type as the initial one and may not be at the same site. Investigate and secure the area where casualties or onlookers congregate during an emergency to exclude the presence of additional devices. For example, a terrorist in Northern Ireland exploded a bomb that caused several casualties. Knowing a crowd would form to unload and assist the casualties, the terrorist exploded a second bomb outside the busy emergency treatment area.

Have a safety plan that addresses specific issues in chemical safety and augments the existing emergency preparedness safety plan for other disasters. An existing safety plan may not include contamination control and casualty decontamination requirements.

Have a safety officer(s) at the medical treatment facility who is knowledgeable in the operations being implemented. The safety officer has specific responsibility for identifying and evaluating hazards and provides direction with respect to the safety of operations. The safety officer's primary function is contamination control to ensure the safety and welfare of the hospital personnel and prevent the contamination of the facility. To avoid contaminating treatment areas, constant vigilance is required to ensure that contaminated casualties, staff, and equipment are not permitted to enter the hospital area prior to decontamination. When positioned at a critical location, the safety officer can help prevent the spread of contamination. Safety officers need to work closely with security staff and police to enforce measures to protect personnel and health care facilities.

In addition to preventing contaminated people and objects from entering the health care facility, the safety officer can observe workers while they are performing decontamination. The officer is able to ensure that work and/or rest cycles are implemented and enforced and that PPE is worn appropriately in hazardous areas. Also, safety officers may be appointed as the final approving authority for decontaminated casualties to enter the hospital.

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Training and Exercises

Training

OSHA cites specific training requirements for first responders and others who will participate at the incident site. Additional information on training requirements may be available at the National Institute for Occupational Safety and Health. Hospital personnel who are designated as safety officers or who are involved in decontamination also should be trained to the appropriate level as specified in the OSHA Hazardous Waste Operations and Emergency Response regulation. Everyone who participates at an incident site or in receiving contaminated casualties must know the basic hazards, signs, and symptoms of exposure to chemical agents.

An effective mass casualty plan relies on all staff members knowing their tasks and responsibilities. Most employees are involved in a specific task in one area of the hospital. Becoming familiar with a particular task may require minimal training. Individual training and small team training are appropriate before integrating multiple teams into a training exercise.

Decontamination is not a routine task, and employees require extensive training. Each specific task is not complex; however, the ability to perform the tasks while wearing PPE requires hours of practice. In addition, cross-train the decontamination team in various tasks, because they must be capable of changing tasks to assist other team members. Building from single tasks to multiple tasks, learning to function in PPE, and learning to work as a team takes time, but rehearsal can produce an effective team. Physicians, physician assistants, and nurse clinicians designated to provide treatment to chemically contaminated casualties require specific training. A cadre of well-trained health care and public health workers should be available at each health care facility.

The US Army requires that physicians assigned to depots storing chemical warfare agents attend courses in the management of chemical agent casualties in addition to attending advanced cardiac life support (ACLS) and advanced trauma life support (ATLS) courses. The Medical Response to Chemical Warfare and Terrorism teleconference course is available to civilian health care providers via the Internet and is offered biannually at many videoconference sites throughout the country. Information on these broadcasts and additional training courses such as the "Medical Management of Chemical and Biological Casualties Course," jointly sponsored by the U.S. Army Medical Research Institute of Infectious Diseases (USAMRIID) and its sister organization, the Institute of Chemical Defense (USAMRICD), can be found at www.nbc-med.org.

Rapid turnover of personnel requires ongoing training programs. Reviewing the appropriate portion of a written plan and viewing a videotape of a previous exercise can facilitate the training process. Videotaping exercises is an excellent method to capture department activities, and videotapes can be produced inexpensively. In addition to being used as training aids, tapes can be observed and critiqued to improve response.

Hospital preparedness for mass casualty incidents increases if hospitals engage in regular, ongoing inservice training programs and in readiness drills. Some of the cost for such training can be defrayed

through the use of videotapes, CD ROMs, and web-based technologies. In addition to being cost effective, these resources are updated frequently and provide new and revised lists of resources. The American College of Emergency Physicians is preparing a standardized 2-phase curriculum and a set of training materials for mass casualty preparedness. Such initiatives to standardize training reduce individual hospital costs and provide acceptable procedures and educational material.

Components of the educational curricula and course outlines have been developed by several agencies. The typical curriculum is divided into multiple levels. The first level, a brief awareness program, is designed for all workers and describes the threat and basics of how to respond while protecting personal safety. A decontamination procedures course is more extensive and is intended for workers designated as members of the decontamination team. The decontamination course includes presentations on PPE, safety issues, required medical screening, stations in the decontamination line, and physical characteristics of chemical agents. Health care providers are presented a course on the recognition and management of chemical agent casualties. The clinician's course may use the Medical Response to Chemical Warfare and Terrorism teleconference.

To supplement training, some hospitals have developed cue cards that identify a set of tasks for a department or unit. Using a checklist facilitates accomplishing all tasks that are necessary for the operation. In the absence of a trained primary member, another member of the unit can ensure that each task is assigned and completed.

Drills

Following individual and unit training, the hospital should integrate a WMD response into one of the two annual drills required by the Joint Commission on Accreditation of Healthcare Organizations (JCAHO). An ideal hospital exercise requires rehearsals through tabletop exercises for supervisors and small unit drills. Both require time and personnel, but in some cases, they can be as simple as a clinic or unit staff meeting designed for a discussion and walk-through of each portion of the emergency preparedness plan.

A tabletop exercise for leadership provides an opportunity for senior personnel to coordinate their activities during a mock exercise. An exercise that involves each department chief and requires synchronization of major activities reveals deficiencies in the emergency preparedness plan at the management level. In addition to the overall plan, the communications component can be tested during the tabletop exercise. Internal hospital communications and contacts with designated agencies within the community are both critical to the response. Once the unit training and the tabletop exercise are completed, schedule an exercise to test the overall system and to meet JCAHO requirements.

Biannual (or more frequent) hospital exercises can be videotaped for review, analysis, and future training. The exercises can be extremely valuable in identifying flaws in and minor adjustments to the response plan. A person who happened to be videotaping in a Tokyo hospital recorded the 1995 sarin incident. That videotape has been used many times as an example in training classes and as a tool to improve hospital response.

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Recovery

Once the event has ended and the patients are within the health care system, the process of returning to normal function begins. Scientifically and logistically, this can be the most difficult aspect of the incident. Numerous areas, equipment, vehicles, and other items may be left contaminated. Identifying the contaminating chemical(s), the preferred methods for decontamination, the chemical's persistency on various surfaces, and the chemical's effect on water and food sources requires considerable review and expertise.

The decontamination area must be cleaned, and contaminated clothing and equipment used during the decontamination process must be cleaned or disposed of appropriately. Final disposition of contaminated articles is in accordance with previously established arrangements and the overall community hazardous waste plan. Patients' valuables are an important issue. During the patient decontamination process, rings, watches, wallets, purses, and other items such as hearing aids are removed and placed in marked plastic bags.

A security person may be the most appropriate person to ensure that these items are stored in a secure manner. To avoid legal difficulties, follow chain-of-custody procedures to ensure that these valuables are not disturbed. As soon as it is practical, check these items for contamination and clean them as necessary for return to their owners. The agency responsible for declaring items safe for return to their owner may be a city, state, or federal organization, depending on local and state capability.

Capture lessons learned early in the recovery phase. An exercise critique must include each participant at the clinic or unit level. Individual workers and unit supervisors can contribute important comments on specific procedures to improve their respective operations. The activities at this basic level are crucial to the overall plan and must be included to refine and improve the complete plan. Department chiefs then must meet to discuss their consolidated recommendations. The incident leader and hospital administrator can incorporate the needed changes.

Lessons learned must be captured within hours after an exercise or incident. Otherwise, hospital staff returns to normal responsibilities, and important information is lost. Allocate a time at the termination of each exercise to obtain these comments and recommendations. The department chiefs can consolidate the comments and recommendations and meet with other chiefs and administrators within 24 hours to discuss their suggestions.

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CBRNE - Cyanides, Cyanogen Chloride

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Synonyms, Key Words, and Related Terms

blood agents, chlorine cyanide, chlorocyan, chlorocyanide, chlorocyanogen, cyanochloride, cyanide gas, ClCN, CK

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Introduction

Background

Cyanogen chloride (North Atlantic Treaty Organization [NATO] designation CK) is one of two volatile cyanide military chemical warfare agents. The other similar agent is hydrogen cyanide, or AC (please see

[CBRNE - Cyanides, Hydrogen](#)). These agents first were used in large quantities by the French and British during World War I. While the US maintained 500-pound and 1000-pound CK bombs, these were not used during World War II. More recently, Iraq is suspected to have used a cyanidelike agent against the Kurds in the 1980s.

While CK and AC are similar in their toxicity, a few important differences exist. Firstly, CK is less volatile than AC, making it more effective at low concentrations. Secondly, by nature of its chlorine moiety, CK causes irritation of the eyes and respiratory tract and potential delayed pulmonary toxicity similar to chlorine or phosgene gases. In high concentrations (eg, in enclosed spaces), CK is a rapidly acting lethal agent, causing death within 6-8 minutes if inhaled at doses at or above its LCt50 (lethal concentration that kills 50% of people; 11,000 mg·min/m³).

CK is synthesized in the US for industry by Matheson Tri-Gas and is used as an organic precursor and in mining and metalworking. Therefore, an emergency physician may be more likely to encounter CK-exposed victims following an industrial accident rather than in a warfare or terrorism scenario.

Pathophysiology

In addition to CK's local irritant effects, systemic toxicity occurs through mechanisms similar to those seen with hydrogen cyanide exposure (see [CBRNE - Cyanides, Hydrogen](#)). CK liberates a cyanide molecule, which enters the blood stream and distributes to tissues. Once inside cells, CK binds to mitochondrial cytochrome aa3, interrupts electron transport, and creates imbalance between ATP synthesis and hydrolysis. Oxygen is unable to be used effectively as the terminal electron acceptor, which forces a shift to anaerobic metabolism. While all organ systems are impacted, the most oxygen-dependent ones are the most affected (ie, brain, heart).

Frequency

- **In the US:** The major source of cyanide poisoning in the US does not come from CK but rather from smoke inhalation during house and/or industrial fires in which plastics (acrylonitriles, polyurethanes), wool, or silk are burned. Cyanide poisoning also is found in association with chemical synthesis, electroplating, mineral extraction, dyeing, photography, and agriculture.

Mortality/Morbidity

In high concentrations, which can be obtained in enclosed spaces, CK is a rapidly acting lethal agent causing death within 6-8 minutes.

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Clinical

History

When taking a history from a patient with possible toxic gas exposure, ask about the smell and color of the gas, onset of symptoms, duration and severity of symptoms, and effect on surroundings (eg, dead animals, other people). CK is a colorless liquid or gas with a pungent biting odor, which may in fact go unnoticed because of discomfort. Symptoms that may be associated with CK exposure include the following:

- In low doses
 - Bronchorrhea
 - Lacrimation
 - Rhinorrhea
- Moderate range doses
 - Onset possibly takes several minutes, except for immediate irritant effects
 - Transient hyperpnea
 - Feelings of anxiety or apprehension
 - Vertigo
 - Nausea and/or vomiting
 - Prolonged prodrome prior to loss of consciousness
 - Seizures
 - Bradypnea followed by apnea
 - Cardiac arrest

Physical

Patients may suffer from any of the above symptoms. Physical findings are generally nonspecific and similar to those of severe hypoxemia.

- Severe prolonged exposure culminates in respiratory distress, convulsions, and apnea.
- Patients may have cherry red or pink skin because of concomitant carbon monoxide poisoning or because of cyanide-induced lack of extraction of oxygen at the tissue level and vasodilation. Arterialization of the venous blood also may be noted at phlebotomy or upon examination of the retinal veins. However, note that bright red skin or absence of cyanosis rarely is described. Cyanosis may be observed and most likely stems from concomitant cardiovascular collapse, seizures, or apnea. Finally, many cyanide victims have normal-appearing skin.
- Patients initially are hypertensive, tachypneic, and bradycardic, but eventually they become hypotensive. They may experience a transient tachycardia before spiraling into bradydysrhythmia that deteriorates into asystole.

Causes

Other than acts of terrorism or war, a mass casualty may develop in an industrial accident in which CK comes in contact with water (eg, during a fire-fighting expedition). Containers of CK may rupture or explode if exposed to high heat or following prolonged storage.

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Differentials

CBRNE - Cyanides, Hydrogen
CBRNE - Lung-Damaging Agents, Chlorine
CBRNE - Nerve Agents, Binary: GB2, VX2
CBRNE - Nerve Agents, G-series: Tabun, Sarin, Soman
CBRNE - Nerve Agents, V-series: Ve, Vg, Vm, Vx
Shock, Septic
Toxicity, Ammonia
Toxicity, Chlorine Gas
Toxicity, Ethylene Glycol
Toxicity, Hydrogen Sulfide
Toxicity, Isoniazid
Toxicity, Organophosphate and Carbamate

Other Problems to be Considered

Consider the diagnosis of CK poisoning in patients with irritation of the eyes, nose, and respiratory tract followed by rapid collapse or seizures, accompanied by metabolic acidosis and decreased oxygen consumption.

Other agents that may have similar features in toxicity include the following:

Methemoglobin-inducing agents

Carbon monoxide

Inert gases (simple asphyxiants)

Hydrogen sulfide

Azides

Arsine

Phosphine

Monomethylhydrazine

Isoniazid

Water hemlock

Strychnine

Organophosphates

Metformin

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Workup

Lab Studies

- Arterial and venous blood gases
 - Cyanide poisoning is characterized by a normal arterial PaO₂ despite symptoms of hypoxia and an abnormally high venous PO₂ (decreased arteriovenous oxygen [A-VO₂] difference).
 - Victims of CK exposure may develop lowered PaO₂ due to irritant effects on the respiratory tract resulting in bronchospasm or pulmonary edema.
 - Depending on the severity of exposure, an arterial blood gas with a mixed respiratory and metabolic acidosis may be present. Metabolic acidosis is a hallmark of significant cyanide toxicity.
- Cyanide levels: These levels generally are not available in time to guide acute treatment but may be confirmatory. The preferred test is an RBC cyanide level.
- Carboxyhemoglobin and methemoglobin levels: Obtain these levels, especially in victims of smoke inhalation. During treatment with sodium nitrite, observing methemoglobin levels over time may help to avoid toxic methemoglobinemia.

Other Tests

- Shortening of the ST segment with eventual fusion of the T wave into the QRS complex has been observed.

Procedures

- Depending on the severity of symptoms, endotracheal intubation may be necessary to optimize oxygen delivery and protect the airway.
- Fluorescein staining and slit lamp examination of the eyes may be necessary following decontamination to assess corneal integrity.

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Treatment

Prehospital Care

- Remove victim from the cyanide source. Rescuers must have the highest level of respiratory protection (Level A).
- Give priority to decontamination of the eyes with water. Remove contaminated clothing and decontaminate the skin as appropriate with soap and water.
- Aggressive oxygenation, airway control, and supportive care with intravenous access and continuous cardiac monitoring are appropriate.
- Cyanide antidotes generally are not available in the prehospital setting but may be on hand at certain industrial sites. Administer antidotes as soon as possible in suggested or known cases of cyanide toxicity.

Emergency Department Care

- Continuation of hemodynamic support and optimization of oxygenation are the mainstays of treatment.
- If not performed at the scene, decontaminate patients by removing and isolating clothing and washing the patient from head to toe with soap and water. Avoid self-contamination of hospital workers. Following ocular decontamination, check for corneal integrity.
- Initiate antidote therapy with nitrites and sodium thiosulfate as soon as possible. Do not delay treatment for confirmatory RBC cyanide levels.
- Aggressive management of seizure activity with benzodiazepines is crucial.

- Consider gastric lavage and administration of charcoal in cases of recent cyanide ingestion. The gastric aspirate may cause secondary contamination and must be treated as hazardous.

Consultations

- Consult with law enforcement authorities and the Federal Bureau of Investigation (FBI) in any suspected terrorist incident.
- Consultation with a medical toxicologist and/or poison control center and intensivist may be useful.

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Medication

The Pasadena (formerly Lilly) Cyanide Antidote Kit contains amyl nitrite, sodium nitrite, and sodium thiosulfate. Theoretically, the nitrite components oxidize iron contained in hemoglobin to methemoglobin. This creates an additional site for cyanide binding and promotes dissociation from cytochrome oxidase. Resultant cyanomethemoglobin then may be converted to less toxic thiocyanate through enzymes such as rhodanese or other sulfurtransferases in the presence of sodium thiosulfate. Only use amyl nitrite perles as a temporizing measure if IV access has not been established, since administration of IV sodium nitrite is more effective in creating therapeutic methemoglobin levels. Do not use sodium nitrite or use it only with extreme caution in the setting of concomitant carbon monoxide poisoning. However, in cases of smoke inhalation in which cyanide toxicity is suspected, administration of sodium thiosulfate is safe.

Unlike carbon monoxide, inhibition of cytochrome oxidase by cyanide is thought to be noncompetitive. Therefore, oxygen has only antidotal efficacy in human cyanide poisoning through uncertain mechanisms. Patients probably should be treated with at least 100% oxygen. Humidified oxygen may be beneficial to victims of CK inhalation who are experiencing airway irritation or those with significant signs of cyanide toxicity. In addition, inhaled beta2 agonists may be used to treat bronchospasm resulting from the irritant effects of CK on the respiratory tract.

Hyperbaric oxygen (HBO) use may be considered for patients with cyanide toxicity that is refractory to other antidotes, especially in the setting of concomitant carbon monoxide poisoning. However, its use in pure cyanide poisoning is controversial, since no human studies have been performed to date, although the animal data are intriguing. Ivanov showed in 1959 that HBO restored normal activity of the brain in mice poisoned with cyanide. In 1966, Skene demonstrated a drop in mortality from 96% to 20% in a group of mice treated with HBO at 2 atmosphere absolute (ATA) compared to those treated at 1 ATA. Finally, Takano showed in 1980 that HBO at 2 ATA reduced the pyridine nuclide fluorescence (which

represents the degree of blockage of the respiratory chain) in the renal cortices of rabbits poisoned with cyanide.

Antidotes

Either directly counteract cyanide's toxicity on the electron transport chain or help the body eliminate the cyanide molecule.

| | |
|-------------------|---|
| Drug Name | Amyl nitrite (Isoamyl Nitrate)- Ampoules can be crushed into gauze and inhaled or broken into an Ambu bag and ventilated into the patient; only a temporary measure until IV access is obtained. |
| Adult Dose | 1 amp (0.2 mL) for 30-60 sec along with 100% oxygen until IV access is obtained |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity; severe anemia; closed-angle glaucoma; head trauma; postural hypertension and hypotension; cerebral hemorrhage |
| Interactions | Coadministration with alcohol may cause severe hypotension and cardiovascular collapse; with calcium channel blockers, may produce symptomatic orthostatic hypotension; aspirin may increase nitrate serum concentrations |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Can cause severe methemoglobinemia in overdose or in those with G-6-PD deficiency; in rare instances has caused hemolytic anemia |

| | |
|-------------------|---|
| Drug Name | Sodium nitrite- DOC if IV access is available. Creates methemoglobinemia more effectively than amyl nitrite. This dose assumes hemoglobin level of 12 mg/dL; dosage adjustment necessary in patients with anemia. Half original dose may be repeated in 1 h if patient continues to exhibit signs of cyanide toxicity. |
| Adult Dose | 300 mg (10 mL 3% sol) IV over 5-20 min; slow infusion if patient develops hypotension |
| Pediatric Dose | 0.33 mL/kg of 10% solution IV over 5-20 min, not to exceed 300 mg |
| Contraindications | Documented hypersensitivity; severe carbon monoxide poisoning |
| Interactions | May potentiate hypotensive effects of other medications |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Can cause severe methemoglobinemia in overdose or in those with G-6-PD deficiency; in rare instances has caused hemolytic anemia |

| | |
|-------------------|--|
| Drug Name | Sodium thiosulfate (Tinver)- Acts as donor of sulfane sulfur, which is used as a substrate by rhodanese and other sulfurtransferases for conversion of cyanide to thiocyanate. DOC for treating cyanide toxicity with concomitant carbon monoxide poisoning. |
| Adult Dose | 12.5 g (50 mL) IV delivered over 10 min; repeat at half initial dose in 1 h if symptoms persist |
| Pediatric Dose | 1.65 mL/kg of 25% solution over 10 min, not to exceed 12.5 g; repeat in 1 h at half initial dose if symptoms persist |
| Contraindications | Documented hypersensitivity |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Rapid IV infusion may cause transient hypotension and ECG changes |

| | |
|-------------------|--|
| Drug Name | Hydroxocobalamin/vitamin B-12a (Crystamine, Cyomin)- Combines with cyanide to form nontoxic cyanocobalamin. Large doses are required for antidotal efficacy, and it is available in the US only in very dilute formulations. |
| Adult Dose | 4 g IV over 30 min, not to exceed 10 g; may be administered more rapidly in cardiac arrest |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity; cobalt allergy |
| Interactions | Orange-red discoloration of the skin, mucous membranes, and urine is seen |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | None reported for this indication |

Anticonvulsants

Cyanide inhibits brain glutamate decarboxylase, which causes a decrease in the inhibitory neurotransmitter GABA and contributes to convulsions. Drugs such as benzodiazepines or barbiturates, which act at the GABA receptor complex, therefore can help control seizures.

| | |
|-------------------|--|
| Drug Name | Lorazepam (Ativan)- First-line drug in controlling seizures related to cyanide toxicity. |
| Adult Dose | 4 mg IV/IM over 2-5 min; repeat prn |
| Pediatric Dose | 0.1 mg/kg IV/IM over 2-5 min; repeat prn |
| Contraindications | Documented hypersensitivity; hypotension; respiratory depression |

| | |
|--------------|---|
| Interactions | May exacerbate hypotension caused by nitrates |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | May cause respiratory depression or hypotension |

| | |
|-------------------|--|
| Drug Name | Phenobarbital (Barbital, Solfoton, Luminal)- Second-line agent for seizures refractory to benzodiazepines. |
| Adult Dose | 15-20 mg/kg IV over 20 min |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity; hypotension; respiratory depression |
| Interactions | May exacerbate hypotension caused by nitrates |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | May cause hypotension and respiratory suppression |

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Follow-up

Further Inpatient Care

- Patients with symptoms beyond minor upper airway irritation and those with abnormal blood gases require admission to the hospital for continued monitoring and support.
 - Perform continuous cardiac monitoring.
 - Optimize oxygenation.
 - Monitor serum lactate levels and arterial and venous blood gases.
 - Monitor for delayed onset of pulmonary edema in those presenting with evidence of respiratory irritation.

Further Outpatient Care

- Reevaluate patients for neurologic sequelae 7-10 days after discharge from the hospital.

Transfer

- Should patients require transfer to a facility with the appropriate level of care, hemodynamically stabilize them prior to transfer. Transfer with an advanced cardiac life support (ACLS) level of service under continuous cardiac monitoring with supplemental oxygen and intravenous access.

Assure cyanide antidote availability prior to transfer.

Complications

- Parkinsonlike syndromes and other neuropsychiatric sequelae are described in survivors of severe intoxication.

Prognosis

- Prognosis is better in patients with low-level exposures whose symptoms resolve after they are removed from exposure.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to consider cyanide toxicity in appropriate circumstances or treatment with oxygen alone
-

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CBRNE - Cyanides, Hydrogen

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Synonyms, Key Words, and Related Terms

AC, hydrocyanic acid, HCN

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Introduction

Background

Hydrogen cyanide (HCN; North Atlantic Treaty Organization [NATO] designation AC) is 1 of 2 cyanide chemical warfare agents. The other is cyanogen chloride (NATO designation CK). Cyanide first was used as a chemical weapon in World War I. The French used approximately 4000 tons without notable military success. This was most likely because of the high volatility of cyanide and the fact that the 1- to 2-lb munitions used could not deliver the amounts of chemical required for biological effects. Other alleged military employments of cyanide include Japanese attacks on China before and during World War II and Iraqi attacks on Kurds in the 1980s.

Cyanide is a rapidly lethal agent when used in enclosed spaces where high concentrations can be achieved easily. In addition, because of the extensive use of cyanide in industry in the US, this agent presents a credible threat for terrorist use. Emergency physicians also may encounter cyanide casualties resulting from industrial accidents. Specific industrial processes involving cyanide include fumigation; metal cleaning, reclaiming, and/or hardening; electroplating; or photoprocessing. Other potential sources of cyanide are fires involving plastics and/or synthetics, acrylic nail removers containing acetonitrile or propionitrile, or nitroprusside infusions. Numerous plants contain amygdalin, which can be hydrolyzed to AC following the ingestion of large quantities.

Pathophysiology

While liquid cyanide can be absorbed through the skin or eyes, the primary route of exposure is by inhalation or ingestion. Following absorption, cyanide is distributed rapidly to all organs and tissues in the body. Cyanide combines with ferric iron in cytochrome a₃ (a component of the cytochrome oxidase complex in mitochondria) and inhibits this enzyme. This prevents intracellular oxygen use and results in imbalance between ATP hydrolysis and production. Metabolic acidosis is a hallmark of cyanide toxicity. It develops as cells are forced to use anaerobic metabolism and accumulate hydrogen ions and lactate.

Frequency

- **In the US:** Emergency physicians are unlikely to encounter casualties from AC used as a weapon except in the setting of a terrorist attack. Typical cyanide exposures are the result of liberation of the chemical during house and/or industrial fires or accidents.

Mortality/Morbidity

The LCt₅₀ (concentration-time product capable of killing 50% of exposed group) for AC is 2500-5000 mg·min/m³. The lethal oral dose of AC and cyanide salts is estimated to be 50 mg and 100-200 mg, respectively. The LD₅₀ (dose capable of killing 50% of exposed group) for skin exposures is estimated at 100 mg/kg. Vapor exposures in high concentrations (at or above the LCt₅₀) typically can cause death in 6-8 minutes.

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Clinical

History

Key historic features for suspected AC casualties include the time and nature of exposure; route of

exposure; presence of smoke; odors and colors of gas; onset, severity, and time course of symptoms; effects on surroundings (dead animals, other human casualties); and evidence of exposure to other chemicals or co-ingestants. As many as 50% of patients exposed to cyanide may describe an odor of bitter almonds.

- Symptoms after high vapor exposure
 - Transient hyperpnea and hypertension 15 seconds after inhalation
 - Convulsions 15-30 seconds later
 - Respiratory arrest 2-3 minutes later
 - Bradycardia, hypotension, and cardiac arrest within 6-8 minutes of exposure
- Exposure to nitriles (acetonitrile and/or propionitrile) - May be associated with a significant delay in onset of symptoms

Physical

Physical findings are nonspecific and are similar to those of severe hypoxemia.

- Severe respiratory distress in an acyanotic patient suggests possible cyanide toxicity.
- When observed, "cherry-red" skin suggests concomitant carbon monoxide poisoning or high venous oxygen content from failure of tissues to extract oxygen. Arterialization of the venous blood also may be noted during phlebotomy or examination of the retinal veins.
- Bright red skin and absence of cyanosis seldom are described in patients with cyanide poisoning, most likely because of concomitant cardiovascular collapse.
- Patients may demonstrate diaphoresis with normal or dilated pupils.
- Initial hypertension and compensatory bradycardia are followed by hypotension and tachycardia.
- Terminal hypotension is accompanied by bradyarrhythmias prior to asystole.

Causes

Causes of cyanide casualties include deliberate use as a chemical warfare agent, industrial exposures, and toxic byproducts of fires.

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Differentials

CBRNE - Cyanides, Cyanogen Chloride

CBRNE - Nerve Agents, Binary: GB2, VX2

CBRNE - Nerve Agents, G-series: Tabun, Sarin, Soman

CBRNE - Nerve Agents, V-series: Ve, Vg, Vm, Vx

Shock, Septic
Toxicity, Carbon Monoxide
Toxicity, Ethylene Glycol
Toxicity, Hydrogen Sulfide
Toxicity, Isoniazid
Toxicity, Organophosphate and Carbamate

Other Problems to be Considered

Consider the diagnosis of cyanide poisoning in patients with rapid collapse or seizures accompanied by metabolic acidosis and decreased oxygen consumption. Other agents that may have similar features in toxicity include the following:

Methemoglobin-inducing agents

Carbon monoxide

Inert gases (simple asphyxiants)

Hydrogen sulfide

Azides

Arsine

Phosphine

Monomethylhydrazine

Isoniazid

Water hemlock

Strychnine

Organophosphates

Metformin

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Workup

Lab Studies

- Arterial and venous blood gases: Cyanide toxicity is characterized by a normal arterial PO₂ and an abnormally high venous PO₂ (decreased arteriovenous oxygen difference [A-VO₂]).
- Serum chemistries and lactate level: Cyanide poisoning classically is associated with high anion gap metabolic acidosis and elevated lactate level.
- Cyanide levels
 - Cyanide levels generally are not available in time to guide acute treatment but may be confirmatory. The preferred test is an RBC cyanide level.
 - With this method, mild toxicity is observed at concentrations of 0.5-1.0 mcg/mL. Concentrations of 2.5 mcg/mL and higher are associated with coma, seizures, and death.
- Methemoglobin level: Obtain methemoglobin level, especially in cyanotic patients. Also, following the treatment of cyanide poisoning with sodium nitrite, methemoglobin levels are useful (see [Treatment](#)).

Other Tests

- Shortening of the ST segment with eventual fusion of the T wave into the QRS complex has been observed.

Procedures

- Endotracheal intubation is indicated for treatment of apneic cyanide casualties.

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Treatment

Prehospital Care

- Appropriate prehospital measures include the following:
 - Rescue from the cyanide source (assuming rescuers have the highest level of respiratory protection [Level A])
 - Removal of contaminated clothing and decontamination of the skin as required with soap

and water

- Administration of high-flow oxygen, airway management, and ventilatory support as required
- Establishment of intravenous access
- Continuous cardiac monitoring
- Advanced cardiac life support (ACLS) measures as indicated for dysrhythmias

Emergency Department Care

- Continue hemodynamic support and monitoring, oxygenation, ventilatory support, and seizure control in the ED.
- Administer cyanide antidotes (sodium nitrite and sodium thiosulfate) as soon as possible, taking care not to create toxic methemoglobinemia. Do not delay treatment for confirmatory RBC cyanide levels.
- Consider gastric lavage followed by the administration of activated charcoal in recent ingestions. The gastric aspirate may cause secondary contamination and should be viewed as hazardous.

Consultations

In any suspected terrorist attack, contact local law enforcement authorities and the Federal Bureau of Investigation. Consultation with a medical toxicologist and/or poison control center and intensivist may be useful.

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Medication

The Pasadena (formerly Lilly) Cyanide Antidote Kit contains amyl nitrite, sodium nitrite, and sodium thiosulfate. Theoretically, the nitrite components oxidize iron contained in hemoglobin to methemoglobin. This creates an additional site for cyanide binding and promotes dissociation from cytochrome oxidase. Resultant cyanomethemoglobin then may be converted to less toxic thiocyanate through enzymes such as rhodanese or other sulfurtransferases in the presence of sodium thiosulfate. Only use amyl nitrite perles as a temporizing measure if IV access has not been established, since administration of IV sodium nitrite is more effective in creating therapeutic methemoglobin levels. In the setting of concomitant carbon monoxide poisoning, do not use sodium nitrate or use only with extreme caution. However, in cases of smoke inhalation where cyanide toxicity is suspected, administration of sodium thiosulfate is safe.

Unlike carbon monoxide, inhibition of cytochrome oxidase by cyanide is noncompetitive. Therefore, oxygen has antidotal efficacy in cyanide poisoning through uncertain mechanisms. Treat patients with

100% oxygen. Hyperbaric oxygen use may be considered for patients with cyanide poisoning refractory to other antidotes; it is especially effective when concomitant carbon monoxide toxicity exists. However, its use in pure cyanide poisoning is controversial.

Antidotes

Administration of antidotes, which counteract the toxic effects of cyanide, is critical for life-threatening intoxication.

| | |
|-------------------|---|
| Drug Name | Amyl nitrite (Isoamyl Nitrate)- Ampules can be crushed and inhaled by a spontaneously breathing patient or ventilated into an apneic patient using a bag valve mask device; temporizing measure until IV access can be established. |
| Adult Dose | 1 ampule (0.2 mL) for 30-60 s administered by inhalation along with 100% oxygen |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity; cerebral hemorrhage |
| Interactions | Coadministration with alcohol may cause severe hypotension and cardiovascular collapse; with calcium channel blockers, may produce symptomatic orthostatic hypotension; aspirin may increase nitrate serum concentrations |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Can cause severe methemoglobinemia in overdose or in those with G-6-PD deficiency; rarely may cause hemolytic anemia |

| | |
|-------------------|--|
| Drug Name | Sodium nitrite- Drug of choice once IV access is established; creates methemoglobinemia more effectively than amyl nitrite. |
| Adult Dose | 300 mg (10 mL of 3% solution) IV over 5-20 min; slow rate of infusion if hypotension develops; this dose assumes hemoglobin level of 12 mg/dL (dosage adjustment necessary in patients with anemia); may repeat up to one-half initial dose in 1 h if required |
| Pediatric Dose | 0.33 mL/kg of 3% solution IV over 5-20 min, not to exceed 300 mg; this dose assumes hemoglobin level of 12 mg/dL (dosage adjustment necessary in patients with anemia) |
| Contraindications | Documented hypersensitivity; concomitant severe carbon monoxide poisoning |
| Interactions | Coadministration with channel blockers may increase symptomatic orthostatic hypotension (adjust dose of either agent) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Excessive methemoglobinemia, hemolysis, or hypotension may occur, especially in patients with G-6-PD deficiency |

| | |
|-------------------|--|
| Drug Name | Sodium thiosulfate (Tinver)- Acts as donor of sulfur, which is used as substrate by rhodanese and other sulfurtransferases for detoxification of cyanide to thiocyanate; DOC for treating cyanide toxicity with concomitant carbon monoxide poisoning. |
| Adult Dose | 12.5 g (50 mL) IV over 10 min; may repeat up to one-half dose at 1 h for patients with persistent symptoms |
| Pediatric Dose | 1.65 mL/kg of 25% solution over 10 min, not to exceed 12.5 g; may repeat up to one-half dose at 1 h for patients with persistent symptoms |
| Contraindications | Documented hypersensitivity |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in asthma; rapid IV infusion may cause transient hypotension and ECG changes |

| | |
|-------------------|--|
| Drug Name | Hydroxocobalamin (vitamin B-12)- Complexes with cyanide to form nontoxic cyanocobalamin (vitamin B-12); disadvantages are large dose required for antidotal efficacy and availability in US only in very dilute solutions. |
| Adult Dose | 4 g IV over 30 min, not to exceed 10 g; may be given more rapidly in cardiac arrest |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | None reported for this indication |

| | |
|-------------------|--|
| Drug Name | Activated charcoal (Insta-Aqua, Actidose-Aqua, Liqui-Char)- Emergency treatment in poisoning caused by drugs and chemicals. Network of pores present in activated charcoal adsorbs 100-1000 mg of drug per g of charcoal. Does not dissolve in water. For maximum effect, administer within 30 min after ingesting poison. |
| Adult Dose | 25-100 g, 1 g/kg, or 10 times the weight of ingested poison; give as susp in 4-8 oz water |
| Pediatric Dose | <1 year: Not recommended >1 year: Administer as in adults |
| Contraindications | Documented hypersensitivity; poisoning or overdosage of mineral acids and alkalis |
| Interactions | May inactivate ipecac syrup if used concomitantly; effectiveness of other medications decreases with coadministration; do not mix with sherbet, milk, or ice cream (decreases absorptive properties of activated charcoal) |

| | |
|-------------|--|
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Not very effective in poisonings of ethanol, methanol, and iron salts; induce emesis before giving activated charcoal; after emesis with ipecac syrup, patient may not tolerate activated charcoal for 1-2 h; can administer in early stages of gastric lavage; without sorbitol, gastric lavage returns are black |

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Follow-up

Further Inpatient Care

- Admit patients who present with more than minimal symptoms that resolve without treatment for observation and supportive care. Also, a 24-hour observation period is necessary for those exposed to nitriles, since delayed onset of toxicity is expected.
- Optimize oxygenation and provide continuous cardiac monitoring.
- Monitor serum lactate levels, chemistries, and arterial and/or venous blood gases.

Further Outpatient Care

- Reevaluate patients for neurologic sequelae 7-10 days after discharge from the hospital.

in/Out Patient Meds

- No additional medications beyond initial antidotes are indicated. Continue to administer oxygen as required.

Transfer

- If a patient requires transfer to a higher level medical facility, the transferring physician should ensure availability of an ACLS emergency medical service unit that can provide continuous cardiac and hemodynamic monitoring and oxygen therapy as well as the availability of cyanide antidotes.

Complications

- Parkinsonian symptoms and other neuropsychiatric sequelae are described in survivors of severe cyanide poisoning. The incidence is unknown, but patients should be given close neurologic

follow-up care.

Prognosis

- Prognosis is good for patients who have only minor symptoms that do not require administration of antidotes.
- Prognosis is poor once cardiovascular collapse occurs in severe cyanide poisoning. These victims are considered expectant in a mass casualty setting.
- Prognosis is fair for patients with seizures or recent-onset apnea if antidotes can be administered rapidly. These victims are considered immediate in a mass casualty setting.

Patient Education

- Educate patients about potential neurologic sequelae and the importance of follow-up evaluation.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to consider diagnosis of cyanide toxicity, thereby delaying administration of antidotes
 - Failure to recognize concomitant carbon monoxide poisoning
 - Failure to admit a patient exposed to nitriles (acetonitrile or propionitrile)
-

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CBRNE - Evaluation of a Biological Warfare Victim

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Introduction

"Rather than invading our beaches or launching bombers, adversaries may â€”deploy compact and relatively cheap weapons of mass destructionâ€”not just nuclear, but also chemical or biological, to use disease as a weapon of war." President William J. Clinton, May 22, 1998

A bioterrorism incident will happen quietly with no explosion and no forewarning. In one possible situation, the emergency physician on shift slowly becomes aware of an unusually large number of patients in the ED with nonspecific complaints. At the end of his or her shift, hours after discharging the first young man with those vague symptoms, the patient returns acutely short of breath, cyanotic, and hypotensive.

Biological weapons (BW) are the terroristâ€™s perfect weapon; they are relatively easy to make, difficult to detect, and a significant threat of morbidity and mortality. A terrorist needs to go no further

than an Internet connection to download the appropriate "recipe" with easily obtainable ingredients.

It is sobering. The question no longer is "What if?" Rather, the question is "When?" As emergency medicine physicians providing acute health care for the nation, the next question, without hesitation, should be "What are we going to do about it?"

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Primary Routes of Exposure of Biological Agents

Potentially more lethal than chemical agents, because they are obtained from nature, more accessible, and easy to produce, biological agents (bacteria, viruses, bacterial toxins) pose a significant threat as weapons of mass destruction.

BWs are devices composed of 4 major components as follows: payload (biological agent), munition (container that keeps the payload intact and virulent during delivery), delivery system (eg, missile, artillery shell, aircraft), and a dispersal mechanism. The two most likely potential methods of dissemination are a line source and a point source.

A line source technique is the most effective dispersal means for biological agents. For example, this may involve a truck or air sprayer moving perpendicular to the wind during an inversion (when air temperature increases with altitude and holds surface air and pollutants down). Inversions normally occur at dawn, dusk, or night.

The point source technique uses small bomblets deployed in a saturation mode. The saturation technique overcomes the meteorologic requirements for line source dissemination. Agents may be introduced into buildingsâ€™ heating-ventilation-air conditioning systems or via food or water contamination. Small packages or envelopes may contain biological agents, but unless they also contain a dispersal device, they are not likely to pose an inhalational threat.

Regardless of the type of dispersal method used, victims are contaminated via 3 potential routes: skin, gastrointestinal (GI), and pulmonary. Cutaneous exposure provides the least potential for significant morbidity and mortality, since intact skin provides an excellent barrier against most of these agents except mycotoxins. However, mucous membranes, abrasions, or other lesions may provide a portal of entry for bacteria, viruses, or toxins.

Contamination of food or water supplies allows for a potentially significant GI exposure. This type of exposure is limited by the direct effects of water dilution and treatment, which inactivates or significantly weakens most microbes and toxins. For this to be a viable method for contamination, the agent must be introduced near the end user and is less likely to result in mass casualties. Exposure via the inhalational

route is the most effective mode of delivery for BW agents. Aerosol clouds with droplet or particle diameters of 1-5 µm containing microbes or toxins are not detectable by the senses. Although limited by environmental constraints (wind, sunlight, temperature, desiccation), the potential impact of widespread illness and death may be catastrophic.

Potential Agents and Routes of Primary Exposure*

| Agent | Biological Weapon | Route |
|------------------------------------|------------------------------------|--------------------------------|
| Bacteria | Plague | Percutaneous (flea), pulmonary |
| Anthrax | Percutaneous, GI, pulmonary | |
| Tularemia | Percutaneous, GI, pulmonary | |
| Q fever | Pulmonary | |
| Viruses | Smallpox | Pulmonary |
| Viral equine encephalitis (VEE) | Percutaneous (mosquito), pulmonary | |
| Viral hemorrhagic fevers (VHF) | Mucous membranes, pulmonary | |
| Toxins | Botulinum | GI, pulmonary |
| Ricin | Percutaneous, GI, pulmonary | |
| Staphylococcal enterotoxin B (SEB) | GI, pulmonary | |

* Specific and additional agents are discussed at length in other articles.

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Epidemiologic Clues

As described in the introductory scenario, the symptoms that develop after a biological attack are likely to be nonspecific initially, making diagnosis difficult. Early diagnosis is key, and emergency medicine physicians should attempt to recognize a number of potential clues that may suggest release of a BW. Rapid realization of a bioterrorist attack affords greater potential for prophylaxis, appropriate treatment, and prevention of secondary spread.

Suspect a BW attack if any of the following are present:

- Large epidemic with unprecedented number of ill or dying
- Immunocompromised individuals demonstrating first susceptibility and rapid progression of disease (although equal affliction of previously healthy individuals also may be a clue)
- Particularly high volumes of patients complaining primarily of similar symptoms that are associated with an escalating mortality rate
- Unusual or impossible vector for transmission for that particular region
- Multiple simultaneous outbreaks
- Epidemic caused by a multidrug-resistant pathogen
- Reports of sick or dying animals or plants
- Single case of disease by an uncommon agent (smallpox, inhalational anthrax, some VHF's)

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Containment and Decontamination

Unlike a chemical terrorism event, where a hazardous materials (HAZMAT) team and local first responders may have the opportunity to decontaminate a significant number of victims prior to their arrival at the hospital, a biological event may be unannounced. As such, the first indication of a release may be the presentation of multiple ill patients to the ED. Decontamination of patients to avoid contaminating unexposed patients and staff only is considered immediately after an announced release of a biological agent and is carried out adequately with soap and water. In the more likely scenario in which the release is unannounced and patients present already ill, the emphasis is not decontamination but rather respiratory isolation of the patient with employment of standard precautions until the agent is known.

To render agents completely harmless on instruments, dry heat requires 2 hours of treatment at 160°C. Using steam at 121°C and 1 atm of pressure can reduce the time to 20 minutes (autoclaving).

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Triage

When a biological attack is suspected, immediately employ necessary steps to ensure the safety of hospital personnel, existing patients, and the facility, thereby maximizing the ability to care for victims (see [CBRNE - Biological Warfare Mass Casualty Management](#)).

One relatively simple triage method relies on a formal color-coded tagging system. For example, black stands for expectant, red for immediate, yellow for delayed, and green for healthy. When the need to triage multiple casualties in a rapid manner presents itself, simplicity is crucial. Additionally, the importance of practicing a triage method to ensure staff familiarity cannot be overstated.

A mass casualty incident (MCI) requires constant retriage of patients and resources. Effective response to an MCI requires that staffing and equipment supplies balance patient care demands, a potentially difficult equilibrium to establish. Nonetheless, this provides the opportunity to maximize the greatest good for the greatest number.

Many difficult decisions must be made under extremely stressful conditions, and using a simple system for evaluation and classification eases the burden of dealing with a BW event. Different triage systems exist and vary by local protocol. The ED physician must become familiar with his or her role within the local system, since the ED likely will serve as the center for medical care for the community during the early hours or days following a biological incident.

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Security

Although not traditionally viewed as a medical priority, securing hospital staff and resources is paramount to the successful delivery of medical care during an MCI. The ED staff, patients, and facility potentially are placed at risk by contaminated casualties seeking medical attention or family members seeking information. In addition, the ED may become a terrorist target through secondary devices, the deployment of which would halt medical care. Hospital entrances should be locked and guarded to prevent the public and injured from entering the facility unnoticed.

Hospital security must be trained to handle the initial moments of an MCI prior to the arrival of the local authorities, whose delay likely is based on the nature of the event. Hospital security personnel have direct exposure to the public. This potential risk mandates that they are trained adequately regarding personal protective equipment and have an understanding of medical triage dynamics. Additionally, they may require training in the areas of public reassurance and guidance; however, a predesignated information officer is essential.

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Prophylaxis

Depending on the agent, prophylactic antibiotics and vaccines may be available for treatment of victims of a bioterrorism incident. Early notification of appropriate authorities (eg, Federal Bureau of Investigations [FBI], local health department, emergency management) and mobilization of resources (first responders, hospitals, health care providers, poison centers, police, fire, media) serve to minimize the number of casualties.

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Appropriate Resources/Conclusion

- [The Centers for Disease Control and Prevention \(CDC\)](#): CDC capabilities are epidemiologic surveillance, biological agent identification, and public health consultation and response.
- [US Army Medical Research Institute of Infectious Diseases \(USAMRIID\)](#): As the Department of Defense's lead laboratory for medical aspects of biological warfare defense, USAMRIID conducts research to develop vaccines, drugs, and diagnostics for laboratory and field use. In addition to developing medical countermeasures, USAMRIID formulates strategies, information, procedures, and training programs for medical defense against biological threats. This website also provides numerous links to other sites involved in biological warfare defense.
- [Federal Emergency Management Agency \(FEMA\)](#)
- [Department of Health and Human Services \(DHHS\)](#) and [Office of Emergency Preparedness \(OEP\)](#)
- [Federal Bureau of Investigations \(FBI\)](#)
- [National Guard Bureau](#)
- [NBC Medical Defense](#)

Although the likelihood of involvement in a bioterrorist incident is low for any given emergency physician, the consequences of unpreparedness are potentially catastrophic. Therefore, it is important that each physician understand his or her role within the hospital and community bioterrorist response plans. The greatest potential to save lives rests on the early recognition of the incident, accurate clinical diagnoses, and the expedient mobilization of appropriate hospital, local, and federal resources.

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CBRNE - Evaluation of a Chemical Warfare Victim

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Synonyms, Key Words, and Related Terms

chemical warfare

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Introduction

The US government has determined that the likelihood of a terrorist attack within the US is increasing. This has prompted governmental domestic preparedness training initiatives targeted at physicians and other healthcare providers in the areas of nuclear, biological, and chemical warfare. This article focuses on the initial approach to the chemical warfare victim. Detailed information on patient decontamination and the appropriate use of personal protective equipment is described elsewhere on this website (see

[CBRNE - Chemical Decontamination](#) for more information).

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Triage of Chemical Casualties

Patient triage only should be carried out in the hospital setting by those wearing appropriate personal protective equipment. In the simplest form of triage, patients or casualties are separated into 3 groups. The first group consists of those for whom the degree of medical care required exceeds that which is available. In this setting, medical assets are insufficient relative to the presenting illness, or an experienced triage officer deems the patient's injuries irreversible. The medical focus is on comfort care, and the casualty is triaged as expectant. A casualty's classification may change as assets become available or when later reevaluation demonstrates that the casualty's condition was not as serious as first anticipated.

The second group consists of casualties who require immediate intervention for survival. In a conventional situation (ie, noncontaminated environment), these casualties usually have treatable injuries affecting the airway, breathing, or circulation (ABCs). Other examples of immediate interventions include administration of antidotes or spot decontamination.

The third group consists of casualties who have injuries that place them in no immediate danger of loss of life. Medical care is needed but not immediately. For example, casualties in this group may include those with minor injuries who merely require laceration repair or those with extensive injuries necessitating long-term hospitalization who are presently stable.

The triage system commonly used by US military medical departments and by civilian medical systems contains the above categories (immediate, delayed, and expectant) and incorporates a fourth category (minimal). In this scheme, minimal injuries are those that require a quick intervention, do not require a physician or evacuation, and result in a rapid return to duty. Occasionally, as in the North Atlantic Treaty Organization (NATO) *Emergency War Surgery Handbook*, a fifth category, urgent, is added to denote a casualty who requires intervention within minutes to survive. Also, in some schemes, the term "chemical immediate" is used for casualties who require immediate administration of antidotes for survival (as in nerve agent or cyanide poisoning).

Immediate support measures

Monitor patients who potentially are exposed to chemical warfare agents for changes in their clinical status and provide appropriate supportive measures. First address the patient's ABCs and coexisting life-threatening or limb-threatening injuries. Reassess these injuries throughout the patient's course.

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Categories for Triage of Chemical Casualties

Immediate

Nerve agents

A casualty of nerve agents who is in severe distress is classified as immediate. The patient may be unconscious, may be in severe respiratory distress, or may have become apneic minutes before reaching the facility. He or she may be convulsing or immediately postictal. Often the contents of 3 MARK I kits (or more) plus diazepam and possibly short-term ventilatory assistance is all that is required to save a life. In addition, classify a casualty with signs in 2 or more systems (eg, neuromuscular, gastrointestinal [GI], respiratory excluding eyes and nose) as immediate and administer the contents of 3 MARK I kits and diazepam.

Cyanide

A casualty of cyanide who is convulsing or who became apneic minutes before reaching the medical facility and has adequate circulation (still has a pulse) is in the immediate group, assuming that cyanide antidotes are available. If the ABCs remain adequate, the administration of antidote may be all that is required for complete recovery. Since death usually occurs within 4-5 minutes of exposure to a lethal amount of cyanide unless treatment is immediate, this type of casualty is unlikely to be seen at the medical facility.

Phosgene and vesicants

Place casualties of phosgene or vesicant agents who have moderate or severe respiratory distress in the immediate group, where intense ventilatory and other respiratory support measures are immediately available.

Incapacitating agents

Those with cardiovascular collapse or severe hyperthermia are placed in the immediate category following exposure to agents such as 3-quinuclidinyl benzilate (BZ) or Agent 15.

Delayed

Nerve agents

Place casualties who require hospitalization but have no immediate threat to life in the delayed group. This generally is limited to a casualty who has survived a severe nerve agent exposure, is regaining consciousness, and has resumed spontaneous respiration. This casualty requires further medical care for the time necessary for recovery.

Vesicants

Triage those with skin injuries of greater than 5% total body surface area but less than 50% as delayed. Other criteria include severe eye involvement or pulmonary symptoms with an onset of more than 4 hours postexposure.

Cyanide

Triage victims of cyanide vapor exposure who are alive after 15 minutes as minimal or delayed.

Pulmonary agents

Following exposure, triage patients with delayed onset of respiratory distress (>4 h) as delayed.

Incapacitating agents

Severe or worsening signs after exposure warrant patient triage to the delayed category.

Minimal

Nerve agents

Patients who are walking and talking can be assessed for the degree of miosis and its potential to interfere with performance prior to return to duty.

Vesicants

Patients with small exposures (<5% total body surface area) or minor eye irritation can be triaged as minimal.

Table 1. Categories for Triage of Chemical Casualties

| Â | Immediate | Delayed | Minimal | Expectant |
|---|-----------|---------|---------|-----------|
| | | | | |

| | | | | |
|--------------------|---|--|---|---|
| <p>Nerve agent</p> | <ul style="list-style-type: none"> • Talking, not walking (severe distress with dyspnea, twitching, and/or nausea, vomiting); moderate-to-severe effects in 2 or more systems (eg, respiratory, GI, muscular); circulation intact • Not talking (unconscious), not walking; circulation intact • Not talking, not walking; circulation not intact (if treatment facilities are available; if not, classify as expectant) | <p>Recovering from severe exposure, antidotes, or both</p> | <p>Casualty walking and talking; capable of self-aid; imminent return to duty</p> | <p>Not talking; circulation failed (with adequate treatment resources, classify as immediate)</p> |
| <p>Cyanide</p> | <p>Severe distress (unconscious, convulsing, or postictal, with or without apnea) with circulation intact</p> | <p>Recovering; has survived more than 15 minutes after vapor exposure</p> | <p>Â</p> | <p>Circulation failed</p> |
| <p>Vesicant</p> | <p>Airway injury; classify as immediate if help obtainable (rare)</p> | <p>Skin injury on greater than 5% but less than 50% (liquid exposure) of body surface area; any body surface area burn (vapor exposure); most eye injuries; airway problems beginning more than 4 hours after exposure</p> | <p>Skin injury on less than 5% of body surface area in noncritical areas; minor eye injuries; minor upper airway injury</p> | <p>Greater than 50% body surface area skin injury from liquid; moderate-to-severe airway injury, particularly with early onset (<4 h after exposure)</p> |
| <p>Phosgene</p> | <p>Acute airway injury: classify as immediate if resources available</p> | <p>Onset of symptoms more than 4 hours postexposure</p> | <p>Â</p> | <p>Moderate-to-severe injury with early onset (<4 h, resource dependent)</p> |

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Chemical Warfare Agents

Nerve agents

Nerve agents are organophosphates and therefore cause the characteristic cholinergic toxidrome. Organophosphates inhibit acetylcholinesterase, which results in an excess accumulation of acetylcholine at muscarinic and nicotinic receptors. Clinically, these agents cause increased bodily secretions (SLUDGE; salivation, lacrimation, urination, defecation, emesis), miosis, bronchospasm, increased airway secretions, sudden loss of consciousness, seizures, apnea, muscle fasciculations that progress to flaccid paralysis, and death. The primary cause of death in patients exposed to nerve agents is respiratory failure caused by respiratory muscle weakness, bronchoconstriction, and increased respiratory secretions. Maintain a high index of suspicion of organophosphate and/or nerve agent poisoning whenever presented with a patient who has pinpoint pupils with seizures, muscle fasciculations, or flaccid paralysis.

Depending on the patient's clinical status, treatment with atropine and pralidoxime may be feasible. Atropine dries secretions and relaxes smooth muscle via antagonism of acetylcholine at its muscarinic receptors. Clinically, this results in decreased SLUDGE and improved ventilation by decreasing bronchoconstriction and bronchorrhea. Pralidoxime improves the patient's muscle strength by removing the nerve agent from acetylcholinesterase. The acetylcholinesterase, when freed from the nerve agent, then resumes breakdown of acetylcholine. Pralidoxime's effectiveness decreases with time, because the enzyme/nerve agent complex can age and form an irreversible bond. The time course for this depends on the specific agent and can vary from minutes to hours.

Exposure to nerve agents occurs from either vapor or liquid forms. A patient's vapor or liquid exposure can be classified as mild, moderate, and severe based on clinical criteria.

Vapor exposure

Mild vapor exposure results in miosis and rhinorrhea. Patients also may develop eye pain, which can be treated with topical homatropine. Patients with only a mild vapor exposure do not require treatment with atropine or pralidoxime. Observe patients with mild vapor exposure and those who are asymptomatic but claim to have been exposed to a nerve agent vapor for 1 hour for development of muscle weakness, respiratory symptoms, or GI symptoms. If these symptoms develop, classify the patient as moderate; he or she requires treatment with atropine and pralidoxime. Severe vapor exposure results in loss of consciousness, seizures, flaccid paralysis, and apnea.

Liquid exposure

Mild liquid nerve agent exposure results in localized fasciculations and sweating. A mild liquid nerve agent exposure requires 1 dose of atropine and pralidoxime. Patients with mild liquid exposure, those who are asymptomatic but claim to have been exposed to a liquid nerve agent, and those with an unknown form of exposure require an observation period of 18 hours. If patients develop respiratory or GI symptoms, classify them as moderate. Loss of consciousness, seizures, flaccid paralysis, and apnea are manifestations of a severe exposure.

Antidotes

Treatment of nerve agent exposure is covered in detail elsewhere (see [CBRNE - Nerve Agents, G-series: Tabun, Sarin, Soman](#), [CBRNE - Nerve Agents, Binary: GB2, VX2](#), [CBRNE - Nerve Agents, V-series: Ve, Vg, Vm, Vx](#)).

Cyanide

Levels of exposure

Cyanide binds to ferric state iron found in mitochondria, which prevents the mitochondria from using oxygen. Because of the subsequent anaerobic metabolism, patients develop lactate accumulation with severe acidosis and cellular asphyxiation.

Cyanide can be found in vapor, liquid, and solid salt forms. Cyanide toxicity from chemical warfare typically occurs by inhalation of vapor, but it also may occur by ingestion or dermal contact with a large amount of the liquid form.

Cyanide poisoning at low concentrations produces anxiety, hyperventilation, headache, dizziness, and vomiting. Patients also may develop a cherry red skin discoloration, although this is rare. Since the body normally metabolizes cyanide into thiocyanate, which then is excreted in the urine, patients with mild exposures should improve when removed from the source of exposure and may not require any other intervention.

Cyanide poisoning at higher concentrations begins to produce respiratory distress. Suspect significant cyanide poisoning whenever confronted with a patient who is experiencing respiratory distress with normal oxygen concentration of the blood. This occurs because cyanide prevents the body from using oxygen. Toxicity screens for cyanide are not timely enough to be useful in patients with toxicity. A high venous oxygen level and lactic acidosis may help confirm suspicion of cyanide poisoning. Patients with this intermediate level of exposure may require treatment with amyl nitrite, sodium nitrite, and sodium thiosulfate.

Cyanide exposure at even higher concentrations can produce dramatic results. Patients can progress

through anxiety and hyperpnea to seizures within 30 seconds. Within 3-5 minutes of exposure, patients may stop breathing. Finally, patients may experience asystole and death within 6-10 minutes.

Treatment

Treatment of cyanide involves removing cyanide from the cells, detoxifying cyanide, and providing supportive measures aimed at minimizing toxic effects. Treatment details are discussed elsewhere (see [CBRNE - Cyanides, Cyanogen Chloride](#)).

Vesicants

Vesicants are chemical agents that cause blistering of the skin. Lewisite and sulfur mustard are the 2 main agents used in chemical warfare. Exposure to either agent can come from a vapor or liquid form. Both agents produce irritation and damage to the eyes, skin, and airway. Chemical decontamination within 30 minutes of exposure is of primary importance, as that is when most of the injury to the patient occurs. Decontamination after that point still is required to prevent cross-contamination of healthcare providers.

Effects of exposure

- **Eyes:** Eye exposure can produce mild conjunctivitis up to corneal perforation depending on the level of exposure. Treat eye exposures beyond decontamination in conjunction with ophthalmology.
- **Skin:** Skin exposures result in vesicles, which coalesce into bullae. Bullae require surgical irrigation, debridement, and a topical antibiotic.
- **Respiratory:** Airway injury initially damages the upper airway, resulting in epistaxis, pharyngitis, laryngitis, and bronchitis. With a high enough level of exposure, damage may progress to the lower airway. Damage to the lower respiratory tract produces dyspnea from bronchiolitis, which can progress to respiratory failure and death. Treatment of respiratory symptoms is supportive, with use of bronchodilators, oxygen, and mechanical ventilation if necessary.
- **Mustard** also can produce bone marrow suppression beginning 3-5 days after exposure, which can contribute to a delayed cause of death. Time to onset of symptoms from exposure to lewisite is immediate. Conversely, mustard exposure does not produce symptoms until 2-48 hours after exposure. For lewisite exposures, a specific antidote of dimercaprol (British antilewisite agent) can be administered in a dose of 2.5-5 mg/kg IM.

Pulmonary Intoxicants

Pulmonary intoxicants cause lung injury via inhalation of a vapor. Chemical warfare agents in this group

include phosgene and chlorine. Phosgene exposure causes a mild transient irritation of the eyes, sinuses, pharynx, and bronchi upon initial exposure. Patients then are asymptomatic until 2-4 hours postexposure, when they begin to develop noncardiogenic pulmonary edema, which should be treated with supplemental oxygen and mechanical ventilation as needed. These patients also may develop hypovolemic shock from loss of fluid into the alveolar space and may require intravenous fluids for hypotension. Since the pulmonary edema is noncardiogenic and the patients are hypovolemic, do not use diuretics.

Chlorine exposure has a pronounced, immediate irritant effect on the upper and lower respiratory tract, which may produce respiratory failure shortly after exposure. If the patient survives the initial exposure, he or she may develop noncardiogenic pulmonary edema and hypovolemic shock within 12-24 hours, similar to that caused by phosgene.

Differential Diagnosis of Chemical Casualties

Chemical agents that may cause rapid onset of respiratory symptoms include nerve agents, cyanide, mustard, lewisite, and phosgene. Additionally, rapid onset of neurologic symptoms is observed following significant nerve agent or cyanide exposures. Treatment considerations in patients with rapid onset of respiratory or neurologic symptoms include the use of atropine, pralidoxime, and diazepam (typically by Autoinjector) followed by the administration of cyanide antidotes if the patient fails to respond to nerve agent antidotes.

Delayed onset of respiratory symptoms may be observed following mustard, lewisite, or phosgene exposure. Care in this setting is discussed in greater detail in other articles (see [CBRNE - Vesicants, Mustard: Hd, Hn1-3, H](#), [CBRNE - Vesicants, Organic Arsenicals: L, ED, MD, PD, HL](#), [CBRNE - Lung-Damaging Agents, Phosgene](#)).

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Summary

Triage of casualties of chemical agents is based on many of the same principles as the triage of conventional casualties. Members of the triage team try to provide immediate care to those who need it for survival; they temporarily set aside or delay treatment of those who have minor injuries or do not need immediate medical intervention; and they do not use limited medical assets on the hopelessly injured. Initial triage and treatment of the chemical-exposed patient pose unique medical challenges, since early care must be rendered within the constraints of personal protective clothing. In addition, decontamination, which may be a time-consuming process, must be carried out before the casualty receives more definitive care. Triage is a matter of judgment on the part of the triage team. Ideally, base this judgment upon knowledge of medical assets, the casualty load, and most importantly, an

understanding of the toxicology of the chemical agent involved and potential complications of exposure.

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CBRNE - Glanders and Melioidosis

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Synonyms, Key Words, and Related Terms

Whitmore disease

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Introduction

Background

Glanders and melioidosis are related diseases produced by bacteria of the *Burkholderia* species, which are gram-negative rods. They produce similar symptoms and have similar pathophysiologic consequences. Glanders and melioidosis are of interest because of significant study for potential weaponization by the US and other countries in the past. During World War I, glanders was believed to have been spread to infect large numbers of Russian horses and mules on the Eastern Front. The Japanese infected horses, civilians, and prisoners of war during World War II. The US studied this agent as a possible biological warfare (BW) weapon in 1943-1944 but did not weaponize it. The former Soviet Union is believed to have been interested in glanders as a potential BW weapon as well.

Burkholderia mallei is the causative agent of glanders, primarily a disease of animals such as horses, mules, and donkeys. Glanders has been only a rare and sporadic disease in humans, and no epidemics have been reported. In China during World War II, 30% of the tested horses were infected with glanders, but human cases were rare. The reason for the low transmission rate is not known. In the human population, it typically is found in those with close and frequent contact with infected animals, such as veterinarians, animal caretakers, abattoir workers, and laboratory personnel.

Melioidosis, also known as Whitmore disease, is caused by the bacterium *Burkholderia pseudomallei*. It is clinically similar to glanders disease, although the epidemiology differs. The bacteria thrive in tropical climates, and the disease is endemic in Southeast Asia. Both humans and other susceptible animals may contract the disease.

Both organisms are considered potential BW agents, especially in the aerosolized form.

Pathophysiology

Glanders is caused by *B mallei* (formerly *Pseudomonas mallei*). The bacteria only exist in infected susceptible hosts and are not found in water, soil, or plants. Once in the host, synthesis and release of certain toxins occur. Usually, the amount is insignificant, and no clinical disease process ensues. However, if a large quantity of the organism is incorporated, the amount of toxin is sufficient to cause specific symptoms. Antibiotics are of little use. The toxins include pyocyanin (blue-green pigment that interferes with the terminal electron transfer system), lecithinase (causes cell lysis by degrading lecithin in certain cell membranes), collagenase, lipase, and hemolysin.

Melioidosis is an infectious disease caused by *B pseudomallei* (formerly *Pseudomonas pseudomallei*). The organism is distributed widely in the soil and water of the tropics. It is spread to humans through direct contact with a contaminated source.

Glanders and melioidosis produce similar clinical syndromes.

Localized form

Bacteria enter the skin through a laceration or abrasion, and a local infection with ulceration develops.

Incubation period is 1-5 days. Swollen lymph glands may develop. Bacteria that enter the host through mucous membranes can cause increased mucus production in the affected areas.

Pulmonary form

When bacteria are aerosolized and enter the respiratory tract via inhalation or hematogenous spread, pulmonary infections may develop. Pneumonia, pulmonary abscesses, and pleural effusions can occur. The incubation period is 10-14 days. With inhalational melioidosis, cutaneous abscesses may develop and take months to appear.

Septicemia

When bacteria is disseminated in the bloodstream in glanders, it usually is fatal within 7-10 days. The septicemia that develops affects multiple systems, and cutaneous, hepatic, and splenic involvement may occur. With melioidosis, bacteremia is observed with chronically ill patients (eg, patients with HIV, patients with diabetes). They develop respiratory distress, headaches, fever, diarrhea, pus-filled lesions on the skin, and abscesses throughout the body. Septicemia may be overwhelming, with a 90% fatality rate and death occurring within 24-48 hours.

Chronic form

The chronic form involves multiple abscesses, which may affect the liver, spleen, skin, or muscles. This form also is known as "farcy" in glanders disease. Melioidosis, in addition to this chronic form, can become reactive many years after the primary infection.

Frequency

- **In the US:** Glanders was eliminated from US domesticated animals in the 1940s. One recent human case of glanders in a laboratory worker occurred in 2000. This is the first human case reported in the US since 1945. A few isolated cases of melioidosis have occurred in the US. Confirmed cases range from 0-5 each year and occur among travelers, immigrants, and intravenous (IV) drug users.

Mortality/Morbidity

- Glanders is primarily zoonotic. It is rare in humans, and no epidemics have been reported.
- Melioidosis is most widespread in Thailand, where in 1 hospital, it was responsible for 19% of community-acquired sepsis and 40% of deaths from community-acquired septicemia.

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Clinical

History

- Glanders is transmitted to humans through direct skin or mucous membrane contact with infected animal tissues. Cases of human-to-human transmission have been reported.
- Melioidosis is transmitted to humans through direct skin contact with contaminated soil or water. Ingestion of contaminated water and inhalation of dust contaminated with the organism are other mechanisms of transmission. Cases of human-to-human transmission are rare but have been documented.
- Generalized symptoms include fever, rigors, night sweats, myalgia, anorexia, and headache. Additional symptoms, which are based on the route of exposure, include chest pain, cough, photophobia, lacrimation, and diarrhea.

Physical

Physical findings may include fever, cervical adenopathy, papular or pustular skin lesions, hepatomegaly, or splenomegaly.

- Severe urticaria has been reported during primary melioidosis.
- During septicemia, flushing, cyanosis, and a disseminated pustular eruption can be seen. Pustules often are associated with regional lymphadenitis, cellulitis, or lymphangitis.
- Rarely, ecthyma gangrenosumlike lesions and cutaneous abscesses (that sometimes ulcerate) may develop.

Causes

- Human cases of glanders have occurred primarily in occupational settings and include laboratorians, veterinarians, and animal caretakers.
- Human cases of melioidosis have occurred from sexual contact and IV drug use. It has been observed in immigrants, military personnel, and travelers.

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Differentials

CBRNE - Anthrax Infection

CBRNE - Plague

CBRNE - Smallpox

Pneumonia, Bacterial

Pneumonia, Mycoplasma

Pneumonia, Viral

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Workup

Lab Studies

- Complete blood count: Complete blood count may reveal a mild leukocytosis with a left shift or leukopenia.
- Gram stain and culture of blood, sputum, urine, and skin lesions
 - Blood cultures usually are negative.
 - Gram stain and culture of sputum, urine, and skin lesions can be performed. Gram stain may reveal small gram-negative bacilli, which stain irregularly with methylene blue.
 - Meat nutrient agar or the addition of 1-5% glucose may accelerate growth of bacteria.
- Complement fixation tests
 - Complement fixation tests are more specific and are considered positive for glanders if the titer is equal to or greater than 1:20.
 - A 4-fold increase in the titer for melioidosis is considered positive.

Imaging Studies

- Chest radiography may demonstrate bilateral bronchopneumonia, miliary nodules, segmental or lobar infiltrates, and cavitating lesions.

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Treatment

Prehospital Care

Use standard precautions (ie, use of disposable surgical masks, face shields, and gowns when

appropriate) to prevent human-to-human transmission.

Emergency Department Care

- Implement barrier protection with secretion precautions.
- Obtain radiography and collect blood, urine, sputum, and skin lesion fluid.
- Initiate rapid administration of supportive care and IV antibiotic therapy for severe disease.

Consultations

- Glanders and melioidosis are reportable diseases; notify local health authorities of suspected cases.
- Occurrence of glanders in the absence of animal attack, occupational exposure, and/or in an epidemic is presumptive evidence of a BW attack. Consultation with an infectious disease specialist, the Centers for Disease Control and Prevention, and the Federal Bureau of Investigation may be warranted.

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Medication

Limited information exists about antibiotic treatment for these conditions in humans, since clinical studies examining antibiotic effectiveness in vivo are rare. For localized disease, a 60- to 150-day course of oral amoxicillin and clavulanate, tetracycline, or trimethoprim and sulfamethoxazole (TMP-SMX) may be used.

For local disease with mild toxicity, combine 2 of the 3 regimens for 30 days; then switch to monotherapy with amoxicillin and clavulanate or TMP-SMX for 60-150 days.

For extrapulmonary suppurative disease, prolong treatment for 6-12 months. Drain abscesses surgically. For severe and/or septicemic disease, initiate parenteral therapy for 2 weeks followed by oral therapy for 6 months (ceftazidime combined with TMP-SMX 8 mg TMP/kg/d and 40 mg SMX/kg/d divided qid). Add streptomycin when initiating treatment if plague cannot be excluded.

Other antibiotics have been effective in treating infections in animals but have not undergone therapeutic evaluation in humans. Currently, no proven preexposure or postexposure prophylaxis is available. Postexposure prophylaxis with TMP-SMX may be attempted. No vaccine is available for human glanders or melioidosis.

Antibiotics

Base choice of antibiotic and route of administration on the clinical setting.

| | |
|-------------------|---|
| Drug Name | Amoxicillin and clavulanate (Augmentin)- Semisynthetic antibiotic with a broad spectrum of bactericidal activity against many gram-positive and gram-negative microorganisms; susceptible to degradation by beta-lactamases, and thus spectrum of activity does not include organisms that produce these enzymes; clavulanic acid is a beta-lactam, structurally related to penicillins, which possesses ability to inactivate a wide range of beta-lactamase enzymes commonly found in microorganisms resistant to penicillins and cephalosporins; formulation of amoxicillin and clavulanic acid protects amoxicillin from degradation by beta-lactamase enzymes and effectively extends antibiotic spectrum of amoxicillin to include many bacteria normally resistant to amoxicillin and other beta-lactam antibiotics; possesses properties of a broad-spectrum antibiotic and a beta-lactamase inhibitor. |
| Adult Dose | 60 mg/kg/d PO divided tid for local disease or mild toxicity |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; history of Augmentin-associated cholestatic jaundice and/or hepatic dysfunction |
| Interactions | Probenecid decreases renal tubular secretion of amoxicillin; concurrent use may result in increased and prolonged blood levels of amoxicillin; coadministration of probenecid cannot be recommended; concurrent administration of allopurinol and ampicillin substantially increases incidence of rashes in patients receiving both drugs compared to patients receiving ampicillin alone; unknown whether this potentiation of ampicillin rashes is due to allopurinol or hyperuricemia present in these patients; no data exist with Augmentin and allopurinol administered concurrently; as with other broad-spectrum antibiotics, may reduce efficacy of oral contraceptives |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Hepatic dysfunction; renal impairment |

| | |
|-------------------|---|
| Drug Name | Tetracycline (Minocin)- Primarily bacteriostatic and believed to exert antimicrobial effect by inhibition of protein synthesis. |
| Adult Dose | 40 mg/kg/d PO divided tid |
| Pediatric Dose | <8 years: Not recommended >8 years: Administer as in adults |
| Contraindications | Documented hypersensitivity |

| | |
|--------------|--|
| Interactions | Concurrent use may render oral contraceptives less effective; demonstrated to depress plasma prothrombin activity; patients receiving anticoagulant therapy may require downward adjustment of anticoagulant dosage; since bacteriostatic drugs may interfere with bactericidal action of penicillin, avoid administering tetracycline-class drugs in conjunction with penicillin; absorption is impaired by antacids containing aluminum, calcium, or magnesium and by iron-containing preparations; concurrent use of methoxyflurane has been reported to result in fatal renal toxicity |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Pseudotumor cerebri (benign intracranial hypertension) in adults has been associated with use; photosensitivity manifested by exaggerated sunburn reaction has been observed in some individuals; persons with impaired renal function may require a lower dose |

| | |
|-------------------|---|
| Drug Name | Sulfamethoxazole and trimethoprim (Bactrim, Bactrim DS, Septra, Septra DS)-SMX inhibits bacterial synthesis of dihydrofolic acid by competing with PABA; TMP blocks production of tetrahydrofolic acid from dihydrofolic acid by binding to and reversibly inhibiting the required enzyme, dihydrofolate reductase; thus blocks 2 consecutive steps in biosynthesis of nucleic acids and proteins essential to many bacteria. |
| Adult Dose | Local or mild disease: 4 mg TMP/kg/d and 20 mg SMX/kg/d PO divided bid Severe disease: 8 mg TMP/kg/d and 40 mg SMX/kg/d IV divided qid for 2 wk; then PO for 6 mo |
| Pediatric Dose | <2 months: Not recommended >2 months: Administer as in adults |
| Contraindications | Documented hypersensitivity; documented megaloblastic anemia due to folate deficiency; pregnant patients and nursing mothers (sulfonamides cross placenta, are excreted in milk, and may cause kernicterus) |
| Interactions | Increased incidence of thrombocytopenia with purpura has been reported in elderly patients concurrently receiving certain diuretics (primarily thiazides); may prolong PT in patients receiving warfarin; may inhibit hepatic metabolism of phenytoin (be alert for possible excessive phenytoin effect); sulfonamides also can displace methotrexate from plasma protein binding sites, thus increasing free methotrexate concentrations |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Renal or hepatic function, folate deficiency, G-6-PD deficiency, and severe allergies or bronchial asthma; local irritation and inflammation due to extravascular infiltration of the infusion have been observed with IV |

| | |
|-------------------|--|
| Drug Name | Ceftazidime (Fortaz)- Bactericidal, exerting effect by inhibition of enzymes responsible for cell-wall synthesis. |
| Adult Dose | 120 mg/kg/d IV divided tid |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Nephrotoxicity has been reported following concomitant administration of cephalosporins with aminoglycoside antibiotics or potent diuretics such as furosemide; chloramphenicol has been demonstrated to be antagonistic to beta-lactam antibiotics, including ceftazidime, based on in vitro studies and time kill curves with enteric gram-negative bacilli; because of possibility of antagonism in vivo, particularly when bactericidal activity is desired, avoid this drug combination |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Reduce total daily dosage in patients with renal insufficiency; cephalosporins may be associated with decreased prothrombin activity; those at risk include patients with renal and hepatic impairment, poor nutritional state, and those receiving a protracted course of antimicrobial therapy; caution in individuals with history of GI disease, particularly colitis |

| | |
|-------------------|---|
| Drug Name | Streptomycin- Aminoglycoside antibiotic recommended when less potentially hazardous therapeutic agents are ineffective or contraindicated. |
| Adult Dose | 30 mg/kg/d IM, not to exceed 2 g/d |
| Pediatric Dose | 25-30 mg/kg/d IM, not to exceed 1-1.5 g/d |
| Contraindications | Documented hypersensitivity; nondialysis-dependent renal insufficiency |
| Interactions | Nephrotoxicity may be increased with aminoglycosides, cephalosporins, penicillins, amphotericin B, and loop diuretics |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Pregnancy; narrow therapeutic index; not intended for long-term therapy; caution in patients with renal failure who are not on dialysis; caution with myasthenia gravis, hypocalcemia, and conditions that depress neuromuscular transmission |

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Follow-up

Deterrence/Prevention

- Biosafety Level 3 containment practices are required for laboratory staff when working with these organisms.
- In countries where glanders is endemic in animals, identification and elimination of the disease in the animal population prevents disease in humans.
- In areas where melioidosis is endemic, persons who have chronic illnesses that lead to an immunocompromised state should avoid contact with soil and standing water. Wearing boots and gloves during agricultural work is advised.

Complications

- Possible complications include septicemia, osteomyelitis, meningitis, and brain, liver, or splenic abscess.

Prognosis

- Untreated patients with septicemia have fatal outcomes. Before antibiotics, the death rate for septicemic disease was 95%. It is greater than 50% for septicemic disease and 20% for localized disease despite treatment. Overall, mortality is 40%.

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Miscellaneous

Medical/Legal Pitfalls

- Several obstacles to understanding these diseases exist. These include lack of awareness among clinicians resulting in delayed diagnosis or misdiagnosis; absence of diagnostic laboratories with adequate, standardized serologic methods to detect these rare organisms; inadequate surveillance systems; and a lack of research. More studies are needed on all aspects of these diseases, as is a heightened level of awareness among health care providers.
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CBRNE - Incapacitating Agents, 3-Quinuclidinyl Benzilate

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Synonyms, Key Words, and Related Terms

QNB, BZ, agent buzz

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Introduction

Background

The chemical warfare agent 3-quinuclidinyl benzilate (QNB, BZ) is an anticholinergic agent that affects both the peripheral and central nervous systems (CNS). It is one of the most potent anticholinergic psychomimetics known, with only small doses necessary to produce incapacitation. It is classified as a hallucinogenic chemical warfare agent. QNB usually is disseminated as an aerosol, and the primary route

of absorption is through the respiratory system. Absorption also can occur through the skin or gastrointestinal tract. It is odorless. QNB's pharmacologic activity is similar to other anticholinergic drugs (eg, atropine) but with a much longer duration of action.

Pathophysiology

QNB acts by competitively inhibiting muscarinic receptors. Muscarinic receptors primarily are associated with the parasympathetic nervous system, which innervates numerous organ systems, including the eye, heart, respiratory system, skin, gastrointestinal tract, and bladder. Sweat glands, innervated by the sympathetic nervous system, also are modulated by muscarinic receptors. The IC₅₀ (concentration in air of QNB necessary to incapacitate 50% of exposed and unprotected individuals through inhalation during a set time) has been reported to be 100 mg·min/m³. Effects of QNB by any route of exposure are slow in onset and long in duration. The onset of action is approximately 1 hour, with peak effects occurring 8 hours postexposure. Symptoms gradually subside over 2-4 days. Most of the QNB that enters the body is excreted by the kidneys, making urine the choice for detection.

Frequency

- **In the US:** Use of QNB against the US has never been reported. Currently, the US government is funding numerous programs to prepare the nation for potential chemical terrorist attacks against citizens and the military.
- **Internationally:** Use of QNB has been suggested in a number of international conflicts. In January 1992, soldiers in Mozambique experienced an explosion above their troop formation. Subsequent symptoms resembled those expected from QNB. In July 1995, approximately 15,000 people attempted to walk from the enclave of Srebrenica to the free territory in Bosnia. Many experienced hallucinations during their march that were suspected to be secondary to QNB.

Mortality/Morbidity

The LC₅₀ of QNB is reportedly 200,000 mg·min/m³. This means, for example, that 50% of an unprotected group would die following inhalation of air that contained 200,000 mg of QNB per cubic meter for 1 minute. By comparison, the highly toxic compound hydrogen cyanide has an LC₅₀ of 5000 mg·min/m³. The LD₅₀ (lethal dose to 50% of an exposed population) for BZ is estimated to be similar to that of atropine, which is approximately 100 mg. Other factors also are important, such as the exposed patient's preexisting health status and the time from exposure to medical care.

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Clinical

History

- An event involving QNB probably would create confusion, panic, multiple seriously ill or dead victims, and a major emergency medical service, police, and/or military response.
 - Large numbers of casualties would overwhelm any community's emergency response.
 - Chaos appropriately would describe events following such an event.
 - In the early phases of an emergency response, the toxin's identification would be unknown, and the history would be misleading and inaccurate.

Physical

After exposure to QNB, the physical examination is consistent with an anticholinergic syndrome. Characteristics of the anticholinergic syndrome have long been taught using the old medical adage, "dry as a bone, blind as a bat, red as a beet, hot as a hare, and mad as a hatter."

- Central nervous system
 - Depending on the dose and time postexposure, a number of CNS effects may manifest. Restlessness, apprehension, abnormal speech, confusion, agitation, tremor, picking movements, ataxia, stupor, and coma are described.
 - Hallucinations are prominent, and they may be benign, entertaining, or terrifying to the patient experiencing them. Exposed patients may have conversations with hallucinated figures, and/or they may misidentify persons they typically know well.
 - Simple tasks typically performed well by the exposed person may become difficult. Motor coordination, perception, cognition, and new memory formation are altered as CNS muscarinic receptors are inhibited.

Causes

Human QNB exposures rarely are reported. Potential causes of exposure to this agent are a laboratory accident, a terrorist event, or a military conflict.

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Differentials

CBRNE - Incapacitating Agents, Agent 15

CBRNE - Incapacitating Agents, LSD

Heat Exhaustion and Heat Stroke

Toxicity, Amphetamine

Toxicity, Anticholinergic
Toxicity, Antidepressant
Toxicity, Antihistamine
Toxicity, Cocaine
Toxicity, Cyclic Antidepressants
Toxicity, Hallucinogen
Toxicity, Lithium
Toxicity, Methamphetamine
Toxicity, Phencyclidine
Toxicity, Salicylate
Toxicity, Sympathomimetic

Other Problems to be Considered

Thyrotoxicosis

Neuroleptic malignant syndrome

Serotonin syndrome

CNS infection

Heat stroke

Sedative-hypnotic withdrawal

Anticholinergic "outbreaks" involving jimsonweed abuse by teenagers, scopolamine-tainted heroin, or alcoholic beverages

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Workup

Lab Studies

- No rapid tests enable a health care provider to diagnose exposure to QNB. Consider QNB if a number of persons arrive after an exposure to an unknown substance and manifest an anticholinergic syndrome.
- Obtaining a complete blood count, electrolytes, clotting studies, and renal and liver function tests

is reasonable in any person who potentially was exposed to a chemical warfare agent.

- If the patient is markedly agitated or comatose, obtaining a urine myoglobin and/or creatine phosphokinase is warranted to exclude rhabdomyolysis. Hyperkalemia, hyperphosphatemia, and hypocalcemia may occur in association with rhabdomyolysis. The agitated patient also may develop an elevated lactate.
- If QNB is considered in the differential, obtain extra blood and urine samples. Tests have been developed to confirm human exposure to QNB.
- Disseminated intravascular coagulation is a potential complication in a patient with marked agitation and/or hyperthermia. Obtain clotting studies (eg, prothrombin time, activated partial thromboplastin time, international normalized ratio) in these patients. If clotting studies are elevated, then fibrinogen, fibrin split products, and a peripheral smear looking for evidence of hemolysis may be necessary.

Imaging Studies

- A patient exposed to QNB who is comatose may be at risk for aspiration pneumonia; obtain a chest x-ray.
- If the etiology of the altered mental status is uncertain, obtaining a head CT scan to exclude other intracranial processes is reasonable.

Other Tests

- ECG: QNB is associated with sinus tachycardia. Patients exposed to QNB who have preexisting cardiac disease may be at risk for cardiac ischemia as their heart rates increase. Other anticholinergic agents are associated with QT prolongation, QRS widening, and various tachydysrhythmias. Obtain an ECG to exclude these potential problems.

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Treatment

Prehospital Care

- Prehospital care providers must place their personal safety before the treatment of potentially contaminated patients.
 - The US military recommends wearing maximum protection when in contact with QNB contamination. These recommendations include wearing an M9 mask and hood, an M3 butyl rubber suit, M2A1 butyl boots, and M3 or M4 butyl gloves.
 - For civilian paramedics, decontamination of the exposed patients prior to transfer must

occur. Dermal absorption and subsequent toxicity is a risk from contact with contaminated patients.

- Off gassing may occur, and paramedics are at risk for toxicity in the closed confines of an ambulance. Caution must be exercised especially for flight crews, since toxicity of the pilot during mid flight can lead to impaired vision and judgment and subsequent risk of crashing the aircraft.
- After the patients have been decontaminated, transport them to the nearest hospital facility.
- Perform general supportive measures (eg, intravenous access, airway management).

Emergency Department Care

Once decontamination has occurred, the primary emphasis simply is supportive care of exposed patients. Emergency department staff must be certain that proper decontamination has occurred. Dermal absorption and off gassing of QNB does occur and can pose a risk to hospital personnel.

- In patients who are not protecting their airway, perform intubation and mechanical ventilation.
- Apply soft restraints to patients at risk of harming themselves or health care workers.
- Intravenous hydration may be necessary; maintain adequate urinary output. If urinary retention is suggested, place a Foley catheter.
- For patients experiencing marked agitation, consider benzodiazepine administration.
- In patients with hyperthermia, cooling measures may be necessary.
 - Completely remove the patient's clothing.
 - Insert a Foley catheter or rectal temperature probe.
 - Administer adequate intravenous fluids.
 - Cooling measures such as evaporative cooling using skin wetting with directed circulating fans, ice water immersion, ice packs, and cooling blankets may be necessary.

Consultations

If an exposure to QNB occurs, consider a number of consultations.

- If the cause of the exposure is a terrorist act against civilians, contact the local health department, poison center, and law enforcement agency immediately. Also contact federal agencies, such as the US Federal Bureau of Investigations (FBI).
- If a patient sustained eye contact with QNB and subsequently developed eye pain, change in vision, or marked conjunctival injection, consultation with an ophthalmologist may be necessary.
- For patients requiring intensive care monitoring, consider early consultation with a physician trained in critical medicine.

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Medication

No specific antidote has been found to reverse the action of QNB definitively. In the past, physostigmine was used to reverse the effects of anticholinergic agents. However, numerous adverse effects associated with its use in reversing poisonings are reported in the literature. Subsequently, the use of physostigmine has diminished greatly in the setting of acute anticholinergic toxicity. Use of physostigmine in QNB poisoning has been studied. However, its efficacy in QNB intoxication and its adverse effect potential have not been delineated definitively. At this time, supportive care is the mainstay of therapy. If the exposed patient is markedly agitated, consider administration of a benzodiazepine.

Benzodiazepines

Consider in patients sustaining exposure to QNB and presenting with marked agitation.

| | |
|-------------------|---|
| Drug Name | Diazepam (Valium, Diastat)- Depresses all levels of CNS (eg, limbic and reticular formation), possibly by increasing activity of GABA. Induces sedation and helps cease seizure activity. |
| Adult Dose | 2 mg IV q15 min until desired level of sedation obtained |
| Pediatric Dose | 0.2 mg/kg IV q15 min until desired level of sedation obtained |
| Contraindications | Documented hypersensitivity |
| Interactions | Increases toxicity of benzodiazepines in CNS with coadministration of phenothiazines, barbiturates, alcohols, and MAOIs |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | May induce hypotension; caution with other CNS depressants, low albumin levels, or hepatic disease (may increase toxicity) |

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Follow-up

Further Inpatient Care

- Inpatient care is no different than that discussed in [Emergency Department Care](#). Keep symptomatic patients who were exposed to QNB in a monitored setting until their symptoms completely resolve. Use of maintenance intravenous fluids and sedatives such as benzodiazepines

may be necessary. Prolonged intoxication may occur depending on the dose of QNB absorbed.

Transfer

- Any health care facility that is unable to adequately monitor a patient intoxicated with an anticholinergic should consider transfer to a facility that can care for such patients.
- Smaller health care facilities may be overwhelmed if a large-scale terrorist attack with multiple victims occurs. Disaster plan implementation and appropriate transfer of patients to less stressed facilities may be necessary.

Complications

- Rhabdomyolysis: If a person exposed to QNB develops marked agitation or profound somnolence, tissue necrosis may occur and rhabdomyolysis may develop. If this remains undiagnosed, myoglobinuric renal failure may develop.
- Anoxic brain injury: If an exposed person becomes comatose and loses his or her ability to maintain ventilatory function, hypoxia may develop and lead to anoxic brain injury.
- Aspiration pneumonia: Inability of exposed patients to maintain their airway may result in aspiration of gastric contents into the lungs.
- Ileus: The prolonged anticholinergic effects of QNB may lead to development of an ileus.
- Angle-closure glaucoma: Those patients predisposed may be at risk due to the mydriasis induced by QNB.
- Bleeding diathesis: Disseminated intravascular coagulation may develop in patients with shock and marked hyperthermia.
- Hepatic injury: Hepatic injury may accompany antimuscarinic agent toxicity that involves hyperthermia or shock.

Prognosis

- The prognosis is good for QNB-exposed patients if they do not develop a secondary injury such as the complications noted above. Once they are removed from the exposure and the absorbed QNB is metabolized, they should become more lucid. Full recovery is expected within 4 days. No long-term effects are expected from QNB itself.

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Miscellaneous

Medical/Legal Pitfalls

- Few pitfalls exist from a medicolegal standpoint. Decontamination of patients and avoidance of contamination of health care workers is paramount. If a physician demonstrates good supportive care as discussed in this article, risk of litigation against the caregiver should be minimal.

Special Concerns

- As noted with other antimuscarinic agents, patients at the extremes of age may be more susceptible to toxicity. Other factors expected to predispose a patient to toxicity include heat stress, volume depletion, and concurrent use of medications with antimuscarinic effects.

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CBRNE - Incapacitating Agents, Agent 15

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Synonyms, Key Words, and Related Terms

chemical warfare, anticholinergic, glycolates, delirium

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Introduction

Background

"Incapacitating agent" is a military term used to denote an agent that temporarily and nonlethally impairs the performance of an enemy by targeting the central nervous system (CNS). Of those substances investigated by the military, anticholinergic agents best fit these criteria and are stable enough for use in war. As far back as 184 BC, Hannibal's army used belladonna plants to induce disorientation in enemies. In 1672, the Bishop of Muenster used belladonna-containing grenades in his campaigns.

Approximately 300 years later, the US Army explored several classes of drugs, as well as noise, microwaves, and photostimulation, and found none to be as promising incapacitating agents as the anticholinergics. Stimulants such as cocaine, amphetamines, and nicotine were tested but did not have the potency to be an airborne threat. Depressants (eg, barbiturates, opiates, neuroleptics) similarly were found to be impractical for battlefield use. The unpredictable behavior incurred by psychedelic agents (ie, lysergic acid diethylamide [LSD], phencyclidine [PCP]) led to an early halt in the testing of that particular class of drugs.

By the mid-1960s, after a decade of tests, the US Army concluded that the long-acting anticholinergic 3-quinuclidinyle benzilate (BZ) was the best candidate for weaponization and deployment. BZ subsequently was stockpiled in American military arsenals from the mid-1960s through the late-1980s. The US military was not alone in its attempt to develop an incapacitating agent in the 20th century. Seven years after the conclusion of the Gulf War, the British Foreign Ministry revealed, in February 1998, the existence of an Iraqi chemical warfare agent believed to be a glycolate anticholinergic, similar, if not identical, to BZ. It was dubbed "Agent 15." Little information is known publicly about Agent 15. For this reason, also refer to [CBRNE - Incapacitating Agents, 3-quinuclidinyl Benzilate](#).

Pathophysiology

BZ is the North Atlantic Treaty Organization (NATO) code for 3-quinuclidinyle benzilate, a glycolate anticholinergic also known as 3-QNB. Both BZ and its Iraqi look-alike, Agent 15, are competitive inhibitors of the effects of acetylcholine at the postsynaptic muscarinic receptors in the peripheral and central nervous systems. In the peripheral nervous system, this inhibition is observed in the smooth muscle, autonomic ganglia, and exocrine glands. BZ's ability to readily cross the blood-brain barrier allows it to wreak havoc on the CNS, causing mental status changes and delirium.

A common problem in developing a chemical warfare agent is finding an effective and reliable chemical. The glycolate anticholinergics (eg, BZ, Agent 15) fit this description. Extremely stable, these chemicals have a half-life of 3-4 weeks in moist air and even longer on surfaces or in soil. Absorption of glycolates can occur following inhalation, ingestion, or cutaneous exposure. Only small doses of this potent drug are needed to produce delirium. The dose of BZ needed to incapacitate 50% of those exposed is 6.2 mcg/kg, compared to 140 mcg/kg for atropine.

Frequency

- **In the US:** With the exception of Army test volunteers in the 1960s, anticholinergic incapacitating agents have not been used in the US. Although many experts believe that most terrorists would opt for a lethal form of chemical attack (eg, nerve agent), use of incapacitating agents cannot be discounted. Other sources of anticholinergic toxicity include clinical medicines such as atropine, antihistamines, and tricyclic antidepressants. Numerous plants commonly found in North America also can cause delirium indistinct from exposure to an incapacitating agent.

Examples include jimsonweed, nightshade, belladonna, and other members of the Solanaceae family.

Mortality/Morbidity

By definition, incapacitating agents are nonlethal. BZ has a high safety ratio. The dose required to produce incapacitating effects is roughly 1000 times less than the fatal dose. Fatalities from this class of drug can result from hyperthermia or from the casualty's delirious behavior. Such a scenario was dramatized in the 1990s movie "Jacob's Ladder," in which a fictitious military unit kills itself after accidental exposure to an incapacitant-type chemical warfare agent.

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Clinical

History

- The patient may complain of dry mouth, a hot feeling, or blurred vision.
- Changes in mental status produced by incapacitating agents may leave some patients delirious to the point that they fail to or are unable to report symptoms.

Physical

Remarkably little variation exists among individuals when anticholinergics are administered.

- Following exposure, typically a latent period of 30 minutes to 24 hours occurs before signs and symptoms appear.
- Anticholinergic toxicity caused by BZ or Agent 15 can last up to 3-4 days, depending on the amount of drug absorbed.
- Peripheral effects usually precede CNS effects and can be summarized by the mnemonic "dry as a bone, hot as a hare, red as a beet, and blind as a bat."
 - "Dry as a bone" results from decreased glandular secretions in the oral pharynx, GI tract, and eccrine and apocrine glands. Urinary retention also is common.
 - "Hot as a hare" refers to hyperthermia caused by decreased sweating.
 - The body attempts to maintain thermoregulation via compensatory cutaneous vasodilatation, hence "red as a beet."
 - Decreased cholinergic stimulation of the pupillary sphincter muscle causes mydriasis. Anticholinergic effects on the ciliary muscles inhibit accommodation, hence "blind as a bat."

- Anticholinergic effects on the heart produce tachycardia. This occasionally is preceded by a bradycardia that results from anticholinergic effects in the brain stem.
- Of final note on the examination is an increase in deep tendon reflexes. Anticholinergic effects on the Renshaw interneurons in the spinal cord cause hyperreflexia.

Causes

- Consider any cause of delirium. Psychiatric disorders such as anxiety reaction also are in the differential diagnosis.
- The presence of peripheral anticholinergic signs suggests another source of anticholinergic such as scopolamine, atropine, jimsonweed, or other anticholinergic source exposure.
- Usually, 6-7 MARK-1 Autoinjectors (ie, 12-14 mg of IM atropine) are needed to cause a significant degree of confusion.
- Nerve agent poisoning can be differentiated by its hyperstimulation of glands.

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Differentials

CBRNE - Incapacitating Agents, 3-quinuclidinyl Benzilate

CBRNE - Incapacitating Agents, LSD

Heat Exhaustion and Heat Stroke

Toxicity, Amphetamine

Toxicity, Anticholinergic

Toxicity, Antidepressant

Toxicity, Antihistamine

Toxicity, Cocaine

Toxicity, Cyclic Antidepressants

Toxicity, Hallucinogen

Toxicity, Lithium

Toxicity, Methamphetamine

Toxicity, Phencyclidine

Toxicity, Salicylate

Toxicity, Sympathomimetic

Other Problems to be Considered

Thyrotoxicosis

Neuroleptic malignant syndrome

Serotonin syndrome

CNS infection

Heat stroke

Sedative-hypnotic withdrawal

Anticholinergic "outbreaks" involving jimsonweed abuse by teenagers, scopolamine-tainted heroin, or alcoholic beverages

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Workup

Lab Studies

- The paucity of distinct diagnostic signs and the lack of any sort of field detector make the diagnosis of Agent 15 toxicity extremely difficult.
- Maintain a high index of suspicion in scenarios in which terrorist or enemy chemical attack is possible. Multiple casualties exhibiting delirium indicate this diagnosis.
- Most standard urine toxicology screens do not detect the presence of Agent 15.
- Confirmatory testing is available at select reference laboratories.
- Routine laboratory tests can be helpful in ruling out other causes of delirium. These include a CBC, electrolytes, BUN and/or creatinine, glucose, LFTs, toxicology screen, ABG, ammonia level, thyroid stimulating hormone, and lumbar puncture for cerebral spinal fluid.

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Treatment

Prehospital Care

- Protection of medical personnel includes removing weapons from victims and using restraints as

necessary.

- Use chemical protective masks if residual aerosolized BZ or Agent 15 is present. The high-efficiency particulate air (HEPA) filter in the gas mask provides adequate protection.
- Protective gowns and gloves are indicated.
- Decontamination of victims is critical and involves removing contaminated clothing and flushing the skin with soap and water. Fine particles can be brushed away gently.
- Anticholinergic poisoning places the victim at high risk for hyperthermia. Thus, remove heavy clothing and initiate intravenous fluids as indicated.

Emergency Department Care

- Ensuring that appropriate decontamination has occurred is paramount to stabilize the patient and to prevent facility contamination. Complete decontamination of the skin and clothing if not already performed in the prehospital setting. Any residual Agent 15 on skin or clothing can be removed effectively with soap and water.
- The 2 greatest risks to the patient are his or her own erratic behavior and hyperthermia.
 - Confiscate weapons and closely observe the patient. Physical restraints may be needed in severely affected patients.
 - Monitor core temperature and maintain adequate fluids orally or intravenously.

Consultations

Additional advice can be acquired by calling the US Army Medical Research Institute of Chemical Defense at the Aberdeen Proving Grounds, Maryland, at (410) 436-3628.

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Medication

In the past, physostigmine was used to reverse the effects of anticholinergic intoxicants. However, numerous adverse effects from its use are reported. For this reason, its role as an antidote is controversial, and benzodiazepines generally are considered to be the safest medications for treating patients with anticholinergic-mediated agitation or delirium. Physostigmine use is reserved for patients with intractable seizures, tachycardia, or agitation. Physostigmine does not shorten the clinical course of anticholinergic toxicity. Neostigmine and pyridostigmine lack the central antimuscarinic activity needed to make them effective antidotes.

Cholinesterase Inhibitors

Physostigmine is a carbamate that increases the concentration of acetylcholine in synapses and neuromuscular junctions through acetylcholinesterase inhibition.

| | |
|-------------------|--|
| Drug Name | Physostigmine (Antilirium)- Increased concentration of acetylcholine can improve patient's delirium dramatically; for reasons that are not entirely clear, appears to have less effect if administered within 4 h postexposure. |
| Adult Dose | 1-2 mg in 10 cm ³ normal saline over 5 min; repeat doses rarely are needed; continuous infusions should not be given |
| Pediatric Dose | 20 mcg/kg or 0.5 mg IV over 5 min |
| Contraindications | Documented hypersensitivity; cardiovascular disease; heart block; bronchospasm; vagotonic symptoms (especially bradycardia); intestinal and/or bladder obstruction; severe peripheral vascular disease (gangrene); diabetes; recent coadministration of succinylcholine |
| Interactions | Concurrent administration with succinylcholine may prolong respiratory depression |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Potential adverse effects include asystole in the setting of conduction disturbance or cyclic antidepressant toxicity, seizures, muscle weakness, cholinergic crisis (bradycardia, salivation, lacrimation, bronchospasm, bronchorrhea, diarrhea); prior to use, confirm presence of a normal ECG (no conduction disturbance), absence of exposure to other cardiotoxic substances, and presence of peripheral and central signs of antimuscarinic toxicity; as added precaution, have atropine at bedside for use if cholinergic symptoms develop |

Benzodiazepines

By binding to specific receptor-sites these agents appear to potentiate effects of GABA and facilitate inhibitory GABA neurotransmission and other inhibitory transmitters.

| | |
|-------------------|---|
| Drug Name | Diazepam (Valium, Diazemuls, Diastat)- Depresses all levels of CNS (eg, limbic and reticular formation), possibly by increasing activity of GABA. |
| Adult Dose | 2 mg IV q15min; titrate to effect |
| Pediatric Dose | 0.2 mg/kg IV q15min; titrate to effect |
| Contraindications | Documented hypersensitivity; narrow-angle glaucoma |
| Interactions | Increases toxicity of benzodiazepines in CNS with coadministration of phenothiazines, barbiturates, alcohols, and MAOIs |
| Pregnancy | D - Unsafe in pregnancy |

Precautions

Caution with other CNS depressants, low albumin levels, or hepatic disease (may increase toxicity)

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Follow-up

Further Inpatient Care

- Monitor victims of Agent 15 or other anticholinergics as inpatients until the drug has cleared from the body. This process can take up to 3-4 days in some patients.

Complications

- Long-term effects from poisoning by Agent 15 or other related glycolate anticholinergics are very unlikely.
- Severe exposures may require several days of observation before the patient is clear of anticholinergic effects.

Prognosis

- Prognosis is excellent, with few neurologic sequelae.

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Miscellaneous

Medical/Legal Pitfalls

- Administration of physostigmine is routine for anticholinergic toxicity. Using this antidote when clinically indicated is sound medicolegal care.

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CBRNE - Incapacitating Agents, Cannabinoids

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Synonyms, Key Words, and Related Terms

hemp, marijuana, pot, Mary Jane, weed

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Introduction

Background

The hemp plant *Cannabis sativa* has been used both recreationally and medicinally for thousands of years. Like other substances that cause predominantly behavioral disruption, cannabinoids potentially can be employed as a biological warfare agent. However, no country or terrorist group is known to have

produced a cannabinoid biological weapon, and the development of one is unlikely. Many other incapacitating agents already available (eg, 3-quinuclidinyl benzilate [BZ]) are superior to potential cannabinoid biological weapons that may be developed. Additionally, military medical personnel need to consider cannabinoid intoxication when assessing personnel with poor performance, stress, lack of motivation, or psychiatric illness.

Most exposures to cannabinoids are intentional. The dried flowering tops and leaves usually are smoked as a cigarette (eg, joint, reefer), although some mix it with foods or brew it as tea. The main active ingredient in cannabis is delta-9-tetrahydrocannabinol (THC), although more than 400 active compounds have been isolated. More than 60 of these active compounds are unique to cannabis and are referred to as cannabinoids. Among them, THC is the most psychoactive in humans, producing euphoria, relaxation, intensification of ordinary sensory experiences, perceptual alterations, diminished pain, and difficulties with memory and concentration. Researchers have discovered that cannabis has other effects throughout the body; this article focuses on the current scientific knowledge of both short-term and long-term morbidity associated with cannabis use.

Pathophysiology

In 1984, THC and several other cannabinoids were demonstrated to directly affect cell function by inhibiting a substance called adenylate cyclase. In the next few years, 2 cell receptor sites on brain cells specific for cannabinoids were identified (CB1, CB2). CB1 receptors were mapped predominantly to the brain, with the highest densities in the hippocampus (memory, cognition), basal ganglion and cerebellum (movement), and striatum (brain reward). Other areas of the brain with these receptors are responsible for anxiety, pain, sensory perception, motor coordination, and endocrine function. These findings demonstrated that the receptor locations in the brain correlate with effects elicited from cannabis. The other receptor, CB2, exists in the spleen and immune cells (macrophages, T lymphocytes, B lymphocytes), suggesting an influence on the immune system.

Since these discoveries, endogenous or naturally occurring cannabinoids have been found in animals. The role of these natural cannabinoids and the effect that cannabis has on the cannabinoid neurochemical system requires more research.

Potency

The average THC potency of cannabis has increased in the last decade as a result of sophisticated plant breeding and cultivation. In the early 1970s, an average marijuana cigarette contained approximately 10 mg of THC; a modern marijuana cigarette may contain 60-150 mg or more. Thus, today's marijuana smoker may be exposed to higher concentrations of THC. Since many of the detrimental effects of THC are dose dependent, current cannabis users may experience greater morbidity than previous users.

Cannabis comes in the following different forms:

- Marijuana is a combination of the *C sativa* flowering tops and leaves. Two preparations are possible for marijuana. Bhang consists of dried leaves and tops, and ganja consists of leaves and tops with a higher resin content (greater potency). The THC concentration is 0.5-5%.
- Hashish is the dried resin collected from flowering tops. Its concentration of THC is 2-20%.
- Sinsemilla, the unpollinated flowering tops from the female plant, and Dutch hemp (Netherweed) both have THC concentrations as high as 20%.

Absorption

Approximately 50% of THC and other cannabinoids in a marijuana cigarette enter the mainstream smoke and are inhaled. The amount absorbed through the lungs depends on the smoking style. Experienced smokers tend to inhale deeply and hold the smoke in for several seconds, allowing virtually all the cannabinoids to enter the bloodstream. Effects are apparent within minutes of smoking. THC concentrations of 2-3 mg produce measurable psychological and physical effects in occasional users. If cannabis is ingested, 25-50% of the cannabinoids are absorbed, with effects occurring 0.5-3 hours later.

Metabolism

After smoking, venous blood levels of THC fall within minutes and 1 hour later are at approximately 5-10% of peak levels. Cannabinoids are lipid soluble and are distributed rapidly throughout the body's tissues, where they are released slowly and metabolized. The elimination half-life of THC is estimated at 20 hours to 10-13 days. Cannabinoids are metabolized in the liver to more than 20 metabolites, many of which are active. Active and inactive metabolites are excreted in the urine and feces.

Tolerance

Repeated use over days to weeks induces considerable tolerance to the behavioral and psychological effects of cannabis. Several studies have noted partial tolerance to the effect on mood, memory, motor coordination, sleep, brain wave activity, blood pressure, temperature, and nausea. The rate of tolerance depends on the dose and frequency of administration. The casual cannabis user experiences more impairment in cognitive and psychomotor function to a particular acute dose than a heavy user. The desired recreational high from cannabis also diminishes with use, prompting many users to escalate the dose.

Dependance and withdrawal

Evidence suggests that with heavy chronic use, individuals experience problems controlling their use, despite adverse personal consequences. One report states that 1 in 10 persons who ever use cannabis becomes dependent on it over the 4-5 years of greatest use. This risk is more like the risk for alcohol (15%) than for nicotine (32%) or opioids (23%). The National Institute on Drug Abuse (NIDA) reports

that 100,000 people per year present for treatment of a primary (or at least self-perceived primary) marijuana abuse problem. Some cannabis users report withdrawal symptoms. Several studies in mostly adult populations demonstrated irritability, restlessness, insomnia, anorexia, nausea, sweating, salivation, increased body temperature, tremors, and weight loss following as little as 1 week of daily use.

Behavioral effects

- The euphoriant potential of cannabis, or the ability to produce a high, is probably the greatest reason for its popularity.
- The euphoriant effect includes feelings of intoxication and detachment, relaxation, altered perception of time and distance, intensified sensory experiences, laughter, and talkativeness, with decreased anxiety, alertness, and depression. These effects depend on a number of factors including the dose, experience, mode of administration, personal expectation, social environment, and personality of the user.
- THC triggers the dopaminergic neurons in the ventral tegmental area of the brain, an area known to be pivotal in mediating the reinforcing (rewarding) effects of most drugs of abuse. This dopaminergic drive elicited by cannabinoids is believed to underlie the reinforcing and addicting properties of marijuana.
- Dysphoric reactions to cannabis are not uncommon, especially in naïve subjects. Such reactions may include severe anxiety or panic, unpleasant somatic sensations, delirium, mania, or paranoid feelings. Anxiety and/or panic reaction is the most common adverse psychological response. These unpleasant effects usually are of sudden onset during or shortly after smoking or appear more gradually 1-2 hours after an oral dose, usually last a few hours (rarely a few days), and completely resolve without intervention.
- Flashbacks occasionally occur in which the original drug experience (usually dysphoria) is relived weeks or months later.

Mental effects

- Cannabis causes acute impairment of cognitive function. Even after small doses, short-term memory is impaired in both naïve and experienced users. Deficits appear to be in acquisition of memory and may result from an attentional deficit combined with an inability to filter out irrelevant information and the intrusion of extraneous thoughts.
- Accumulating research indicates that chronic cannabis use may be associated with subtle impairment in cognitive function. These changes are related to dose and duration of use. In a study of 12th-grade students, 144 chronic heavy cannabis users who abstained for 24 hours were compared with 72 nonusers previously matched for intelligence quotient (IQ) in the fourth grade. The users demonstrated deficits in mathematical skills, verbal expression, and memory retrieval. Other studies of heavy users also revealed deficits in memory, attention, and executive function. One study of ex-cannabis users demonstrated

impaired attentional skills compared to nonusers 3 months to 6 years after discontinuing the drug.

Immune system effects

- Animal studies have demonstrated that cannabinoids impair the immune system's capacity to ward off microbial and viral infection.
- Probably through a combination of cannabinoid receptor and nonspecific mechanisms, lung macrophage function including phagocytosis, migration, and cytokine production appears to be compromised by dose-dependent cannabis smoke. This has been demonstrated in limited human in vitro studies.
- Although cannabinoid receptors are present on T lymphocytes and B lymphocytes, the effect of cannabis on their numbers or humoral function in the human body have not been demonstrated conclusively.

Cardiovascular effects

- To the naïve user, marijuana causes a sudden 20-100% rise in heart rate lasting up to 2-3 hours.
- Peripheral vasodilatation usually causes postural hypotension, often leading the user to feel dizzy or faint when standing up. Cardiac output increases by as much as 30%. This is accompanied by increased cardiac oxygen demand. These changes are of little concern in the healthy, young user. Moreover, tolerance develops within a few days of regular use.
- Naïve cannabis use has precipitated angina, myocardial infarction, congestive heart failure, and stroke in older users with preexisting coronary artery or cerebrovascular disease.

Respiratory effects

- Marijuana smoking causes transient bronchodilatation after acute exposure.
- With chronic heavy smoking, users experience increased cough, sputum production, and wheezing. These complaints are greater than among current tobacco smokers.
- In one study over an 8-year period, the rate of decline in respiratory function was greater among marijuana smokers than among tobacco smokers.
- A marijuana cigarette contains the same constituents (apart from nicotine) as tobacco smoke, including bronchial irritants, tumor initiators (mutagens), and tumor promoters. The amount of tar in a marijuana cigarette is 3 times the amount in a tobacco cigarette when smoked, with one-third greater deposition in

the respiratory tract.

- Chronic cannabis use is associated with illnesses including bronchitis, squamous metaplasia of the tracheobronchial epithelium, and emphysema. These problems have been reported more frequently in cannabis-only users than in tobacco-only users.
- Several case reports strongly suggest a link between cannabis smoking and cancer of the aerodigestive system including the oropharynx and tongue, nasal and sinus epithelium, and larynx. More case-controlled studies addressing the role of cannabis smoking in these cancers are needed.
- Most illegally obtained marijuana is contaminated with *Aspergillus* species and has caused invasive pulmonary aspergillosis in immunocompromised people.

Reproductive effects

- High-dose THC in animals causes a drop in testosterone levels, decreased sperm production, and compromised sperm motility and viability. It alters the normal ovulatory cycle.
- Cannabis administration during pregnancy reduces birthweight in animals; studies are equivocal in humans. No evidence exists that cannabis increases the risk of birth defects.
- Three studies have demonstrated a possible increased risk of nonlymphoblastic leukemia, rhabdomyosarcoma, and astrocytoma in children whose mothers reported using cannabis during their pregnancies.

Psychosis association

- Large doses of THC may produce confusion, amnesia, delusions, hallucinations, anxiety, and agitation; most episodes are rapidly remitting.
- A clear relationship exists between cannabis use and mental health.
 1. Substance-abusing adolescents commonly suffer 1 or more comorbid health or behavioral problems. Several studies have demonstrated marijuana abuse to coexist with attention deficit hyperactivity disorder, other learning disabilities, depression, and anxiety.
 2. An association exists between cannabis use and schizophrenia. A prospective study of 50,000 Swedish conscripts found a dose-response relationship between the frequency of cannabis use by age 18 and the risk of a diagnosis of schizophrenia over the subsequent 15 years. Although research argues that cannabis does not cause schizophrenia, prospective evidence exists that continued use predicts more psychotic symptoms in people with schizophrenia.
 3. Most youth with conduct disorders such as stealing without confrontation, truancy, breaking and entering, and running away also are dependent on marijuana.

- Cannabis use and other drug use are related. The greater the involvement with marijuana, the more likely a young person will use other illicit drugs now and over his or her lifetime. Similarly, the proportion of youth who smoke daily increases sharply with frequency of cannabis use. No such relationship exists between alcohol and cannabis.

Frequency

- **In the US:** Marijuana became the major drug of abuse in the 1960s, peaking in use in the late 1970s. According to the NIDA-funded Monitoring the Future survey, the peak year of use occurred in 1979, with 60.4% of 12th-grade students having used cannabis in their lifetimes, 50.8% in the preceding year, and more than 10.3% on a daily basis. Cannabis use began a continuous decline, with the lowest use occurring in 1992. At that time, 32.6% of 12th-grade students reported ever using cannabis, 21.9% reported use in the preceding year, and 1.9% reported using on a daily basis. The decline in use is attributed to perceived risk and to personal disapproval of drugs. From 1992-1997, marijuana use increased dramatically and then leveled off in the last 2 years. Figures from 1999 reveal that 22.0% of 8th-grade students and 49.7% of 12th-grade students reported ever using cannabis. Daily use was 1.4% and 6.0%, respectively. In 1998, the NIDA-sponsored Community Epidemiology Work Group investigated the rates of emergency department mentions of marijuana use in 20 metropolitan areas. Cities with the highest rates included Dallas (63.9%), Boston (44.1%), Denver (40.0%), San Diego (35.1%), and Atlanta (31.1%).

Mortality/Morbidity

No cases of mortality are reported from any cannabis product. Presumably, this is because cannabinoid receptors are scant in the lower brain stem, where cardiovascular and respiratory function is controlled. Despite this, do not consider cannabis an innocuous drug. The pharmacologic actions of cannabinoids are many and complex; they include a unique combination of some of the effects of alcohol, tranquilizers, opioids, and hallucinogens. Cannabis use also has somatic consequences.

Race

No differences are reported in patterns of cannabis use according to racial or ethnic background.

Sex

Little information is available regarding gender differences in cannabis use. In a 1995 US study, 6.5% of females and 10.5% of males aged 12 years and older reported marijuana use in the previous year.

Age

- Most cannabis users begin when younger than 20 years (peak of onset occurring at approximately 16-18 y), use it intermittently, and stop by their mid- to late-20s. Only approximately 10% become daily users.
- The Community Epidemiology Work Group sponsored by NIDA studied the rates of marijuana mention in emergency departments of 20 metropolitan areas. The highest increase in mention was among adolescents aged 12-17 years.
- Epidemiologic information suggests that the age of initiation of use is declining.

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Clinical

History

- Since overt clinical effects of cannabis use are rare, determining if a person uses cannabis primarily depends on self-admission.
- People often fail to admit cannabis use. They may deny the effect it has on their personal and professional life. Also, they may be ashamed and fear being discovered.
- Physicians rarely diagnose dependence on cannabis. It often is deemed an insignificant drug compared to other substances. Physicians may have a negative attitude toward use of illicit substances and marijuana for the amotivational effect of heavy use characterized by poor attention and goal-directed thinking and behavior.
- Physicians may not know how to screen for cannabis use. Screening questions may help determine if marijuana and other substances are used. A quick and well-studied screening tool, CAGE, is designed to evaluate alcohol dependence and has been encouraged for use with cannabis. The acronym CAGE stands for the main words of the 4 questions: cut, annoyed, guilty, and eye-opener. Two or more positive responses suggest dependence and the need for further evaluation. The questions are as follows:
 - Have you ever felt you ought to cut down on your use?
 - Have people annoyed you by criticizing your use?
 - Have you felt bad or guilty about your use?
 - Have you ever had a drink in the morning to steady your nerves or get rid of a hangover ("eye opener")?

Physical

- Two major clinical signs of acute cannabis intoxication are reddened conjunctiva and increased

heart rate.

- Other less objective findings include the following:
 - Possible dose-related constriction of the pupils
 - Difficulty with balance or motor coordination as indicated by a positive Romberg test or unsteady gait
 - Possible impaired performance of cognitive function tasks (eg, serial addition and/or subtraction, object recall [decreased accuracy and increased response time])
 - Sensory function and pain perception that appear to remain intact

Causes

- Several factors are believed to contribute to the resurgence of cannabis use.
 - Perceived health risk and addiction from cannabis use among teenagers is falling.
 - Disagreeing authorities have different beliefs about the risks of cannabis use. Some physicians are calling for loosened sanctions against prescribing cannabis for medical purposes, and many experts debate the issues of cannabis dependence and withdrawal.

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Differentials

CBRNE - Incapacitating Agents, 3-quinuclidinyl Benzilate

CBRNE - Incapacitating Agents, Agent 15

CBRNE - Incapacitating Agents, LSD

Panic Disorders

Toxicity, Barbiturate

Toxicity, Benzodiazepine

Toxicity, Cyclic Antidepressants

Toxicity, Mushrooms

Toxicity, Sedative-Hypnotics

Withdrawal Syndromes

Other Problems to be Considered

Conduct disorder

Attention deficit hyperactivity disorder

Dysthymic disorder

Major depression

Other substance abuse

Narcotics

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Workup

Lab Studies

- Urine screening is a sensitive measure for cannabinoid metabolites, but a positive test only indicates probable prior use and does not correlate well with the amount used or with psychomotor impairment.
 - A low urine concentration may result from a large dose taken some time previously or a recent small dose.
 - The enzyme-multiplied immunoassay technique (EMIT dau) detects urine levels of approximately 20-100 ng/mL.
 - Cannabinoids are detected for an average of 1-2 days and for as long as 7 days after a single marijuana cigarette. An individual who smokes an occasional marijuana cigarette tests positive at 500-1000 ng/mL or more for 3-4 days. Chronic users have a positive test an average of 27 days (maximum 46 d) after cessation.
 - Passive inhalation of smoke is highly unlikely to result in a positive urine test and does not cause psychomotor impairment.

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Treatment

Emergency Department Care

- The first step in treatment is addressing the problem. Tell the patient about concerning cannabis use. Emphasize the negative consequences of cannabis use, using examples from the patient's professional or social life. Present this without judgment and with empathy.
 - If the patient appreciates his or her problem with cannabis and desires help, refer him or

her to treatment.

- If the patient does not appreciate a problem, educate him or her about health consequences of cannabis use, including physical effects on the cardiorespiratory system, risk of cancers, and possible impairment of cognition. Provide follow-up treatment information.

Consultations

- If a patient is deemed to have a probable cannabis dependency problem, refer him or her to a social services counselor.
- Treatment programs are different for adolescents and adults.
 - Treatment for adolescents commonly uses a family-based approach with well-defined but flexible interventions. Both outpatient and residential treatment is effective with youth. If adolescents and parents will not come to treatment, home-based programs are an alternative.
 - Adult treatment interventions, including teaching coping skills to deal with situations presenting a risk of use, group discussions related to cessation, and group support, are more effective than no intervention in reducing cannabis use.

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Follow-up

Patient Education

- Always warn patients using cannabis about adverse effects and encourage them to stop. Important health advice includes the following:
 - Impaired attention, memory, and psychomotor performance occur while intoxicated. With chronic use, subtle changes in attention and memory may not be reversible after prolonged abstinence.
 - Increased risk of an accident is possible if a person drives a motor vehicle while intoxicated with cannabis.
 - Chronic bronchitis and histopathologic changes may be precursors to the development of malignant disease in the oral cavity, throat, or chest.

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Miscellaneous

Medical/Legal Pitfalls

- The Drug Enforcement Administration issued a final order in March 1992, stating that the plant marijuana has no currently accepted medical use. It remains a Schedule I controlled substance. Despite this, advocates in the scientific community argue that marijuana is both efficacious and safe for use in treatment of a number of medical conditions. In 1999, the Institute of Medicine concluded that scientific data support certain therapeutic uses for cannabinoid-based drugs including pain relief, nausea and vomiting, and appetite stimulation. They declared that smoking marijuana is a crude cannabinoid delivery system that also delivers harmful substances.
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Pictures



Picture 1: Flowering top of cannabis plant

Picture type: Photo

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CBRNE - Incapacitating Agents, Lsd

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Synonyms, Key Words, and Related Terms

D-lysergic acid diethylamide, LSD-25, CAS No 50-37-3, Chemical Abstracts Service No 50-37-3, Registry of Toxic Effects of Chemical Substances No KE4100000, RTECS No KE4100000, cid, acid, boomers, yellow sunshine, beast, heavenly blue

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Introduction

Background

Lysergic acid diethylamide (LSD) is a synthetic hallucinogen derived from the naturally occurring alkaloid lysergic acid, which is produced by the rye fungus *Claviceps purpurea*. Other natural sources of lysergic acid include morning glory seeds and the Hawaiian baby woodrose. LSD is a white, odorless,

crystalline material that first was synthesized by the Swiss chemist Albert Hofmann in 1938. During the 1950s and early 1960s, the US Army's Chemical Corps investigated the use of LSD as an incapacitating agent, one that caused temporary behavioral changes of such magnitude that soldiers were unable to perform their regular duties for hours to days. However, it ultimately was felt to have no practical military application.

The same mind-altering and mind-expanding properties that made LSD appealing as an incapacitating agent led to its use as a psychotherapeutic agent during the 1950s. It was used to help patients overcome inhibitions and explore their subconscious mind. However, its application in psychotherapy decreased as it became a popular drug of abuse in the 1960s. It currently is categorized as a schedule I drug, indicating high abuse potential, no known medical application, and questionable safety. LSD use is currently on the rise, and it has emerged as a rave party or club drug.

Pathophysiology

As a hallucinogen, LSD is 100 times more potent than psilocybin and 5000 times more potent than mescaline. It is believed to induce hallucinations by acting as a partial agonist at the serotonin (5-hydroxytryptamine or 5-HT) receptor 2A subtype in the cerebral cortex. LSD also has sympathomimetic properties.

The most common route of exposure is oral, and it is absorbed rapidly from the GI tract. Dermal absorption has not been well documented. LSD can be aerosolized and is absorbed by the lungs provided the particle diameter is 5 micrometers or less. Metabolism is primarily via hydroxylation and conjugation in the liver, with conjugates excreted in the stool. Tolerance develops after 3-4 daily doses. Full sensitivity returns after a similar drug-free interval. No physical dependence and no withdrawal occur.

Frequency

- **In the US:** According to National Institute on Drug Abuse (NIDA) data, 13-17 million Americans have used a hallucinogen at least once. NIDA data from 1999 indicate that 8.1% of high school seniors used LSD within the previous 12 months. In one study reported by Johnston and Schwartz of high school-aged and college-aged students, LSD was the third most commonly abused substance, after alcohol and marijuana.
- **Internationally:** No data on international use are available.

Mortality/Morbidity

Very few deaths are attributed exclusively to the pharmacologic effects of LSD. Deaths associated with LSD use are often from trauma resulting from risk-taking behavior while intoxicated.

Race

In the US, LSD is used predominantly by whites. Use by African Americans is uncommon.

Sex

Males use LSD more frequently than females. The typical LSD user is a middle-class, risk-taking, white male in high school or college.

Age

Of LSD users seen in emergency departments, 50% are younger than 20 years.

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Clinical

History

The most common route of exposure is oral. LSD frequently is sprayed onto small squares of paper (ie, "blotter acid") that are decorated with a variety of patterns. Both the drug and paper are eaten. Current street doses typically are 20-80 mcg, considerably less than those used in the 1960s and 1970s. LSD also is sold as tiny tablets (microdots), thin squares of gelatin (windowpanes), liquid, or powder. It may be insufflated, smoked, injected, used sublingually, or instilled into the conjunctiva.

- Mental effects develop in 30-90 minutes, peak in 3-5 hours, and last 8-12 hours. These include the following:
 - Feeling of inner tension, often relieved by laughing or crying
 - Multiple, simultaneous emotions, such as joy, rage, terror, or panic
 - Religiosity and a feeling of "oneness with the universe"
 - Possible distorted perception of the passage of time
 - Possible magnification or distortion of sounds
 - Illusions (or hallucinations with high doses)
 - Moving patterns of bright colors on people and objects
 - Geometric images within larger images
 - Trails behind moving objects
 - Halos around objects
 - Shapes blending together or melting like wax

Physical

- Predominantly sympathomimetic effects develop within 5-10 minutes of ingestion. Findings include the following:
 - Profound mydriasis
 - Hyperactive reflexes
 - Tachycardia
 - Hypertension
 - Tremors
 - Vomiting
 - Diarrhea
 - Piloerection
 - Mild pyrexia
 - Seizures (rare and typically with doses >10 mcg/kg)
 - Intact orientation and cognition

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Differentials

Panic Disorders

Other Problems to be Considered

Toxins

Other psychedelics or entactogens

N,N-dimethyltryptamine (DMT)

4 methyl-2,5-dimethoxyamphetamine (STP, DOM, serenity, tranquility, peace)

3,4-methylenedioxyamphetamine (MDA)

3,4-methylenedioxymethamphetamine (MDMA, ecstasy, Adam)

3,4-methylenedioxyethamphetamine (MDEA, Eve)

Mescaline

Psilocybin

Phencyclidine (PCP)

Anticholinergics (atropine, Jimsonweed)

Sympathomimetics (cocaine, amphetamines)

Others - Steroids, yohimbine, bufotenine (toads), nutmeg, or other hallucinogenic mushrooms

Sedative-hypnotic withdrawal (ethanol, benzodiazepines, barbiturates, gamma-hydroxybutyrate [GHB])

Metabolic causes

Hypoxia

Hypoglycemia

Electrolyte abnormalities

CNS structural lesions

Trauma

Neoplasms

Functional

Schizophrenia

Psychosis

Other

Heat-related illnesses

CNS Infection

Thyrotoxicosis

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Workup

Lab Studies

- Diagnosis is made predominantly by history and physical examination. Direct diagnostic testing at identifying complications or excluding comorbidity.
 - Routine urine or serum drug screens do not detect LSD. Specialized methods for confirmation exist but are not performed in most hospital laboratories. A radioimmunoassay is available for detecting LSD and its major metabolite, 2-oxy-LSD, in the urine. Urine remains positive for LSD up to 24-36 hours after ingesting 200-400 mg. However, given an accurate history, laboratory confirmation of LSD intoxication rarely is necessary. Levels in the urine do not correlate with severity of symptoms.
 - Coagulation studies, total creatine phosphokinase, or serum electrolytes may be indicated in patients with seizures, coma, or a neuroleptic malignant syndromelike presentation to identify coagulopathy or rhabdomyolysis or to exclude other diagnoses. Platelet dysfunction and associated bleeding have been reported in patients with large LSD overdoses.

Imaging Studies

- Imaging studies such as radiographs or CT scans rarely are necessary. However, they may aid in identifying complications of LSD use or in excluding other diagnoses.

Other Tests

- ECG may be appropriate if co-ingestion is possible or to exclude other causes of tachycardia.

Procedures

- Lumbar puncture may be indicated to exclude meningitis or encephalitis.

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Treatment

Prehospital Care

Direct prehospital care toward supporting the vital signs. Obtaining vascular access, administering oxygen, and cardiac monitoring may be appropriate in severely intoxicated patients. Make an attempt to provide a quiet environment. Prehospital providers should obtain as thorough a history as possible and examine the patient for signs of co-ingestion.

Emergency Department Care

Most patients evaluated by medical personnel after using LSD are experiencing a "bad trip." Emergency department care is primarily supportive and directed at alleviating the patient's symptoms.

- Specifically, management priorities include searching for other causes of altered mental status, attending to the patient's safety, and achieving adequate sedation to prevent complications such as rhabdomyolysis or hyperthermia.
- Gut decontamination rarely is appropriate, unless the patient presents early after co-ingesting a potentially life-threatening substance. Administration of activated charcoal may be indicated to treat co-ingestants.
- Place the patient in a quiet room to minimize sensory input. In many cases, establishing verbal rapport with patients makes it possible to "talk them down," eliminating the need for pharmacologic intervention. Attempt to define reality for the patient, making it clear that the patient's hallucinations are from the drug and are not real.

Consultations

The need for consultation is dictated by the clinical situation. Most patients can be treated supportively and discharged. In some situations, consultation with a medical toxicologist or regional poison control center may be appropriate.

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Medication

Pharmacologic intervention may be required when a quiet environment and reassurance cannot control the patient's behavior or symptoms.

Anxiolytics

Benzodiazepines are indicated to control behavior and other autonomic signs and symptoms refractory to

a quiet environment and "talking the patient down." Unlike antipsychotics, benzodiazepines do not lower the seizure threshold, a theoretical advantage in patients manifesting severe LSD toxicity. Sedation should be titrated carefully by the physician at the bedside.

| | |
|-------------------|---|
| Drug Name | Diazepam (Valium)- Successfully used for decades to treat patients with signs and symptoms of severe LSD toxicity; no head-to-head study has compared the efficacy of diazepam with lorazepam in this setting; either is considered an appropriate agent. |
| Adult Dose | 5-10 mg IV or 10-20 mg PO; should not be given IM |
| Pediatric Dose | 0.05-0.1 mg/kg IV; should not be given IM |
| Contraindications | Documented hypersensitivity, acute angle-closure glaucoma (theoretical issue; no documented cases exist) |
| Interactions | Elimination decreased by drugs that inhibit hepatic metabolism, including cimetidine, disulfiram, fluoxetine, fluvoxamine, isoniazid, ketoconazole, propoxyphene, and valproic acid |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Although pregnancy class D, no specific fetal malformations are attributed to diazepam (concern is that potentially decreased alertness and floppiness in the neonate may occur if given close to time of delivery); caution in respiratory insufficiency, hypotension, elderly patients, or those with liver disease |

| | |
|-------------------|--|
| Drug Name | Lorazepam (Ativan)- Effective alternative to diazepam for treatment of patients with signs and symptoms of severe LSD toxicity. |
| Adult Dose | 1-2 mg IV/IM |
| Pediatric Dose | 0.05 mg/kg IV/IM |
| Contraindications | Documented hypersensitivity, acute angle-closure glaucoma (theoretical issue; no documented cases exist), preexisting CNS depression, hypotension |
| Interactions | Toxicity of benzodiazepines in CNS increases when used concurrently with alcohol, phenothiazines, barbiturates, and MAOIs; clearance increased by oral contraceptives |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Although pregnancy class D, no specific fetal malformations are attributed to diazepam (concern is primarily potential decreased alertness and floppiness in the neonate if given close to time of delivery); caution in respiratory insufficiency and hypotension |

Antihypertensives

The antihypertensive agent clonidine has been shown to attenuate some signs and symptoms of LSD

toxicity.

| | |
|-------------------|---|
| Drug Name | Clonidine (Catapres)- Recently demonstrated to decrease severity of flashbacks and attenuate increased sympathetic activity associated with LSD use. |
| Adult Dose | 0.025 mg PO tid for 2 mo |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | TCAs inhibit hypotensive effects; coadministration with beta-blockers may potentiate bradycardia; TCAs may enhance hypertensive response associated with abrupt clonidine withdrawal; hypotensive effects are enhanced by narcotic analgesics |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Abrupt discontinuation can lead to severe rebound hypertension; caution in coronary artery disease, recent MI or stroke, or chronic renal insufficiency |

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Follow-up

Further Inpatient Care

- Admission is warranted in the setting of serious comorbidity or if the patient is severely intoxicated, requires prolonged observation, or is suicidal.

Further Outpatient Care

- Most patients are discharged after a period of observation. Discharge patients into the care of a responsible adult. Carefully explain discharge instructions to the patient and accompanying friends or family members. In appropriate cases, refer patients for outpatient drug counseling.

in/Out Patient Meds

- Admitted patients may warrant continued administration of anxiolytics or other medications directed at specific symptoms. Outpatient medications rarely are necessary.

Transfer

- As most patients only require a period of observation, transfer rarely is necessary. However, transfer may be justified in situations of serious complications or comorbidity or when management of behavioral symptoms exceeds the capability of the facility.

Deterrence/Prevention

- The only feasible deterrence is abstinence.
- "Mind Over Matter," an educational tool designed to encourage young people in grades 5-9 to learn about the effects of drug abuse on the body and brain, is available at www.nida.nih.gov/mom/momindex.html.

Complications

- Complications include persistent or recurrent affective disorders (eg, depression), although these are usually reversible.
- LSD exacerbates preexisting psychiatric illness (eg, psychosis).
- The drug disrupts personality.
- Hallucinogen persistent perceptual disorder (HPPD) or "flashbacks" are reported in 15-77% of LSD users, last minutes to hours, and tend to occur during times of psychological stress. With cessation of LSD use, flashbacks tend to stop with time. However, in one study by Abraham, 50% of subjects experienced flashbacks 5 or more years after they stopped using LSD. Flashbacks are characterized by visual disturbances including the following:
 - Geometric hallucinations
 - Flashes of color
 - Moving light
 - Terrifying illusions of people decomposing, crawling bugs or skulls, Satan's face superimposed on the faces of friends, or objects melting
 - Impaired color perception

Prognosis

- The long-term prognosis for LSD users is good, provided they stop using the drug.

Patient Education

- Counsel patients on the potential dangers of LSD use, including driving automobiles while intoxicated or combining LSD ingestion with ethanol, marijuana, or other illicit drugs.

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Miscellaneous

Medical/Legal Pitfalls

- In most patients, the diagnosis of LSD use is made on the basis of history and physical examination. However, if the clinical picture is unclear, seek other explanations for the patient's symptoms.

Special Concerns

- Pregnant patients: Despite early reports of LSD-related fetal malformations, inadequate evidence exists to establish causality.
- Pediatric patients: The relatively low cost of a dosage unit of LSD blotter paper (\$5), its widespread availability, and the artful designs make it very popular with adolescents. However, LSD intoxication in very young children suggests the possibility of child abuse.
- Geriatric patients: LSD potentially may exacerbate comorbid conditions in elderly patients.
- Patients taking selective serotonin reuptake inhibitors or lithium have greater potential for LSD-related complications such as seizures or flashbacks.

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CBRNE - Incendiary Agents, Magnesium and Thermite

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Introduction

Background

Two major types of metal incendiaries exist, those that are magnesium based and those of the thermite/thermate type. Most types generally are encountered only in the military or industrial setting.

Magnesium, a silvery white metal of atomic weight 24.32, ignites at 632°C and burns at 1982°C, with magnesium oxide (MgO) as its combustion product. In an exothermic reaction, metallic magnesium can ignite to produce magnesium dihydroxide, ie, Mg(OH)₂, and hydrogen. Magnesium is used in either powdered or solid form as an incendiary agent for both illumination and antipersonnel purposes. Various alloys of magnesium (eg, aluminum/zinc/magnesium alloy found in US M126 round) are mechanically sturdier but also can be ignited easily.

Thermite is a mixture of powdered or granular aluminum and powdered iron oxide. When combined with other substances, such as binders, the material is termed a "thermate." All such materials react vigorously when heated to the combustion temperature of aluminum. This reaction produces aluminum oxide, elemental iron, and sufficient heat to melt the iron. The reaction temperature is approximately 2200°C.

Since the burning temperature of these chemicals is so high, standard hazardous-materials clothing (even Level A self-contained and chemical-proof clothing) is not protective.

Pathophysiology

Burning thermite or magnesium produces predominantly thermal injury, but residual particles (especially of magnesium) may produce chemical injury to the eyes, skin, and respiratory tract.

Most injuries are thermal burns, which may be considered identical to deep partial or full-thickness thermal burns (see [Burns, Thermal](#)).

Mortality/Morbidity

Outcome of thermite or ignited magnesium burns is essentially the same as for identical thermal burns. [Top](#)

Clinical

History

The history usually makes the nature of the exposure evident, as the patient or rescuer describes the circumstances leading to exposure to thermite or magnesium incendiaries. In the event that a patient presents with burn injury and is unable to give a history, consider exposure to magnesium, thermite, or other hazardous materials.

Obtain the patient's relevant medical history. In decision making, consider diseases that may affect healing (eg, diabetes mellitus, vascular disease) as well as drug allergies.

Physical

Incendiary agents produce predominantly dermatologic and respiratory effects.

- Vital signs
 - As with all resuscitations, first priority is to maintain and support airway, breathing, and circulation (ABC). Patients with airway burns or significant fume exposure may require endotracheal intubation and ventilatory support. Acute respiratory distress syndrome

(ARDS) may develop.

- Patients with significant dermal burns require aggressive fluid resuscitation, following a formula such as the Parkland burn resuscitation guidelines, and require monitoring of urinary output and other vital signs.
- Thermal burns
 - Dermal exposure to incendiary agents produces thermal burns. Thermite burns, being predominantly due to molten iron, essentially are thermal burns with minimally reactive metal particles embedded in the tissue. Assume that these burns are deep partial thickness or full thickness until proven otherwise.
 - Magnesium particle reactions with tissue fluid also may produce magnesium dihydroxide, which produces an alkali chemical burn.
 - Retained magnesium particles in skin may produce a lesion that mimics gas gangrene, with tissue death and intratissue gas bubbles due to hydrogen gas formed from the same reaction.

Causes

Exposure to thermite or burning magnesium likely would occur in the context of military or paramilitary actions (including terrorist activities) or as a result of an industrial or scientific laboratory accident. Exposure potentially could occur as a result of a transportation accident.

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Differentials

Acute Respiratory Distress Syndrome
Burns, Chemical
Burns, Ocular
Burns, Thermal
CBRNE - Chemical Warfare Agents
Corneal Abrasion
Gas Gangrene
Hazmat

Other Problems to be Considered

Metal fume fever

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Workup

Lab Studies

- Order lab studies as needed to manage thermal burns and associated lung injury. No specific studies are required for thermite or ignited magnesium exposure.

Imaging Studies

- Perform chest radiography on patients with possible pulmonary involvement.

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Treatment

Prehospital Care

- Remove patients from the burning environment, with appropriate attention to personal safety.
- Flush thermite burns with water and debride them to remove contaminating particles.
- Initial care for magnesium burn wounds should include removal of all unburned particles by mechanical means, including wound debridement, if needed. If particles are present, do not flush with water until particles have been removed. If water irrigation is needed for burn treatment or other decontamination, use copious amounts to rapidly flush away residual magnesium before the resulting chemical reaction can cause harm.
- Treat burns with standard thermal burn techniques. Undertake standard support of the ABCs, including intubation and fluid resuscitation if needed.
- Cover burned areas with dry sterile dressings. Avoid large areas of wet dressings due to the risk of hypothermia.
- Narcotic analgesia may be useful if the patient's hemodynamic status permits.

Emergency Department Care

- Institute airway support.
- Start fluid resuscitation, guided by formulas for similar thermal burns.
- Perform wound debridement to remove residual particles of magnesium or iron.
- Aggressively seek and treat associated traumatic injuries (eg, from blast).

- Institute analgesia.
- Consider all incendiary burns tetanus prone and administer appropriate tetanus prophylaxis.

Consultations

- A burn surgeon or other appropriate surgeon (eg, plastics, trauma) should be involved in care.
- Consult an ophthalmologist if eye injury has occurred.
- Continuing critical care expertise may be required if injury severity is high.

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Medication

Major drugs of use are fluids for resuscitation, oxygen for respiratory support, tetanus prophylaxis, and analgesia. Follow standard therapeutic protocols for [thermal burn](#) injury. Antibiotic therapy, including topical agents (eg, silver sulfadiazine) and IV or oral agents, may be needed.

Gases

To support respiration and metabolism.

| | |
|-------------------|---|
| Drug Name | Oxygen- Used to support respiration and metabolism. |
| Adult Dose | 100% oxygen inhaled; reduce to 60% or less as soon as tolerated to minimize oxygen toxicity |
| Pediatric Dose | Administer as in adults |
| Contraindications | COPD, oxygen toxicity |
| Interactions | None reported |
| Pregnancy | A - Safe in pregnancy |
| Precautions | Inspired oxygen concentrations from 50-100% carry a substantial risk of lung damage |

Electrolytes

Used to maintain hydration and salt balance.

| | |
|-------------------|--|
| Drug Name | Lactated Ringer with normal saline- Usually crystalloids such as normal saline or Ringer lactate; little indication for colloid use in acute burn management. |
| Adult Dose | For resuscitation, 2-4 cm ³ /kg per percent of TBSA burned to partial thickness or deeper; administer one half of this amount over 8 h and one half over next 16 h; adjust based on central venous pressure, systolic blood pressure, and urine output |
| Pediatric Dose | Administer as in adults |
| Contraindications | Major complication of isotonic fluid resuscitation is interstitial edema; edema of extremities is unsightly but not a significant complication; edema in brain or lungs is potentially fatal; major contraindication to isotonic fluid resuscitation is pulmonary edema; added fluid promotes more edema and may lead to development of ARDS |
| Interactions | None reported |
| Pregnancy | A - Safe in pregnancy |
| Precautions | Isotonic fluids administered during resuscitation of septic shock require close monitoring of cardiovascular and pulmonary function; stop fluids when desired hemodynamic response is seen or pulmonary edema develops |

Topical Burn Treatment

Topical burn-healing and antimicrobial properties.

| | |
|-------------------|---|
| Drug Name | Silver sulfadiazine (Silvadene)- Contains both a sulfa antibiotic and a silver ion, which is an antibacterial; speeds burn healing and eases debridement. |
| Adult Dose | Topical application to burned area q12h |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity, G-6-PD deficiency |
| Interactions | Effect of proteolytic enzymes is reduced when used concomitantly with this product |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in G-6-PD deficiency and renal insufficiency; may cause "tattooing" on the face and should not be used on facial burns in most situations |

Topical Antibiotics

Antibacterial and to aid in burn healing.

| | |
|-----------|---|
| Drug Name | Bacitracin (AK-Tracin, Baciquent)- Mild topical antibiotic, usually in an ointment base, for use on facial burns not deep enough to require grafting. |
|-----------|---|

| | |
|-------------------|--|
| Adult Dose | Apply topically qid |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; vaccinia, varicella, epithelial herpes simplex keratitis, mycobacterial infections, and fungal diseases of the eye; patients using steroid combinations after uncomplicated removal of a corneal foreign body |
| Interactions | None reported |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Ophthalmic ointments may delay healing of corneal epithelia; in deep-seated eye infections, supplement with systemic medications; prolonged use may result in overgrowth of nonsusceptible organisms |

Immunizing Agents

Used to immunize patients against tetanus.

| | |
|-------------------|---|
| Drug Name | <p>Tetanus toxoid- Used to induce active immunity.</p> <p>Immunizing agents of choice for most adults and children >7 y are tetanus and diphtheria toxoids. Necessary to administer booster doses to maintain tetanus immunity throughout life.</p> <p>Pregnant patients should receive only tetanus toxoid, not a diphtheria antigen-containing product.</p> <p>In children and adults, may administer into deltoid or midlateral thigh muscles. In infants, preferred site of administration is the mid thigh laterally.</p> |
| Adult Dose | <p>Primary immunization: 0.5 mL IM; give 2 injections 4-8 wk apart and a third dose 6-12 mo after second injection</p> <p>Booster dose: 0.5 mL q10y</p> |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; a history of any type of neurologic symptoms or signs following administration of this product; FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are an important cause of provocative poliomyelitis |

| | |
|--------------|--|
| Interactions | Patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization due to poor immune response; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent use of medication with systemic chloramphenicol since it may impair amnestic response to tetanus toxoid; concurrent use of tetanus immune globulin may delay development of active immunity by several days (interaction is nevertheless clinically insignificant and does not preclude concurrent use) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Do not use to treat actual tetanus infections or for immediate prophylaxis of unimmunized individuals (use instead tetanus antitoxin, preferably human tetanus immune globulin); diminished antibody response to active immunization may be seen in patients receiving immunosuppressive therapy; better to defer primary diphtheria immunization until immunosuppressive therapy discontinued; routine immunization of symptomatic and asymptomatic HIV-infected persons is recommended |

| | |
|-------------------|---|
| Drug Name | Tetanus immune globulin (Hyper-Tet)- Used for passive immunization of any person with a wound that may be contaminated with tetanus spores. |
| Adult Dose | Prophylaxis: 250-500 U IM in opposite extremity to tetanus toxoid lesion Clinical tetanus: 3000-10,000 U IM |
| Pediatric Dose | Prophylaxis: 250 U IM in opposite extremity to tetanus toxoid Clinical tetanus: 3000-10,000 U IM |
| Contraindications | Since antibodies in globulin preparation may interfere with immune response to vaccination, do not administer within 3 mo of live virus immune globulin administration; may be necessary to revaccinate persons who received immune globulin shortly after live virus vaccination |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Persons with isolated IgA deficiency have potential for developing antibodies to IgA and may have anaphylactic reactions to subsequent administration of blood products that contain IgA; do not perform skin testing since intradermal injection of concentrated gamma globulin may cause localized area of inflammation and can be misinterpreted, causing medication to be withheld from a patient not allergic to this material; true allergic responses to human gamma globulin given in prescribed IM manner are extremely rare; do not admix with other medications since usually incompatible |

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Follow-up

Further Inpatient Care

- Inpatient care is identical to care for other thermal burns, and it usually involves topical antibiotics (eg, silver sulfadiazine) and surgical debridement. Skin grafting may be needed; institute life-support measures as necessary.

Further Outpatient Care

- Outpatient care is identical to care for other thermal burns. A physician experienced in burn management usually should provide follow-up care for patients. Treatment may include dressings, topical antibiotics, analgesia, and grafting.

in/Out Patient Meds

- Medications are standard therapies for thermal burn care and analgesia.

Transfer

- Transfer patients with thermal burns to a burn center if they meet any of the following burn center criteria:
 - Partial thickness burns over 20% body surface area
 - Full-thickness burns over 10% body surface area
 - Burns involving hands, feet, eyes, ears, and/or perineum
 - Airway involvement
 - Significant underlying illness
 - Age younger than 1 year or older than 65 years

Prognosis

- Prognosis depends on the extent of the burn injury, the underlying medical history of the victim, and the extent of care available.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to involve an appropriate burn specialist early in the care of patients with burn wounds may result in poor prognosis and poor functional or cosmetic outcome.
- Failure to seek associated traumatic injuries (eg, from blast) can lead to significant morbidity or mortality.
- Failure to investigate underlying conditions or drug allergies.
- Failure to administer tetanus prophylaxis as needed.
- Failure to perform adequate fluid resuscitation.

Special Concerns

- Special concerns mirror those for any burn injury. Patients with poor nutrition, underlying illness retarding healing, immunocompromise, or of extremes of age have a poorer prognosis from such injuries.

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CBRNE - Incendiary Agents, Napalm

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Introduction

Background

Napalm, invented by Fieser in 1942, is an incendiary substance made by the simple procedure of adding a "gelling" powder, composed of naphthenate and palmitate (hence "napalm"), to gasoline in varying concentrations to form a sticky, combustible substance.

This white, cloudy, jellylike substance has unique properties that render it an effective incendiary agent. Napalm is extremely stable, tolerating temperatures well above 150°F (effective in the tropics) and as low as -40°F (bomb shelters, cold weather environments). It is not shattered easily by explosives and can be stored for long periods without significant breakdown. Gelation of this substance occurs in 3-20 minutes. Gel formation enhances its effectiveness by allowing for a controlled, contained, and prolonged burn. Gelation also enhances its stability, with napalm requiring much higher temperatures to ignite than gasoline. There is no "off-sourcing" of hydrocarbon fumes associated with the nonignited compound. In fact, ignition requires the use of trinitrotoluene (TNT) to explode and ignite white phosphorus, the ignited temperature of which is high enough to result in the combustion of napalm.

Napalm has been used primarily in the form of incendiary bombs, firebombs, land mines, and flamethrowers. During World War II, firebombs, in the form of 165-gallon containers, were the primary method for the disbursement of napalm. One firebomb released from a low-flying airplane was capable of producing damage to a 2500-yd² area. During the Korean War, the US dropped approximately 250,000 pounds of napalm per day. Napalm's increased viscosity resulted in the enhanced efficacy of flamethrowers, frequently used in World War II. Because of gasoline's increased instability, volatility, and its rapid burning and self-consumption, its effectiveness was limited to within 30 yards. Napalm, through its unique properties, extended the effective range of flamethrowers to 150 yards.

After World War II, the US conducted an intensive effort to enhance the properties and effectiveness of napalm as an incendiary agent. This effort resulted in the development of napalm B, which substituted polystyrene and benzene for naphthenate and palmitate. The resulting substance continued to bear the name "napalm" although it lacked the 2 components of its namesake. Napalm B provided the US with an incendiary substance with enhanced stability and controllability and as such, became the weapon of choice during the Vietnam War. White phosphorus, as the igniting agent, was replaced by thermitite, which burns at a higher temperature of 4532°F.

Frequency

- **In the US:** Napalm has not been used as an agent of war since the Vietnam War.

Mortality/Morbidity

Morbidity and mortality are related directly to the extent of injuries received from trauma and extensive burns from exposure. No cases have been reported of systemic poisoning of individuals in contact with nonignited napalm.

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Clinical

History

- Exposure history usually is quite obvious, with the individual recounting the sounds of an explosion and the unbearable pain associated with the burns of exposure.
- Napalm produces carbon monoxide as a by-product of combustion. Thus, also evaluate individuals exposed to burning napalm for carbon monoxide exposure. In particular, consider individuals who are found with altered levels of consciousness in the vicinity of burning napalm

to have been exposed to toxic levels of carbon monoxide until proven otherwise.

Physical

- Severe burns (second and/or third degree) in areas exposed to burning napalm frequently are found. Injuries related to the thermal elevation of the air temperature may result in respiratory embarrassment.
- Burning napalm raises the ambient environmental temperature and has been known to cause the deaths of individuals in raid shelters as a result of radiant heat and dehydration. This was a frequent cause of death in the bombing raids carried out over Hamburg, Germany, during World War II. The result of this phenomenon frequently was referred to as Bombenbrandschrumpfleichen (incendiary-bomb-shrunken bodies).

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Differentials

Burns, Thermal
Pediatrics, Pharyngitis
Pediatrics, Pneumonia
Pediatrics, Respiratory Distress Syndrome
Pharyngitis
Pneumonia, Bacterial
Respiratory Distress Syndrome, Adult
Sunburn
Toxicity, Hydrocarbons
Toxicity, Toluene

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Workup

Lab Studies

- Patients exposed to napalm represent individuals with severe burns; perform laboratory evaluation as for any burn patient.

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Treatment

Prehospital Care

Give care to extinguishing flames and removing smoldering napalm from the skin. Remove contaminated clothing to prevent continued burning from hot napalm. If possible carbon monoxide exposure is a concern, provide 100% oxygen via nonrebreather mask en route.

Emergency Department Care

Rapid intervention to stop cutaneous burning from napalm is of paramount importance. As with all burn patients, provide respiratory support and multiorgan evaluation.

- Follow the standard ABC approach to resuscitation, paying special attention to respiratory evaluation, since patients may experience severe respiratory injury secondary to elevated ambient air temperature.
- Perform full exposure and removal of the offending agent.
- Evaluate burns and calculate the exposed area. This can be done by either 1 of 2 common methods. The first involves using an affected individual's palmar surface, which roughly represents 1% body surface area (BSA) of that individual. The second uses the "rule of nines" method.
 - Percentage of BSA involved assists in determining disposition and/or transfer of the patient to a regional burn center. The American Burn Association has developed criteria for burn center admission that include third-degree burns over 5% BSA; second-degree burns over 10% BSA; any second-degree and/or third-degree burns involving critical areas (eg, face, hands, feet, genitals); circumferential burns of thorax or extremities; inhalational injuries; and significant chemical injuries, electrical burns, trauma, or significant preexisting medical conditions.
 - Base fluid resuscitation on the Parkland formula (2-4 cc/kg/h of intravenous crystalloid). Maintain urine output at 1-2 cc/kg/h.
- Take care to evaluate patients for carbon monoxide exposure.

Consultations

- Consult the burn team for evaluation and management of burns.
- Consult the trauma team for the evaluation and management of traumatic injuries received as a consequence of explosions associated with napalm disbursement.

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Medication

Implement medical therapy as indicated by the patient's medical condition. Remember to administer tetanus prophylaxis.

Toxoid

Used for immunization; a booster injection in previously immunized individuals is recommended.

| | |
|-------------------|---|
| Drug Name | Tetanus toxoid- Used to induce active immunity against tetanus in selected patients. The immunizing agents of choice for most adults and children >7 y are tetanus and diphtheria toxoids. Necessary to administer booster doses to maintain tetanus immunity throughout life. Pregnant patients should receive only tetanus toxoid, not a diphtheria antigen-containing product. In children and adults, may administer into deltoid or midlateral thigh muscles. In infants, preferred site of administration is the mid thigh laterally. |
| Adult Dose | Primary immunization: 0.5 mL IM; give 2 injections 4-8 wk apart; third dose 6-12 mo after second injection Booster dose: 0.5 mL q10y |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; history of any type of neurologic symptoms or signs following administration; FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are an important cause of provocative poliomyelitis |
| Interactions | Patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization due to poor immune response; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent use of medication with systemic chloramphenicol, since it may impair amnestic response to tetanus toxoid; concurrent use of tetanus immune globulin may delay development of active immunity by several days (interaction nevertheless is clinically insignificant and does not preclude concurrent use) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|--|
| Precautions | Do not use to treat actual tetanus infections or for immediate prophylaxis of unimmunized individuals (use instead tetanus antitoxin, preferably human tetanus immune globulin); diminished antibody response to active immunization may be observed in patients receiving immunosuppressive therapy; better to defer primary diphtheria immunization until immunosuppressive therapy discontinued; routine immunization of symptomatic and asymptomatic HIV-infected persons is recommended |
|-------------|--|

Analgesics

Ensure patient comfort, promote pulmonary toilet, and have sedating properties, which are beneficial for patients who have sustained injuries.

| | |
|-------------------|---|
| Drug Name | Morphine sulfate (Duramorph, Astramorph, MS Contin, MSIR, Oramorph)- DOC for analgesia due to reliable and predictable effects, safety profile, and ease of reversibility with naloxone. Various IV doses are used; commonly titrated until desired effect obtained. |
| Adult Dose | Starting dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h Relatively hypovolemic patients: Start with 2 mg IV/IM/SC; reassess hemodynamic effects of dose |
| Pediatric Dose | Infants and children: 0.1-0.2 mg/kg dose IV/IM/SC q2-4h prn; not to exceed 15 mg/dose; may initiate at 0.05 mg/kg/dose |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway where establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects of opiate agonists; TCAs, MAOIs, and other CNS depressants may potentiate adverse effects of morphine |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

| | |
|----------------|--|
| Drug Name | Meperidine (Demerol)- Analgesic with multiple actions similar to those of morphine; may produce less constipation, smooth muscle spasm, and depression of cough reflex than similar analgesic doses of morphine. |
| Adult Dose | 50-150 mg PO/IV/IM/SC q3-4h prn |
| Pediatric Dose | 1-1.8 mg/kg (0.5-0.8 mg/lb) PO/IV/IM/SC q3-4h prn; not to exceed adult dose |

| | |
|-------------------|--|
| Contraindications | Documented hypersensitivity; MAOIs; upper airway obstruction or significant respiratory depression; during labor when delivery of premature infant is anticipated |
| Interactions | Monitor for increased respiratory and CNS depression with coadministration of cimetidine; hydantoins may decrease effects of meperidine; avoid with protease inhibitors |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients with head injuries since meperidine may increase respiratory depression and CSF pressure (use only if absolutely necessary); caution when using postoperatively and with history of pulmonary disease (suppresses cough reflex); substantially increased dose levels, due to tolerance, may aggravate or cause seizures even if no history of convulsive disorders; monitor closely for morphine-induced seizure activity if seizure history |

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

Have analgesic, antiinflammatory, and antipyretic activities. Mechanism of action is not known, but may inhibit cyclooxygenase activity and prostaglandin synthesis. Other mechanisms may exist as well, such as inhibition of leukotriene synthesis, lysosomal enzyme release, lipoxygenase activity, neutrophil aggregation, and various cell-membrane functions.

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Motrin, Ibuprin)- DOC for patients with mild to moderate pain. Inhibits inflammatory reactions and pain by decreasing prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h while symptoms persist; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 4-10 mg/kg/dose PO tid/qid >12 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and possibly toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT when taking anticoagulants (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Naproxen (Aleve, Naprelan, Naprosyn, Anaprox)- For relief of mild to moderate pain; inhibits inflammatory reactions and pain by decreasing activity of cyclo-oxygenase, which results in a decrease of prostaglandin synthesis. |
| Adult Dose | 500 mg PO followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and possibly toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT when taking anticoagulants (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

Topical Antibiotics

Therapy must be comprehensive and cover all likely pathogens in the context of this clinical setting.

| | |
|-------------------|--|
| Drug Name | Neomycin and polymyxin B (Neosporin)- Used in treatment of minor infections. Inhibits bacterial protein synthesis and growth. Polymyxin B disrupts bacterial cytoplasmic membrane, permitting leak of intracellular constituents and causing inhibition of bacterial growth. |
| Adult Dose | Apply qd/qid to affected areas |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|--|
| Precautions | Caution in treating extensive burns (>20% BSA) since absorption of neomycin is possible and may cause nephrotoxicity and ototoxicity; prolonged use may result in overgrowth of nonsusceptible organisms |
|-------------|--|

| | |
|-------------------|--|
| Drug Name | Silver sulfadiazine (Silvadene, Thermazene, SSD, SSD-AF)- Useful in prevention of infections from second-degree or third-degree burns. Has bactericidal activity against many gram-positive and gram-negative bacteria, including yeast. |
| Adult Dose | Apply qd/bid to a thickness of 1/16; burned area should be covered with medication continuously |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; neonates and infants <2 y |
| Interactions | Effect of proteolytic enzymes is reduced when used concomitantly with this product |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in G-6-PD deficiency and renal insufficiency |

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Follow-up

in/Out Patient Meds

- Administer medications as indicated by the patient's medical condition.

Transfer

- If indicated, transfer patients to a regional trauma and/or burn center.

Prognosis

- Prognosis is dictated by extent of physical injury, burns, and existing metabolic complications.

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Miscellaneous

Medical/Legal Pitfalls

- Legal issues are associated with inadvertent exposure to the ignited agent and to its inappropriate use (detonation) in situations other than wartime deployment.
 - Military personnel should be cognizant of the medical issues associated with exposure to ignited napalm and maintain an appropriate safe distance from any military use.
-

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CBRNE - Incendiary Agents, White Phosphorus

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Synonyms, Key Words, and Related Terms

yellow phosphorus, Willy P

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Introduction

Background

White phosphorus has been used commonly by the military as an incendiary agent or as an igniter for munitions. It commonly is found in hand grenades, mortar and artillery rounds, and smoke bombs.

Munitions-quality white phosphorus commonly is found in solid form. When exposed to air, it spontaneously ignites and is oxidized rapidly to phosphorus pentoxide. Such heat is produced by this reaction that the element bursts into a yellow flame and produces a dense white smoke. Phosphorus also becomes luminous in the dark, and this property is conveyed to "tracer bullets." This chemical reaction continues until either all the material is consumed or the element is deprived of oxygen.

Most injuries associated with white phosphorus are the result of accidents due to either human or mechanical error.

Pathophysiology

White phosphorus results in painful chemical burn injuries. The resultant burn typically appears as a necrotic area with a yellowish color and characteristic garliclike odor. White phosphorus is highly lipid soluble and as such, is believed to have rapid dermal penetration once particles are embedded under the skin. Because of its enhanced lipid solubility, many have believed that these injuries result in delayed wound healing. This has not been well studied; therefore, all that can be stated is that white phosphorus burns represent a small subsegment of chemical burns, all of which typically result in delayed wound healing.

Few studies have investigated the degree of tissue destruction associated with white phosphorus injuries. In the experimental animal model, most tissue destruction appears to be secondary to the heat generated by oxidation.

Systemic toxicity has been described extensively in the animal model. Pathologic changes have been documented in the liver and kidney. These changes result in the development of progressive anuria, decreased creatinine clearance, and increased blood phosphorus levels. Depression of serum calcium with an elevation in the serum phosphorus level (reversed calcium-phosphorus ratio) with electrocardiographic changes including prolongation of the QT segment, ST segment depression, T wave changes, and bradycardia also have been observed. Oral ingestion of white phosphorus in humans has been demonstrated to result in pathologic changes to the liver and kidneys. The accepted lethal dose is 1 mg/kg, although the ingestion of as little as 15 mg has resulted in death. Individuals with a history of oral ingestion have been noted to pass phosphorus-laden stool ("smoking stool syndrome").

Mortality/Morbidity

Morbidity and mortality are related directly to trauma and burns sustained from exposure.

- Burns usually are limited to areas of exposed skin (upper extremities, face). Burns frequently are second and third degree because of the rapid ignition and highly lipophilic properties of white phosphorus.
- Trauma usually is a combination of blunt and penetrating. Blunt trauma results from the percussion and force of the blast, and penetrating trauma results from projectiles produced from

the explosion.

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Clinical

History

- Since most exposures occur in the military setting with the use of munitions, history frequently is obtained easily.
- Be aware of unconscious individuals with a history of percussion injuries from white phosphorusâ€ containing munitions who may pose an exposure hazard to the health care provider.

Physical

- Direct the physical examination toward the identification of traumatic and burn injuries. Pay particular attention to areas where phosphorus may be embedded as a result of explosion.
- Fully expose the patient for the primary survey. Exercise care when handling potentially contaminated clothing to prevent secondary exposure and burns to the health care provider.

Causes

Most exposures to white phosphorus are accidental in etiology.

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Differentials

Acute Respiratory Distress Syndrome

Burns, Chemical

Burns, Ocular

Burns, Thermal

Dermatitis, Contact

Sunburn

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Workup

Lab Studies

- Obtain a basic trauma panel (CBC, basic metabolic panel, prothrombin time and/or activated partial thromboplastin time, type and crossmatch) with the addition of calcium, phosphorus, and magnesium levels.

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Treatment

Prehospital Care

Direct prehospital management toward the evaluation and management of trauma.

- Secure the scene, since live munitions may be in the area.
- Perform ABCs of resuscitation.
- Terminate further oxidation of phosphorus by irrigation or placement of saline-soaked and/or water-soaked pads on areas of exposure.
 - Do not use oily or greasy dressing, since the element is lipid soluble and can penetrate into the tissue.
 - Remove contaminated clothing, since it may re-ignite and cause more extended and worsened burns than those sustained with white phosphorus alone.

Emergency Department Care

Continue a trauma management approach to the patient.

- Avoid contact with ignited white phosphorus. Such contact may result in a chemical burn injury to the health care provider.
- Continue irrigation; do not allow areas of exposure to dry, as this may result in re-ignition of white phosphorus.
- Grossly debride as much white phosphorus as possible. The use of a Wood lamp (ultraviolet light) results in the fluorescing of the white phosphorus and may facilitate its removal.
- Solutions of copper sulfate traditionally have been used as a neutralizing agent. Copper sulfate

reacts with phosphorus to form cupric phosphate, which is black and assists in visualizing phosphorus. Stereomicroscopically, phosphorus particles have been observed to become covered with cupric phosphate, and this may facilitate their removal. This treatment has fallen out of favor because of reports of massive intravascular hemolysis associated with its use. This phenomenon is believed to be due to copper's activity as an inhibitor of several enzymes of the erythrocyte hexose monophosphate shunt.

- Take care to ensure that tetanus immunization is up to date as a standard component of burn therapy.

Consultations

Consultation with a burn team is mandatory for most patients. In addition, obtain trauma consultation for all patients with a history of significant trauma, especially those who may require surgical debridement of injuries.

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Medication

Direct medical therapy to the treatment of any underlying condition. As always, provide tetanus prophylaxis.

Toxoid

Used for immunization; a booster injection in previously immunized individuals is recommended.

| | |
|----------------|---|
| Drug Name | Tetanus toxoid- Used to induce active immunity against tetanus in selected patients. The immunizing agents of choice for most adults and children >7 y are tetanus and diphtheria toxoids. Necessary to administer booster doses to maintain tetanus immunity throughout life. Pregnant patients should receive only tetanus toxoid, not a diphtheria antigen-containing product. In children and adults, may administer into deltoid or midlateral thigh muscles. In infants, preferred site of administration is mid thigh laterally. |
| Adult Dose | Primary immunization: 0.5 mL IM; administer 2 injections 4-8 wk apart; third dose 6-12 mo after second injection Booster dose: 0.5 mL q10y |
| Pediatric Dose | Administer as in adults |

| | |
|-------------------|---|
| Contraindications | Documented hypersensitivity; history of any type of neurologic symptoms or signs following administration; FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are an important cause of provocative poliomyelitis |
| Interactions | Patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization due to poor immune response; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent use of medication with systemic chloramphenicol, since it may impair anamnestic response to tetanus toxoid; concurrent use of tetanus immune globulin may delay development of active immunity by several days (interaction nevertheless is clinically insignificant and does not preclude concurrent use) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Do not use to treat actual tetanus infections or for immediate prophylaxis of unimmunized individuals (use instead tetanus antitoxin, preferably human tetanus immune globulin); diminished antibody response to active immunization may be observed in patients receiving immunosuppressive therapy; better to defer primary diphtheria immunization until immunosuppressive therapy discontinued; routine immunization of symptomatic and asymptomatic HIV-infected persons is recommended |

Analgesics

Pain control is essential to quality patient care. Analgesics ensure patient comfort, promote pulmonary toilet, and have sedating properties, which are beneficial for patients who have sustained trauma.

| | |
|-------------------|---|
| Drug Name | Morphine sulfate (Duramorph, Astramorph, MS Contin, MSIR, Oramorph)- DOC for analgesia due to reliable and predictable effects, safety profile, and ease of reversibility with naloxone. Various IV doses are used; commonly titrated until desired effect obtained. |
| Adult Dose | Starting dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h Relatively hypovolemic patients: Start with 2 mg IV/IM/SC; reassess hemodynamic effects of dose |
| Pediatric Dose | Infants and children: 0.1-0.2 mg/kg dose IV/IM/SC q2-4h prn; not to exceed 15 mg/dose; may initiate at 0.05 mg/kg/dose |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |

| | |
|--------------|---|
| Interactions | Phenothiazines may antagonize analgesic effects of opiate agonists; TCAs, MAOIs, and other CNS depressants may potentiate adverse effects of morphine |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in hypotension, respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

| | |
|-------------------|--|
| Drug Name | Meperidine (Demerol)- Analgesic with multiple actions similar to those of morphine; may produce less constipation, smooth muscle spasm, and depression of cough reflex than similar analgesic doses of morphine. |
| Adult Dose | 50-150 mg PO/IV/IM/SC q3-4h prn |
| Pediatric Dose | 1-1.8 mg/kg (0.5-0.8 mg/lb) PO/IV/IM/SC q3-4h prn; not to exceed adult dose |
| Contraindications | Documented hypersensitivity; MAOIs; upper airway obstruction or significant respiratory depression; during labor when delivery of premature infant is anticipated |
| Interactions | Monitor for increased respiratory and CNS depression with coadministration of cimetidine; hydantoins may decrease effects of meperidine; avoid with protease inhibitors |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients with head injuries since meperidine may increase respiratory depression and CSF pressure (use only if absolutely necessary); caution when using postoperatively and with history of pulmonary disease (suppresses cough reflex); substantially increased dose levels due to tolerance may aggravate or cause seizures even if no history of convulsive disorders; monitor closely for morphine-induced seizure activity if seizure history |

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

Have analgesic, anti-inflammatory, and antipyretic activities. Mechanism of action is not known, but may inhibit cyclooxygenase activity and prostaglandin synthesis. Other mechanisms may exist as well, such as inhibition of leukotriene synthesis, lysosomal enzyme release, lipoxygenase activity, neutrophil aggregation, and various cell-membrane functions.

| | |
|----------------|---|
| Drug Name | Ibuprofen (Motrin, Ibuprin)- DOC for patients with mild to moderate pain. Inhibits inflammatory reactions and pain by decreasing prostaglandin synthesis. |
| Adult Dose | 200-400 mg PO q4-6h while symptoms persist; not to exceed 3.2 g/d |
| Pediatric Dose | 6 months to 12 years: 4-10 mg/kg/dose PO tid/qid >12 years: Administer as in adults |

| | |
|-------------------|--|
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency; high risk of bleeding |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and possibly toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT when taking anticoagulants (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

| | |
|-------------------|--|
| Drug Name | Naproxen (Aleve, Naprelan, Naprosyn, Anaprox)- For relief of mild to moderate pain; inhibits inflammatory reactions and pain by decreasing activity of cyclooxygenase, which results in decrease of prostaglandin synthesis. |
| Adult Dose | 500 mg PO followed by 250 mg q6-8h; not to exceed 1.25 g/d |
| Pediatric Dose | <2 years: Not established >2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d |
| Contraindications | Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency |
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and possibly toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT when taking anticoagulants (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Category D in third trimester of pregnancy; acute renal insufficiency, interstitial nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug |

Topical Antibiotics

Therapy must be comprehensive and cover all likely pathogens in the context of this clinical setting.

| | |
|-------------------|--|
| Drug Name | Neomycin and polymyxin B (Neosporin)- Used in treatment of minor infections. Inhibits bacterial protein synthesis and growth. Polymyxin B disrupts bacterial cytoplasmic membrane, permitting leak of intracellular constituents and causing inhibition of bacterial growth. |
| Adult Dose | Apply qd/qid to affected areas |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in treating extensive burns (>20% BSA) since absorption of neomycin is possible and may cause nephrotoxicity and ototoxicity; prolonged use may result in overgrowth of nonsusceptible organisms |

| | |
|-------------------|--|
| Drug Name | Silver sulfadiazine (Silvadene, Thermazene, SSD, SSD-AF)- Useful in prevention of infections from second-degree or third-degree burns. Has bactericidal activity against many gram-positive and gram-negative bacteria, including yeast. |
| Adult Dose | Apply qd/bid to a thickness of 1/16 inch; burned area should be covered with medication continuously |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; neonates and infants <2 y |
| Interactions | Effect of proteolytic enzymes is reduced when used concomitantly with this product |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in G-6-PD deficiency and renal insufficiency |

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Follow-up

Further Inpatient Care

- Direct inpatient care toward further trauma management and burn care. Consider scar revisions associated with burns later in the patient's hospitalization.

Transfer

- Transfer the patient to a trauma center with burn care capabilities if such facilities are not available initially.

Deterrence/Prevention

- Care in handling and use of munitions should serve as the primary prevention of injuries and burns associated with white phosphorus.

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Miscellaneous

Medical/Legal Pitfalls

- Legal issues are associated with inadvertent exposure to the agent and to its inappropriate use in situations other than wartime deployment.
-

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CBRNE - Irritants: Cs, Cn, Cnc, Ca, Cr, Cnb, Ps

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Synonyms, Key Words, and Related Terms

tear gas, tear gases, riot control agents, lacrimators, pepper gas

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Introduction

Background

The sole purpose of irritants, also known as tear gas, riot control agents, and lacrimators, is to produce immediate discomfort and eye closure to render the victim incapable of fighting or resisting. Police forces use them for crowd control, and military forces currently use them mainly for training. They were used prior to World War I, and during the war they were the first chemical agents employed, well before the better-known chlorine, phosgene, and mustard gas. The US used them during the Vietnam War to deny tunnel access to its enemies. The US excludes these agents from the 1925 Geneva Convention banning other chemical and biological weapons. Dispersal is allowed in specific US military operations but only by presidential order.

Tear gas (CS) and chloroacetophenone (CN) are by far the most important pulmonary irritants. CN was the primary pulmonary irritant after World War I until Corson and Stoughton developed CS in 1928. CS was found to be more potent but less toxic. In approximately 1959, CS replaced CN as the principal military and law enforcement riot control agent. CS gas is the familiar tear gas most often used by police for crowd control. CN is available as Mace over-the-counter for personal protection. Capsaicin, or pepper spray, has to some extent replaced CN as a personal protective agent, with less dangerous effects.

Although CS and CN are the most important agents in this class, several others require mention. Chloropicrin (PS) and bromobenzene cyanide (CA) were developed before World War I. Both largely have been replaced, as they were too lethal for their intended effects but not lethal enough to compete with the more effective blistering and nerve agents. PS still is seen occasionally as a soil sterilant or grain disinfectant. The creation of CNB (CN, carbon tetrachloride, and benzene), chloroacetophenone in chloroform (CNC), and CNS (CN, chloroform, and PS) attempted to make CN more effective. However, CS proved more effective and less toxic than any of the CN series and largely has replaced them. Dibenz-(b,f)-1,4-oxazepine (CR) is a more recent tear gas, first synthesized in 1962. It reportedly is more potent and less toxic than CS but still is not used widely.

Pathophysiology

Riot control agents are solids with low vapor pressures that are dispersed as fine particles or in solution. CS and CN are SN_2 alkylating agents and react at nucleophilic sites. Although presently unclear, injuries caused by this class of agents may be caused by inactivation of sulfhydryl-containing enzymes such as lactic dehydrogenase and a specific coenzyme in the pyruvate decarboxylase system (disulfhydryl form of lipoic acid).

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Clinical

History

- Most pulmonary irritant exposures are self-limited. Onset of symptoms occurs in seconds to several minutes. Duration is 15-30 minutes after clothing is removed and the patient is in an open space.
- Typically, the eyes, nose, mouth, and even airway feel a burning sensation.
- The eye is the most sensitive organ involved.
 - Eye tearing and severe blepharospasm are common.
 - More serious and even permanent eye injuries (eg, corneal abrasions, foreign bodies) can occur. Tear gas particles, other foreign particles, or the blast injury itself causes these injuries.
 - Patients may complain of blindness because of the intense tearing and blepharospasm, but patients who physically can open their eyes have no significant change in visual acuity.
- Patients also may report cough, chest tightness, and dyspnea, but pulmonary function tests typically are not changed. These agents can exacerbate a chronic pulmonary condition such as asthma or chronic obstructive pulmonary disease.
- Cardiovascular function may demonstrate an increased blood pressure and heart rate, but this effect is believed to be most likely a psychological response to the situations in which tear gases typically are used.
- Exercise exacerbates symptoms.
- Patients develop tolerance to tear gas symptoms with chronic low-grade exposures.
- Psychological effects (eg, anxiety) also provoke increased response.
- Once the skin comes in contact with a riot control agent, erythema, tingling, and burning occur. These symptoms occur within minutes of exposure and last up to 1 hour after termination of exposure.
 - More severe skin injuries can occur in hot, humid environments with heavily sweating or wet patients or with prolonged or close-range exposures.
 - Patients can develop first-degree or second-degree burns and allergic dermatitis.
- Unintentional oral ingestions can occur, specifically in children.
 - Abdominal cramps and diarrhea are common, but the ultimate course usually is uneventful.

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Differentials

Acute Respiratory Distress Syndrome

Anaphylaxis

Toxicity, Phosgene

Ultraviolet Keratitis

Anxiety

Burns, Chemical

Burns, Ocular

Burns, Thermal

CBRNE - Chemical Warfare Agents

CBRNE - Incendiary Agents, Magnesium and Thermite

CBRNE - Lung-Damaging Agents, Chlorine

CBRNE - Urticants, Phosgene Oxime

CBRNE - Vesicants, Mustard: Hd, Hn1-3, H

CBRNE - Vesicants, Organic Arsenicals: L, ED, MD, PD, HL

Chronic Obstructive Pulmonary Disease and Emphysema

Congestive Heart Failure and Pulmonary Edema

Conjunctivitis

Pediatrics, Croup or Laryngotracheobronchitis

Pediatrics, Pneumonia

Pediatrics, Reactive Airway Disease

Pediatrics, Respiratory Distress Syndrome

Pneumonia, Aspiration

Pneumonia, Bacterial

Pneumonia, Empyema and Abscess

Pneumonia, Immunocompromised

Pneumonia, Mycoplasma

Pneumonia, Viral

Respiratory Distress Syndrome, Adult

Smoke Inhalation

Toxicity, Chlorine Gas

Toxicity, Hydrocarbons

Toxicity, Hydrogen Sulfide

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Workup

Lab Studies

- Arterial blood gases allow for confirmation of adequate ventilation.

Imaging Studies

- A chest x-ray may be indicated in patients with significant respiratory complaints, especially if the offending agent is not known.

Other Tests

- Perform slit lamp examination with fluorescein on patients with significant eye complaints, especially if the patient experienced a close-range exposure.

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Treatment

Prehospital Care

Most people exposed to pulmonary irritants do not seek medical care, and effects are self-limited.

- When patients seek care, first withdraw them from exposure. Then, decontaminate patients.
 - Acceptable decontaminating solutions are water, soap and water, or bicarbonate solution.
 - Do not use hypochlorite. This relatively caustic solution may worsen the condition of skin injuries already suffered from exposure to irritants.
 - Warn patients that the pain worsens during decontamination.

Emergency Department Care

Initiate or continue care in the emergency department as discussed above.

- Flush the eyes of patients with eye complaints with normal saline or water to remove any particulate matter before fluorescein slit lamp examination for corneal abrasion.
- Treat more severe injuries, which occur in fewer than 1% of patients, in the usual fashion.
- Corneal abrasions can be treated with local antibiotics, oral analgesics, and close follow-up care.
- The rare eye foreign body may merit ophthalmologic consultation.
- Treat burns based on the severity and location of injury.
- The patient with significant respiratory damage is rare and may require oxygen supplementation, bronchodilator therapy (if bronchospasm is present), and admission to the hospital, possibly a critical care unit.

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Medication

In general, only decontamination with water is necessary when a patient's skin has become grossly exposed. Bronchodilators, analgesics, and pulmonary support may be needed depending upon severity of injury.

Bronchodilator

Use only for patients with evidence of significant bronchospasm after exposure.

| | |
|-------------------|---|
| Drug Name | Albuterol 0.5% (Proventil, Ventolin)- Beta-agonist for bronchospasm refractory to epinephrine. Relaxes bronchial smooth muscle by action on beta 2-receptors with little effect on cardiac muscle contractility. |
| Adult Dose | 0.5 cm ³ (2.5 mg) mixed with 2.5 cm ³ normal saline solution and used as nebulizer |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Beta-adrenergic blockers antagonize effects; inhaled ipratropium may increase duration of bronchodilatation by albuterol; cardiovascular effects may increase with MAOIs, inhaled anesthetics, TCAs, and sympathomimetic agents |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution with possible cardiac ischemia; caution in hyperthyroidism, diabetes mellitus, and cardiovascular disorders |

Nonsteroidal Anti-Inflammatory Agents (NsAIDs)

NSAIDs have analgesic, anti-inflammatory, and antipyretic activities. Their mechanism of action is not known, but they may inhibit cyclooxygenase activity and prostaglandin synthesis. Other mechanisms may exist as well, such as inhibition of leukotriene synthesis, lysosomal enzyme release, lipoxygenase activity, neutrophil aggregation, and various cell-membrane functions.

| | |
|-------------------|--|
| Drug Name | Ibuprofen (Motrin, Ibuprin)- Acts as an analgesic, antipyretic, and anti-inflammatory agent. |
| Adult Dose | 2400 mg PO divided tid/qid prn pain for 7 d |
| Pediatric Dose | 10 mg/kg q6h prn pain for 7 d |
| Contraindications | Documented hypersensitivity; active bleeding; use in patients with aspirin hypersensitivity, rhinitis or nasal polyps, and asthma may precipitate bronchospasm |

| | |
|--------------|--|
| Interactions | Coadministration with aspirin increases risk of inducing serious NSAID-related adverse effects; probenecid may increase concentrations and possibly toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta-blockers; may decrease diuretic effects of furosemide and thiazides; monitor PT closely (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Category D in third trimester of pregnancy; caution in congestive heart failure, hypertension, and decreased renal and hepatic function; caution in anticoagulation abnormalities or during anticoagulant therapy |

Analgesics

Pain control is essential to quality patient care. Analgesics ensure patient comfort, promote pulmonary toilet, and have sedating properties, which are beneficial for patients who have sustained trauma or injuries.

| | |
|-------------------|---|
| Drug Name | Oxycodone/acetaminophen (Percocet, Roxicet, Roxilox)- Drug combination indicated for relief of moderate to severe pain. |
| Adult Dose | 1-2 tab PO q4h prn pain |
| Pediatric Dose | Recommend oxycodone without acetaminophen for children Oxycodone dose: 5 mg/5 cm ³ syrup, 0.10 mg/kg PO q4h prn pain; single dose not to exceed 10 mg |
| Contraindications | Documented hypersensitivity |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | May cause respiratory depression or change in mental status, especially in older patients |

Antibiotics

Therapy must cover all likely pathogens in the context of the clinical setting.

| | |
|------------|--|
| Drug Name | Gentamicin 0.3 % solution (Genoptic, Ocu-Mycin)- Indicated for corneal abrasions. Aminoglycoside antibiotic used for gram-negative bacterial coverage. |
| Adult Dose | 1-2 gtt q4h while awake to affected eye for 7 d |

| | |
|-------------------|---|
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; mycobacterial, viral, and fungal infections of the eye; avoid using this product with steroid combinations after uncomplicated removal of a foreign body from cornea |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Do not use to treat ocular infections that may become systemic; prolonged or repeated antibiotic therapy may result in bacterial or fungal overgrowth of nonsusceptible organisms and may lead to secondary infection |

| | |
|-------------------|--|
| Drug Name | Erythromycin 0.5% eye ointment- Indicated for corneal abrasions and infections caused by susceptible strains of microorganisms and for prevention of corneal and conjunctival infections. |
| Adult Dose | Apply 0.5-inch ribbon to lid of eye tid for 7 d |
| Pediatric Dose | Apply as in adults |
| Contraindications | Documented hypersensitivity; viral, mycobacterial, or fungal infections of eye; patients using steroid combinations after uncomplicated removal of a foreign body from cornea should avoid using this product |
| Interactions | None reported |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Do not use topical antibiotics to treat ocular infections that may become systemic; prolonged or repeated antibiotic therapy may result in bacterial or fungal overgrowth of nonsusceptible organisms and may lead to secondary infection (take appropriate measures if superinfection occurs) |

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Follow-up

Further Inpatient Care

- Most patients can be discharged safely. The rare patient with significant respiratory findings may merit admission.

Prognosis

- Prognosis is excellent. The few reported dangerous effects occur rapidly.

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Miscellaneous

Medical/Legal Pitfalls

- Significant injuries, although rare, can occur with pulmonary irritant exposure. Screen patients for significant eye, skin, or respiratory injuries as discussed above.
-

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CBRNE - Lung-Damaging Agents, Chlorine

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Synonyms, Key Words, and Related Terms

chemical warfare agent, mucous membrane irritant, respiratory tract irritant, noncardiogenic pulmonary edema, ICD-9-CM 983-9 corrosive aromatics, International Classification of Diseases, Ninth Revision, Clinical Modification 983-9 corrosive aromatics

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Introduction

Background

(The views expressed in this article are those of the author(s) and do not reflect the official policy or

position of the Department of the Navy, Department of Defense, or the US Government.)

The respiratory and mucous membrane irritant effects of chlorine have been well known for many years. John Doughty, a New York City schoolteacher, first suggested use of chlorine gas as a chemical warfare agent during the American Civil War. This proposal never was acted upon during that war. Chlorine gas officially was introduced into the chemical warfare arsenal in 1915 at Ypres, Belgium, during World War I. A total of 93,800 tons of chlorine gas is estimated to have been produced during World War I, with more than half produced by Germany.

Accounts of chlorine attacks at Ypres describe an olive-green cloud rolling over the Allied positions, following the ground contours, and sinking into the trenches. Soldiers seeking safety in those trenches were overcome by the gas and experienced tearing eyes, vomiting, and difficulty breathing. They abandoned their trenches to suffer great losses from artillery and rifle fire.

Chlorine was abandoned as a warfare agent when the use of gas masks was introduced and more effective compounds were created and deployed. Total gas casualties in World War I were estimated at almost 1.3 million troops.

Chlorine liquid presently is used in cleaning agents such as bleach, disinfectants, and water purification and in the manufacture of items such as plastics. It is used in the following industries: pesticide, refrigerant, paper and pulp, textile, metallurgy, pharmaceutical, cosmetic, battery, water and sewage purification, and food processing.

Pathophysiology

Chlorine is a greenish yellow gas that is heavier than air in its pure form. It is an oxidizing agent that is highly reactive with water and liberates hypochlorous acid, hydrochloric acid, and oxygen-free radicals, which are toxic to tissues. The most common site of injury is at the mucous membrane, where water concentrations are highest. The specific sites of chlorine effect include the eye conjunctiva, nasal mucosa, pharynx, larynx, trachea, and bronchi.

Chlorine has a strong, pungent odor and stinging, burning effect on the skin and mucous membranes. Because its odor threshold of 0.08 ppm occurs below that associated with toxicity, the sense of smell usually provides adequate warning that chlorine is in the vicinity. This may allow escape and avoidance of serious toxicity. Chlorine also is soluble in alkalis, alcohols, and chlorides. Chlorine is not combustible.

Inhalation toxicity is a function of the dose received and is dependent on the concentration of gas and duration of exposure. The Occupational Safety and Health Administration (OSHA) permissible exposure level is 1 ppm. The immediately dangerous to life or health concentration (IDLH) determined by the National Institute for Occupational Safety and Health (NIOSH) is 25 ppm. Exposure to 15 ppm causes throat irritation. Exposure over 50 ppm is dangerous, and exposure to 1000 ppm is fatal even with short

exposures. The lowest reported lethal concentration is 430 ppm for 30 minutes. In a study by D' Alessandro et al, exposure to 1 ppm for 60 minutes created significant changes in forced expiratory volume in 1 second (FEV1), forced expiratory flow (FEF) after 25-75% of vital capacity has been expelled, and specific airway resistance in normal subjects and in subjects with airway hyperresponsiveness. These changes appear to be transient.

Household mixing of sodium hypochlorite (bleach) cleaning agents with ammonia produces chloramine gas. This gas then interacts with water in the mucous membranes, producing ammonia and hypochlorous or hydrochloric acid. Typically, this occurs in an enclosed environment such as a restroom, which may allow greater exposure to occur. Chlorine gas also may be released in the household by mixing sodium hypochlorite with acidic cleaning agents (toilet bowl cleaners).

Frequency

- **In the US:** Chlorine and chloramine poisoning are common in the US. Chlorine is listed as the most common inhalational irritant, and in San Francisco Bay Area Regional Poison Control Center data from 1989, chlorine was cited in almost one third of the morbidity cases following acute irritant exposure involving both adults and children. Toxic effects after inhalation exposure usually are mild to moderate, and death is uncommon. Large amounts of chlorine are produced in the industrial sector, and the potential for accidental or deliberate release exists.
- **Internationally:** The same potential for release that exists in the US is present worldwide. In addition, chlorine can be used in sabotage, warfare, and terrorist actions. A disgruntled employee introduced chlorine liquid into the air filtration system in a department store, causing evacuation of the store, but no injuries occurred. One terrorist incident involved the release of chlorine on board a ferry.

Mortality/Morbidity

Mortality is a rare consequence of chlorine gas exposure. During the first battle at Ypres, 800 deaths were reported and almost 3000 personnel were incapacitated out of 15,000 troops. A study of the American Expeditionary Force in World War I revealed a total of 1843 patients exposed to chlorine gas with an average admission time of 60 days.

Morbidity from moderate and severe exposures typically is caused by noncardiogenic pulmonary edema. This may occur within 2-4 hours of exposure to moderate chlorine concentrations (25-50 ppm) and within 30-60 minutes of severe exposures (>50 ppm).

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Clinical

History

The patient may experience complaints based on the exposure. Exposure possibilities include acute low levels, acute high levels, and chronic low levels.

- Acute exposure (low levels): Most poisonings fall into this category and are caused by household exposure to low-concentration cleaning products.
 - Eye tearing, nose and throat irritation
 - Sneezing
 - Excess salivation
 - General excitement or restlessness
- Chronic exposure
 - Acne (chloracne)
 - Chest pain
 - Cough
 - Sore throat
 - Hemoptysis

Physical

- Tachypnea
- Cyanosis (most prevalent during exertion)
- Tachycardia
- Wheezing
- Intercostal retractions
- Decreased breath sounds
- Rales (pulmonary edema)
- Nasal flaring
- Aphonia, stridor, or laryngeal edema
- Ulceration or hemorrhage of the respiratory tract
- Rhinorrhea
- Lacrimation, salivation, and blepharospasm
- Chloracne or tooth enamel corrosion (with chronic exposure)
- Redness, erythema, and chemical burns to the skin from dose-dependent exposure to liquid

Causes

- Occupational exposures constitute the highest risk for serious exposure to high-concentration chlorine.
- Other exposures occur during industrial or transportation accidents.

- Wartime exposure is rare but always possible.
- Household exposure occurs during swimming pool maintenance or inappropriate mixing of bleach cleaning agents with acids or ammonia products.

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Differentials

Pediatrics, Anaphylaxis
Pediatrics, Apnea
Pediatrics, Bronchiolitis
Pediatrics, Croup or Laryngotracheobronchitis
Pediatrics, Epiglottitis
Pediatrics, Pharyngitis
Pediatrics, Pneumonia
Pediatrics, Reactive Airway Disease
Pediatrics, Respiratory Distress Syndrome
Pneumonia, Aspiration
Pneumonia, Bacterial
Pneumonia, Mycoplasma
Pneumonia, Viral
Pulmonary Embolism
Respiratory Distress Syndrome, Adult
Smoke Inhalation
Sunburn
Toxicity, Ammonia
Toxicity, Chlorine Gas
Toxicity, Nitrous Dioxide

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Workup

Lab Studies

- Arterial blood gas: The typical abnormality is hypoxia from bronchospasm or pulmonary edema.
- Obtain serum electrolytes, BUN, and creatinine after significant exposure, as metabolic acidosis and hyperchloremia may occur.

Imaging Studies

- Chest x-ray
 - Chest x-ray frequently is normal initially but may exclude other causes of hypoxia in the differential.
 - Chest x-ray findings commonly lag behind the clinical findings of pulmonary edema.

Other Tests

- Pulse oximetry can be used as a measure of oxygenation.
- Handheld peak flow meters can measure and follow the degree of bronchospasm and response to treatment.
- Pulmonary function testing may be helpful to measure the degree of airway obstruction or restriction.

Procedures

- Laryngoscopy or bronchoscopy can be used to evaluate the degree of damage caused by exposure.

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Treatment

Prehospital Care

- Take precautions to minimize exposure to toxins. In areas of large concentrations or enclosed environments, providers should use self-contained breathing apparatus.
- Remove victims from the toxic environment. Begin initial decontamination at the scene if eye or skin involvement is found. Copious amounts of water may be used. Remove the patient's clothing if it has been contaminated with liquid chlorine.
- Properly sealed chemical containers or material safety data sheets (MSDS) should accompany the patient if available.

Emergency Department Care

The most important aspect of treating patients exposed to chlorine gas is the provision of good supportive care. No antidotes are available. The medications listed below are adjuncts to rigorous attention to the

airway patency, breathing, and circulation.

- Initial assessment
 - Remove the patient's clothing if it has been contaminated with liquid chlorine.
 - Evaluate airway, breathing, and circulation. Provide supplemental oxygen (humidified if possible), as required, by nasal cannula, face mask, nonrebreather mask, noninvasive positive pressure ventilation, or intubation. Poor oxygenation or laryngospasm may necessitate intubation. Positive pressure ventilation with positive end-expiratory pressure (PEEP) set at 5-10 mm Hg may improve oxygenation in patients with noncardiogenic pulmonary edema and allow for lower fraction of inspired oxygen settings to minimize the risk of oxygen toxicity.
- Bronchospasm
 - Treat initial bronchospasm with beta agonists such as albuterol. Ipratropium may be added to the treatment.
 - Poor responses may require terbutaline or aminophylline.
 - Nebulized lidocaine (4% topical solution) may provide analgesia and reduce coughing.
- Corticosteroids: Inhaled and parenteral steroids have been used with many patients exposed to chlorine gas, but no strong clinical evidence supports their use.
- Fluid management
 - Closely monitor the patient's input and output because of the potential of pulmonary edema.
 - Fluid restriction may be required and diuretics may be used to treat impending pulmonary edema.

Consultations

- Request critical care or pulmonary consultation for most admissions.
- Toxicology or poison control center consultation is recommended.
- Obtain ophthalmologic consultation for patients with ocular involvement.

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Medication

The goal of pharmacotherapy is to reduce morbidity and prevent complications.

Bronchodilators

Beta-receptor agonists that relax airway smooth muscles, causing an increase in airway diameter;

specifically, the beta 2 receptor is targeted.

| | |
|-------------------|---|
| Drug Name | Albuterol (Proventil, Ventolin)- Beta-agonist useful for treatment of bronchospasm; preferred choice for initial treatment because of rapid actions. |
| Adult Dose | 0.5 mL of 5% solution in 3 cm ³ NS nebulized; repeat prn or until cardiac effects make continued use undesirable; maintenance prn to maintain adequate oxygenation Metered dose inhaler: 2 puffs (90 mcg/puff) prn to maintain adequate oxygenation |
| Pediatric Dose | 0.03-0.05 mL/kg in 3 cm ³ NS nebulized, not to exceed 0.5 mL; repeat prn or until cardiac effects make continued use undesirable; maintenance with treatments prn to maintain adequate oxygenation Metered dose inhaler: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Beta-adrenergic blockers antagonize effects; inhaled ipratropium may increase duration of bronchodilatation by albuterol; cardiovascular effects may increase with MAOIs, inhaled anesthetics, TCAs, and sympathomimetic agents |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients diagnosed with hyperthyroidism, diabetes mellitus, or cardiovascular disorders; may decrease serum potassium levels |

| | |
|-------------------|---|
| Drug Name | Terbutaline (Brethair, Brethine)- Selective beta 2-agonist relieves bronchospasm by acting on beta 2 receptors to relax bronchial smooth muscle. |
| Adult Dose | 0.25 mg SC q15-30min prn |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; cardiac arrhythmias |
| Interactions | Concomitant use with beta blockers may inhibit bronchodilating, cardiac, and vasodilating effects of beta agonists; concomitant administration of MAOIs with beta sympathomimetics may result in a hypertensive crisis; concurrent administration of oxytocic drugs such as ergonovine may result in severe hypotension |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Through intracellular shunting, may decrease serum potassium levels, which can produce adverse cardiovascular effects; decrease is usually transient and may not require supplementation |

Anticholinergics

Believed to work synergistically with bronchodilators.

| | |
|-------------------|--|
| Drug Name | Ipratropium (Atrovent)- Inhibits secretions from some respiratory mucosa; historically atropine was used in asthma, but ipratropium has fewer adverse effects. |
| Adult Dose | 0.5 mg nebulized qid 2-3 puffs (18 mcg/puff) q4-6h |
| Pediatric Dose | 0.25-0.5 mg nebulized qid |
| Contraindications | Documented hypersensitivity |
| Interactions | Drugs with anticholinergic properties (eg, dronabinol) may increase toxicity; albuterol increases effects |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Not indicated for acute episodes of bronchospasm; caution in narrow-angle glaucoma, prostatic hypertrophy, and bladder neck obstruction |

Methylxanthines

Historically used to treat asthma but lost favor because of newer treatment strategies, toxic effects, and narrow therapeutic windows.

| | |
|-------------------|---|
| Drug Name | Theophylline (Theo-Dur, Aminophylline)- Believed to potentiate exogenous catecholamines administered, stimulate endogenous catecholamine release, and relax diaphragmatic musculature. |
| Adult Dose | Aminophylline: 6 mg/kg lean body weight IV over 20-30 min, followed by a drip at 0.5-0.7 mg/kg/h Theo-Dur: 600-900 mg/d PO divided bid/tid Monitor serum levels and adjust dosages accordingly |
| Pediatric Dose | Aminophylline: 1 mg/kg/h IV drip Theo-Dur: 3-4 mg/kg PO qid Monitor serum levels and adjust dosages accordingly |
| Contraindications | Documented hypersensitivity; uncontrolled arrhythmias; peptic ulcers; hyperthyroidism; and uncontrolled seizure disorders |
| Interactions | Aminoglutethimide, barbiturates, carbamazepine, ketoconazole, loop diuretics, charcoal, hydantoin, phenobarbital, phenytoin, rifampin, isoniazid, and sympathomimetics may decrease effects of theophylline; theophylline effects may increase with allopurinol, beta-blockers, ciprofloxacin, corticosteroids, disulfiram, quinolones, thyroid hormones, ephedrine, carbamazepine, cimetidine, erythromycin, macrolides, propranolol, and interferon |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|---|
| Precautions | Caution in peptic ulcer, hypertension, tachyarrhythmias, hyperthyroidism, and compromised cardiac function; do not inject IV solution faster than 25 mg/min; patients diagnosed with pulmonary edema or liver dysfunction are at greater risk of toxicity because of reduced drug clearance |
|-------------|---|

Topical Anesthetic

Inhaled topical anesthetics have been used to reduce cough and may reduce pain associated with chlorine inhalations.

| | |
|-------------------|---|
| Drug Name | Lidocaine hydrochloride topical solution 4%- Stabilizes neuronal membrane by inhibiting ionic fluxes required for initiation and conduction of impulses; provided by nebulizer, acts in areas exposed to chlorine injury. |
| Adult Dose | 4 mL of 4% lidocaine topical solution nebulized, not to exceed 8 mg/kg Airway absorption estimated to be 7-12% of total dose administered |
| Pediatric Dose | 4.5 mg/kg of 4% lidocaine topical solution nebulized; if <3 mL, normal saline may be added to make the total amount 3 mL Studies exist using a maximum of 8 mg/kg with no adverse results, but the number of subjects is low |
| Contraindications | Documented hypersensitivity; avoid use in Adams-Stokes syndrome and Wolf-Parkinson-White syndrome |
| Interactions | Repeated use or use with other forms of lidocaine increases risk for toxicity, which manifests with seizures and cardiac depression |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | For external or mucous membrane use only; do not use in eyes |

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Follow-up

Further Inpatient Care

- Pulmonary edema may present in a delayed fashion after chlorine gas exposure.
 - Patients who present asymptomatic and remain asymptomatic 6 hours after exposure may be discharged with appropriate instructions and in the presence of reliable family members.
 - Admit patients who present with symptoms that continue for 6 hours after exposure for an

observation period of at least 24 hours. If asymptomatic at 24 hours, patients may be discharged with appropriate follow-up care.

- Consider patients exposed to large concentrations in an enclosed environment, those with underlying cardiopulmonary disorders, and children for admission and observation, even if initially asymptomatic.

Further Outpatient Care

- Only discharge asymptomatic patients from the emergency department, eliminating the need for discharge medications.
- Literature exists that describes cases of chronic reactive airway disease after acute exposures to chlorine gas. Consider referring patients for pulmonary function testing.

Transfer

- Consider transfer to a higher level of care when patients cannot be treated locally. Treatment of noncardiogenic pulmonary edema that may require positive pressure ventilation is the major concern.

Deterrence/Prevention

- Prevention is improved by proper labeling and by avoiding the mixing of chemicals. Household cleaning products should not be mixed. Proper precautions when handling swimming pool chemicals reduce risks. Adequate ventilation is necessary when handling any potentially noxious chemical.
- On a larger scale, deterrence is improved with chemical warfare treaties between countries and the safe transportation and handling of industrial chlorine compounds.
- Training prehospital and hospital providers in the management of chemical casualties can improve treatment provided to exposed personnel while minimizing personal risks. Hospitals can establish mass casualty plans and perform drills to ensure preparations are adequate in the event of a large-scale industrial accident.

Complications

- Short-term effects
 - Bacterial superinfection resulting in bronchitis or pneumonia may present 3-5 days after chlorine gas exposure. Search for infection if the patient fails to recover from chlorine gas toxicity in 3-4 days.
 - Pleural effusions associated with pulmonary edema are possible.

Prognosis

- Most individuals exposed to chlorine gas recover without significant sequelae. Even exposure to high-concentration chlorine gas is unlikely to result in significant, prolonged pulmonary disease.

Patient Education

- Educate patients in the risks associated with the improper handling of chlorine pool chemicals or the improper mixing of household cleaning chemicals.

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Miscellaneous

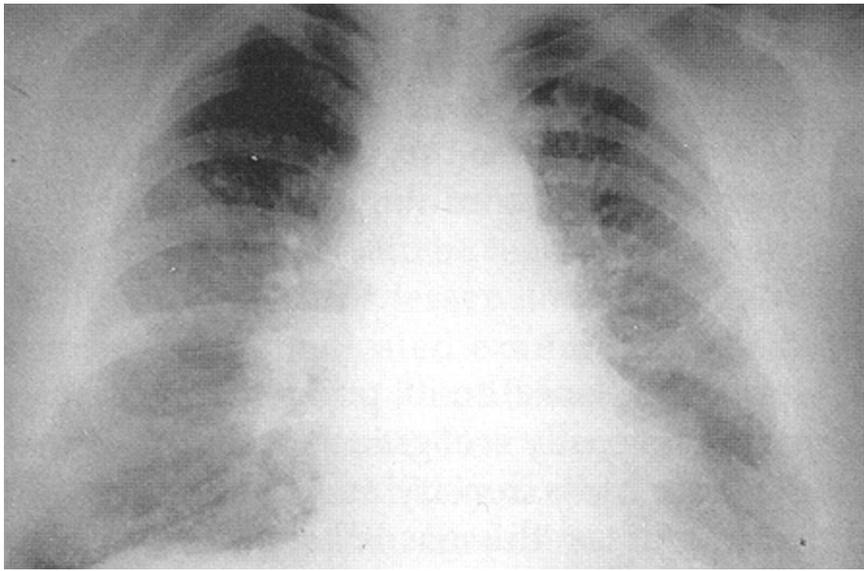
Medical/Legal Pitfalls

- Discharging a patient prior to resolution of symptoms that may warn of impending pulmonary edema
- Failure to ascertain a possibility of chlorine gas exposure through occupational history or the household mixing of chemicals
- Failure to consider bacterial superinfection in patients not responding to several days of appropriate therapy
- Failure to monitor patient in a setting in which respiratory support is available immediately or failure to transfer to a facility with appropriate respiratory support capability

Special Concerns

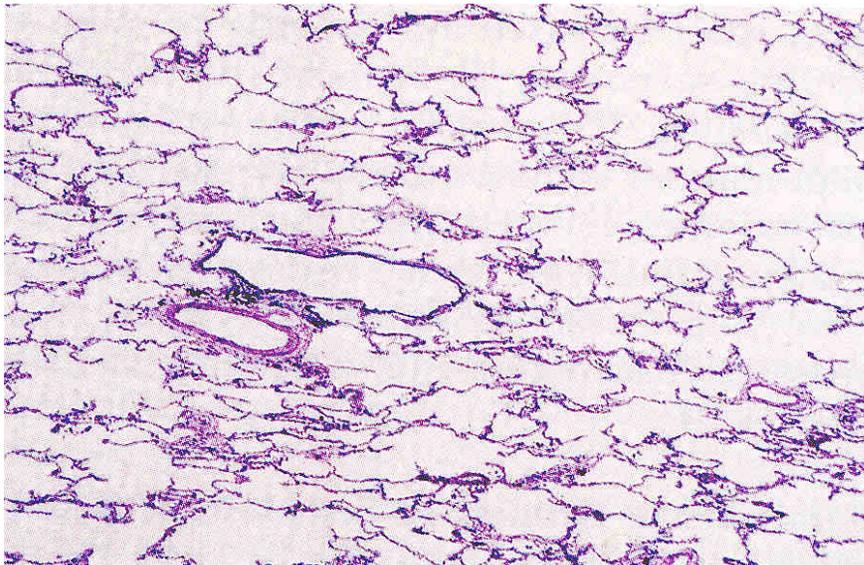
- Superheated chlorine gas from an industrial fire or chemical warehouse explosion may carry the danger of direct thermal injury to the mucous membranes of the eyes, mouth, and respiratory tract in addition to the chemical effects.
-

Pictures



Picture 1: Chest radiograph of a 36-year-old chemical worker 2 hours postexposure to chlorine inhalant. She had severe resting dyspnea during the second hour, diffuse crackles/rhonchi on auscultation, and a partial pressure of oxygen of 32 mm Hg breathing room air. The radiograph shows diffuse pulmonary edema without significant cardiomegaly (used with permission from Medical Aspects of Chemical and Biological Warfare, Textbook of Military Medicine. 1997: 256).

Picture type: X-RAY



Picture 2: A section from a lung biopsy (hematoxylin and eosin stain; original magnification X 100) from a 36-year-old chemical worker taken 6 weeks postexposure to chlorine. At that time, the patient had no clinical abnormalities and a partial pressure of oxygen of 80 mm Hg breathing room air. The section shows normal lung tissues without evidence of interstitial fibrosis and/or inflammation (used with permission from Medical Aspects of Chemical and Biological Warfare, Textbook of Military Medicine. 1997: 256).

Picture type: Photo

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CBRNE - Lung-Damaging Agents, Chloropicrin

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Synonyms, Key Words, and Related Terms

nitrochloroform, nitrotrichloromethane

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Introduction

Background

Chloropicrin is a soil fumigant used for its broad biocidal and fungicidal properties, primarily in high-value crops such as strawberries, peppers, onions, tobacco, flowers, tomatoes, and nursery crops. John Stenhouse, a Scottish chemist and inventor, synthesized chloropicrin in 1848. Because chloropicrin is toxic by all routes of entry, it has the potential for widespread destruction as a chemical warfare agent.

Properties, stability, and reactivity

Chloropicrin is a colorless-to-light green oily liquid with an intense and penetrating odor. Even though chloropicrin is not flammable, it is a significant explosion hazard if involved in a fire. Bulk containers of this liquid are shock sensitive and can detonate. Chloropicrin is an irritant to all body surfaces. This liquid decomposes in the environment. Photochemical reactions with chloropicrin produce phosgene; other decomposition products include nitrogen oxides and chlorine compounds. Chloropicrin photodegrades, with a half-life of 20 days. It is known to undergo violent reactions with aniline, 3-bromopropyne, sodium hydroxide/alcohol solutions, sodium methoxide, and propargyl bromide. Hazardous polymerization does not occur with chloropicrin.

Detection

The odor is a distinctive warning property of this liquid compound.

Table 1. Symptoms According to Concentrations

| | |
|--------|--|
| 1 ppm* | Irritation with pain in the eyes |
| 4 ppm | Incapacitates exposed individuals |
| 20 ppm | Causes definite bronchial or pulmonary lesions |

*Concentrations expressed in parts of material per million parts of air or water.

Pathophysiology

Inhalation

Overexposure leads to irritation of the nose and throat. Chloropicrin is a lacrimator. Exposure to vapors leads to coughing, labored breathing, sore throat, dizziness, bluish skin, vomiting, and in some instances, chemical pneumonitis and pulmonary edema.

Contact with skin or eyes

Contact with chloropicrin can lead to chemical burns or dermatitis manifested by red, cracked, irritated skin. The extent of skin injury depends on the concentration and duration of exposure. Contact with the eyes can cause pain, redness, and tearing. Prolonged eye exposure to chloropicrin can cause blindness. Entrance through damaged skin causes similar symptoms as those seen in overexposure through inhalation.

Ingestion

If ingested, chloropicrin can cause burns to the mouth, throat, and esophagus. Other symptoms are similar to those of overexposure through inhalation. Ingestion of large quantities of chloropicrin liquid can be fatal.

Injection

Overexposure to chloropicrin by injection can lead to redness and irritation of surrounding tissues. Other symptoms are similar to those of overexposure through inhalation.

Chronic exposure

Dermatitis may result from repeated exposure to chloropicrin.

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Clinical

History

Clinicians should attempt to elicit an accurate history to involve the setting of exposure. This may occur as an occupational exposure, as intentional and unintentional industrial release, or as a terrorist attack.

Physical

Physical manifestations depend on the route of exposure.

- Skin - Evidence of chemical burns or dermatitis manifested by red, cracked, irritated, or bluish skin
- Eyes - Pain, redness, and tearing
- Respiratory - Coughing, labored breathing, rales, and rhonchi

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Differentials

Acute Respiratory Distress Syndrome

Anaphylaxis
Smoke Inhalation
Sunburn
Toxicity, Chlorine Gas
Toxicity, Phosgene
Ultraviolet Keratitis
Anxiety
Bronchitis
Burns, Chemical
Burns, Ocular
Burns, Thermal
CBRNE - Chemical Warfare Agents
CBRNE - Incendiary Agents, Magnesium and Thermite
CBRNE - Irritants: Cs, Cn, Cnc, Ca, Cr, Cnb, PS
CBRNE - Lung-Damaging Agents, Chlorine
CBRNE - Urticants, Phosgene Oxime
Chronic Obstructive Pulmonary Disease and Emphysema
Corneal Abrasion
Corneal Ulceration and Ulcerative Keratitis
Hazmat
Pediatrics, Bronchiolitis
Pediatrics, Croup or Laryngotracheobronchitis
Pediatrics, Epiglottitis
Pediatrics, Foreign Body Ingestion
Pediatrics, Pneumonia
Pediatrics, Reactive Airway Disease
Pediatrics, Respiratory Distress Syndrome
Pneumonia, Aspiration
Pneumonia, Bacterial
Pneumonia, Empyema and Abscess
Pneumonia, Immunocompromised
Pneumonia, Mycoplasma
Pneumonia, Viral
Respiratory Distress Syndrome, Adult

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Treatment

Prehospital Care

- General considerations
 - The rescuer's protective equipment should be Level A (eg, triple gloves [polyethylene gloves and nitrile gloves over latex gloves], fully encapsulating chemical resistant suit and boots, hard hat, self-contained breathing apparatus).
 - Standard organic vapor filters used with gas masks or air-purifying respirators do not remove chloropicrin effectively.
- Eye exposure
 - If possible, open victim's eyes while under gentle running water. Use sufficient force to open the eyelids. The victim must "roll" the eyes.
 - Flush for a minimum of 15 minutes.
- Ingestion
 - Do not induce vomiting.
 - Rinse mouth immediately with water.
 - Have the victim drink milk, egg whites, or large quantities of water if available.

Emergency Department Care

- Skin exposure: If not completed in the field, continue decontamination with running water for at least 15 minutes.
- Eye exposure: If not completed in the field, continue flushing for at least 15 minutes.
- Inhalation
 - Continue assisted ventilation and initiate artificial ventilation as needed to support pulmonary function.
 - In severe respiratory compromise, ventilatory support is mandatory. If a PaO₂ cannot be maintained greater than 60 mmHg with a fraction of inspired oxygen (FIO₂) less than or equal to 0.6, then add positive end-expiratory pressure in attempts to open previously closed alveoli.

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Medication

Albuterol and aminophylline may be beneficial in cases involving signs of bronchoconstriction. Use supplemental humidified oxygen in cases of respiratory compromise.

In severe respiratory compromise, ventilatory support is mandatory. If a PaO₂ cannot be maintained greater than 60 mmHg with a fraction of inspired oxygen (FIO₂) less than or equal to 0.6, then add positive end-expiratory pressure in attempts to open previously closed alveoli. No specific drug therapy is available for chloropicrin toxicity.

Sympathomimetic (Adrenergic) Agents

These agents relieve reversible bronchospasm by relaxing smooth muscles of the bronchi.

| | |
|-------------------|--|
| Drug Name | Albuterol (Ventolin)- Stimulates beta-adrenergic receptors. Main effect following oral inhalation is bronchodilation resulting from smooth muscles of bronchial tree. In event of chloropicrin intoxication, use nebulizer route of administration. |
| Adult Dose | Deliver solution over approximately 5-15 min; doses of 5 mg or even 10 mg q4-6h may be required in severe toxicity |
| Pediatric Dose | Not recommended <5 years: Suggested dose 1.25-2.5 mg nebulized; may be administered q4-6h prn with close monitoring for adverse systemic affects 5-12 years: Suggested dosing regimens include nebulized doses of 2.5-5 mg (0.5-1 mL of 0.5% solution) |
| Contraindications | Documented hypersensitivity |
| Interactions | Beta-adrenergic blockers antagonize effects; inhaled ipratropium may increase duration of bronchodilatation by albuterol; cardiovascular effects may increase with MAOIs, inhaled anesthetics, TCAs, and sympathomimetic agents |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hyperthyroidism, diabetes mellitus, and cardiovascular disorders |

Respiratory Smooth Muscle Relaxant

Aminophylline relieves bronchospasm through smooth muscle relaxation of respiratory tract, thereby increasing flow rates and vital capacity.

| | |
|----------------|---|
| Drug Name | Aminophylline (Aminophyllin)- Theophylline compound with ethylenediamine; structurally classified as xanthine derivative. Directly relaxes smooth muscle of respiratory tract. |
| Adult Dose | Loading dose of 6 mg/kg (lean body weight) diluted in 25-50 mL of saline may be given IV over 20 min in event of severe bronchospasm; maintenance infusion of 0.6-0.8 mg/kg may be beneficial |
| Pediatric Dose | 1-9 years: Loading dose is 6 mg/kg IV; maintenance infusion is 1-1.2 mg/kg/h 10-16 years: 0.8 to 1 mg/kg/h IV >16 years: Administer as in adults |

| | |
|-------------------|---|
| Contraindications | Documented hypersensitivity; uncontrolled arrhythmias; peptic ulcers; hyperthyroidism; uncontrolled seizure disorders |
| Interactions | Aminoglutethimide, barbiturates, carbamazepine, ketoconazole, loop diuretics, charcoal, hydantoin, phenobarbital, phenytoin, rifampin, isoniazid, and sympathomimetics may decrease effects of theophylline; theophylline effects may increase with allopurinol, beta-blockers, ciprofloxacin, corticosteroids, disulfiram, quinolones, thyroid hormones, ephedrine, carbamazepine, cimetidine, erythromycin, macrolides, propranolol, and interferon |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in peptic ulcer, hypertension, tachyarrhythmias, hyperthyroidism, and compromised cardiac function; do not inject IV solution faster than 25 mg/min; patients diagnosed with pulmonary edema or liver dysfunction are at greater risk of toxicity because of reduced drug clearance; monitor theophylline levels on a regular basis |

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Miscellaneous

Medical/Legal Pitfalls

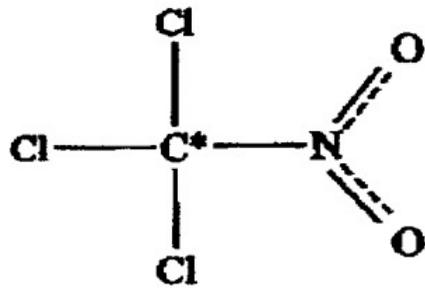
- As chloropicrin is toxic by all routes, failure to recognize exposure may result in long-term disability or death.
- Failure to educate users of chloropicrin in the danger of its use and importance of proper protective equipment can result in unnecessary morbidity or mortality from routine use.

Special Concerns

- Chloropicrin as a chemical weapon of war
 - Chloropicrin is toxic by all routes of entry, especially by inhalation and ingestion.
 - Short overexposures may cause fatal lung damage.
 - Although not flammable, chloropicrin presents a significant explosion hazard if involved in a fire.
 - Bulk containers are shock sensitive and can detonate.
 - Standard organic vapor filters used with gas masks or air-purifying respirators do not remove chloropicrin effectively.
 - In 1887, while the Germans apparently were considering using lacrimators for military purposes, the French began a chemical warfare program. The French developed a tear gas grenade containing ethyl bromoacetate and proposed artillery shells filled with

chloropicrin. In addition, the US War Department's Chemical Warfare Service (CWS) produced chloropicrin-filled artillery shells at the Edgewood Arsenal in Aberdeen, Maryland, as part of the war effort during World War I.

Pictures



CHLOROPICRIN
(Trichloronitromethane)

Picture 1: Chemical structure of chloropicrin

Picture type: Graph



Picture 2: Level A suit (DuPont Tychem 10,000)

Picture type: Photo

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CBRNE - Lung-Damaging Agents, Diphosgene

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Synonyms, Key Words, and Related Terms

choking agents, DP, trichloromethylchloroformate

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Introduction

Background

Diphosgene (DP, trichloromethylchloroformate) was a product of the chemical weapons race in World

War I. It belongs to a class of chemicals termed lung-damaging agents or choking agents. These agents attack lung tissue directly, causing pulmonary edema. The mechanism of action is not well understood, but the chemical is believed to react directly upon the alveolar and capillary walls, resulting in pulmonary edema. The Germans staged the first major successful chemical attack of the war using chlorine. Chlorine then was replaced by phosgene, which caused greater casualties. Gas masks of the era were designed to filter out phosgene. DP was created by combining phosgene with chloroform, which destroyed the gas filters. Blistering and nerve agents largely have replaced the pulmonary agents chlorine, phosgene, and DP.

In the field, DP rapidly vaporizes and breaks down into phosgene and chloroform. It is a colorless gas under standard temperatures and pressures. Clinically, DP behaves in essentially the same manner as phosgene. The chloroform does not reach levels sufficient to cause toxicity, even of the liver, during tactical employment. DP is heavier than air and remains in low-lying areas for longer periods. Doses are cumulative, since DP is not detoxified in the body.

DP deployment almost surely indicates a purposeful, not an accidental, event. Industrial accidents have occurred with both chlorine and phosgene but not with DP, which is not a normal product of manufacturing processes. It also is relatively unstable and degrades easily into phosgene and chloroform. DP must be transported in glass (instead of metal) containers. No automatic detectors are available for use in the field.

Pathophysiology

Like phosgene, the principal feature of DP is delayed pulmonary edema. Although the mechanism is not entirely clear, edema may be caused by direct alveolar damage when DP breaks down into hydrochloric acid and carbon dioxide in the presence of water. DP also causes irritation of the upper respiratory tract and rarely can cause airway obstruction. Respiratory effects occur at doses of 1-10 ppm. Doses greater than 25 ppm can be rapidly fatal. Toxicity varies with both the concentration of vapor and the length of exposure. Because of DP's low water solubility, patients often inhale significant amounts of vapor before symptoms appear.

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Clinical

History

- History likely reveals multiple casualties with respiratory complaints.
- Some patients also may report irritation of the eyes or skin.
- Victims likely will have been involved in a mass gathering or military event within the last 24

hours. Patients may report explosions, smoke, or a gas cloud. Patients inconsistently report the odor of newly mown hay.

- Exposure to low concentrations of DP causes chest discomfort or dyspnea.
- Exposure to somewhat higher concentrations tends to cause lacrimation and irritation of the eyes and skin.
- Exposure to high concentrations quickly can cause pulmonary edema with cough, dyspnea, and production of frothy sputum. Signs include tachypnea, rales, and decreased oxygen saturations.
- Further cardiopulmonary decompensation can occur from noncardiogenic pulmonary edema, with respiratory failure, hypotension, and death.
- Direct eye exposure to DP liquid can cause corneal abrasions, ulcers, or perforation. Direct skin exposure to DP liquid can cause burns.

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Differentials

Dermatitis, Contact
Hazmat
Pediatrics, Anaphylaxis
Pediatrics, Bronchiolitis
Pediatrics, Croup or Laryngotracheobronchitis
Pediatrics, Epiglottitis
Pediatrics, Pneumonia
Pediatrics, Reactive Airway Disease
Pediatrics, Respiratory Distress Syndrome
Pneumonia, Aspiration
Pneumonia, Bacterial
Pneumonia, Empyema and Abscess
Pneumonia, Immunocompromised
Pneumonia, Mycoplasma
Pneumonia, Viral
Respiratory Distress Syndrome, Adult
Smoke Inhalation
Sunburn
Toxicity, Chlorine Gas
Toxicity, Phosgene
Ultraviolet Keratitis

Other Problems to be Considered

Noncardiogenic pulmonary edema, etiology other than DP (distinguish from DP by history of mass casualties, knowledge of direct terrorist or military attack, history of gas exposure, or delayed onset of pulmonary edema following symptom onset)

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Workup

Lab Studies

- An arterial blood gas may be useful to determine the degree of hypoxemia or acidemia in patients where endotracheal tube placement is in question. No laboratory test confirms DP exposure.

Imaging Studies

- A chest x-ray initially may appear normal but eventually reveals pulmonary edema with significant exposure.

Other Tests

- An ECG can be obtained to help ensure that the patient does not have cardiogenic pulmonary edema.

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Treatment

Prehospital Care

- Scene responders need to ensure their own safety when possible to prevent becoming victims themselves.
- Remove patients from the scene to fresh air or administer oxygen if necessary.
- Terminate exposure by removing clothing. Begin skin decontamination with soap and water.
- Pulmonary edema may be precipitated by exertion. Enforce strict bedrest if possible.

Emergency Department Care

- Begin or continue care as discussed above.
- Administer standard resuscitation measures.
 - Patients can present with airway obstruction, although this situation is rare.
 - Acute pulmonary edema is common, and patients may require positive end-expiratory pressure if they are clinically in respiratory distress or frank failure.
 - Patients also can present with hypotension; perform standard resuscitation with crystalloid fluids as first-line agents and vasopressors as second-line agents.
- Antibiotics are unnecessary, prophylactically or therapeutically, unless a secondary infection is present.
- Patients with skin irritation can be decontaminated with soap and water.
- Irrigate the eyes of patients with eye irritation with copious amounts of normal saline solution and check for corneal abrasions or ulcers.

Consultations

Obtain an ophthalmology consult for a significant eye injury.

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Medication

In general, follow decontamination with soap and water by symptomatic treatment.

Bronchodilator

Used to relieve significant bronchospasm.

| | |
|-------------------|---|
| Drug Name | Albuterol 0.5% (Proventil, Ventolin)- Used to relieve bronchospasm after DP exposure. Beta-agonist for bronchospasm refractory to epinephrine. Relaxes bronchial smooth muscle by action on beta2-receptors with little effect on cardiac muscle contractility. |
| Adult Dose | 0.5 cm ³ (2.5 mg) mixed with 2.5 cm ³ normal saline solution and used as a nebulizer |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity |

| | |
|--------------|---|
| Interactions | Beta-adrenergic blockers antagonize effects; inhaled ipratropium may increase duration of bronchodilatation by albuterol; cardiovascular effects may increase with MAOIs, inhaled anesthetics, TCAs, and sympathomimetic agents |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hyperthyroidism, diabetes mellitus, and cardiovascular disorders |

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Follow-up

Further Inpatient Care

- Admit patients requiring resuscitation or oxygen supplementation.
- For at least 12 hours, observe patients with likely exposure who have minor symptoms or are asymptomatic, since delayed pulmonary edema is the classic feature of DP exposure.
- Counsel all patients with significant exposure to avoid strenuous activities for 72 hours and to return if significant respiratory symptoms develop.

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Miscellaneous

Medical/Legal Pitfalls

- Early discharge with patient return in respiratory failure is the major pitfall in DP exposure. Patients with significant toxicity reportedly have presented up to 72 hours after exposure.
-

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Synonyms, Key Words, and Related Terms

CG

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Introduction

Background

Phosgene gas (military designation CG) is a toxic inhalant that directly damages the lungs. Sir Humphrey Davey, a British chemist, first synthesized it in 1812. Although phosgene has not been deployed as a chemical weapon since 1918, its continued use in common industrial processes, such as dye or plastic manufacturing, makes it a potential industrial hazard today. Phosgene exposure also can occur in fires associated with organochlorine compounds (eg, vinyl chloride), the use of carbon tetrachloride fire extinguishers, and during arc welding procedures.

Phosgene gas has the appearance of a white cloud and the characteristic odor of newly mown hay. Odor alone is insufficient for the detection of phosgene exposure, since toxic exposures may occur at concentrations below the olfactory threshold. Although phosgene is one of the most volatile chemical warfare agents, its density is greater than air, and it tends to accumulate in low areas.

Pathophysiology

Phosgene exerts a direct toxic effect on the respiratory tract, causing extensive cellular damage to the alveolar-capillary membrane. Phosgene reacts with intraalveolar water to form hydrochloric acid, which injures the alveoli.

The clinical effects of phosgene are dose dependent. At low concentrations, victims may complain only of a mild cough, dyspnea, and chest discomfort. At moderate concentrations, they also may complain of tearing. At high concentrations, victims rapidly develop noncardiogenic pulmonary edema within 2-6 hours of exposure, producing a clinical picture similar to adult respiratory distress syndrome. Laryngospasm also occurs at higher concentrations, which in turn may cause sudden death. Physical exertion within 72 hours of exposure can trigger dyspnea and pulmonary edema in otherwise asymptomatic patients. Toxic manifestations often are clinically silent at rest, because patients are able to compensate for pulmonary damage in the absence of stress. Death from phosgene inhalation often is caused by latent noncardiogenic pulmonary edema.

Mortality/Morbidity

As reported by Polednak, a 1943-1945 study of 106 male uranium-processing plant workers who were acutely exposed to high levels of phosgene found that 25 patients (24%) developed radiologic evidence of acute pneumonitis, while 1 patient (<1%) died within 24 hours.

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Clinical

History

- Respiratory
 - Cough
 - Dyspnea
 - Dyspnea on exertion
 - Wheezing

- Chest discomfort

Physical

- Respiratory
 - Tachypnea
 - Rales
 - Wheezes

Causes

Phosgene has not been used as a chemical warfare agent since World War I. Accordingly, the risk of phosgene exposure during military action is extremely low. Conversely, because phosgene is used in a number of manufacturing processes, exposure as a consequence of an industrial chemical accident is more likely.

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Differentials

Pediatrics, Pneumonia
Pediatrics, Reactive Airway Disease
Pediatrics, Respiratory Distress Syndrome
Pneumonia, Aspiration
Pneumonia, Bacterial
Pneumonia, Empyema and Abscess
Pneumonia, Immunocompromised
Pneumonia, Mycoplasma
Pneumonia, Viral
Pneumothorax, Iatrogenic, Spontaneous and Pneumomediastinum
Pneumothorax, Tension and Traumatic
Pulmonary Embolism
Smoke Inhalation
Toxicity, Chlorine Gas
Tuberculosis

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Workup

Lab Studies

- Pulse oximetry: Measure oxygen saturation in all symptomatic patients, since hypoxemia is a common consequence of exposure to a lung-damaging agent.
- Arterial blood gas: In patients with severe phosgene exposure, an arterial blood gas is indicated (when resources allow).
- Lactate dehydrogenase: Lactate dehydrogenase is the only consistently elevated lab test in patients with serious phosgene inhalation.

Imaging Studies

- Chest x-ray
 - Symptomatic patients in whom a possible inhalant exposure is suggested also require a chest x-ray early in their evaluation to look for stigmata of noncardiogenic pulmonary edema.
 - Radiologic abnormalities include bilateral perihilar fluffy infiltrates ("batwing" infiltrates), diffuse interstitial infiltrates, and a normal cardiac silhouette.
 - Radiologic changes lag behind clinical changes in phosgene inhalation.
 - Since noncardiogenic pulmonary edema in these patients can worsen rapidly over the first few hours, a repeat chest x-ray is warranted if the patient's status deteriorates.

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Treatment

Prehospital Care

- Emergency medical service personnel entering the phosgene hot zone require full personal protective equipment for toxic vapor exposures, including Level A suits and self-contained breathing apparatus.
- Since phosgene is deployed as a highly volatile gas, transport of victims beyond the exposure site is not expected to pose a significant risk of contamination. Consider disrobing victims exposed to gas as an effort to minimize the potential for any significant off-gassing.
- Appropriate prehospital care of patients with phosgene exposure consists of attention to the ABCs (airway, breathing, circulation), including the provision of supplemental oxygen.
- Inhalational bronchodilator treatment may be initiated in the field for patients with significant

wheezing.

Emergency Department Care

- Appropriate ED care also depends on careful attention to the ABCs.
- Ongoing reassessment is a key component of the early treatment of patients with suspected inhalation injury. Toxic effects may not be apparent in the first few hours, and noncardiogenic pulmonary edema may develop 4-6 hours after exposure.
- Patients with noncardiogenic pulmonary edema require positive end-expiratory pressure via a bilevel positive airway pressure mask or, in more severe cases, endotracheal intubation with positive pressure ventilation. The frothy secretions of pulmonary edema can make endotracheal intubation quite challenging when it is performed late in the patient's course.
- In animal studies, aminophylline has been demonstrated to decrease pulmonary edema following exposure to phosgene; however, human evidence is lacking.

Consultations

In the domestic US, more detailed information about the management of phosgene exposure can be obtained by consulting a regional poison control center.

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Medication

No specific antidote exists for phosgene inhalation.

Diuretics play a limited role in the treatment of phosgene inhalation injury, since they tend to reduce fluid loss into the lungs via the damaged alveolar-capillary membrane. Conversely, diuretics may cause hypotension in hypovolemic patients receiving positive pressure ventilation.

Steroids are not effective in phosgene inhalation injury. Patients with hyperactive airways usually benefit from aerosolized bronchodilator therapy.

Diuretics

These agents are beneficial in the treatment of fluid retention.

| | |
|-------------------|--|
| Drug Name | Furosemide (Lasix)- Loop diuretic that inhibits sodium reabsorption in the ascending loop of Henle. |
| Adult Dose | 10-20 mg IV for patients not already taking diuretics 40-80 mg IV for patients already taking diuretics 80-120 mg IV in 1 h for patients not responding to first dose |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity, hepatic coma, anuria, and severe electrolyte depletion |
| Interactions | May interact with aminoglycosides to increase ototoxicity; may interfere with hypoglycemic effects of oral hypoglycemic agents; may increase anticoagulant activity of warfarin; may lead to increased serum lithium levels |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Resulting volume loss may lead to hypotension; may cause electrolyte imbalance; may increase lithium levels, causing lithium toxicity; caution with nephrotoxic medications; may cause varying degrees of hearing loss; caution with other ototoxic medications; may lead to increased anticoagulant effects of warfarin |

Aerosolized Bronchodilator Therapy

Patients with hyperactive airways usually benefit from aerosolized bronchodilator therapy.

| | |
|-------------------|---|
| Drug Name | Albuterol- Relaxes bronchial smooth muscle by action on beta 2-receptors with little effect on cardiac muscle contractility. |
| Adult Dose | Nebulizer: Dilute 0.5 mL (2.5 mg) of 0.5% inhalation solution in 1-2.5 mL normal saline; administer 2.5-5 mg q4-6h, diluted in 2-5 cc sterile saline or water |
| Pediatric Dose | <5 years (nebulizer): Dilute 0.25-0.5 mL (1.25-2.5 mg) of 0.5% inhalation solution in 1-2.5 mL normal saline and administer q4-6h in equally divided doses >5 years (nebulizer): Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Beta-adrenergic blockers antagonize effects; inhaled ipratropium may increase duration of bronchodilatation by albuterol; cardiovascular effects may increase with MAOIs, inhaled anesthetics, TCAs, and sympathomimetic agents |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hyperthyroidism, diabetes mellitus, and cardiovascular disorders |

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Follow-up

Further Inpatient Care

- Patients with noncardiogenic pulmonary edema following inhalational phosgene exposure require further care in an intensive care unit setting. Initiate or continue standard treatment for noncardiogenic pulmonary edema, including the reversal of hypoxemia with supplemental oxygen and positive pressure ventilation as outlined in [Emergency Department Care](#).

Further Outpatient Care

- Patients with possible phosgene exposure may be discharged home if they remain symptom free 6 hours postexposure. No further treatment is necessary in asymptomatic individuals.

in/Out Patient Meds

- Inpatient treatment of phosgene-induced noncardiogenic pulmonary edema may require judicious use of diuretics such as furosemide.
- Wheezing typically requires aerosolized bronchodilator therapy.
- No recommended outpatient treatment of patients with inhalational phosgene exposure is available, since symptomatic patients require hospitalization and treatment until symptoms resolve.

Transfer

- Transfer of symptomatic patients with inhalational phosgene exposure may be necessary to provide a higher level of care at a second facility. Take care to transfer such patients only after the ABCs are secured.
- Because noncardiogenic pulmonary edema may evolve during transfer, accompanying personnel must be qualified and accompanying resources must be sufficient to provide definitive airway control and positive pressure ventilation if required.

Complications

- Toxic effects of phosgene inhalation are short lasting. Evaluate patients who continue to have symptoms 3-4 days later for the presence of another superimposed disease process, such as bacterial pneumonia.
- Patients commonly complain of exertional dyspnea and reduced exercise tolerance for several months to years after exposure. Although permanent structural lung damage is rare, some recommend follow-up chest x-ray and pulmonary function tests 2-3 months after exposure.

Prognosis

- Onset of pulmonary edema within 2-6 hours of exposure predicts severe injury. Still, the prognosis in phosgene inhalation is good, especially with recent advances in critical care.

Patient Education

- Educate the public that the most significant risk of phosgene exposure arises from industrial chemical accidents and not from the deployment of chemical warfare agents during acts of war or terrorism.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to observe patients for the full 6 hours that noncardiogenic pulmonary edema may take to develop
 - Failure to distinguish noncardiogenic pulmonary edema from cardiogenic pulmonary edema, which may result in over reliance on diuretics in the patient with phosgene-induced noncardiogenic pulmonary edema and lead to excessive intravascular volume loss with consequent hypotension
-

Pictures



Picture 1: Anteroposterior portable chest radiograph in a male patient who developed phosgene-induced adult respiratory distress syndrome. Notice the bilateral infiltrates and ground-glass appearance (Image courtesy of Fred P. Harchelroad, MD, and Ferdinando L. Mirarchi, DO).

Picture type: X-RAY

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CBRNE - Lung-Damaging Agents, Toxic Smokes: Nox, Hc, Rp, Fs, Fm, Sgf2, Teflon

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Introduction

Background

Smokes and obscurants long have been used by the military as a means of hiding troops, equipment, and certain areas from view of the opposing forces. In the past, smoke also has been a form of communication and identification. Smokes are not unique to the military but also are produced in industry by explosion, mechanical generation, or as a by-product of a chemical interaction. Smoke is made of solid particles of varying sizes that are suspended in air. Although smokes typically are not used as direct chemical agents, they may produce toxic injury to skin, eyes, and all parts of the respiratory tract.

Although most smokes used for obscuring purposes are not concentrated enough to be hazardous, any

smoke can be hazardous to health if the concentration is sufficient or if the exposure is long enough. The smoke itself can be directly toxic, or it may carry, adsorbed to the particulate surface, any of a variety of toxic gaseous substances that interact with mucosa, skin, or any surfaces of the airway.

This article reviews the pathophysiology and toxic effects of lung and airway injury caused by different smokes: the oxides of nitrogen (NO_x), zinc oxide (HC), red phosphorus (RP), sulfur trioxide (FS), titanium tetrachloride (FM), standard gas fuel-2 (ie, fog oil [SGF2]), and the pyrolysis of Teflon.

Oxides of nitrogen

NO_x are components of photochemical smog, usually approximately 0.053 ppm. Nitrogen dioxide exists as a mixture of nitrogen dioxide, a reddish brown gas, and nitrogen tetroxide, a colorless gas. Other forms of nitrogen oxide include nitrous oxide, which is a common anesthetic or (when given without oxygen) asphyxiant, and nitric oxide, which quickly decomposes to nitrogen dioxide in the presence of moisture.

Zinc oxide

HC smoke is a mixture of equal amounts of hexachloroethane, zinc oxide, and approximately 7% grained aluminum or aluminum powder. Upon combustion, the mixture produces zinc chloride, which rapidly absorbs moisture from the air to form a grayish white smoke. More humid air results in thicker smoke. Other chemicals also are released in the combustion process, such as chlorinated hydrocarbons (eg, phosgene), chlorine gas, carbon monoxide, and several other compounds.

HC smoke resulted from the French and US Chemical Warfare Service, which, after World War I, sought an obscurant that was not fraught with as many difficulties as white phosphorus. HC has a sweetish acrid odor, even at moderate concentrations. Although HC can irritate the upper airway mucous membranes, it probably is studied most for its role in fume fever.

Red phosphorus

After World War II, RP smoke was developed as an attempt to avoid the toxicity associated with the manufacturing of white phosphorus. RP is 95% phosphorus in a 5% butyl rubber base and provides an adequate tank screen on the battlefield. When RP is oxidized, it forms a mixture of phosphorus acids. When these acids are exposed to water vapor, they in turn form polyphosphoric acids, which may be responsible for the toxic injuries to the upper airways. Most of these injuries are mild irritations. No human deaths have been reported from exposure to either white phosphorous or RP smokes.

Sulfur trioxide

FS, also known as sulfuric oxide, chlorosulfonic acid, or sulfuric anhydride, is typically a colorless liquid, which can exist as ice, fiberlike crystals, or gas. When it is exposed to air, it rapidly takes up water and forms white fumes. The smoke consists of 50% sulfur trioxide and 50% chlorosulfonic acid. It

usually is dispersed by spray atomization. The sulfur trioxide evaporates from spray particles, reacts with surrounding moisture, and forms sulfur acid. The sulfur acid condenses into droplets that produce a dense white cloud. FS is extremely corrosive, which led to its disuse in the army.

Titanium tetrachloride

FM is a colorless-to-pale yellow liquid that has fumes with a strong odor. Once it comes in contact with water, it rapidly forms hydrochloric acid and titanium compounds. It is used to make titanium metal, white pigment in paints, and other products. It breaks down rapidly in the environment.

FM readily hydrolyzes in the presence of water or moist air via an exothermic reaction that occurs in 2 stages. First, FM reacts to form a highly dispersed particulate smoke. This smoke reacts with more moisture in the air to form hydrolytic products of FM such as hydrochloric acid, titanium oxychlorides, and titanium dioxide. Generation of the smoke has been used as screens in military operations. The formation of hydrochloric acid makes it irritating and corrosive.

When FM liquid is exposed to the air, it produces white fumes. These white fumes can come into contact with skin, where a mild epithelial irritation results and usually subsides within 24 hours. When it is mixed with water, it generates a vigorous exothermic reaction that produces both heat and hydrochloric acid, which can work synergistically to produce deep thermal burns.

Oil fog

SGF2 is one type of chemical smoke obscurant used in the military. SGF2 is generated by injecting a light petroleum-based lubricating oil onto a heated engine exhaust manifold, causing the oil to vaporize and eventually recondense in the atmosphere. Any industry that generates an oil mist also may produce similar exposures. Petroleum oil smokes are the least toxic smokes. They seldom produce ill effects even after prolonged or multiple exposures.

Teflon particles

Teflon (polytetrafluoroethylene [PTFE]) is used widely in a variety of industrial and commercial settings. Its lubricity, high dielectric constant, and chemical inertness make it a desirable component in military vehicles such as tanks and aircraft. Closed-space fires in such settings have prompted studies of the toxicity of exposure to the by-products created from incinerated organofluorines. Pyrolysis of PTFE produces a particulate smoke, which if inhaled, produces a constellation of symptoms termed "polymer fume fever."

Pathophysiology

Oxides of nitrogen

Inhalation of nitric oxide causes the formation of methemoglobin. Inhalation of nitrogen dioxide results in the formation of nitrite, which leads to a fall in blood pressure, production of methemoglobin, and cellular hypoxia. Inhalation of high concentrations causes rapid death without the formation of pulmonary edema. Milder yet still severe exposures may result in death with production of yellow frothy fluid in the nasal passages, mouth, and trachea and marked pulmonary edema. The symptoms following the inhalation of NO_x are mostly due to nitrogen dioxide.

Zinc oxide

HC is probably the most acutely toxic of the military smokes and obscurants. HC's toxicity mainly is attributed to the irritating effects of zinc chloride. Most likely, carbon monoxide, phosgene, hexachloroethane, and other products contribute to the observed respiratory effects. Primary damage largely is confined to the upper respiratory tract, where zinc chloride acts much like a corrosive irritant.

Studies have demonstrated that HC exposure can produce a gradual decrease in total lung capacity, vital capacity, and diffusion capacity of carbon monoxide (DLCO). It also is associated with the presence of pulmonary edema, increased airway resistance, and decreased compliance. When these episodic exposures were stopped, the changes were reversible.

In a study by Conner et al performed with guinea pigs, exposure to ultrafine HC particles (0.05 μm) in increasing degrees was associated with a dose-response elevation in protein, neutrophils, and angiotensin-converting enzyme found in lavage fluid. A direct relationship also was observed with alkaline phosphatase, acid phosphatase, and lactate dehydrogenase in lavage fluid. Centriacinar inflammation was seen histologically, indicating evidence of pulmonary damage.

An interesting study by Marrs et al involving mice, rats, and guinea pigs demonstrated a positive association of alveogenic carcinoma in a dose-response trend to HC smoke, as well as a variety of inflammatory changes. The article states that hexachloroethane and zinc, as well as carbon tetrachloride (which may be present in HC smoke), may be animal carcinogens in certain circumstances. This raises the suspicion of HC as a potential carcinogen.

Metal fume fever is a well-documented acute disease induced by intense inhalation of metal oxides, especially zinc oxide. The exact pathology is not really understood, but the clinical syndrome is well described and has been studied at length. A study by Kuschner et al on human volunteers showed that pulmonary cytokines such as tumor necrosis factor (TNF), interleukin 6 (IL-6), and interleukin 8 (IL-8) may play important initial roles in mediating metal fume fever.

Red phosphorus

Most of the pathologic consequences associated with phosphorus are from elemental white phosphorus fumes or vapor. Contact with elemental phosphorus can cause burns to body surfaces. A well-described condition termed "phossy jaw" is associated with longer-term occupational exposures to airborne

phosphorus fumes. This disease is a degenerative condition affecting the entire oral cavity including soft tissue, teeth, and bones. Massive necrosis of teeth, bone, and soft tissue can lead to life-threatening infections. Treatment typically consists of soft tissue and bone debridement, abscess drainage, and reconstructive surgery.

White phosphorus and RP smokes may cause respiratory tract irritation after 2-15 minutes of exposure. This probably is caused by the polyphosphoric acids that react with moist mucosal membranes. Respiratory tract irritation has been observed at concentrations of 187 mg phosphorus pentoxide equivalents/m³ for 5 minutes or longer. Intense congestion, edema, and hemorrhages were observed in lung tissue following a 1-hour exposure at varying concentrations in studies using rats, mice, and goats.

Sulfur trioxide

Since FS is an intermediate used to produce sulfuric acid upon its reaction with moisture, the resulting toxicity is that of an acidic irritation to mucosal membranes and even skin. The corrosive effect of acid on mucosa and keratinized skin causes significant irritations and chemical burns.

Titanium tetrachloride

The same pathophysiologic effects that occur with FS smoke occur with FM smoke, since both are associated with the production of corrosive and irritating acids.

Oil fog

Concentrations of oil mists in industrial settings vary over a wide range (0.8-50 mg/m³), with most at 3 mg/m³. The particle sizes also vary more than 1.0-5.0 µm in median diameter. They typically have a high molecular weight and are saturated hydrocarbons derived from distilled petroleum. Exposures to such smoke are likely to last for many hours in a single day or repeatedly over consecutive days.

Animal studies have demonstrated, after chronic exposure, that pulmonary function endpoints such as total lung capacity, vital capacity, residual volume, DLCO, compliance, and end-expiratory volume were unaffected by oil fog. One exception exists; male rats exposed at 1.5 mg/L had decreased end-expiratory volume.

Bronchiolar lavage and histopathology showed changes consistent with a mild inflammatory edema (ie, increased protein content, total cells, polymorphonuclear leukocytes [PMNs], macrophages).

Teflon particles

Pyrolysis of Teflon occurs at approximately 450°C. The mixture of particles that is produced contains a substance called perfluoroisobutylene (PFIB), which appears to be the main cause of toxicity in polymer fume fever. The ultrafine particles initiate a severe inflammatory response at low inhaled particle mass

concentrations, which suggests an oxidative injury. PMNs may regulate the inflammatory process with cytokine and antioxidant expression.

PFIB particles cause an extremely rapid toxic effect on pulmonary tissues. Evidence of microscopic perivascular edema is observed within 5 minutes. Less intense exposures are followed by a latent period during which normal physiologic compensatory measures to control developing pulmonary edema ensue. Once these mechanisms are overcome, the time frame of which depends upon the degree of exposure, the clinical syndrome of fume fever follows. More intense exposures also may produce a chemical conjunctivitis. Hemorrhagic inflammation of the lungs also can occur.

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Clinical

History

- Oxides of nitrogen: Because of their insolubility in water, NO_x tend not to cause immediate upper airway irritation. Unfortunately, this may allow a significant exposure to remain undetected for prolonged periods. As with most toxic inhalations, severity of disease and presentation are related to the concentration of the smoke or fumes, length of time of exposure, manner in which the exposure was delivered, and underlying health of the exposed individual.
 - Mild exposure to nitrogen dioxide results in upper airway and ocular irritation such as itching or burning eyes. Cough, dyspnea, fatigue, chest tightness, throat tightness, nausea, vomiting, vertigo, somnolence, and loss of consciousness also may occur from mild exposure. At weaker concentrations, the individual may experience very little discomfort, quickly accommodating to the cough, mild choking, or upper airway irritation. Because of this, symptoms may appear quickly or remain unnoticed for a few hours. Although the symptoms of mild exposure may become quite dramatic, once the patient is removed from the exposure, complete recovery is expected within 24 hours.
 - In more severe exposures, the clinical response may be described as triphasic.
 - During phase I, an intense respiratory symptom complex may occur. Severe cough, dyspnea, and rapid onset of pulmonary edema suddenly may arise. Physical exertion actually may be a precipitating factor, quickening the progression to pulmonary edema. If the patient survives this episode, spontaneous remission occurs within 48-72 hours postexposure. Fiercer exposures can cause acute bronchiolitis with severe cough, dyspnea, and weakness. This typically resolves 3-4 days postexposure.
 - Phase II lasts from 2-5 weeks and is relatively uneventful. A mild residual cough with malaise and perhaps dyspnea may linger, but the chest radiograph (CXR) typically remains clear.
 - In phase III, symptoms may recur 3-6 weeks after the exposure. Severe cough,

fever, dyspnea, and cyanosis may develop in the setting of rales and increasing carbon dioxide retention.

- Many studies have evaluated effects of NO_x on individuals with healthy lungs and those with asthma or chronic obstructive bronchitis. Concentrations of 0.5 ppm or less generally have not affected people with preexisting airway disease. Levels from 0.5-1.5 ppm begin to bother patients with asthma, who notice minor airway irritation. With concentrations greater than 1.5 ppm, people with healthy lungs experience decreases in pulmonary function tests and decreased DLCO with widening of the alveolar-arterial gradient on arterial blood gas.
- Red phosphorus: Individuals with toxic inhalation usually have a history of exposure to the smoke either on the battlefield or in some other setting where phosphorus smokes are used.
 - Complaints of eye, nose, and throat irritation are common.
 - A severe exposure can be associated with an explosive persistent cough. If a person has come in contact with unoxidized phosphorus, chemical burns to the skin can cause pain and erythema.
 - Most often the cough and irritating symptoms resolve after the individual is removed from the exposure source.
- Titanium tetrachloride: Although several industrial exposures have occurred with FM liquid and smoke, only 1 death has been reported. This was a worker who accidentally was splashed over his entire body with liquid FM. He died from complications resulting from inhalation of FM fumes and overwhelming superinfection.
- Oil fog: Individuals exposed to SGF2 or other oil mists may report mild irritation or slight cough, a sensation of shortness of breath, or headache. Those who have underlying pulmonary disease such as asthma or chronic obstructive pulmonary disease (COPD) may have symptoms triggered after exposure to SGF2.
- Teflon particles: Clinical complaints of exposed individuals closely mimic influenzalike symptoms.
 - The individual complains of malaise, fever (at times to 104°F), chills, sore throat, sweating, and chest tightness 1-4 hours postexposure. These symptoms usually resolve 24-48 hours after the patient is removed from the source.
 - More intensely exposed individuals complain of dyspnea on exertion, orthopnea, and later, dyspnea at rest. Cough productive of bloody sputum occasionally is seen.
 - Some animal studies have demonstrated disseminated intravascular coagulation and other organ involvement, but this may be due to global hypoxia, since this only occurred in animals with severe lung damage.

Physical

- Oxides of nitrogen: The severity of physical examination findings depends on the severity of exposure.
 - In a mild exposure, an individual may have injected conjunctiva and normal to mildly erythematous-appearing mucous membranes.
 - After a more severe exposure, signs may range from mild respiratory distress (eg,

tachypnea, accessory muscle use) to more severe signs of wheezes and rales, yellow frothy sputum, and yellow staining of the mucous membranes. This may be followed by cyanosis, lethargy, convulsions, coma, and death.

- Red phosphorus: Physical examination findings are those associated with irritation of mucosal surfaces. A cough or chemical burns to exposed skin surfaces from direct contact with unoxidized phosphorus may be present.
- Sulfur trioxide
 - Conjunctivitis, corneal erosion with uptake of fluorescein, and lacrimation may be present. Erythema of exposed skin surfaces and an inflammatory reaction of mucosal surfaces also may be present. Intense salivation may follow. The individual may have an explosive cough with bloody sputum, dyspnea, hypoxia, rales, or wheezes.
 - Obviously, physical examination findings vary due to length of exposure, concentration of FS smoke, environment of the exposure, and underlying health of the exposed individual.
 - FS smoke is known to exacerbate symptoms of asthma or COPD and significantly worsen pulmonary function test numbers in these patients
- Oil fog: After an intense and prolonged exposure, a patient may have mild dyspnea, basilar rales, or evidence of bronchoconstriction (eg, wheezing, prolonged expiratory phase).
- Teflon particles
 - Physical examination is similar to that of patients with chemical inhalation injury, but fever often is present as well. Dyspnea, increased work of breathing, and rales are common. Pulmonary edema usually is mild and typically does not require oxygen supplementation.
 - More intense toxicity and hypoxia may be seen, requiring more invasive methods of oxygenation and ventilation. Pulmonary edema is also worse if the individual exercises postexposure.
 - CXR findings of pulmonary edema worsen for up to 12 hours and then typically clear by 72 hours.
 - Deaths have been reported with severe pulmonary edema, hypotension, and gram-negative superinfection.

Causes

- Oxides of nitrogen
 - At ground level, NO_x are produced during electric or arc welding, combustion of fuels, detonation of nitrate-based explosives, combination of nitrogen-containing products, and decomposition of organic matter. Recently filled farm silos have high nitrogen dioxide levels for approximately 10 days, peaking at 4000 ppm. Significant quantities of nitrogen dioxide also are found in diesel engine exhaust.
 - Severe pulmonary reactions have been reported after accidental exposures in unventilated farm silos, welding in confined spaces, detonating nitrogen-based explosives in enclosed spaces (tanks, ships), handling nitric acid, resurfacing ice arenas, using anesthesia, and in missile fuel oxidizer spills. Any person engaged in associated occupations or environments is at risk.
- Red phosphorus: Phosphorus smokes are used in military formulations for smoke screens,

incendiaries, smoke markers, colored flares, and tracer bullets. People also can be exposed to phosphorus smoke at phosphorus loading plants.

- Sulfur trioxide: One may become exposed to FS on the job in a chemical or metal plating industry. FS exposure also may occur in the production of detergents, soaps, fertilizers, or lead-acid batteries (car batteries), in printing and publishing, or in photography shops. Since the army does not use FS much anymore, military exposures are less common.
- Titanium tetrachloride: Since FM smoke breaks down so rapidly in the environment, those who work with it in industry seem to be most at risk. Since titanium tetrachloride is extremely irritating and corrosive in both the liquid and smoke formulations, its use has diminished.
- Oil fog: Military personnel can be exposed to fine-particle oil fog when it is used in training or in combat. Industrial settings where oil mists are created may produce similar exposures (eg, metalworking, automobile and textile industries, pressrooms, mining, die and mould lubrication).
- Teflon particles: As mentioned in the [Background](#) section, exposure to these fumes is common in closed-space fires where Teflon is pyrolyzed. Also, polymer fume fever has been observed in those smoking Teflon-contaminated cigarettes.

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Differentials

Acute Respiratory Distress Syndrome

Other Problems to be Considered

Any process that presents with pulmonary symptoms or signs

Pneumonia

Congestive heart failure

Pulmonary embolism

COPD exacerbation

Precipitant of noncardiogenic pulmonary edema, myocardial infarction, pleurisy, or tuberculosis

Any toxic inhalation exposure (eg, cyanide, carbon monoxide, other toxic fumes)

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Workup

Lab Studies

- In the workup of inhalation injuries caused by toxic smokes, the primary investigation is toward the pulmonary system. Other tests should be clinically indicated based on history, physical examination, and underlying health problems.
- Arterial blood gas aids in the evaluation of the degree of hypoxia and alerts the health care provider to other possible toxins such as carbon monoxide and methemoglobin.
- Carbon dioxide levels also may be monitored, since patients with prior lung disease such as asthma and COPD may be affected more severely and are at greater risk to retain carbon dioxide.
- Perform baseline pulmonary function tests (PFTs) once the patient is stable. This may be difficult in the emergency department, but serial peak flow readings may be helpful. Later, PFTs allow evaluation and comparison of lung function and reversibility with bronchodilators and potentially steroids. If the patient develops dyspnea on exertion, then perform PFTs with exertion if PFTs at rest cannot explain the symptoms.

Imaging Studies

- Chest x-ray
 - CXR can help in evaluating the presence of hyperinflation that may suggest injury of the smaller airways and air trapping. Noncardiogenic pulmonary edema also is a clue to toxic inhalation.
 - CXR changes may lag behind clinical changes by hours or days, so if it is normal it may be of limited value.
 - Individuals with fume fever often are sent home after 4 hours observation and with a clear CXR, only to return after the initial recovery and latent phase with more severe dyspnea and florid noncardiogenic pulmonary edema.
 - CXR in a significant HC exposure may not show anything abnormal until 4-6 hours postexposure. CXR findings slowly may improve with supportive care or advance to a long-standing diffuse interstitial fibrosis.
 - In phase III of NO_x exposure, a noncardiogenic pulmonary edema pattern may be seen on CXR. Pathologic findings may demonstrate classic bronchiolitis fibrosa obliterans, which may mimic miliary tuberculosis on CXR. Fibrotic changes either may clear spontaneously or proceed to severe respiratory failure.

Other Tests

- ECG and serial cardiac enzymes also are important in the setting of chest pain as clinically

indicated to evaluate underlying cardiac ischemia, which may be precipitated by hypoxia or increased oxygen demand.

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Treatment

Prehospital Care

- Prehospital care is directed toward securing the airway as needed, administering oxygen, and obtaining intravenous (IV) access. Cardiac monitoring also is important for any patient with respiratory distress. Beta-agonists such as albuterol may be given as a nebulized treatment to those who demonstrate signs of bronchoconstriction.
- Although the care is mostly supportive, prompt delivery to the emergency department should be a priority for prehospital care providers.
- As always, the prehospital care providers must do all in their power to remove the patient from ongoing exposure without becoming casualties themselves.

Emergency Department Care

Treatment for inhalation injuries caused from toxic smokes is based on clinical presentation and involves primarily supportive care directed at the cardiopulmonary system.

- Begin treatment by removing the patient from the source of exposure and providing appropriate detoxification.
- Provide IV access, cardiac monitor, and supplemental oxygen in the setting of hypoxia.
- Begin bronchodilators in a patient with bronchoconstriction.
- Subcutaneous epinephrine has been used in HC exposures.
- Steroids remain controversial but have been suggested as having some value in NO_x, HC, RP, FS, FM, and PTFE exposures.
- HC exposures may require British anti-Lewisite (BAL) administration and the chelating agent calcium ethylenediaminetetraacetic acid (CaEDTA).
- If the patient was exposed to particulate RP and burns are present, a topical bicarbonate solution to neutralize phosphoric acids may be used. Mechanical removal and debridement of contaminated wounds helps diminish toxicity to elemental phosphorus.
- FS and FM exposures require washing of irritated skin with water and then a sodium bicarbonate solution. Any eye involvement should prompt generous irrigation and examination with fluorescein. Obtain ophthalmology follow-up care. Mydriasis with atropine sulfate has been suggested as potentially helpful.

- Some animal studies suggest that in the setting of PTFE exposure, increasing concentrations of pulmonary oxygen free radical scavengers containing thiol groups may be valuable. *N*-acetyl cysteine has been found effective.

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Medication

The goals of pharmacotherapy are to reduce morbidity and prevent complications.

Bronchodilators

Important in the setting of bronchoconstriction and bronchorrhea. Toxic smokes can cause bronchoconstriction, especially if the exposed individual has underlying asthma or COPD.

| | |
|-------------------|--|
| Drug Name | Albuterol (Proventil, Ventolin)- Beta-agonist for bronchospasm refractory to epinephrine; relaxes bronchial smooth muscle by action on beta2-receptors with little effect on cardiac muscle contractility. |
| Adult Dose | Nebulizer: Dilute 0.5 mL (2.5 mg) of 0.5% inhalation solution in 1-2.5 mL normal saline; administer 2.5-5 mg q4-6h, diluted in 2-5 cm ³ sterile saline or water; may administer above unit dose q5min 3-5 times or opt to use a continual dose nebulizer for patients with more severe symptoms; use cardiac monitor in the setting of continuous albuterol nebulizer to monitor heart rate |
| Pediatric Dose | Nebulizer: Dilute 0.25-0.5 mL (1.25-2.5 mg) of 0.5% inhalation solution in 1-2.5 mL normal saline and administer q4-6h in equally divided doses >5 years (nebulizer): Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Beta-adrenergic blockers antagonize effects; inhaled ipratropium may increase duration of bronchodilatation by albuterol; cardiovascular effects may increase with MAOIs, inhaled anesthetics, TCAs, and sympathomimetic agents |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hyperthyroidism, diabetes mellitus, and cardiovascular disorders |

Anti-Inflammatory Agents

Although somewhat debated, steroids are believed to be helpful in toxic smoke inhalation, especially in

metal fume fever, which is believed to be mediated by an inflammatory cascade of events involving cytokines and histamine release.

| | |
|-------------------|---|
| Drug Name | Methylprednisolone (Medrol, Solu-Medrol, Depo-Medrol)- Decreases inflammation by suppressing migration of PMNs and reversing increased capillary permeability. |
| Adult Dose | 20-60 mg/d PO in 1-4 divided doses followed by gradual reduction to lowest level that maintains clinical response Some have administered IV form known as Solu-Medrol as much as 60-250 mg IV q6h |
| Pediatric Dose | 0.5-1.7 mg/kg/d or 5-25 mg/m ² /d PO/IV/IM divided q6-12h Some have administered the IV form Solu-Medrol 1-2 mg/kg bid/qid |
| Contraindications | Documented hypersensitivity; viral, fungal, or tubercular skin infections |
| Interactions | Coadministration with digoxin may increase digitalis toxicity secondary to hypokalemia; estrogens may increase levels; phenobarbital, phenytoin, and rifampin may decrease levels (adjust dose); monitor patients for hypokalemia when administering medication concurrently with diuretics |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Hyperglycemia, edema, osteonecrosis, peptic ulcer disease, hypokalemia, osteoporosis, euphoria, psychosis, growth suppression, myopathy, and infections are possible complications of glucocorticoid use |

Chelating Agents

No reports exist as to the efficacy of chelating agents; however, BAL and CaEDTA have been suggested because of their ability to reduce serum zinc levels.

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| Drug Name | Dimercaprol (BAL in oil)- DOC for treatment of mercury toxicity; administered IM q4h, mixed in a peanut oil base; although not formally indicated for zinc toxicity, has been suggested in the setting of severe HC inhalation since it lowers serum zinc levels. |
| Adult Dose | 3-5 mg/kg IM q4h for 2 d followed by 3-5 mg/kg IM q6-12h until stable |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; G-6-PD deficiency; concurrent iron supplementation therapy |
| Interactions | Toxicity may increase when coadministered with selenium, uranium, iron, or cadmium |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

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| Precautions | May be nephrotoxic and may cause hypertension; caution when administering to patients with oliguria or G-6-PD deficiency; may induce hemolysis in G-6-PD deficient patients; excreted in urine and bile; may be administered to patients with renal failure |
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| Drug Name | CaEDTA (Disotate, Endrate, Chealamide)- Second drug used in lead toxicity; although mostly used in lead chelation, use has been associated with lowering serum zinc levels; begin therapy 4 h after BAL is given; only administered IV; continuous infusion is recommended. Zinc toxicity may be treated with a combination of BAL and EDTA or with EDTA alone; Llobet et al studied 16 chelating agents as possible antidotes for acute zinc exposure in mice; BAL and CaEDTA remain most commonly used in zinc toxicity; the combination approach has a higher incidence of nausea, vomiting, and elevated liver enzymes. |
| Adult Dose | Symptomatic encephalopathic adult patient: 1500 mg/m ² /d administered as continuous IV infusion |
| Pediatric Dose | For severe exposure only: 1500 mg/m ² /d continuous IV infusion Symptomatic nonencephalopathic patient: 1000 mg/m ² /d continuous IV infusion |
| Contraindications | Documented hypersensitivity; renal failure |
| Interactions | Enhances hypoglycemic effects of insulin in patients with diabetes |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Because of potential for renal toxicity, patient should be well hydrated; EDTA may worsen CNS toxicity if given prior to BAL therapy; not recommended for patients in renal failure; to prevent hypocalcemia, use only calcium disodium salt of EDTA for chelation in heavy metal toxicity |

Adrenergic Agonists

In profound bronchoconstriction and wheezing, SC epinephrine has been helpful in stabilizing mast cells and halting or reversing potentially fatal bronchoconstriction.

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| Drug Name | Epinephrine (Adrenalin, EpiPen, Bronitin)- DOC for treating anaphylactoid reactions; has alpha-agonist effects that include increased peripheral vascular resistance, reversed peripheral vasodilatation, systemic hypotension, and vascular permeability; beta-agonist effects include bronchodilatation, chronotropic cardiac activity, and positive inotropic effects. |
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| Adult Dose | Initial dose: 0.01 mL/kg IM/SC of 1:1000 solution, not to exceed 0.5 mL of 1:1000 solution (0.5 mg) Fraction of total dose (0.1-0.2 mL) at site of antigenic exposure if accessible Severe anaphylaxis (laryngeal edema, respiratory failure, shock): 10 mL of 1:100,000 dilution of aqueous epinephrine IV over 10 min If no improvement seen, establish continuous infusion starting at 1 mcg/min of 4 mcg/mL concentration; increase to 4 mcg/min if necessary |
| Pediatric Dose | 0.1 mcg/kg/min SC q15 min for 2 doses, then q4h with increments of 0.1 mcg/kg/min prn; not to exceed 1.5 mcg/kg/min |
| Contraindications | Documented hypersensitivity; cardiac arrhythmias or angle-closure glaucoma; local anesthesia in areas such as fingers or toes (vasoconstriction may produce sloughing of tissue); do not use during labor (may delay second stage of labor) |
| Interactions | Increases toxicity of beta-blocking and alpha-blocking agents and of halogenated inhalational anesthetics |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in elderly patients and those with prostatic hypertrophy, hypertension, cardiovascular disease, diabetes mellitus, hyperthyroidism, and cerebrovascular insufficiency; rapid IV infusions may cause death from cerebrovascular hemorrhage or cardiac arrhythmias |

Mydriatic Agents

These agents relax any ciliary muscle spasm that can cause deep aching pain and photophobia.

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| Drug Name | Atropine (Atropisol, Atropair, Isopto)- Acts at parasympathetic sites in smooth muscle to block response of sphincter muscle of iris and muscle of ciliary body to acetylcholine, causing mydriasis and cycloplegia. |
| Adult Dose | Solution (1%): 1-2 gtt qid; compress lacrimal sac by digital pressure for 1-3 min after instillation Ointment: Apply 0.5-inch ribbon in conjunctival sac tid |
| Pediatric Dose | Solution (0.5%): 1 or 2 gtt into eye(s) bid/tid Ointment: Not established |
| Contraindications | Documented hypersensitivity; thyrotoxicosis; narrow-angle glaucoma; tachycardia |
| Interactions | Coadministration with other anticholinergics has additive effects; pharmacologic effects of atenolol and digoxin may increase with atropine; antipsychotic effects of phenothiazines may decrease; TCAs with anticholinergic activity may increase effects of atropine |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

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| Precautions | Avoid in Down syndrome and/or children with brain damage to prevent hyperreactive response; avoid in coronary heart disease, tachycardia, congestive heart failure, cardiac arrhythmias, and hypertension; caution in peritonitis, ulcerative colitis, hepatic disease, and hiatal hernia with reflux esophagitis; in prostatic hypertrophy, prostatism can have dysuria and may require catheterization |
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Alkalinizing Agents

Indicated for FS and FM cutaneous exposure.

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| Drug Name | Sodium bicarbonate solution 5% solution- Rinse affected skin thoroughly before applying sodium bicarbonate solution. Potential exists for exothermic reaction (burns) whenever a base is mixed with an acid; therefore, after titanium chloride or FS exposure, rinse affected skin thoroughly and copiously with water or saline. Pharmacists at Walter Reed Medical Center recommend a 5% solution of sodium bicarbonate to rinse over affected area, followed by rinsing copiously with water or saline. The author feels that copious irrigation alone with water or saline should be sufficient, along with proper wound care, rather than introducing another chemical onto an already irritated area of skin. |
| Adult Dose | 5% solution applied topically; rinse thoroughly with saline or water before and after application |
| Pediatric Dose | Apply as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Potential for exothermic reactions causing additional burns to skin if any acid solution remains prior to rinsing with bicarbonate solution |

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Follow-up

Further Outpatient Care

- Although most individuals recover without lasting sequelae, some relapse into an acute pulmonary syndrome or develop chronic changes in pulmonary function. Obtain serial PFTs to evaluate progression or deterioration in lung function.

Deterrence/Prevention

- In the military setting, the mission-oriented protective posture (MOPP) gear ensemble provides adequate protection against all smokes. In the industrial setting, guidelines have been established for the protection of the worker as well as any person who may come in contact with toxic smokes. Aim preventive efforts at decreasing the concentration of the smoke and the time of exposure and recognizing underlying health problems that may be exacerbated by exposure to toxic smokes.

Prognosis

- The prognosis for mild-to-moderate exposures of toxic smokes is generally very good, with the usual outcome return to full recovery without sequelae.
- With more severe exposures, lungs may become severely damaged and develop chronic pulmonary fibrosis.

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Miscellaneous

Medical/Legal Pitfalls

- Perhaps the primary medicolegal pitfall in evaluating and treating toxic smoke inhalation and fume fever is overlooking or ignoring the delayed reactions and clinical deterioration associated with many of these exposures. Acute respiratory distress usually responds very well to aggressive initial management. Normal laboratory values and imaging studies, coupled with clinical improvement, usually give the health care provider a false sense of security. The patient then may be discharged from the health care center only to deteriorate as delayed pulmonary edema ensues. Anyone with significant exposure to toxic smokes should be observed for 24-48 hours and imaged with serial CXRs. Difficulty arises in defining a significant exposure, since the clinical response is so varied.
-

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CBRNE - Nerve Agents, G-Series: Tabun, Sarin, Soman

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Synonyms, Key Words, and Related Terms

GA, GB, GD, GF

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Introduction

Background

The organophosphate nerve agents tabun (GA), sarin (GB), soman (GD), and cyclosarin (GF) are among the most toxic chemical warfare agents known. Together they comprise the G-series nerve agents, thus named because German scientists first synthesized them, beginning with GA in 1936. GB was discovered next in 1938, followed by GD in 1944 and finally the more obscure GF in 1949. The only other known nerve agent, O-ethyl S-(2-diisopropylaminoethyl) methylphosphonothioate (VX), is discussed in a separate article of this text. (see [CBRNE - Nerve Agents, V-series: Ve, Vg, Vm, Vx](#)).

G-series nerve agents share a number of common physical and chemical properties. At room temperature, the G-series nerve agents are volatile liquids, making them a serious risk for 2 types of exposure: dermal contact with liquid nerve agent or inhalation of nerve agent vapor. GB is the most volatile of these agents and evaporates at the same rate as water; GD is the next most volatile. Dispersal devices or an explosive blast also can aerosolize nerve agents. Nerve agent vapors are denser than air, making them particularly hazardous for persons in low areas or underground shelters. GB and GD are colorless, while GA ranges from colorless to brown. GB is odorless, while GA and GD smell fruity.

Because nerve agents are soluble in fat and water, they are absorbed readily through the eyes, respiratory tract, and skin. Vapor agents penetrate the eyes first, producing localized effects, then pass into the respiratory tract, with more generalized effects when the exposure is greater. Liquid agents penetrate the skin at the point of contact, producing localized effects followed by deeper penetration and generalized effects if the dose is large enough. Accordingly, the lethality of these agents varies with the route of exposure. For inhalational exposures to GB, the lethal concentration time product in 50% of the exposed population is 75-100 mg·min/m³. For dermal exposures, the lethal dose in 50% of the exposed population is 1700 mg.

Pathophysiology

Nerve agents act by first binding and then irreversibly inactivating acetylcholinesterase (AChE), producing a toxic accumulation of acetylcholine (ACh) at muscarinic, nicotinic, and CNS synapses. Excessive ACh at these cholinergic receptors may account for the spectrum of clinical effects observed in nerve agent exposure. At muscarinic receptors, nerve agents cause miosis, glandular hypersecretion (salivary, bronchial, lacrimal, bronchoconstriction, vomiting, diarrhea, urinary and fecal incontinence, bradycardia). At nicotinic receptors in skin, nerve agents cause sweating, and on skeletal muscle, they cause initial defasciculation followed by weakness and flaccid paralysis. At CNS cholinergic receptors, nerve agents produce irritability, giddiness, fatigue, lethargy, amnesia, ataxia, seizures, coma, and respiratory depression.

Nerve agents also cause tachycardia and hypertension via stimulation of the adrenal medulla. They also appear to bind nicotinic, cardiac muscarinic, and glutamate *N*-methyl-d-aspartate (NMDA) receptors directly, suggesting that they may have additional mechanisms of action yet to be defined. Nerve agents also antagonize gamma-aminobutyric acid (GABA) neurotransmission, which in part may mediate seizures and CNS neuropathology.

Clinical effects of nerve agents depend on the route and amount of exposure. The effect of inhalational exposure to nerve agent vapor in turn depends on the vapor concentration and the time of exposure. Exposure to low concentrations of nerve agent vapor produces immediate ocular symptoms, rhinorrhea, and in some patients, dyspnea. These ocular effects are secondary to the localized absorption of GB

vapor across the outermost layers of the eye, causing lacrimal gland stimulation (tearing), pupillary sphincter contraction (miosis), and ciliary body spasm (ocular pain). As the exposure increases, dyspnea and gastrointestinal symptoms ensue.

Exposure to high concentrations of nerve agent vapor causes immediate loss of consciousness, followed shortly by convulsions, flaccid paralysis, and respiratory failure. These generalized effects are caused by the rapid absorption of nerve agent vapor across the respiratory tract, producing maximal inhibition of AChE within seconds to minutes of exposure. Nerve agent vapor is expected to have had its full effect by the time victims present to the emergency care system.

The effect of dermal exposure to liquid nerve agent depends on the anatomic site exposed, ambient temperature, and dose of nerve agent. Percutaneous absorption of nerve agent typically results in localized sweating caused by direct nicotinic effect on the skin, followed by muscular fasciculations and weakness as the agent penetrates deeper and a nicotinic effect is exerted on underlying muscle. In moderate dermal exposures, vomiting and/or diarrhea occur. Vomiting and/or diarrhea soon after exposure are ominous signs. With further absorption, full-blown systemic or remote effects occur.

Because percutaneous absorption takes time, the onset of symptoms in dermal exposures usually is delayed. Even with thorough decontamination, symptoms may not occur until several hours have elapsed if some agent was absorbed prior to decontamination. A small droplet of GB liquid on the skin may not produce any clinical effects for as long as 18 hours postexposure. A large droplet of GB liquid rapidly causes loss of consciousness, seizures, paralysis, and apnea but only after a brief asymptomatic period typically lasting 10-30 minutes.

Miosis cannot be used as a marker for the severity of nerve agent exposure, because it depends on the route and time course of exposure. In inhalational exposures, miosis occurs early and frequently. In such exposures, normal pupil size is predictive of nontoxicity. However, in dermal exposures at sites distinct from the eye, miosis occurs later in the progression of toxicity and depends on whether significant systemic absorption has occurred.

Nerve agents cause death via respiratory failure, which in turn is caused by increased airway resistance (bronchorrhea, bronchoconstriction), respiratory muscle paralysis, and most importantly, loss of central respiratory drive.

Two chemical properties of nerve agents also provide the rationale for effective measures against them. First, nerve agents are hydrolyzed readily by alkaline solutions, which explains why soap and water or hypochlorite solutions are effective skin decontaminants. Second, the bond between the nerve agent and AChE takes time to chemically mature and become a stable covalent bond. During the period immediately after nerve agent binding to enzyme, the bond is vulnerable to disruption by agents called oximes. This aging phenomenon forms the pharmacologic basis for the effective use of the antidote, pralidoxime, during this early window of opportunity before the bond becomes permanent.

Frequency

- **In the US:** Nerve agent exposure is extremely rare in the US.
- **Internationally:** Despite international attempts to control the proliferation of chemical weapons, nerve agents reportedly still are stockpiled by the militaries of several countries.

To date, no large-scale military deployment of a nerve agent has occurred during war, although indirect evidence exists that the Iraqi military used GB against Kurdish villagers in 1988 as well as during the Iraq-Iran War.

In 1994, the Japanese terrorist cult, Aum Shinrikyo, synthesized and then deployed GB against civilians at Matsumoto, Japan, killing 8 people. The following year, the same terrorist group released GB again in the infamous Tokyo Subway sarin attack, killing 13 and sending 5500 persons to local hospitals.

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Clinical

History

Symptoms of nerve agent toxicity vary with the type of cholinergic receptor affected, muscarinic, nicotinic, or CNS.

- Respiratory - Dyspnea, cough, chest tightness, wheezing
- Neurologic - Headache, weakness, fasciculations, extremity numbness, decreased level of consciousness (LOC), vertigo, dizziness, convulsions
- Ophthalmic - Eye pain, blurred vision, dim vision, conjunctival injection, tearing
- Ear, nose, throat - Rhinorrhea
- Gastrointestinal - Nausea, vomiting, diarrhea, tenesmus, fecal incontinence
- Genitourinary - Urinary incontinence
- Dermal - Sweating
- Psychological - Agitation
- General - Fatigue

Physical

Signs of nerve agent toxicity also vary with the type of cholinergic receptor affected.

- Respiratory - Tachypnea, wheezing, respiratory failure
- Cardiovascular - Bradycardia, tachycardia
- Neurologic - Decreased LOC, weakness, fasciculations, seizure
- Ophthalmic - Miosis, tearing, conjunctival injection
- Dermal - Sweating

Causes

Nerve agent exposure may occur as a result of an industrial accident involving nerve agent production, accidental release from a military stockpile, chemical warfare, and chemical terrorism.

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Differentials

CBRNE - Nerve Agents, Binary: GB2, VX2

CBRNE - Nerve Agents, V-series: Ve, Vg, Vm, Vx

Toxicity, Organophosphate and Carbamate

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Workup

Lab Studies

- While no laboratory test exists to directly measure nerve agent levels in serum or urine, the acute effects of nerve agents can be estimated by measuring the percent reduction in the activity of erythrocytic (RBC) cholinesterase.
 - RBC cholinesterase and plasma cholinesterase (pseudocholinesterase) appear to have a physiologic role as buffers for the tissue AChEs found in the nervous system. These 2 enzymes are clinically important, because their activities can be assayed directly in blood, whereas the tissue cholinesterases cannot. Activity of RBC cholinesterase is considered a more sensitive indicator of nerve agent toxicity than that of plasma cholinesterase.
 - Despite the clinical use of RBC cholinesterase, keep certain limitations in mind when using the activity of RBC cholinesterase to interpret nerve agent effects.
 - Activity of RBC cholinesterase is subject to some individual variation.
 - Without establishing the baseline value of RBC cholinesterase in individuals, measuring the percent reduction in enzyme activity is difficult.

- Poor correlation exists between clinical effects of nerve agents and the percent reduction of RBC cholinesterase activity at low-dose exposures. Accordingly, RBC cholinesterase activity always must be correlated with the patient's clinical status and never should determine patient disposition alone.
- A rising RBC cholinesterase level indicates that no further nerve agent absorption is occurring and that the enzyme is regenerating. RBC cholinesterase is replaced fully every 120 days at the natural regeneration rate of RBCs (approximately 1%/d).
- Draw blood for RBC cholinesterase activity level prior to administering oxime antidotes.
- Hypokalemia has been reported in GB intoxication, although the mechanism is unclear.

Imaging Studies

- Chest x-ray may be helpful in treating patients with significant pulmonary symptoms.

Other Tests

- A number of electrocardiographic changes have been reported in nerve agent intoxication, including bradycardia and varying degrees of atrioventricular block (first through third degree) from the direct muscarinic effect on the heart and tachycardia and ventricular dysrhythmias from hypoxia. Nerve agent toxicity has been associated with PR interval prolongation, QT prolongation, and torsade de pointes.
- Bedside EEG monitoring is recommended for patients paralyzed from nerve agent exposure, because paralysis from nicotinic effects of these agents may mask seizures from CNS effects.

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Treatment

Prehospital Care

- Personal protective equipment
 - A key consideration in prehospital care is protection of emergency medical service personnel from exposure to the nerve agent until victims are decontaminated thoroughly or the need for decontamination is excluded.
 - Personnel should wear personal protective equipment including protective suits, heavy butyl rubber gloves, and air-supplied respirators (eg, self-contained breathing apparatus) when entering a scene posing a nerve agent vapor risk or when treating victims exposed to liquid nerve agents.
- Airway, breathing, and circulation

- Patients with signs and symptoms of moderate nerve agent toxicity require supplemental oxygen, pulse oximetry, cardiac monitoring, and intravenous (IV) access.
- Early endotracheal intubation and ventilatory support are critical in patients with manifestations of severe toxicity (eg, unconsciousness, seizures, paralysis, apnea), since respiratory failure is the principle cause of death in nerve agent exposure.

Emergency Department Care

- Personal protective equipment: Emergency department (ED) personnel should wear personal protective equipment similar to that worn by prehospital care personnel until adequate decontamination of victims is assured or the need for decontamination is eliminated.
- Decontamination
 - Goals of decontamination are to prevent further absorption of nerve agent by victims and to prevent the introduction of nerve agent into the clean ED environment.
 - Liquid nerve agent exposure requires formal decontamination as outlined in [Prehospital Care](#) before victims enter the ED.
 - No decontamination is necessary in vapor exposure.
 - Previously reported terrorist episodes have demonstrated that victims who physically can flee the scene frequently bypass emergency medical services (EMS) and go directly to the nearest ED.

Consultations

Consultation with a toxicologist via a regional poison control center may be helpful.

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Medication

Reversal of nerve agent toxicity depends on the prompt parenteral administration of the 2 antidotes, atropine and pralidoxime.

Although IV administration of these antidotes is preferred, this may not be practical in combat situations or civilian mass casualty incidents. The US military Mark I kit contains 2 IM Autoinjectors, 1 with atropine 2 mg and the other with pralidoxime 600 mg, to be administered simultaneously in the event of nerve gas exposure. The recommended number of MARK I kits to be administered varies from 1-3 and depends on the route of exposure, severity of clinical effects, and elapsed time after exposure. US military personnel deployed during the Persian Gulf War carried 3 Mark I kits per person.

While seizures complicating nerve agent exposure often respond to IV atropine and pralidoxime, they also may require IV benzodiazepines titrated to effect.

Another common complication of vapor nerve agent exposure is ocular pain, which may be treated effectively with a mild, mydriatic-cycloplegic ophthalmic solution (eg, 0.5% tropicamide). Atropine or homatropine ophthalmic solution also can be used to treat ocular pain, but these agents tend to exacerbate visual impairment.

Pretreatment with pyridostigmine before exposure to GA, GD, and GF may improve survival. No evidence supports the chemoprophylactic use of pyridostigmine against GB or VX.

A number of other novel treatments currently are under investigation. Newer H-series oximes and dioximes (HI-6, HLo7) have greater ability to reactivate phosphorylated AChE. These agents demonstrate greater efficacy against all nerve agents (particularly GD) in animal studies and have direct antimuscarinic and antinicotinic actions to antagonize the effects of nerve agents. Other promising treatments currently under investigation include exogenous cholinesterase and the use of human monoclonal antibodies against nerve agents, both of which scavenge nerve agents and prevent them from binding to tissue AChE.

Anticholinergics

Act directly on smooth muscles and secretory glands innervated by cholinergic nerves to block muscarinic effects of excess ACh.

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|-------------------|--|
| Drug Name | Atropine (Isopto, Atropair, Atropisol)- Initial DOC for symptomatic victims of nerve agent exposure; acts via muscarinic receptors to reverse bronchoconstriction, bronchorrhea, abdominal pain, nausea, vomiting, and bradycardia; appears to be involved in stopping seizure activity. Because atropine does not act on nicotinic receptors, has no effect on muscle weakness or paralysis. The most important therapeutic endpoints are drying of respiratory secretions, reversal of bronchoconstriction, and reversal of bradycardia; pupillary response and tachycardia are not useful measures of adequate atropinization; >20 mg rarely is needed in first 24 h, unlike in organophosphate insecticide poisoning where up to 200 mg may be required; atropine almost never is required beyond 24 h postexposure. |
| Adult Dose | 2 mg IV q2-5min, titrated to effect; although IV is preferred, also may be administered IM/ETT in similar doses |
| Pediatric Dose | 0.02 mg/kg IV q2-5min, titrated to effect; 0.1 mg minimum dose |
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with other anticholinergics or TCAs may have an additive anticholinergic effect |

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| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients with coronary artery disease, dysrhythmias, congestive heart failure, hypertension, peritonitis, ulcerative colitis, hiatal hernia with reflux esophagitis, prostatic hypertrophy, and Down syndrome In setting of true nerve agent toxicity, benefits of antidotal atropine are expected to outweigh any risks |

Oximes

Reactivate AChEs, which have been inactivated from phosphorylation by nerve agents (or other compounds, such as organophosphate pesticides).

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|-------------------|--|
| Drug Name | Pralidoxime chloride (2-PAM Cl, Protopam)- Reverses skeletal muscle weakness by reactivating AChE; acts by disrupting covalent bond between nerve agent and AChE before it becomes permanent. Bonds between different nerve agents and AChE have various aging periods. The half-time of the aging reaction for GD is approximately 2 min, for GB it is 5 h, and for GA it is 13 h. Accordingly, administer pralidoxime IV as early as possible (ideally concurrently with atropine). Excreted rapidly and almost completely unchanged by the kidneys. Administration over 30-40 min minimizes adverse effects (eg, hypertension, headache, blurred vision, epigastric pain, nausea, vomiting). |
| Adult Dose | 1-2 g IV; although absorption is slower, also may administer IM |
| Pediatric Dose | 15-25 mg/kg IV |
| Contraindications | Documented hypersensitivity |
| Interactions | Use barbiturates with caution because action of barbiturates is potentiated by AChE inhibitors; antagonism with neostigmine, pyridostigmine, and edrophonium; morphine, theophylline, aminophylline, succinylcholine, reserpine, and phenothiazines can worsen condition of patients poisoned by organophosphate insecticides or nerve agents (do not administer) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Rapid injection can cause tachycardia, laryngospasm, muscle rigidity, pain at injection site, blurred vision, diplopia, impaired accommodation, dizziness, drowsiness, nausea, tachycardia, hypertension, and hyperventilation; can precipitate myasthenia crisis in patients with myasthenia gravis and muscle rigidity in normal volunteers; decrease in renal function increases drug levels in blood because 2-PAM is excreted in urine; can produce transient elevation in creatine phosphokinase; 1 of 6 patients has an elevation in SGOT and/or SGPT |

Benzodiazepines

Believed to exert antiseizure effect by enhancing binding of the major CNS inhibitory neurotransmitter, GABA, to A-type GABA receptors in the CNS, reducing depolarization of neurons and preventing generation and spread of seizures.

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|-------------------|---|
| Drug Name | Diazepam (Valium, Diazemuls, Diastat)- Indicated for treatment of seizures associated with nerve agent toxicity. Depresses all levels of CNS function by increasing activity of the inhibitory neurotransmitter GABA. |
| Adult Dose | 5-10 mg IV q10-20min, titrated to effect; may repeat in 2-4 h prn; not to exceed 30 mg/8 h |
| Pediatric Dose | 0.05-0.3 mg IV over 2-3 min q15-30min, titrated to effect; may repeat in 2-4 h prn; not to exceed 10 mg |
| Contraindications | Documented hypersensitivity |
| Interactions | Coadministration with alcohol, barbiturates, phenothiazines, and MAOIs increases CNS toxicity and respiratory depression |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Use diazepam with caution in setting of nerve agent toxicity or CNS depressants, since may lead to further respiratory depression; caution in hepatic failure or hypoalbuminemia, since may result in toxic diazepam levels |

Mydriatic-Cycloplegics

Dilate iris and relax ciliary muscle, reversing ocular pain and miosis of nerve agent toxicity.

| | |
|-------------------|---|
| Drug Name | Tropicamide (Mydracyl, Tropicacyl)- Anticholinergic compound that reverses miosis and relieves ocular pain in nerve agent toxicity. Acts by blocking cholinergic stimulation of sphincter muscle of iris and ciliary muscle. When applied as weaker preparation (0.5%), causes pupillary dilation (mydriasis); when applied as stronger preparation (1%), results in loss of accommodation (cycloplegia). Acts rapidly; effect is relatively short lasting. |
| Adult Dose | 1-2 gtt of 0.5% solution to eye; may repeat in 5 min; patients with heavily pigmented irides may require larger doses |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity; in patients with primary glaucoma or patients with narrow anterior chamber angles |
| Interactions | None reported |

| | |
|-------------|---|
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in older patients, since increased intraocular pressure is more likely to be encountered in this age group; estimate depth of angle of anterior chamber before administration; advise patients not to engage in hazardous activity (ie, driving) while pupils are dilated; anticholinergic effects may cause CNS disturbances in infants and children; compression of lacrimal sac with a finger for 2-3 min after administration decreases systemic absorption |

Cholinesterase Inhibitors

Temporarily bind and inhibit AChE, thus blocking subsequent binding of certain nerve agents to AChE. Although usually used to treat myasthenia gravis or reverse nondepolarizing neuromuscular blockade, also may be useful as chemoprophylactic agents when administered before exposure to certain nerve agents.

| | |
|-------------------|---|
| Drug Name | Pyridostigmine (Mestinon, Regonol)- Orally available cholinesterase inhibitor, which may be useful as chemoprophylactic agent when administered prior to exposure to GA, GD, and GF. This recommendation is based on animal studies; little information is available regarding the efficacy of pyridostigmine chemoprophylaxis in humans. Only effective in preventing peripheral (non-CNS) effects of nerve agents; since it exists in an ionized form (quaternary amine), does not readily pass into CNS and thus cannot prevent nerve agent-induced CNS injury; no evidence demonstrates that pretreatment before exposure to GB or VX is effective. |
| Adult Dose | 30 mg PO q8h prior to nerve agent exposure for 3 wk total |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity; bronchial asthma; mechanical intestinal obstructions; mechanical urinary obstructions |
| Interactions | Increases effects of depolarizing neuromuscular blockers; increases toxicity of edrophonium |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

Precautions

Inhibits breakdown of ACh; resulting cholinergic excess may lead to muscarinic and nicotinic adverse effects in dose-dependent manner, similar to spectrum of toxicity observed with nerve agents; muscarinic adverse effects include nausea, vomiting, diarrhea, abdominal cramping, hypersalivation, bronchorrhea, and miosis; approximately 50% of military personnel taking prophylactic pyridostigmine during the Gulf War at the dose listed above experienced flatus, loose stools, and abdominal cramping; 5-30% experienced urinary frequency and urgency; <5% suffered headaches, rhinorrhea, diaphoresis, and paresthesias; muscarinic adverse effects are reversible with atropine; potential nicotinic adverse effects include diaphoresis, muscle cramps, fasciculations, and weakness; effects of cholinergic excess can be controlled to some extent by careful selection of dose; bromide component may cause skin rash; long-term effects of pyridostigmine administration to healthy individuals is unclear

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Follow-up

Further Inpatient Care

- Severely poisoned patients in respiratory arrest may need ventilatory assistance for several hours despite aggressive antidotal therapy. Patients in critical condition caused by complications of nerve agent poisoning, such as hypoxic brain injury, may require prolonged intensive care.

Further Outpatient Care

- Toxic effects of GB usually peak within minutes to hours and resolve within 24 hours.
 - Patients who inhale nerve agent vapor suffer peak toxic effects before arriving in the ED.
 - Patients who present to the ED with only ocular findings following vapor exposure can be discharged home safely. Refer patients discharged home with miosis or other eye complaints to an ophthalmologist.
- A variety of neurobehavioral symptoms may persist in patients exposed to nerve agents. Such patients may benefit from neurologic consultation.

Transfer

- Transfer patients only after performing appropriate decontamination and appropriately addressing the need for an airway and ventilation.

Complications

- Little data are available describing long-term effects of nerve agent exposure.
- Structural brain damage in animals has been attributed to nerve agent–induced seizures. A consensus panel of experts concluded that structural brain damage does not occur until seizures have lasted longer than 45 minutes.
- Miosis-related visual problems in dim light and mental lapses have been reported as long as 6-8 months after nerve agent exposure.
- Some information about long-term sequelae has emerged from studies of victims of the Tokyo Subway GB attack. Postural imbalance has been reported 8 months after exposure to GB. Fatigue, asthenia, nausea, shoulder stiffness, and blurred vision have been reported 3 years after exposure to GB.

Prognosis

- Patients who survive nerve agent exposure have a good prognosis.

Patient Education

- Counsel patients who are discharged home with miosis to avoid driving at night.

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Miscellaneous

Medical/Legal Pitfalls

- A number of pitfalls may occur in the assessment and treatment of patients with exposure to nerve agents.
- The most serious mistake is failure to recognize signs and symptoms of cholinergic excess as being caused by nerve agent toxicity. This may lead to further contamination of emergency care personnel and life-threatening delays in emergency medical care of the primary victims.
- Once nerve agent poisoning is diagnosed, another pitfall lies in using the ocular finding of miosis to interpret the severity of exposure or to guide atropine therapy (except when exposure is clearly via vapor and miosis is absent). Similarly, overreliance on the reduction in RBC cholinesterase activity levels can lead to false impressions about the severity of exposure.
- Another pitfall in assessment may occur when emergency care personnel fail to suspect occult seizures in paralyzed patients. Since prolonged seizures lead to structural brain injury, these

patients require bedside EEG monitoring.

- Major mistakes also may occur in treatment. The most devastating error is for first responders to fail to adequately protect themselves from nerve agent exposure before entering the scene, turning these individuals into victims. EMS personnel always must follow the edict of first ensuring that the scene is safe.
 - Failure to adequately decontaminate victims of liquid nerve agent exposure at the scene can lead to contamination of both prehospital and hospital personnel and equipment.
 - When emergency care personnel fail to recognize the rapidity with which nerve agents act, critical interventions (eg, airway management) may be delayed.
 - Another potential error occurs when emergency care providers fail to appreciate the time course of liquid nerve agent exposure. They may fail to recognize that in low-dose exposure to liquid nerve agent, signs and symptoms of cholinergic excess may not appear for up to 18 hours. Conversely, in high-dose liquid nerve agent exposure, a brief asymptomatic period after exposure of 10-30 minutes may occur before the patient acutely deteriorates.
 - Administration of the antidote atropine before hypoxemia is treated may cause ventricular fibrillation. Administration of succinylcholine as part of a rapid sequence intubation protocol may lead to markedly prolonged paralysis. Always administer the antidote pralidoxime as early as possible, since it is ineffective after the aging period has elapsed.
-

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CBRNE - Nerve Agents, V-Series: Ve, Vg, Vm, Vx

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Synonyms, Key Words, and Related Terms

persistent agents, G agents, VX, O-ethyl S-(2-diisopropylaminoethyl) methylphosphonothioate

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Introduction

Background

Nerve agents are compounds that have the capacity to inactivate the enzyme acetylcholinesterase (AChE). The first of these compounds are known as the G agents ("G" stands for German). These compounds were discovered and synthesized by German scientists during World War II, led by Dr Gerhard Schrader.

V agents are part of the group of persistent agents, which are nerve agents that can remain on clothes and other surfaces for long periods. The British first synthesized O-ethyl S-(2-diisopropylaminoethyl) methylphosphonothioate (VX) in 1954. The most important agent in the series was coded in the US as VX. The other agents in the series are less known, and the information available about them is fairly limited. The other agents also have coded names, including VE, V-

gas, VG, and VM (see [Table 1](#)). V agents are approximately 10-fold more poisonous than sarin (GB). The consistency of these agents is similar to oil; thus the inhalation hazard is less than with G agents. This consistency renders them toxic by dermal exposures. Since many of the agents in this series have not been studied extensively, this article discusses VX as the prototype of the series.

Table 1. Code and Chemical Names for the V-Series Agents

| Code Name | Chemical Name |
|-----------|---|
| VX | O-Ethyl-S-[2(diisopropylamino)ethyl] methylphosphonothioate |
| VE | O-Ethyl-S-[2-(diethylamino)ethyl] ethylphosphonothioate |
| VG | O,O-Diethyl-S-[2-(diethylamino)ethyl] phosphorothioate |
| VM | O-Ethyl-S-[2-(diethylamino)ethyl] methylphosphonothioate |
| V-gas | Russian equivalent of VX |

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Pathophysiology

V agents bind to AChE much more potently than the organophosphate and carbamate insecticides. AChE is the enzyme that mediates the degradation of acetylcholine (ACh). ACh is an important neurotransmitter of the peripheral nervous system. It activates 2 types of receptors, muscarinic and nicotinic. Nicotinic ACh receptors are found at the skeletal muscle and at the preganglionic autonomic fibers. Muscarinic receptors are found mainly in the postganglionic parasympathetic fibers. In addition, ACh is believed to mediate neurotransmission in the central nervous system (CNS).

ACh is released when an electrical impulse reaches the presynaptic neuron. It travels in the synaptic cleft and reaches the postsynaptic membrane. In the postsynaptic membrane, ACh binds to the receptor (muscarinic or nicotinic). This interaction leads to activation of the ACh receptor and signal transmission in the postsynaptic side of the cleft. Normally, after this interaction between ACh and its receptor, ACh is degraded into choline and acetic acid by AChE. This regenerates the receptor and renders it active again. The choline moiety undergoes reuptake into the presynaptic cell and is used to regenerate ACh.

Nerve agents act by inhibiting the hydrolysis of ACh by AChE. Nerve agents bind to the active site of AChE, rendering it incapable of deactivating ACh. ACh that is not hydrolyzed still can interact with the receptor, resulting in persistent and uncontrolled stimulation of that receptor. After persistent activation of the receptor, fatigue occurs. This is the same principle used by the depolarizing neuromuscular blocker succinylcholine. Clinical effects of nerve agents are the result of this persistent stimulation and subsequent fatigue at the ACh receptor.

For nerve agents, including V agents, inactivation of AChE eventually becomes permanent (irreversible). This phenomenon of irreversible inactivation of AChE is known as aging. Once aging occurs, the AChE enzyme cannot be reactivated. For the clinical effect to be reversed, new enzyme has to be produced. This new enzyme production is a very slow process. This irreversible binding is one important difference between organophosphate compounds (including nerve agents) and carbamates. For carbamates, AChE binding is always reversible. With VX, a small degree of spontaneous enzyme reactivation occurs, which has been found to be approximately 6% per day for the first 3-4 days and then 1% per day.

Frequency

- **Internationally:** Although G agents were synthesized during World War II, no evidence exists that they ever were used as part of the chemical warfare arsenal. They were tested in concentration camps but not in the battlefield. The only instance in which nerve agents were used is believed to be during the Iran-Iraq war. The Iraqis also allegedly used them in the war against the Kurds.

Mortality/Morbidity

Toxicity of nerve agents is measured in 2 forms, the LCt50 and LD50. LCt50 refers to the inhalational toxicity of the vapor form. "Ct" refers to the concentration of the vapor or aerosol in the air (measured as mg/m³) multiplied by the time the individual is exposed (measured in minutes). At 10 mg·min/m³, VX is the most toxic of the nerve agents (see Table 2). VX also is the least volatile of the nerve agents. This characteristic makes VX a hazard by percutaneous and dermal routes. By contrast, G agents tend to volatilize instead of penetrating the skin.

Table 2. Toxicity of Nerve Agents

| Agent | LCt50 (mg·min/m ³) | LD50 (mg) |
|------------|--------------------------------|-----------|
| Tabun (GA) | 400 | 1000 |
| Sarin (GB) | 100 | 1700 |
| Soman (GD) | 50 | 100 |
| VX | 10 | 10 |

Race

Sensitivity to nerve agents varies with the individual, but no studies have addressed this differential in susceptibility.

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Clinical

History

- The onset of symptoms after exposure to a nerve agent varies according to the route of exposure.
 - After inhalation, onset is rapid due to the high vascularity of the lungs and because the lungs are primary target organs.
 - After cutaneous exposure, systemic symptoms may be delayed for minutes to hours.
- In many situations, history of exposure to a nerve agent is absent. In case of a terrorist attack, suspect the diagnosis when multiple patients present with symptoms of cholinergic excess.
- Occupational history may aid in making the diagnosis. Military personnel and laboratory workers may be at particular risk for exposure.

Physical

Clinical signs and symptoms are related to excessive stimulation at the cholinergic nicotinic and muscarinic receptors. Central effects may be mediated by cholinergic receptors, as well as by effects on *N*-methyl-D-aspartate-ergic and GABA-ergic systems. See Table 3 for a summary of the clinical effects of nerve agents.

Table 3. Pharmacologic Effects of Nerve Agents*

| Receptor Involved | Clinical Effect |
|---|---|
| Acetylcholine, GABA, <i>N</i> -methyl-D-aspartate: Central (CNS) | Anxiety, restlessness, seizures, failure to concentrate, depression, coma, apnea |
| Acetylcholine: Muscarinic <ul style="list-style-type: none"> Postganglionic parasympathetic | "DUMBELS" (commonly used mnemonic) D - Diarrhea U - Urination M - Miosis B - Bronchorrhea, bronchoconstriction E - Emesis L - Lacrimation S - Salivation |
| Acetylcholine: Nicotinic <ul style="list-style-type: none"> Motor endplate Sympathetic and parasympathetic ganglia | Pallor, tachycardia, hypertension, muscle weakness and/or paralysis, fasciculations |

* Adapted from Marrs, Maynard, and Sidell

- Eyes
 - The most common effects of nerve agents on the eyes are conjunctival injection and pupillary constriction, known as miosis. The patient complains of eye pain, dim vision, and blurred vision. This is most likely from direct contact between the agent and eye.
 - Miosis may persist for long periods and may be unilateral. Severe miosis results in the complaint of dim vision. Ciliary spasm also may cause eye pain.
 - Patients exposed to VX may not have miosis. This is probably because the eye usually is not exposed directly to the agent, unlike with G agents. Miosis may be a delayed sign of VX exposure.
- Lungs
 - Shortness of breath is a common complaint. Patients may describe chest tightness, respiratory distress, or gasping and even may present in apnea. Bronchoconstriction and excessive bronchial secretions cause these important life-threatening symptoms.
 - With severe exposures, death may result from central respiratory depression and/or complete paralysis of the muscles of respiration. Respiratory failure is the major cause of death in nerve agent poisoning.
- Skin: With small liquid exposures, localized sweating can be observed with the fasciculations. Generalized diaphoresis can be observed with larger exposures.
- Gastrointestinal: Abdominal cramping can be observed. With larger exposures, nausea, vomiting, and diarrhea are more prominent.
- Heart
 - The patient may present with either bradycardia or tachycardia. Heart rate depends on the predominance of adrenergic stimulation (resulting in tachycardia) or of the parasympathetic tone (causing bradycardia via vagal stimulation). Heart rate is an unreliable sign of nerve agent poisoning.

- Many disturbances in cardiac rhythm have been reported after both organophosphate and nerve agent poisonings. Heart blocks and premature ventricular contractions can be observed. The 2 arrhythmias of greatest concern that have been reported include torsade de pointes and ventricular fibrillation.
- Most signs and symptoms are related to the excessive activation and subsequent fatigue at the cholinergic receptor. Some authors have divided exposures into minimal, moderate, and severe toxicity. Signs and symptoms associated with each exposure are summarized in Table 4.

Table 4. Severity of Toxicity from Liquid and Vapor Exposures

| Severity of Exposure | Signs and Symptoms - Liquid | Signs and Symptoms - Vapor |
|-----------------------------|---|---|
| Onset of symptoms | Possibly delayed toxicity | Rapidly manifesting toxicity |
| Minimal | Localized sweating at site Localized fasciculations at site | Miosis Rhinorrhea Mild dyspnea |
| Moderate | Fasciculations Diaphoresis Nausea, vomiting, and diarrhea Generalized weakness | Above symptoms and the following: Moderate-to-marked dyspnea (bronchorrhea and/or bronchoconstriction) |
| Severe | Above symptoms and the following: Loss of consciousness Seizures Generalized fasciculations Flaccid paralysis and apnea | Above symptoms and the following: Loss of consciousness Seizures Generalized fasciculations Flaccid paralysis and apnea |

Causes

Nerve agents are not readily available. Suspect nerve agent exposures in military or research laboratory workers who may have access to these substances. Also, suspect nerve agent poisoning when several patients present with signs of cholinergic overstimulation. This presentation would be typical during a terrorist attack.

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Differentials

CBRNE - Chemical Warfare Agents
 CBRNE - Nerve Agents, Binary: GB2, VX2
 CBRNE - Nerve Agents, G-series: Tabun, Sarin, Soman
 Toxicity, Organophosphate and Carbamate

Other Problems to be Considered

Diagnosis of toxicity from a nerve agent is suggested when several persons present with the symptoms discussed in [Clinical](#).

Differential diagnosis mainly includes poisoning by organophosphate or carbamate insecticides.

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Workup

Lab Studies

- Exposure to VX in both the vapor and liquid forms has been studied since the 1950s. Laboratory tests do not aid in the acute treatment of patients exposed to nerve agents. AChE testing is most useful in treating chronic exposures when the clinician is able to compare values to an individual's baseline. If a clinician is caring for a single patient or a small group of patients, sending AChE levels for documentation and ongoing treatment is nevertheless prudent. Never withhold treatment while waiting for laboratory results.
- Red blood cell cholinesterase (RBC-AChE) level: This is believed to be the most reliable indicator of the tissue cholinesterase status.
- Plasma cholinesterase (butyrylcholinesterase) levels: These also are referred to as pseudocholinesterase levels, because they are less predictive of CNS cholinesterase activity. This often is the earliest enzyme to be inhibited by organophosphates, but this is not true for some nerve agents, particularly VX and GB.
- Other laboratory studies: Order basic laboratory tests for all but minimally symptomatic patients. Electrolytes and arterial blood gases may aid in the evaluation of the fluid status and the acid/base balance.

Imaging Studies

- Chest x-ray: Typically, request a chest x-ray for dyspneic and intubated patients.

Other Tests

- Electrocardiogram - For palpitations, irregular rhythm, or dysrhythmia noted on monitor

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Treatment

Prehospital Care

An important concept to keep in mind is that rescue personnel, if not properly protected, can become victims. The

cornerstones of prehospital management are based on rapid termination of the exposure, treating the life-threatening emergencies, and administration of antidotes whenever indicated and available.

- Ideally, decontaminate prior to transportation of the victim. Move decontaminated victims to a clean area to prevent cross-contamination of patients and medical personnel. Decontamination techniques vary with the extent and route of exposure.
 - With a vapor, removal of the victim and provision of fresh air is the most important step.
 - If the exposure is dermal, undress the patient. If droplets can be seen, blot them away without forceful wiping. Abrading the skin increases absorption of the agent. Agents also can be neutralized with alkaline solutions such as soap and water or 0.5% hypochlorite solution, which releases chlorine, followed by a water rinse. Avoid unnecessary delays of decontamination while looking for hypochlorite solution if simple soap and water is available.
- During a mass casualty incident, most patients arrive to the emergency department (ED) without the benefit of emergency medical services (EMS) or HAZMAT team treatment. In the Tokyo subway sarin attack, 85% of patients arrived by private car. This emphasizes the importance of proper decontamination facilities, training, and personnel at the ED, since most victims are likely to be contaminated fully upon their arrival at the hospital.

Emergency Department Care

- If decontamination has not occurred, ED personnel should be able to provide this intervention prior to the patient's entrance to the hospital. If weather permits, decontamination stations can be set up outside.
- All hospital personnel in contact with contaminated individuals must wear full personal protective equipment (PPE) at either the A or B levels.
 - Level A PPE refers to the highest level of respiratory protection and protective clothing. It is a fully encapsulated, chemical-resistant, vapor-protective suit that provides vapor protection to the respiratory and mucous membranes and skin. A self-contained breathing apparatus (SCBA) with a full face piece must be worn inside the suit.
 - Level B still provides the highest level of respiratory protection with SCBA but with a lesser level of skin protection. Level B suits are not encapsulated and do not protect the skin from vapor exposures.

Consultations

Whenever the diagnosis of nerve agent exposure is suspected, contact the regional poison center for advice. In a multiple casualty incident, activate the hospital emergency plan and notify local authorities.

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Medication

[Table 5](#) summarizes different agents used to treat nerve agent-poisoned patients. [Table 6](#) provides an overview of general treatment guidelines.

Gases

All but the mildest exposures have some degree of respiratory compromise. For this reason, oxygen should be readily available. Most of these symptoms result from bronchorrhea and bronchoconstriction and improve after administration of antidotes. In the severely poisoned patient, respiratory muscle paralysis adds to the problem. Intubation and mechanical ventilation are required for these patients.

| | |
|-------------------|---|
| Drug Name | Oxygen- Assists patients with respiratory compromise. |
| Adult Dose | Supplement as needed |
| Pediatric Dose | Supplement as needed |
| Contraindications | None reported |
| Interactions | None reported |
| Pregnancy | A - Safe in pregnancy |
| Precautions | Inspired oxygen concentrations of 50-100% carry a substantial risk of lung damage |

Anticholinergic

Antagonizes ACh at the muscarinic receptor.

| | |
|-------------------|---|
| Drug Name | Atropine (Isopto, Atropair, Atropisol)- Antagonizes ACh at its receptor; acts only at muscarinic receptor, leaving nicotinic receptors unaffected; in contrast to organophosphate insecticides, nerve agents rarely require >20 mg; administer until excess muscarinic symptoms improve; this can be gauged by improved ease of breathing in conscious patient or improvement in ease of ventilation of intubated patient; airway patency is critical, life-saving endpoint in treatment. |
| Adult Dose | Usual starting dose: 2 mg IV/IM/ETT; dose can be repeated after 5-10 min in boluses of 2-4 mg |
| Pediatric Dose | Usual starting dose: 0.02 mg IV/ETT (minimal dose 0.1 mg); dose can be repeated q5-10min, titrated to clinical response |
| Contraindications | Documented hypersensitivity; thyrotoxicosis; narrow-angle glaucoma; tachycardia |
| Interactions | Coadministration with other anticholinergics has additive effects; pharmacologic effects of atenolol and digoxin may increase with atropine; antipsychotic effects of phenothiazines may decrease with this medication; TCAs with anticholinergic activity may increase effects of atropine |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Caution in Down syndrome and/or children with brain damage to prevent hyperreactive response; caution also in coronary heart disease, tachycardia, congestive heart failure, cardiac arrhythmias, hypertension, peritonitis, ulcerative colitis, hepatic disease, and hiatal hernia with reflux esophagitis; in prostatic hypertrophy, prostatism can have dysuria and may require catheterization |

Oximes

Reactivators of AChE; 2-PAM Cl, also known as pralidoxime, is widely available in the US; administer concomitantly with atropine. After aging (irreversible binding of agent with AChE enzyme) occurs, usefulness of pralidoxime is negligible. VX has a slow aging process (aging half-life has been calculated at 48 h or more), so delayed treatment with oximes may be beneficial. Pralidoxime has a half-life of 1 hour and is excreted renally. Another subset of oximes termed the H oximes (H is for Hagedorn) include agents such as HI-6, HGG-12, and HGG-42; studies exist using these

antidotes in the military setting, but the drugs currently are not available for use in the US.

| | |
|-------------------|---|
| Drug Name | Pralidoxime (2-PAM Cl, Protopam)- Reactivators of AChE. |
| Adult Dose | Recommended dose: 15-25 mg/kg IV/IM (military Autoinjectors are IM); infuse IV over 20 min to prevent hypertension, one of the most common complications; can repeat in 1 h if needed |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Use barbiturates with caution because action of barbiturates is potentiated by AChE inhibitors; antagonism with neostigmine, pyridostigmine, and edrophonium; morphine, theophylline, aminophylline, succinylcholine, reserpine, and phenothiazines can worsen condition of patients poisoned by organophosphate insecticides or nerve agents (do not administer) |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Infuse IV dose over 20 min to prevent one of the most common complications, hypertension, which usually is transient but can be treated with phentolamine (5 mg IV) if severe; rapid injection can cause tachycardia, laryngospasm, muscle rigidity, pain at injection site, blurred vision, diplopia, impaired accommodation, dizziness, drowsiness, nausea, tachycardia, hypertension, and hyperventilation; can precipitate myasthenia crisis in patients with myasthenia gravis and muscle rigidity in normal volunteers; decrease in renal function increases drug levels in the blood because 2-PAM is excreted in urine; can produce transient elevation in creatine phosphokinase; 1 of 6 patients has an elevation in SGOT and/or SGPT |

Benzodiazepines

Seizures can result from severe nerve agent poisoning; for this reason, treatment with benzodiazepines has been advocated as part of the antidotal armamentarium. Experts advocate use in moderately-to-severely poisoned patients, even prior to seizure onset, as well as in actively seizing patients.

| | |
|-------------------|---|
| Drug Name | Diazepam (Valium, Diazemuls)- Belongs to benzodiazepine family, the members of which act by stimulating GABA, the main inhibitory neurotransmitter in the CNS. Stimulation of GABA results in sedation and increased seizure threshold. |
| Adult Dose | 2-5 mg IV or 10 mg IM |
| Pediatric Dose | 0.2-0.4 mg/kg IV |
| Contraindications | Documented hypersensitivity; narrow-angle glaucoma |
| Interactions | Increases toxicity of benzodiazepines in CNS with coadministration of phenothiazines, barbiturates, alcohols, and MAOIs |
| Pregnancy | A - Safe in pregnancy |
| Precautions | Large doses can result in excessive sedation and potential airway compromise; caution with other CNS depressants, low albumin levels, or hepatic disease (may increase toxicity) |

Table 5. Drugs Used to Treat Nerve Agent-Poisoned Patients*

| Drug | Dose | Route | Indications | Contraindications |
|---|--|-----------|---|--|
| Atropine | 2 mg q5-10min prn | IV/IM/ETT | Excessive muscarinic symptoms | Relative - IV route in hypoxia has been associated with ventricular fibrillation |
| 2-PAM Cl (pralidoxime chloride, Protopam) | 15-25 mg/kg over 20 min; can be repeated after 1 h | IV/IM | Symptomatic nerve agent poisoning | Rapid infusion may result in hypertension |
| Diazepam (Valium) | 2-5 mg IV or 10 mg IM | IV/IM | Active seizures; administer as prophylaxis if moderate or severe signs of poisoning are present | None |

FACE="Arial">*Adapted from Sidell

Table 6. Summary of Treatment Modalities According to Severity of Exposure*

| Severity/Route of Exposure | Atropine | 2-PAM Cl | Diazepam | Other |
|----------------------------|---|---|--|---|
| Suspected | No | No | No | Decontamination and 18-h observation for liquid exposures |
| Mild | 2 mg for severe rhinorrhea or dyspnea; may repeat prn | Administer if patient has nonimproving dyspnea or GI symptoms | No | Decontamination and 18-h observation for liquid exposures; oxygen |
| Moderate | 6 mg; may require repeat doses | Administer with atropine | Administer even in absence of seizures | Decontamination, oxygen |
| Severe | Start with 6 mg; may need to repeat | Administer with atropine; should repeat once or twice | Administer even in absence of seizures | ABCs, decontamination |

* Adapted from Sidell

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Follow-up

Further Inpatient Care

- Admit patients with liquid exposures for observation. Onset of symptoms with these exposures has been observed to be delayed as long as 18 hours. After vapor exposure with only minimal symptoms, the patient usually can be discharged home. Admit patients who are more than minimally symptomatic for observation and further inpatient care.

Further Outpatient Care

- Patients who are discharged from the hospital generally do not require further care. Nerve agents have not been associated with organophosphate-induced delayed neuropathy. Advise patients with miosis not to drive at night until this symptom resolves.

in/Out Patient Meds

- Generally, none are needed.

Complications

- Patients with status epilepticus may suffer from anoxic brain injury.

Prognosis

- If patients recover from the acute effects of exposure, chronic effects should not occur. Subtle behavioral and cognitive changes have been noted to persist for days to weeks after the initial exposure. Patients may have permanent sequelae if they suffered from anoxia during the acute phase of poisoning.

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Miscellaneous

Medical/Legal Pitfalls

- Careful documentation of physical findings, response to treatment, and laboratory parameters is important.
 - In a terrorist attack, any evidence collected can be used to prosecute the perpetrators.
 - In occupational accidents, data are needed to make recommendations for follow-up care and for determining dates for possible return to work. Documentation of an occupational exposure to a nerve agent such as VX also helps to improve safety in the workplace.

Special Concerns

- Special concerns (pregnant, pediatric, geriatric): Information from nerve agents has been gathered mainly from accidental exposures or volunteer studies in military personnel. Little information exists regarding effects or

outcome for children or other special populations.

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CBRNE - Personal Protective Equipment

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Synonyms, Key Words, and Related Terms

PPE, protective respiratory devices, chemical protective clothing, universal precautions

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Introduction

Personal protective equipment (PPE) refers to the respiratory equipment, garments, and barrier materials used to protect rescuers and medical personnel from exposure to biological, chemical, and radioactive hazards. The goal of PPE is to prevent the transfer of hazardous material from patients or the environment to health care workers. Different types of PPE may be used depending on the hazard present.

The types of hazards addressed in this article include biological warfare agents (BWAs), chemical warfare agents (CWAs), and radioactive agents. The most common routes of exposure to these hazards include inhalation, dermal contact, and ingestion.

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Routes of Exposure to Hazards

Routes of exposure to biological warfare agents

Exposure to BWAs is most likely to occur by inhalation of biological aerosols. BWA particles of 1-5 μm in diameter are inhaled most efficiently into the pulmonary alveoli. Mucous membranes or abraded skin also are vulnerable and require protection against BWAs. Conversely, dermal contact does not pose a significant risk, since intact skin provides an effective barrier to all BWAs except trichothecene mycotoxins. Insignificant amounts of aerosolized BWA particles adhere to clothing or skin. Secondary aerosols are not generated efficiently. Ingestion is a minor route of exposure but inadvertently may occur with hand-to-mouth contact or by swallowing contaminated secretions.

Routes of exposure to chemical warfare agents

Exposure to chemicals and CWAs occurs by inhalation of chemical gas or vapor. Exposure also occurs by direct contact of the eyes or skin to chemical vapor or liquid. Mucous membranes are particularly vulnerable, since moisture promotes the absorption of many chemicals. Ingestion is a minor route of exposure.

Routes of exposure to radioactive agents

Patients exposed to beams of ionizing radiation (eg, patients receiving diagnostic x-rays) do not emit radiation and therefore pose no radiation danger to others. In the setting of an explosion, fire, or spill of radioactive material, victims can become contaminated with radiation-emitting material. External contamination occurs when radioactive material gets on a victim's clothing, skin, or hair. Victims also can become contaminated internally if radioactive material enters the body through the gastrointestinal tract, an open wound, or less likely, inhalation of highly radioactive dust. In any situation, the goal of PPE is to prevent the transfer of radioactive material from the victim to the rescuer until the victim is decontaminated.

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Civilian Personal Protective Equipment

Civilian PPE refers to the PPE typically worn by civilian rescue or emergency care workers. The goal of civilian PPE is to protect emergency personnel while they perform essential response functions in contaminated environments or with contaminated patients. Various types of emergency personnel require PPE, including first responders working in the hot zone (exclusion zone or contaminated area), emergency medical personnel involved in field decontamination, and hospital personnel involved in decontamination at the hospital. Physicians rarely require PPE unless they are participating in prehospital response (usually as part of a specialized team) or providing medical care to contaminated patients at the hospital.

Many types of PPE are currently available, ranging from maximum protection with a positive pressure respirator and total body encapsulation to minimum protection with a simple surgical mask and a pair of latex gloves. The various types of protective respiratory devices and clothing are described below.

Protective Respiratory Devices

Two basic types of respirators exist: atmosphere supplying (self-contained breathing apparatus [SCBA], supplied-air respirator [SAR]) and air purifying (APR).

Self-contained breathing apparatus

SCBA consists of a full facepiece connected by a hose to a portable source of compressed air. The open-circuit, positive-pressure SCBA is the most common type. This SCBA provides clean air under positive pressure from a cylinder; the air then is exhaled into the environment. Negative-pressure SCBAs are prohibited by Occupational Safety and Health Administration (OSHA) regulations for hazardous materials (HAZMAT) incidents. SCBA provides the highest level of respiratory protection.

Supplied-air respirator

SAR consists of a full facepiece connected to an air source away from the contaminated area via an airline. Because SARs are less bulky than SCBA, they can be used for longer periods. SARs also are easier for most hospital personnel to use. Although negative-pressure SARs exist, positive-pressure SARs are recommended for HAZMAT incidents. SARs, like SCBA, provide the highest level of respiratory protection.

Air-purifying respirator

An APR consists of a facepiece worn over the mouth and nose with a filter element that filters ambient air before inhalation. Three basic types of APRs exist: powered, disposable, and chemical cartridge or canister. Powered air-purifying respirators (PAPRs) deliver filtered air under positive pressure to a

facepiece mask, helmet, or hood, which provides respiratory and ocular protection. Nonpowered APRs operate under negative pressure, depending on the inspiratory effort of the wearer to draw air through a filter. Because PAPRs function under positive pressure, they provide the greatest degree of respiratory protection. A variety of chemical cartridges or canisters, which eliminate a variety of chemicals including organic vapors and acid gases, are available.

Disposable APRs usually are half masks, which do not provide adequate eye protection. This type of APR depends on a filter, which traps particulates. The use of a high-efficiency particulate air (HEPA) filter or use in combination with a chemical cartridge enhances disposable APRs.

One measure of respiratory filtration efficiency relevant to BWA exposures is the percent penetration of droplet nuclei into the facepiece. For exposures to biological aerosols, PAPRs with HEPA filters are most efficient, followed by elastomeric half-mask HEPA filter respirators and non-HEPA disposable APRs.

All APRs are limited by the adequacy of their face seals. Accordingly, APRs do not provide adequate respiratory protection in environments immediately dangerous to life or health (IDLH).

High-efficiency particulate air filter

HEPA filters remove particles of 0.3-15 μm diameter with an efficiency of 98-100%, efficiently excluding aerosolized BWA particles in the highly infectious 1- to 5- μm range. HEPA filters are incorporated into a variety of protective respiratory devices including PAPRs and elastomeric half-mask respirators (see [Air-purifying respirator](#)).

Surgical mask

Surgical masks are designed to protect the sterile field of the patient from contaminants generated by the wearer. While surgical masks filter out large-size particulates, they offer no respiratory protection against chemical vapors and little against most biological aerosols.

Protective Clothing

Most protective clothing is aimed at protection against chemicals and CWAs, since intact skin provides an effective barrier against all BWAs except the trichothecene mycotoxins.

Chemical-protective clothing

Chemical-protective clothing (CPC) consists of multilayered garments made out of various materials that protect against a variety of hazards. Since no single material can protect against all chemicals, multiple layers of various materials usually are used to increase the degree of protection. Aluminum-lined, vapor-

impermeable garments increase the level of protection. Protection is maximized by total encapsulation. An assortment of types of chemical-protective hats, hoods, gloves, and boot covers complements the garments.

Barrier gown and latex gloves

Barrier gowns are waterproof and protect against exposure to biological materials, including body fluids, but do not provide adequate skin or mucous membrane protection against chemicals. Latex gloves also protect wearers from biological materials but are inadequate against most chemicals. Barrier gowns, surgical masks, latex gloves, and leg and/or shoe covers together comprise "universal precautions."

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Military Personal Protective Equipment

Military PPE refers to the protective respiratory devices, garment ensembles, gloves, and footwear covers worn by military personnel. The purpose of military PPE is to protect military personnel from chemical, biological, and radioactive hazards, while enabling these personnel to accomplish their assigned missions. In general, military PPE used for CWA exposures also protects against BWAs. The PPE used by the US military is not available for civilian use.

M40 mask

The M40 mask is a full-face chemical and biological protective mask that protects the respiratory tract, eyes, and mucous membranes in a manner similar to a nonpowered APR. Available in 3 sizes, the M40 mask combines the protective mechanisms of a charcoal filter against CWA vapors (especially nerve agents and vesicants) and a HEPA filter against BWA particles in 1 screw-on filter canister. Maintenance of this filter canister is critical. Filter canisters must be replaced every 30 days, whenever filter elements are damaged physically or immersed in water, or when excessive breathing resistance is encountered. Other features include 2 voicemitters for communication, optical inserts for visual correction, and a drinking tube.

Battledress overgarments

Battledress overgarments (BDOs) are 2-layered chemical protective overgarments that contain an inner layer of activated charcoal to adsorb penetrating chemical liquids and vapors. BDO also protects against BWAs and radioactive alpha and beta particles. Available in 8 sizes and woodland or desert camouflage patterns, BDOs may be worn up to 24 hours in a contaminated environment. Contaminated BDOs must be incinerated or buried.

Chemical-protective gloves

Chemical-protective glove sets consist of a protective outer glove made out of butyl rubber and an inner glove for absorption of perspiration. Glove sets are available in 4 sizes and 3 thicknesses (7, 14, and 25 mL) with varying tactile sensitivities. Gloves may be worn for 12 hours in the contaminated environment. After visual inspection, gloves may be reused for another 12 hours. After use, gloves may be decontaminated and reused.

Chemical-protective footwear covers

Chemical-protective footwear covers (CPFC) are single-sized butyl rubber footwear covers that protect combat boots against all agents. Vinyl overboots also are available.

Patient protective wraps

Patient protective wraps (PPWs) or casualty wraps are chemical-protective and biological-protective wraps for casualties in contaminated environments in which personnel are unable to wear BDOs. The top of the PPW has a charcoal lining similar to the BDO, while the bottom is constructed of impermeable rubber. Breathing occurs through the permeable PPW top, which functions as a protective respiratory mask.

Wartime personal protective equipment for civilians

The chemical infant protective system (CHIPS) is a semiclosed hoodlike system designed to protect infants in contaminated environments. This protective device delivers filtered air via a battery-operated blower. CHIPS is available for civilian use in Israel.

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Levels of Personal Protective Equipment

Civilian Personal Protective Equipment

The US Environmental Protection Agency has graded PPE into 4 levels based on the degree of protection provided. Each level of PPE consists of a combination of the protective respiratory equipment and clothing, which protects against varying degrees of inhalational, ocular, or dermal exposure.

Level A

Level A PPE consists of a SCBA and a totally encapsulating chemical-protective (TECP) suit. Level A PPE provides the highest level of respiratory, eye, mucous membrane, and skin protection.

Level B

Level B PPE consists of a positive-pressure respirator (SCBA or SAR) and nonencapsulated chemical-resistant garments, gloves, and boots, which guard against chemical splash exposures. Level B PPE provides the highest level of respiratory protection with a lower level of dermal protection.

Level C

Level C PPE consists of an APR and nonencapsulated chemical-resistant clothing, gloves, and boots. Level C PPE provides the same level of skin protection as Level B, with a lower level of respiratory protection. Level C PPE is used when the type of airborne exposure is known to be guarded against adequately by an APR.

Level D

Level D PPE consists of standard work clothes without a respirator. In hospitals, Level D consists of surgical gown, mask, and latex gloves (universal precautions). Level D PPE provides no respiratory protection and only minimal skin protection.

Military Personal Protective Equipment

Military PPE also has been graded into levels, which are known as mission-oriented protective postures (MOPP). Seven levels of MOPP have been defined, ranging from MOPP ready (prepared to use MOPP gear within 2 h) to MOPP 4 (maximum protection in protective respiratory mask and BDO). The higher the level of MOPP, the greater is the level of protection (and greater is the negative impact on individual performance).

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Choice of Personal Protective Equipment

Emergency care personnel who provide medical care to victims of hazardous incidents have the responsibility of first protecting themselves by wearing adequate PPE. Whenever possible, select the level of PPE based on the known properties of the hazard. When the type of hazard is unknown, assume a "worst case" exposure and use the highest level of adequate PPE.

The primary consideration in selecting appropriate PPE is whether it will be worn in the hot zone (exclusion zone or contaminated area) or in the warm zone (contamination reduction zone or area where

decontamination of patients takes place). Since patients and equipment should be decontaminated thoroughly before leaving the warm zone, PPE is unnecessary in uncontaminated areas (except as noted below).

Hot Zone

The hot zone is IDLH. Accordingly, Level A PPE with SCBA or SAR is required for first responders or other personnel working inside the hot zone, where contact with HAZMAT is likely, including chemical gas or vapors, biological aerosols, or chemical and/or biological liquid or powder residua. Incidents occurring in enclosed spaces with poor ventilation increase the risk of inhalation.

Warm Zone

The warm zone is an uncontaminated environment into which contaminated victims, first responders, and equipment are brought. In classic HAZMAT response, the warm zone is adjacent to and upwind from the hot zone. However, experience with previous disasters indicates that contaminated victims capable of fleeing the hot zone are likely to bypass emergency medical services (EMS) and go directly to the nearest hospital, in which case the warm zone may occur outside the emergency department or even inside the hospital.

Accordingly, the warm zone poses the risk of contaminated victims and equipment, which in turn depends on the type and route of exposure. In general, early recognition of the type of exposure is based on the clinical presentation of victims. The PPE required depends on whether victims were exposed to a BWA, CWA, radiological agent, or agent(s) of unknown identity. The route of exposure may be inferred from the presence of contaminant on the clothing and skin of victims. Vapor or aerosol exposure leaves no or minimal contaminant on victims, and off gassing from the lungs does not occur. Liquid or powder exposures leave visible residua. For example, in the Tokyo subway sarin attack in 1995, approximately 90% of victims exposed to sarin vapor reported to medical facilities by private or public transportation without notable contamination of others. Secondary injury to hospital staff was minimal (mostly miosis) and did not necessitate specific treatment. In a similar manner, handling patients

exposed to biological aerosols poses little risk to emergency care personnel outside the hot zone.

Known biological warfare agent hazards

- Personnel handling patients contaminated with BWAs require respiratory protection. Dermal protection is largely unnecessary, since BWAs are not dermally active (with the single exception of the mycotoxins).
- Personnel handling victims who have been exposed to a known BWA aerosol are not required to wear PPE since secondary aerosolization of residual agent from clothing, skin, or hair is insignificant.

- When victims are contaminated with a known BWA liquid or powder, Level D PPE (universal precautions) and PAPR with HEPA filter are required until decontamination is complete. Level C PPE and PAPR with HEPA filter may be considered if residues on victims is suspected of containing mycotoxins.

Known chemical warfare agent hazards

- Personnel handling patients contaminated with CWAs require respiratory and dermal protection.
- When victims are exposed to a known CWA gas at standard temperature and pressure (STP; eg, chlorine, phosgene, oxides of nitrogen, cyanide), no PPE is required, since off gassing is insignificant.
- When victims are exposed to a known CWA vapor from volatile liquid (eg, nerve agent, vesicant vapor), PPE is required, since off gassing may result in low-level exposure of responders.
- When victims are contaminated with a known CWA volatile liquid (eg, nerve agent liquid, vesicant liquid), Level C PPE with PAPR and chemical cartridge is required until decontamination is complete. In general, Level C PPE is used when the inhalation risk is known to be below the concentration-time product expected to harm personnel and when eye, mucous membrane, and skin exposures are unlikely.

Known radiation hazards

- When victims are exposed to external radiation but not contaminated with a radiation-emitting source, no PPE is required. If any doubt exists whether victims or their clothing are contaminated, they should be surveyed with a Geiger-Müller counter.
- When victims are contaminated externally with radioactive material (skin, hair, wounds, clothes), use Level D PPE (ie, waterproof barrier materials, such as surgical gown, mask, gloves, leg, and/or shoe coverings; universal precautions) until decontamination is complete. Double layers of gloves and frequent changes of the outer layer help reduce the spread of radioactive material. Handle radioactive materials with tongs whenever possible. Lead aprons are cumbersome and do not protect against gamma or neutron radiation. For this reason, experts currently recommend against their use when caring for a radiation-contaminated patient. Health care workers also should wear radiological dosimeters while working in a contaminated environment. The health care facility radiation safety officer usually supplies these devices.
- When victims are contaminated internally with radioactive material, wear latex gloves when handling body fluids (urine, feces, wound drainage). The health care facility radiation safety officer or health physicist can determine when the amount of radioactivity in the patient's body secretions has fallen to a nondangerous level.

Unknown hazards (BWA, CWA, or both)

According to current US OSHA regulations, Level B PPE is required for emergency medical personnel responding to an unknown hazard. For hospital personnel using Level B PPE, SAR is recommended, since SCBA is more cumbersome to use. Some experts maintain that Level C PPE with PAPR (with organic vapor cartridge and HEPA filter) provides adequate protection until decontamination is complete. Unfortunately, no single ensemble of PPE can protect emergency care personnel against all hazards.

Cold Zone

By definition, the cold zone should be completely uncontaminated. Nevertheless, patients exposed to certain BWAs may develop transmissible disease, which then poses a risk of secondary spread to medical personnel. The type of PPE required depends on the route of transmission of these infectious diseases.

Respiratory droplet/airborne particles

PAPR with HEPA filter provides the greatest degree of respiratory protection against BWA-associated disease spread by respiratory droplet (ie, smallpox, pneumonic plague) or airborne particles (possibly smallpox) when treating patients with overt disease. Disposable HEPA filter masks also suffice. Evidence exists that smallpox may be transmitted by airborne particles under certain circumstances. Some patients develop a very dense confluent rash and severe cough when infected with variola. These patients are also likely to have many lesions involving the oral mucosa and pharynx. During bouts of severe cough, they may shed virus as an airborne aerosol. One well-documented episode of this form of transmission occurred at the Meschede Hospital in Germany in January 1970. Medical personnel should wear latex gloves while handling the skin of patients with smallpox, since smallpox also is transmitted by contact with pox lesions that have not yet crusted over.

Blood or body fluid

While in contact with patients with BWA-associated disease spread by blood or body fluid contact (ie, hemorrhagic fever viruses), wear Level D PPE (universal precautions).

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Limitations of Personal Protective Equipment

PPE is associated with a number of potential limitations, as listed below. In general, higher levels of PPE are more difficult to use.

- Takes time to put on: Level A PPE takes the longest time to put on.
- Impaired dexterity: Some first responders or emergency care personnel may experience difficulty in performing some life-saving interventions.
- Impaired mobility: Mobility decreases with weight. Mobility also is limited by using a SAR, since the wearer must retrace his or her steps along the supplied airline to exit hot zone.
- Impaired communication: Wearing a facepiece or mask commonly results in poor speech intelligibility.
- Impaired vision: Facepieces also may limit the wearer's visual field.
- Heat stress: Encapsulation and moisture-impermeable CPC material lead to heat stress.
- Increased weight: Level A with SCBA is the heaviest PPE.
- Psychological stress: Encapsulation increases the psychological stress to wearers and patients.
- Limited duration of use: Wearing Level A PPE for longer than 30 minutes is difficult.
- Limited oxygen availability: SCBAs only can be used for the period of time allowed by the air in the tank. APRs only can be used in environments in which the ambient air provides sufficient oxygen.

PPE also is associated with potential "hazards" or risks to wearers, as follows:

- Improper use: Protective respiratory devices and CPC must be properly fitted, tested, and periodically checked before use. An improper fit is an avoidable cause of penetration.
- Penetration: Penetration refers to the process by which HAZMAT may penetrate openings in protective respiratory equipment or clothing. The risk of penetration increases with the use of negative-pressure respirators.
- Permeation: Permeation refers to the process by which HAZMAT cross through protective barriers. Permeation depends on both the properties of the protective garment (or equipment) and concentration of chemical at surface. Permeation is measured in terms of the breakthrough time.
- Degradation: Degradation refers to the process by which structural characteristics of PPE are degraded by contact with chemical substances. Degradation allows permeation or penetration.
- Recontamination: Wearers may become contaminated during PPE removal unless decontamination

and PPE removal protocols are followed systematically.

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Personal Protective Equipment Training, Regulation, and Conclusion

Personal Protective Equipment Training

The use of any type of PPE requires adequate training. The overall goals of PPE training are to protect the wearer from physical hazards (biological, chemical, radioactive) and to prevent injury from improper use or equipment malfunction. Appropriate training topics include hazard identification, medical monitoring, environmental surveillance, and the selection, use, maintenance, and decontamination of PPE.

Personal Protective Equipment and US Regulatory Agencies

Occupational Safety and Health Administration

US OSHA requires that hospitals participating in community emergency response plans to HAZMAT incidents comply with hazardous waste operations emergency response (HAZWOPER) standards. Accordingly, emergency medical personnel responding to a HAZMAT incident in the US are required to wear an appropriate level of PPE. Furthermore, OSHA regulations require that a minimum of Level B PPE be used for emergency medical personnel responding to an unknown hazard. New OSHA standards also require that employees who serve as first responders to HAZMAT incidents receive 8 hours of initial training in the use of PPE.

Joint Commission on Accreditation of Healthcare Organizations

The Joint Commission on Accreditation of Healthcare Organizations (JCAHO) requires that accredited healthcare institutions in the US have emergency procedures that define the use of PPE during HAZMAT exposures. JCAHO specifically requires that every institution with an emergency department have a plan for treating at least 1 contaminated patient.

National Institute for Occupational Safety and Health

The US National Institute for Occupational Safety and Health (NIOSH) establishes the technical criteria for certification of respiratory protective equipment and makes recommendations for its use.

Conclusion

This article is intended as an introduction to the types and levels of PPE currently available. The optimal choice of PPE remains challenging, since little scientific evidence is available to guide selection. Even OSHA regulations and expert recommendations may disagree at times, and neither is supported by demonstrations of increased safety or improved outcomes. Furthermore, higher levels of protection also increase costs, physical stress, and training requirements. Nevertheless, 2 important principles remain to guide the optimal choice of PPE. Whenever possible, choose the level of PPE based on the known properties of the hazard. When the types or properties of the hazard are unknown, assume a "worst case" exposure and use the highest level of adequate PPE.

Pictures





Picture 1: Rescuer wearing Occupational Safety and Health Administration (OSHA) Level A protection. Note that he is encapsulated completely with a self-contained breathing apparatus (SCBA). This type of suit provides the highest degree of both dermal and respiratory protection and is appropriate for wear in an immediate danger to life and health (IDLH) environment (ie, hot zone). However, the garment severely limits communication and provides a great deal of heat stress (photo credit: Tom Blackwell, MD).

Picture type: Photo



Picture 2: Rescuer wearing Occupational Safety and Health Administration (OSHA) Level A protection, rear view. By definition, Level A protection incorporates either a self-contained breathing apparatus (SCBA, shown here) or a supplied-air respirator (SAR). The wearer is encapsulated completely (photo credit: Tom Blackwell, MD).

Picture type: Photo



Picture 3: Rescuer wearing Occupational Safety and Health Administration (OSHA) Level B protection. This type of suit provides excellent splash protection from the front, but the wearer is not encapsulated completely. Air is supplied by a self-contained breathing apparatus (SCBA, shown here) or supplied-air respirator (SAR). Level B protection is appropriate for workers performing patient care and decontamination in the warm zone, in which the victims and their clothing possibly are contaminated with a chemical that could evaporate or be absorbed through the skin (photo credit: Tom Blackwell,

MD).

Picture type: Photo





Picture 4: Rescuer wearing Occupational Safety and Health Administration (OSHA) Level C protection. The dermal protection is the same as with Level B, but the rescuer now is breathing filtered air from a powered air-purifying respirator (PAPR) rather than supplied air from a tank. Because it avoids the weight and complexity of a self-contained breathing apparatus (SCBA) system, Level C protection is much easier to wear and causes less heat stress. Level C protection is appropriate for most activities in the warm zone, unless droplet and/or vapor levels are very high (photo credit: Tom Blackwell, MD).

Picture type: Photo

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CBRNE - Plague

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Introduction

Background

The plague has caused more fear and terror than perhaps any other infectious disease in the history of humankind. It has laid claim to nearly 200 million lives and has brought about monumental changes, such as the end of the Dark Ages and the advancement of clinical research in medicine.

Although still debated by historians, the plague has been responsible for at least 3 great pandemics and multiple epidemics in history. The first spread occurred from the Middle East to the Mediterranean basin during the fifth and sixth centuries AD, killing approximately 50% of the population in these areas. The second pandemic afflicted Europe between the 8th and 14th centuries, destroying nearly 40% of the population. The third pandemic started in approximately 1855 in China and spread to every major continent.

Alexandre Yersin isolated the plague bacillus, developed an antiserum to combat the disease, and postulated its connection with fleas and rats during the epidemic of 1894. The plague bacillus was named

Yersinia pestis in his memory.

Pandemics have succeeded in entrenching the plague in every major continent, with the possible exception of Australia. Unlike smallpox, the plague never will be eradicated. It lives in millions of animals and on billions of fleas that reside on them. It is a disease of the desert, the steppes, the mountains, and the forest.

Pathophysiology

The exact pathophysiology of the plague is unknown. The etiologic agent is *Y pestis*, a facultative anaerobic, intracellular, gram-negative bacillus.

The organism can be transmitted from a host to a human via the bite of a vector. More than 200 different rodents and species can serve as hosts. These include domestic cats and dogs, squirrels, chipmunks, marmots, deer mice, rabbits, hares, rock squirrels, camels, and sheep.

The vector is usually the rat flea, *Xenopsylla cheopis*. Thirty different flea species have been identified as able to carry the plague bacillus. Other carriers of plague include ticks and human lice.

Rodents resistant to the infection form an enzootic stage that ensures the long-term survival of the bacillus. Occasionally, the infected animals are not resistant to the disease and die. This is known as an epizootic stage and ensures the spread of the organism to new territory.

A sylvatic stage occurs when humans are infected from wild animals. Transmission is not only vector mediated but may occur via inhalation of aerosolized bacilli or close contact with infected tissue or fluid.

The bacillus proliferates in the flea's esophagus, preventing food entry into the stomach. To overcome starvation, the flea begins a blood-sucking rampage. Between its attempts to swallow, the distended bacillus-packed esophagus recoils, depositing the bacillus into the victim's skin.

The bacillus invades nearby lymphoid tissue, producing the famous bubo, an inflamed, necrotic, and hemorrhagic lymph node. Spread occurs along the lymphatic channels toward the thoracic duct, with eventual seeding of the vasculature. Bacteremia and septicemia ensue. The bacillus potentially seeds every organ, including the lungs, liver, spleen, kidneys, and rarely, the meninges. Direct inhalation of the bacillus results in pneumonic plague with subsequent bacteremia and septicemia. It causes a multilobar hemorrhagic and necrotizing bronchopneumonia.

The third type of plague is a primary septicemic plague. This is hypothesized to occur when the bacillus is deposited in the vasculature, bypassing the lymphatics. Early dissemination with sepsis occurs but without the formation of a bubo. This usually is observed in bites to the oral, tonsillar, and pharyngeal area and is believed to occur because of the vascularity of the tissue and short lymphatic distance to the thoracic duct.

Frequency

- **In the US:** An average of 18 cases per year has been reported during the last few decades. Repopulation of prairie dogs of the southwestern plains, which had been depleted by an epizootic stage, is nearly complete. Some evidence suggests that a new epizootic stage is beginning, with higher sylvatic infections being reported than anytime since 1992. One of the largest animal foci of the plague worldwide is found west of the 100th parallel, in states such as New Mexico, Arizona, Colorado, Utah, and California. Only one case of imported plague has been reported since 1926.
- **Internationally:** From 1967-1993, an annual average of 1666 cases of the plague has been reported by the World Health Organization. The number of actual cases is probably much higher, given the failure of many countries to diagnose and report the plague. In decreasing order, the following countries have reported the most cases of the plague since 1979: Tanzania, Vietnam, Zaire, Peru, Madagascar, Burma, Brazil, Uganda, China, and the US.

Mortality/Morbidity

- Bubonic plague has a 1-15% mortality rate in treated cases and a 40-60% mortality rate in untreated cases.
- Septicemic plague (primary or secondary) has a 40% mortality rate in treated cases and 100% mortality in untreated cases.
- Pneumonic plague (primary or secondary) has 100% mortality if not treated within the first 24 hours of infection.

Sex

More than 50% of cases occur in males.

Age

Approximately 50% of cases occur in persons younger than 20 years.

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Clinical

History

- Recent travel in the Southwestern and Pacific Coast regions of the US, particularly in New

Mexico, Arizona, California, and Utah, should raise suspicion of a fleabite. Although imported plague is rare, similar suspicion should exist for any recent travel to endemic areas outside the US. Fewer than 10% of patients recall a prior fleabite.

- Close contact with any potentially infected host or rural environment should raise suspicion for the plague. Historically, the rat has been believed to be the main plague host; however, currently in the US, the ground and rock squirrels are the most common hosts. In recent years, the domestic cat has emerged as a prominent host that transmits the plague to veterinarians.
- Symptoms
 - Fever
 - Chills
 - Myalgias
 - Sore throat
 - Headache
 - Weakness
 - Malaise
 - Enlarged, painful, swollen lymph node
 - Abdominal pain - Only presenting symptom more common in a patient presenting with septicemic plague (primary blood-borne plague) versus one presenting with bubonic plague
 - Nausea, vomiting (bloody at times)
 - Constipation, diarrhea, and black or tarry stools
 - Gastrointestinal complaints (may precede a bubo)
 - Cough, which may be productive of bloody sputum
 - Shortness of breath
 - Stiff neck (if meningitic infiltration by plague bacillus has occurred)

Physical

- Temperature of 37-40.9°C, tachycardia, tachypnea, and hypotension, if in late septic shock
- Inguinal bubo (60%), axillary (30%), cervical (10%), or epitrochlear (10%) (usually no greater than 5 cm, extremely tender, erythematous, and surrounded by a boggy hemorrhagic area; Patient often flexes, abducts, and externally rotates the hip near an involved inguinal node to reduce pain at the site.)
- Dermatologic findings
 - A maculopapular lesion may be found at the site of the fleabite; however, such lesions commonly are found at autopsy implying that in the US the diagnosis often is not determined until it is too late.
 - Acral cyanosis, ecchymosis, petechiae, and digital gangrene are seen with *Y pestis* septicemia (from disseminated intravascular coagulation [DIC]).
 - The medieval epithet "Black Death" is thought to have originated from the deeply cyanotic skin, ecchymoses, and/or acral necrosis associated with terminal septicemic and pneumonic plague.
 - The initially rose-colored purpuric lesions most likely gave rise to the child's nursery rhyme "Ring Around the Rosy."

- "Ring around the rosy" - Rose-colored purpuric macules (may be caused by the *Y pestis* enzyme that acts alternately as a plasminogen activator or coagulase at various temperatures or may be due to DIC)
 - "Pocket full of posies" - Sweet-smelling flowers that those tending the sick would carry to ward off the stench of disease
 - "Ashes, ashes" - Impending mortality *or* "A-choo, a-choo" - The sneezing and coughing of pneumonic plague
 - "All fall down" - Death
- Diffuse abdominal tenderness, with or without guarding, splenomegaly, hematochezia, or heme-positive stools
 - Nuchal rigidity and diffuse muscle and joint tenderness
 - Various degrees of mental status changes, ranging from mild confusion or agitation to delirium and coma
 - Seizures
 - Bleeding from any body site or cavity (eg, hematemesis, hematochezia, hemoptysis)
 - Gangrene and necrosis of areas such as the digits, penis, and nares (ascribed to peripheral thrombosis secondary to DIC)
 - Pharyngitis culture positive for *Y pestis* has been seen in endemic areas in household contacts of those with bubonic plague. These patients also have associated cervical lymphadenopathy.

Causes

The etiologic agent is *Y pestis*, a facultative anaerobic, intracellular, gram-negative bacillus. The following are some epidemiologic factors that suggest an increased likelihood of infection with the plague:

- Rural or nonurban residency, especially in geographic areas with known plague foci
- Contact with sick animals, small rodents, or other possible hosts
- Wilderness activities (eg, camping, hiking, sleeping on ground, hunting)
- Fleabite
- Recent plague in the community
- Occupation as a veterinarian
- Summer months

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Differentials

Acute Respiratory Distress Syndrome
Cat scratch Disease

Cellulitis
Disseminated Intravascular Coagulation
Gas Gangrene
Necrotizing Fasciitis
Pediatrics, Pneumonia
Pediatrics, Respiratory Distress Syndrome
Pediatrics, Scarlet Fever
Pneumonia, Aspiration
Pneumonia, Bacterial
Pneumonia, Empyema and Abscess
Pneumonia, Immunocompromised
Pneumonia, Mycoplasma
Respiratory Distress Syndrome, Adult
Scarlet Fever
Shock, Septic

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Workup

Lab Studies

- Complete blood count
 - WBC count usually is markedly elevated to levels of 20,000 or greater.
 - Usually a shift to the left is noted. In late septic shock, the WBC count may be low.
- Arterial blood gas: Arterial blood gas may reveal hypoxia and/or acidosis.
- *Y pestis* coccobacillus identified in peripheral smear (in up to 20% of patients according to some studies)
- Gram stain
 - Gram stain may identify the gram-negative, pleomorphic coccobacillus.
 - Gram stain can be performed on bubo aspirate, sputum, and blood.
 - In 70% of patients, the gram-negative, bipolar-stained coccobacillus is visualized if present. When stained with Wayson or Giemsa stain, a bipolar safety pin structure may be identified.

Imaging Studies

- Chest radiograph
 - Pneumonic plague should produce alveolar infiltrates with or without hilar lymphadenopathy.
 - Bilateral consolidation may be evidenced.

Other Tests

- *Y pestis* fluorescent antibody stain
 - This stain is performed on blood or sputum samples.
 - It may provide rapid diagnosis if available.
 - If unavailable, send specimens to the Centers for Disease Control (CDC), Plague Branch, PO Box 2087, Fort Collins, CO 80522.

Procedures

- Needle aspiration of a bubo
 - The diagnosis may be made by Gram stain and culture of the aspirate.
 - Attempt aspiration even if the lymph node is hard and nonfluctuant.
 - Infusion of 1-3 cm³ of normal saline in the aspiration site prior to aspiration may prove beneficial.
 - Perform this procedure under universal precautions to avoid contamination of health care personnel and the environment.

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Treatment

Prehospital Care

- Provide supportive care.
 - Crystalloid infusion to maintain normal vital signs and clinical hydration state may be necessary.
 - Administer oxygen via nasal cannula, nonrebreather mask, or intubation as determined by the respiratory distress of the patient. Use pulse oximetry to monitor the degree of respiratory compromise.

Emergency Department Care

- Depending on the stage of presentation, supportive care varies. Early presentation may require only crystalloid administration with monitoring of vital signs, clinical state, and urine output.
- Septic shock requires invasive hemodynamic monitoring with crystalloid and vasopressor agents. Airway management may require intubation and mechanical ventilation with positive end-

- expiratory pressure (PEEP).
- Empiric antibiotic coverage is discussed in the [Medication](#) section.
- Use strict isolation precautions.
 - If respiratory symptoms are present, institute universal precautions with strict respiratory isolation for the first 72 hours of therapy.
 - If no respiratory symptoms are present, only 48 hours of isolation or isolation until purulent drainage from the bubo ceases is required.
 - Incinerate or autoclave all contaminated material.

Consultations

- Consult an infectious disease specialist.
- Early notification of the CDC allows samples to be sent to the headquarters in Colorado for diagnosis by fluorescent antibody testing. The CDC, in conjunction with the Department of Health, will attempt to identify the source of the plague and implement early epidemiologic measures to control a potential epidemic.
- Consult a medical intensivist as indicated.
 - In most patients with plague, some degree of septic shock is present.
 - Invasive hemodynamic monitoring and close observation of fluid and cardiac status requires admission to a medical intensive care unit.

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Medication

Medical management of plague can involve a myriad of supportive medications, including crystalloids, colloids, medications used for intubation, vasopressor agents, and antiulcer and antipyretic agents. This section only describes antibiotic management of plague. Early administration of antibiotics is essential, after samples for diagnostic purposes have been obtained.

Antibiotics

Drugs that cover *Y pestis* should be empirically given to any patient with predisposing risk factors and signs and symptoms of the plague. For prophylaxis, all contacts of the patient with the bubonic or septicemic plague should be placed under surveillance. At first sign of illness (eg, fever, adenopathy), begin regular antibiotic treatment. Household contacts of patients with the bubonic or septicemic plague may have been exposed to the same fleas, thus antibiotic prophylaxis is recommended. Prophylaxis also is indicated for all contacts of patients with pneumonic plague (eg, ED and EMS personnel). See discussion on tetracycline below for concrete recommendations.

| | |
|-------------------|--|
| Drug Name | Streptomycin sulfate- DOC in combination with tetracycline or chloramphenicol; due to toxicity, usually is discontinued after first 5 d of treatment, with tetracycline or chloramphenicol continued alone for remaining course; given high mortality of plague, streptomycin and chloramphenicol are antibiotics of choice for treatment of plague in a pregnant patient. |
| Adult Dose | 30 mg/kg/d IM divided bid/qid; not to exceed 2 g/d |
| Pediatric Dose | 20-30 mg/kg/d IM divided bid/qid Newborn infants with transplacental infection by plague should receive gentamicin instead |
| Contraindications | Documented hypersensitivity; those with nondialysis-dependent renal insufficiency |
| Interactions | Nephrotoxicity may be increased with aminoglycosides, cephalosporins, penicillins, amphotericin B, and loop diuretics |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Due to narrow therapeutic index and toxic hazards associated with extended administration, not intended for long-term therapy; adjust dose in patients with renal impairment; caution in myasthenia gravis, renal failure (not on dialysis), hypocalcemia, and conditions that depress neuromuscular transmission |

| | |
|-------------------|--|
| Drug Name | Tetracycline (Sumycin, Tetracyn IV)- Treats susceptible bacterial infections of both gram-positive and gram-negative organisms as well as mycoplasmal, chlamydial, and rickettsial infections; inhibits bacterial protein synthesis by binding with 30S and possibly 50S ribosomal subunit(s) of susceptible bacteria; DOC for use with streptomycin for first 5 d of treatment (or until patient is afebrile) and alone for remaining course. |
| Adult Dose | Loading dose: 15 mg/kg PO; not to exceed 1 g Day 1: 40-50 mg/kg PO q4h Thereafter: 30 mg/kg PO q6h for 10-14 d Loading dose: 5 mg/kg IV Day 1: 15 mg/kg IV q4h Thereafter: 5 mg/kg IV q6h; may switch to PO at any time if patient can tolerate Prophylactic dosing: 25-50 mg/kg/d PO divided qid |
| Pediatric Dose | If suspicion of plague is high, some authors recommend similar dosages and regimens for all pediatric patients, even children <8 y Prophylactic dosing >8 years: 250 mg PO qid for 5-10 d |
| Contraindications | Documented hypersensitivity; those diagnosed with severe hepatic dysfunction |

| | |
|--------------|---|
| Interactions | Bioavailability decreases with antacids containing aluminum, calcium, magnesium, iron, or bismuth subsalicylate; can decrease effects of oral contraceptives, causing breakthrough bleeding and increased risk of pregnancy; can increase hypoprothrombinemic effects of anticoagulants |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Photosensitivity may occur with prolonged exposure to sunlight or tanning equipment; reduce dose in renal impairment; consider drug serum level determinations in prolonged therapy; use during tooth development (last one half of pregnancy through age 8 y) can cause permanent discoloration of teeth; Fanconilike syndrome may occur with outdated tetracyclines |

| | |
|-------------------|--|
| Drug Name | Chloramphenicol (Chloromycetin)- DOC to be used instead of tetracycline in plague meningitis (better CNS penetrations), profound hypotension, pleural or pericardial involvement, and in pregnant patients; binds to 50S bacterial ribosomal subunits and inhibits bacterial growth; effective against gram-negative and gram-positive bacteria. |
| Adult Dose | 50-100 mg/kg/d IV divided q6h 30 mg/kg/d PO divided q6h may be substituted for IV in last 5 d of therapy |
| Pediatric Dose | 0-7 days: 25 mg/kg PO/IV qd >7 days: 50 mg/kg/d PO/IV divided q12h |
| Contraindications | Documented hypersensitivity |
| Interactions | Concurrently with barbiturates, chloramphenicol serum levels may decrease while barbiturate levels may increase, causing toxicity; manifestations of hypoglycemia may occur with sulfonylureas; rifampin may reduce serum chloramphenicol levels, presumably through hepatic enzyme induction; may increase effects of anticoagulants; may increase serum hydantoin levels, possibly resulting in toxicity; chloramphenicol levels may be increased or decreased |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Use only for indicated infections or as prophylaxis for bacterial infections; serious and fatal blood dyscrasias (aplastic anemia, hypoplastic anemia, thrombocytopenia, granulocytopenia) can occur; evaluate baseline and perform periodic blood studies approximately every 2 d while in therapy; discontinue upon appearance of reticulocytopenia, leukopenia, thrombocytopenia, anemia, or findings attributable to chloramphenicol; adjust dose in liver or kidney dysfunction; caution in pregnancy at term or during labor because of potential toxic effects on fetus (gray syndrome) |

| | |
|-----------|--|
| Drug Name | Cotrimoxazole (Bactrim, Septra)- DOC for prophylaxis of pregnant women and children <8 y; inhibits bacterial synthesis of dihydrofolic acid by competing with PABA, inhibiting folic acid synthesis and resulting in the inhibition of bacterial growth. |
|-----------|--|

| | |
|-------------------|--|
| Adult Dose | 1 DS tab PO bid for 5-10 d |
| Pediatric Dose | <2 months: Do not administer 8 mg/kg/d trimethoprim and 40 mg/kg/d sulfamethoxazole PO divided bid for 5-10 d |
| Contraindications | Documented hypersensitivity; those diagnosed with megaloblastic anemia due to folate deficiency |
| Interactions | May increase PT when used with warfarin (perform coagulation tests and adjust dose accordingly); coadministration with dapsone may increase blood levels of both drugs; coadministration of diuretics increases incidence of thrombocytopenia purpura in elderly patients; phenytoin levels may increase with coadministration; may potentiate effects of methotrexate in bone marrow depression; hypoglycemic response to sulfonylureas may increase with coadministration; may increase levels of zidovudine |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Discontinue at first appearance of skin rash or sign of adverse reaction; obtain CBCs frequently; discontinue therapy if significant hematologic changes occur; goiter, diuresis, and hypoglycemia may occur with sulfonamides; prolonged IV infusions or high doses may cause bone marrow depression (if signs occur, give 5-15 mg/d leucovorin); caution in folate deficiency (eg, chronic alcoholics, elderly patients, those receiving anticonvulsant therapy, or those with malabsorption syndrome); hemolysis may occur in individuals with G-6-PD deficiency; patients with AIDS may not tolerate or respond to TMP-SMZ; caution in renal or hepatic impairment (perform urinalyses and renal function tests during therapy); give fluids to prevent crystalluria and stone formation |

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Follow-up

Further Inpatient Care

- Take care to isolate all infected individuals.

Transfer

- Whenever possible, patients suspected of having plague should not be transferred.
- All transfers must comply with Consolidated Omnibus Budget Reconciliation Act (COBRA) regulations.
- Transfers must be performed with the patient in strict isolation.

Deterrence/Prevention

- Use prophylactic antibiotics in contacts of patients with pneumonic plague and in household contacts of patients with bubonic and/or septicemic plague.
- A plague vaccine exists.
 - Its use is recommended only for health personnel who may come into contact with *Y pestis*.
 - The vaccine may be useful for specialized care and for agricultural personnel who work in areas with endemic plague and are unable to minimize contact with wild animals.
 - The amount of protection the vaccine provides is unknown. Studies have demonstrated a decrease in the incidence of the plague and severity of disease.

Complications

- Meningitis
- Septic shock
- DIC
- Skin necrosis
- Pericarditis
- Death

Prognosis

- In those treated for bubonic plague, the mortality rate is 1-15%.
- Primary or secondary septicemic plague has a 40% mortality rate, even in treated patients.
- Pneumonic plague has 100% mortality if not treated within the first 24 hours.

Patient Education

- Avoid contact with wild animals.
- Reduce rat and flea population.

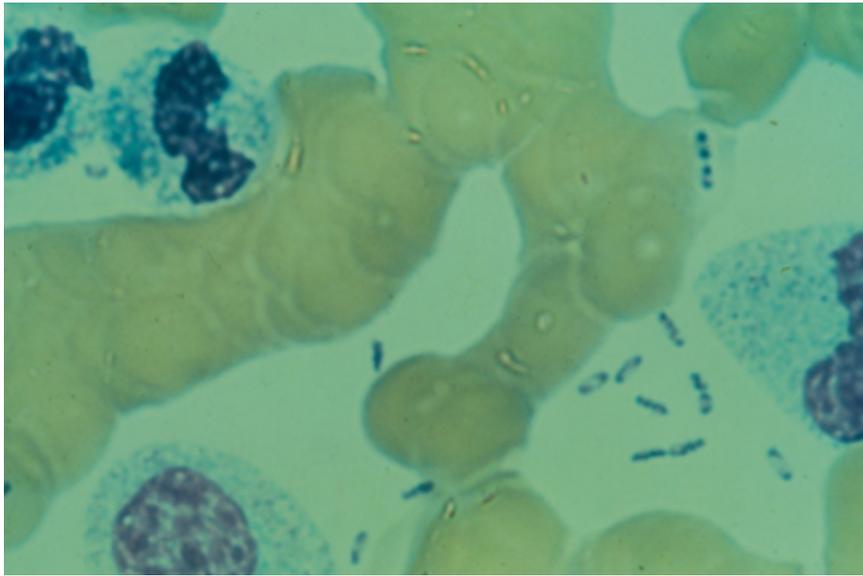
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Miscellaneous

Medical/Legal Pitfalls

- Failure to recognize early plague
 - Failure to institute early antibiotic therapy or empiric therapy as diagnostic studies proceed
 - Failure to ensure strict isolation of infected individuals and prophylactic antibiotics for the contacts
 - Failure to obtain proper consultation from an infectious disease specialist, medical intensivist, the CDC, and the Department of Health
-

Pictures



Picture 1: Wright stain peripheral blood smear of patient with septicemic plague demonstrating bipolar, safety pin staining of *Yersinia pestis*. While Wright stain often demonstrates this characteristic appearance, Giemsa and Wayson stains most consistently highlight this pattern. (Courtesy Jack Poland, PhD, CDC, Fort Collins, CO)

Picture type: Photo



Picture 2: Here a flea is shown with a blocked proventriculus, equivalent to the gastroesophageal region in man. In nature, this flea would develop a ravenous hunger because of its inability to digest the fibrinoid mass of blood and bacteria. Ensuing a biting of the nearest mammal results in clearing of the proventriculus through regurgitation of thousands of bacteria into the bite wound. (Courtesy United States Army Environmental Hygiene Agency)

Picture type: Photo



Picture 3: A suppurative, bubo of the femoral lymph node (a), the most common site of the erythematous, tender, swollen, nodes in a plague victim. The next most common lymph node regions involved are the inguinal, axillary (b), and the cervical areas. The child in (b) has an erythematous, eroded, crusting, necrotic ulcer at the presumed primary inoculation site on the left upper quadrant. This type of lesion is uncommonly found in patients with plague. Bubo location is primarily a function of the region of the body in which an infected flea inoculates plague bacilli. (Photos courtesy Jack Poland, PhD, CDC, Fort Collins, CO)

Picture type: Photo



Picture 4: A suppurative, bubo of the femoral lymph node (a), the most common site of the erythematous, tender, swollen, nodes in a plague victim. The next most common lymph node regions involved are the inguinal, axillary (b), and the cervical areas. The child in (b) has an erythematous, eroded, crusting, necrotic ulcer at the presumed primary inoculation site on the left upper quadrant. This type of lesion is uncommonly found in patients with plague. Bubo location is primarily a function of the region of the body in which an infected flea inoculates plague bacilli. (Photos courtesy Jack Poland, PhD, CDC, Fort Collins, CO)

Picture type: Photo



Picture 5: Ecchymoses at the neck base of a girl with plague. Bandage is over the site of a prior bubo aspirate. These lesions probably gave rise to the title line of the children's nursery rhyme 'Ring around the rosy.' (Courtesy Jack Poland, PhD, CDC, Fort Collins, CO)

Picture type: Photo



Picture 6: Right-side middle and lower lobe involvement in a patient with plague pneumonia. No chest x-ray pattern is characteristic of plague, but bilateral interstitial infiltrates are most commonly seen. (Courtesy Jack Poland, PhD, CDC, Fort Collins, CO)

Picture type: Photo



Picture 7: Rock squirrel in extremis coughing of blood-streaked sputum of pneumonic plague. (Courtesy Ken Gage, PhD, CDC, Fort Collins, CO)

Picture type: Photo



Picture 8: Acral necrosis of nose, lips, fingers (a) and toes (b) and residual ecchymoses over both forearms in a patient recovering from bubonic plague that disseminated to blood and lungs. At one time, the patient's entire body was ecchymotic. (Reprinted from McGovern TW, Friedlander AM. Plague. In: Sidell FR, Takafuji ET, Franz DR, eds. *Medical Aspects of Chemical and Biological Warfare*. Chapter 23 in: Zajтчuk R, Bellamy RF, eds. *Textbook of Military, Medicine*. Washington, DC: US Department of the Army, Office of the Surgeon General, and Borden Institute; 1997:493.) Government publication, no copyright on photos.

Picture type: Photo



Picture 9: Acral necrosis of nose, lips, fingers (a) and toes (b) and residual ecchymoses over both forearms in a patient recovering from bubonic plague that disseminated to blood and lungs. At one time, the patient's entire body was ecchymotic. (Reprinted from McGovern TW, Friedlander AM. Plague. In: Sidell FR, Takafuji ET, Franz DR, eds. *Medical Aspects of Chemical and Biological Warfare*. Chapter 23 in: Zajtchuk R, Bellamy RF, eds. *Textbook of Military, Medicine*. Washington, DC: US Department of the Army, Office of the Surgeon General, and Borden Institute; 1997:493.) Government publication, no copyright on photos.

Picture type: Photo

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CBRNE - Q Fever

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Introduction

Background

First described in Australia in 1935, Q fever is a rickettsial disease with acute and chronic stages. Q fever differs from other rickettsial diseases in that it is caused by inhalation of infected particles, not by a tick bite.

Pathophysiology

The respiratory system is the main organ system affected, although GI and cardiac systems also are affected. Incubation period varies from 9-40 days, with an average range of 18-21 days.

Frequency

- **In the US:** Frequency is difficult to ascertain because Q fever is not a reportable disease. Dairy and slaughterhouse workers are most at risk.
- **Internationally:** Incidence is worldwide and varies in frequency and presentation from country to country.

Mortality/Morbidity

Mortality with acute infection is reportedly as high as 2.4% but generally is less than 1%.

Sex

Males are affected more than females.

Age

Adults are affected more than children.

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Clinical

History

- Patients initially present with influenzalike symptoms.
- Respiratory symptoms appear 4-5 days after initial onset of illness (most prominently a dry nonproductive cough and pleuritic chest pain).
- Symptoms include the following:
 - Fever
 - Severe headache
 - Myalgias
 - Anorexia
 - Cough
 - Pleuritic chest pain
 - Sweats
 - Chills
 - Nausea, vomiting, and diarrhea (rare)

Physical

Often in acute Q fever, specific findings may not exist. In chronic Q fever, findings consistent with endocarditis and hepatitis more frequently are found.

- Findings in endocarditis include the following:

- Vegetations on any valve (although aortic and prosthetic valves are favored)
- Clubbing of digits
- Hepatomegaly and splenomegaly in approximately one half of patients
- Arterial emboli in approximately one third of patients
- Purpuric rash in approximately 20% of patients
- Aseptic meningitis/encephalitis occurs in approximately 1% of acute and chronic Q fever cases.

Causes

Coxiella burnetii, a pleomorphic coccobacillus that is much smaller than other rickettsias, is the etiologic agent of Q fever. Humans are infected by inhalation of *C burnetii*. Why chronic Q fever develops in certain patients is unknown.

- Q fever is extremely virulent; 1 bacterium can cause infection.
- Q fever is extremely resistant to inactivation; it can survive for months in dust and feces particles.
- Tick species, naturally infected, infect domestic and small mammals (eg, cats).
- *C burnetii* localizes in the mammary glands, uterus, and feces of these animals. Exposure to feces can lead to disease.
- Laboratory outbreaks have occurred. Only 1 case of documented human-to-human transmission exists.
- *C burnetii* exists in 2 antigenic states, phase I (virulent) and phase II (avirulent).

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Differentials

Endocarditis

Hepatitis

Mononucleosis

Pneumonia, Mycoplasma

Pneumonia, Viral

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Workup

Lab Studies

- Acute Q fever
 - Complete blood count is normal in 70% of patients with acute Q fever, but an elevated white blood count (WBC) is present in 30% of patients.
 - Liver function tests often show a slight elevation (2-3 times) of transaminases, but bilirubin usually is normal.
 - Serologic tests are not helpful acutely but later may confirm diagnosis. A 4-fold rise in complement-fixing antibody titer against phase II antigen occurs. This requires a baseline sample and another sample in 3-4 weeks.

Imaging Studies

- Acute Q fever
 - Chest x-ray is variable (normal-to-widespread pneumonitis).
 - Hepatic ultrasound may show granulomatous hepatitis.

Procedures

- Perform lumbar puncture if Q fever meningoencephalitis is suspected.

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Treatment

Emergency Department Care

- Recognize disease and start appropriate antibiotic therapy.
- Provide supportive care.

Consultations

Consult infectious disease, especially if chronic Q fever is suspected.

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Medication

Antibiotics are necessary for acute and chronic Q fever. However, in chronic Q fever, exact antibiotics recommended for use are in a state of flux. Consult infectious disease prior to starting antibiotics in this situation. Best studied are combinations of doxycycline plus an additional antibiotic (eg, fluoroquinolone, rifampin, trimethoprim-sulfamethoxazole).

Antibiotics

Therapy must cover all likely pathogens in the context of the clinical setting.

| | |
|-------------------|--|
| Drug Name | Doxycycline (Doryx, Bio-Tab, Vibramycin)- Interferes with bacterial cell wall synthesis during active multiplication, causing cell wall death, resulting in bactericidal activity against susceptible bacteria. For severe cases, administer IV; for outpatients, PO is preferred. |
| Adult Dose | 100 mg PO q12h |
| Pediatric Dose | <8 years: Not recommended >8 years: 3 mg/kg/d PO q12h; not to exceed 200 mg/d |
| Contraindications | Documented hypersensitivity; severe hepatic dysfunction |
| Interactions | Bioavailability decreases with antacids containing aluminum, calcium, magnesium, iron, or bismuth subsalicylate; tetracyclines can increase hypoprothrombinemic effects of anticoagulants; tetracyclines can decrease effects of oral contraceptives, causing breakthrough bleeding and increased risk of pregnancy |
| Pregnancy | D - Unsafe in pregnancy |
| Precautions | Photosensitivity may occur with prolonged exposure to sunlight or tanning equipment; reduce dose in renal impairment; consider drug serum level determinations in prolonged therapy; tetracycline use during tooth development (last one half of pregnancy through age 8 y) can cause permanent discoloration of teeth; Fanconilike syndrome may occur with outdated tetracyclines |

| | |
|-------------------|---|
| Drug Name | Chloramphenicol (Chloromycetin)- Binds to 50S bacterial-ribosomal subunits and inhibits bacterial growth by inhibiting protein synthesis; is effective against gram-negative and gram-positive bacteria. In severe cases, administer IV; for outpatients, administer PO. |
| Adult Dose | 500-750 mg PO/IV q6h; not to exceed 4 g/d |
| Pediatric Dose | 50 mg/d PO/IV divided qid |
| Contraindications | Documented hypersensitivity |

| | |
|--------------|--|
| Interactions | Concurrently with barbiturates, chloramphenicol serum levels may decrease, while barbiturate levels may increase, causing toxicity; manifestations of hypoglycemia may occur with sulfonyleureas; rifampin may reduce serum chloramphenicol levels, presumably through hepatic enzyme induction; may increase effects of anticoagulants; may increase serum hydantoin levels, possibly resulting in toxicity; chloramphenicol levels may be increased or decreased |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Use only for indicated infections or as prophylaxis for bacterial infections; serious and fatal blood dyscrasias (aplastic anemia, hypoplastic anemia, thrombocytopenia, granulocytopenia) can occur; evaluate baseline and perform periodic blood studies approximately q2d while in therapy; discontinue upon appearance of reticulocytopenia, leukopenia, thrombocytopenia, anemia, or findings attributable to chloramphenicol; adjust dose in liver or kidney dysfunction; caution in pregnancy at term or during labor because of potential toxic effects on fetus (gray syndrome) |

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Follow-up

Further Inpatient Care

- Valve replacement may be required, depending on cardiac status.

Further Outpatient Care

- Antibiotic therapy should last 10-14 days for acute Q fever.
- Antibiotic therapy lasts a minimum of 2 years for chronic Q fever.

Deterrence/Prevention

- Identify infections in domesticated animal populations.
- Birthing should take place in indoor facilities.
- Properly dispose of placentae, fetal membranes, and aborted material.
- Human Q fever vaccine is available in Australia and Eastern Europe but not in the US.

Prognosis

- Acute Q fever, if properly diagnosed and treated, has very low morbidity and mortality.

- Chronic Q fever requires long-term antibiotic therapy including close follow-up care with an infectious disease specialist.

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Miscellaneous

Medical/Legal Pitfalls

- Obtain an occupational history from patients.
-

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CBRNE - Ricin

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Introduction

Background

Ricin is a potent toxin that has potential to be used as an agent of biological warfare and as a weapon of mass destruction (WMD). Ricin is widely available, easily produced, and derived from the beans of the castor plant (*Ricinus communis*).

In attempting to evaluate and discuss agents that can be used as WMDs, the question, "What can cause a maximum credible event?" hopefully is answered. A maximum credible event is one that could cause a large loss of life in addition to disruption, panic, and overwhelming use of civilian healthcare resources. For an agent to be considered capable of causing a maximum credible event, it should be highly lethal, inexpensively and easily produced in large quantities, stable in aerosol form, and have the ability to be dispersed (1-5 μm). The ideal agent also is communicable from person to person and has no treatment or vaccine.

When ricin's characteristics are applied to this model, its use appears limited but should not be underestimated. Ricin is produced easily and inexpensively, is highly toxic, is stable in aerosolized form,

and has no treatment or vaccine. Its toxicity when compared to living replicating biological agents limits ricin's use. A large amount of ricin is necessary to produce the desired effect of a WMD. For example, the amount of ricin necessary to cover a 100-km² area and cause 50% lethality, assuming aerosol toxicity of 3 mcg/kg and optimum dispersal conditions, is approximately 4 metric tons, whereas only 1 kg of *Bacillus anthracis* is required. Ricin, however, would have efficacy as a disabling agent. Its use as a food and water contaminant easily could incapacitate many and overwhelm local healthcare resources. Thus, its use as a food and water contaminant is a major concern because of ricin's ease of availability.

Ricin can be disseminated as an aerosol, by injection, or as a food and water contaminant.

Pathophysiology

Ricin is a widely available potential toxin that is produced easily. It is a potent protein derived from the beans of the castor plant (*R communis*). Castor beans are used in the production of castor oil, a brake and hydraulic fluid constituent. The aqueous phase of the process, termed the "waste mash," is 5-10% ricin. Separating this 66,000-dalton protein requires chromatography, a common undergraduate chemistry skill. Ricin's ease of availability and its lethality make it an attractive agent for use in biological warfare and for potential use as a WMD. Routes of exposure are respiratory (inhaled aerosol), gastrointestinal (GI [ingested]), and parenteral (injected). Clinical manifestations depend on the route of exposure and the amount of absorption.

Ricin is composed of 2 hemagglutinins and 2 toxins. The toxins RCL III and RCL IV are dimers of approximately 66,000 daltons in molecular weight. The toxins have an "A" and a "B" chain, which are polypeptides and joined by a disulfide bond. The B chain binds to cell surface glycoproteins and affects entry into the cell by an unknown mechanism. The A chain acts on the 60S ribosomal subunit and prevents the binding of elongation factor-2. This inhibits protein synthesis and leads to cell death. This basic structure of ricin is similar to those of the botulinum toxin, cholera toxin, diphtheria toxin, tetanus toxin, and insulin.

Frequency

- **In the US:** From 1991-1997, 3 cases were related to ricin. In 1991 in Minnesota, 4 members of the Patriots Council, an extremist group that held antigovernment and antitax ideals and advocated the overthrow of the US government, were arrested for plotting to kill a US marshal with ricin. The ricin was produced in a home laboratory. They planned to mix the ricin with the solvent dimethyl sulfoxide (DMSO) and then smear it on the door handles of the marshal's vehicle. The plan was discovered, and the 4 men were convicted. In 1995, a man entered Canada from Alaska on his way to North Carolina. Canadian custom officials stopped the man and found him in possession of several guns, \$98,000, and a container of white powder, which was identified as ricin. Lastly, in 1997, a man shot his stepson in the face. Investigators discovered a makeshift laboratory in his basement and found agents such as ricin and nicotine sulfate.

Mortality/Morbidity

Mortality and morbidity depend on the route and amount of exposure.

- Dermal exposure
 - Dermal exposure of ricin is of little concern because the absorption amount is insignificant.
 - To be absorbed dermally, ricin must be enhanced with a strong solvent such as DMSO.
 - Dermal symptoms depend on the type of solvent and length of exposure. Dermal exposure probably is unable to achieve toxicity.
- Parenteral exposure
 - Parenteral exposures can be rapidly fatal, with an LD50 similar to aerosol exposure.
 - The highly publicized case of Georgi Markov is evidence of the rapidly fatal nature of parenteral exposure. Markov, an exiled Bulgarian broadcaster, was waiting for a bus in 1978 when he was jabbed with an umbrella in the lower extremity. He then developed severe gastroenteritis and high fevers and died 3 days later. At autopsy, a small 1.5-mm metallic sphere was found at the wound site. It had 2 tiny holes and could hold a volume of 0.28 mm³. No toxin was isolated. Because of the small volume and rapid demise of the patient, ricin was believed to be the only capable inciting agent. The coroner recreated the scenario by injecting a pig with a similar dose of ricin. The pig died in a similar manner 26 hours later.
 - Ricin, if injected, can cause severe local necrosis of muscle and regional lymph nodes with organ involvement and death.

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Clinical

History

- In the case of an isolated attack such as an assassination attempt, no historical markers may be present.
- A victim may relate the pain of an antecedent injection, but this may be overlooked during the history.
- A patient is unlikely to be aware of contamination of ingested foods or beverages.
- If a number of patients are affected simultaneously, by either ingestion or inhalation, the subsequent cluster of patients presenting with similar symptoms over a brief time may alert an astute clinician to the possibility of an intentional act. This is especially true in the case of an inhalation incident (ingestion initially may mimic food poisoning).

Physical

Perform a complete physical examination with any exposure.

- In parenteral exposure, inspect the site for induration, erythema, and the possibility of a retained foreign body. These physical findings may be present prior to or at the time of systemic manifestations.
- In aerosol exposure, the presentation is that of a rapidly progressive acute lung injury, with findings consistent with the stage of progression from a physical examination with normal findings through hypoxia, cyanosis, labored breathing, tachypnea, tachycardia, and progressive respiratory failure.
- In GI exposure, physical examination should be consistent with that for gastroenteritis and volume depletion. If the dose was sufficient and the disease had progressed, frank hematemesis and/or bloody diarrhea or melena may be present.

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Differentials

CBRNE - Anthrax Infection

CBRNE - Q Fever

CBRNE - Staphylococcal Enterotoxin B

Cellulitis

Other Problems to be Considered

Pneumonic plague

Salmonella

Shigella

Cholera

Necrotizing fasciitis

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Workup

Lab Studies

- Obtain baseline laboratory information. Useful testing includes a complete blood count (which may reveal leukocytosis), electrolytes, BUN, creatinine, glucose, prothrombin time, activated partial thromboplastin time, international normalized ratio, type and screen, fibrinogen, liver enzymes, amylase, and lipase. An arterial blood gas may reveal hypoxemia.
- Specific enzyme-linked immunosorbent assay (ELISA) testing on serum and immunohistochemical techniques for direct tissue analysis are under development.
- Collect acute and convalescent serum to determine measurements of antibody response.

Imaging Studies

- Chest x-ray
 - A chest x-ray may reveal infiltrates or an acute respiratory distress syndrome (ARDS) picture.
 - X-ray also may be useful in parenteral exposures to evaluate for retained foreign body.

Procedures

- Bronchoscopy: If performed, bronchial aspirate may be rich in protein compared to plasma, as observed in any condition causing high-permeability pulmonary edema.

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Treatment

Prehospital Care

Strictly adhere to universal precautions at all times, although secondary dermal absorption to prehospital providers is not expected. The risk of secondary aerosolization is minimal. Use protective masks, which are effective in preventing toxicity, when an overt aerosol attack is suspected.

Emergency Department Care

- Management
 - ED management begins with universal precautions and the ABCs. Add a "D" for decontamination (including the removal of garments). If ingestion is possible, based on the history and presenting findings, consider gut decontamination as well.
 - Management also involves the ability to recognize, diagnose, and treat a possible biological event.
- Diagnosis
 - Diagnosis of an aerosolized attack or food and water contaminant with ricin is similar to that of any of the biological or chemical agents that serve as WMDs. It primarily depends on the clinical and epidemiologic setting. In cases of isolated injection, the diagnosis is extremely difficult.
 - The clinical presentation of acute lung injury in a large number of patients in a particular area should suggest a pulmonary irritant. The clinical presentation of severe gastroenteritis or hemorrhagic gastroenteritis in a large number of patients in a particular area should suggest a food and water contaminant.
 - Include ricin and other agents (eg, staphylococcal enterotoxin B, Q fever, tularemia, pneumonic plague, inhalational anthrax, chemical agents such as phosgene) in the differential diagnosis.
 - Ricin is expected to progress despite antibiotic therapy. Chest x-ray exhibits no evidence of mediastinitis, as would be expected with pulmonary anthrax.
 - Staphylococcal enterotoxin B does not progress to a life-threatening syndrome, and phosgene produces ARDS, which is mediated by exertion. Phosgene also has the characteristic odor of newly mown hay or grass and is quite irritating to mucous membranes in lethal amounts.
- Treatment
 - Treatment and toxicity depend on the route of exposure. Treatment is supportive, and no antidote is available for ricin.
 - Emergency department employees should obey strict universal precautions at all times.
 - For dermal exposure, a weak sodium hypochlorite solution (0.1%) and/or soap and water suffice to decontaminate the skin.
 - For GI exposure, include gastric decontamination with superactivated charcoal, volume replacement, and H₂ blockers in treatment. Include chemistry panels, complete blood count, liver function panel, BUN and creatinine, urinalysis, and type and screen in the laboratory evaluation.
 - For percutaneous exposure, base treatment on excision of the injection site, if possible, within the shortest amount of time. Obtain baseline laboratory information, including arterial blood gas and fibrinogen. Although antibiotics serve no role in the treatment of ricin, withholding such therapy in an acutely septic-appearing patient would be difficult. Antibiotics may serve to prevent infection resulting from the percutaneous mechanism. Update tetanus immunization status if unknown.
 - For aerosol or pulmonary exposure, provide standard critical care treatment directed

toward acute lung injury and pulmonary edema. Maintain a low threshold to secure the patient's airway and ensure adequate oxygenation and ventilation. Obtain a chest x-ray, which may show infiltrates. The clinical course progresses despite antibiotic therapy.

- The only effective treatment for ricin toxicity is prevention. Current investigations are underway with candidate vaccines and ricin inhibitors as antidotes or to facilitate immunotoxin treatment. Pteric acid, neopterin, pterin tautomer, and guanine tautomer are particularly useful .

Consultations

Surgical consultation for local excision and removal is warranted for parenteral exposures when a retained foreign body is located.

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Medication

Update tetanus status if unknown. If exposure is via parenteral route, antibiotics may be helpful in preventing secondary bacterial infection.

Antibiotics

With regard to ricin toxicity, the only possible indication for antibiotics is for the parenteral mechanism of exposure. Direct the choice of antibiotic to cover skin flora.

| | |
|-------------------|--|
| Drug Name | Cefazolin (Ancef)- First-generation semisynthetic cephalosporin that arrests bacterial cell wall synthesis, inhibiting bacterial growth. |
| Adult Dose | 1-2 g IV/IM q6-8h |
| Pediatric Dose | 25-50 mg/kg/d divided q6-8h |
| Contraindications | Documented hypersensitivity; relative contraindication for patients who have a true anaphylactic reaction to penicillin-type agents; cross-reaction is reportedly 3-8% |
| Interactions | Probenecid prolongs effect of cefazolin; coadministration with aminoglycosides may increase renal toxicity; may yield false-positive urine-dip test for glucose |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Adjust dose in renal impairment; superinfections and promotion of nonsusceptible organisms may occur with prolonged use or repeated therapy |

Vasopressor Agents

Perform adequate volume resuscitation of patients with isotonic fluids and packed red blood cells prior to using or in conjunction with these agents; do not use in place of volume resuscitation. Choice of agent usually is determined by physician preference.

| | |
|-------------------|--|
| Drug Name | Dopamine (Intropin)- Probably most well-known and used pressor agent. Standard mixture of 200 mg in 250 cm ³ produces a concentration of 800 mcg/cm ³ ; administer IV. |
| Adult Dose | Low dose: 0.5-5 mcg/kg/min IV Medium dose: 5-10 mcg/kg/min IV High dose: >10 mcg/kg/min IV |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; relative contraindications are tachycardia and myocardial ischemia; increases myocardial demand and oxygen consumption; reportedly causes sudden cardiac death in conjunction with dilantin (diphenylhydantoin); incidence is rare and never was studied adequately; should not deter use |
| Interactions | Phenytoin, alpha- and beta-adrenergic blockers, general anesthesia, and MAOIs increase and prolong effects of dopamine |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Closely monitor urine flow, cardiac output, pulmonary wedge pressure, and blood pressure during infusion; prior to infusion, correct hypovolemia with either whole blood or plasma, as indicated; monitoring central venous pressure or left ventricular filling pressure may be helpful in detecting and treating hypovolemia |

| | |
|-------------------|--|
| Drug Name | Norepinephrine (Levophed)- Often a second-line agent but can be used as a first-line agent; can be used with dopamine. Standard mixture of 4 mg in 250 cm ³ produces a concentration of 16 mcg/cm ³ ; administer IV. |
| Adult Dose | 2-4 mcg/min IV; can be increased by 2-4 mcg/min q5-10min prn |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; relative contraindications are tachycardia and myocardial ischemia; increases myocardial demand and oxygen consumption |
| Interactions | Effects increase when administered concurrently with TCAs, MAOIs, antihistamines, guanethidine, methyldopa, and ergot alkaloids; atropine may block reflex tachycardia caused by norepinephrine and enhances pressor response |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|--|
| Precautions | Correct blood-volume depletion, if possible, before giving norepinephrine therapy; extravasation may cause severe tissue necrosis and thus should be administered into a large vein; caution in occlusive vascular disease |
|-------------|--|

Immunization

Update tetanus status if unknown.

| | |
|-------------------|---|
| Drug Name | Tetanus and diphtheria toxoids- Used to induce active immunity against tetanus in selected patients. |
| Adult Dose | 0.5 mL IM |
| Pediatric Dose | <7 years: Not recommended >7 years: Administer as in adults |
| Contraindications | Documented hypersensitivity; a history of any type of neurologic symptoms or signs following administration of this product; FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are an important cause of provocative poliomyelitis |
| Interactions | Patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization due to poor immune response; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent use of medication with systemic chloramphenicol since may impair amnestic response to tetanus toxoid; concurrent use of tetanus immune globulin may delay development of active immunity by several days (interaction is nevertheless clinically insignificant and does not preclude concurrent use) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Do not use to treat actual tetanus infections or for immediate prophylaxis of unimmunized individuals (use instead tetanus antitoxin, preferably human tetanus immune globulin); diminished antibody response to active immunization may be seen in patients receiving immunosuppressive therapy; better to defer primary diphtheria immunization until immunosuppressive therapy discontinued; routine immunization of symptomatic and asymptomatic HIV-infected persons is recommended |

H2 Blockers

Reversible competitive blockers of histamine at H₂ receptors, particularly those in the gastric parietal cells where they inhibit acid secretion. The H₂ antagonists are highly selective, do not affect H₁ receptors, and are not anticholinergic agents.

| | |
|-------------------|---|
| Drug Name | Famotidine (Pepcid)- Competitively inhibits histamine at H2 receptor of gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and reduced hydrogen concentrations. |
| Adult Dose | Normal renal function: 20 mg IV q12h Renal failure: 20 mg IV qd |
| Pediatric Dose | Normal renal function: 0.5 mg/kg IV q12h Renal failure: No standard recommendations; consider decreasing dose to 0.5 mg/kg IV qd |
| Contraindications | Documented hypersensitivity |
| Interactions | May decrease effects of ketoconazole and itraconazole |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | If changes in renal function occur during therapy, consider adjusting dose or discontinuing treatment |

Antidotes

Used to inhibit or reduce absorption of the toxin.

| | |
|-------------------|---|
| Drug Name | Activated charcoal (Liqui-Char, Insta-Aqua, Actidose Aqua)- Emergency treatment in poisoning caused by drugs and chemicals. Network of pores present in activated charcoal adsorbs 100-1000 mg of drug per gram of charcoal. Does not dissolve in water. For maximum effect, administer within 30 min after ingesting poison. |
| Adult Dose | 25-100 g, 1 g/kg, or 10 times weight of ingested poison; give as susp in 4-8 oz of water |
| Pediatric Dose | <1 year: Not recommended >1 year: Administer as in adults |
| Contraindications | Documented hypersensitivity; poisoning or overdosage of mineral acids and alkalies |
| Interactions | May inactivate ipecac syrup if used concomitantly; effectiveness of other medications decreases with coadministration; do not mix charcoal with sherbet, milk, or ice cream (decreases adsorptive properties of activated charcoal) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Not very effective in poisonings of ethanol, methanol, and iron salts; induce emesis before giving activated charcoal; after emesis with ipecac syrup, patient may not tolerate activated charcoal for 1-2 h; can administer in early stages of gastric lavage; without sorbitol gastric lavage, returns are black |

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Follow-up

Further Inpatient Care

- Perform adequate volume resuscitation of patients. First treat hypotension with isotonic fluids and packed red blood cells as needed. Use a pressor-type agent such as dopamine or norepinephrine when needed.

Deterrence/Prevention

- The only effective treatment or prevention against a biological attack with ricin is prophylaxis; unfortunately, no prophylaxis exists. Currently, investigations are ongoing with candidate vaccines and ricin inhibitors. In an aerosol attack, protective masks are effective in preventing toxicity and should be used.

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Miscellaneous

Medical/Legal Pitfalls

- Investigate any ricin exposure that involves multiple victims. Weaponized use of ricin is a reportable offense and mandates reporting to local and federal authorities.

Special Concerns

- Conclusion: Although ricin is not the ideal biological warfare agent, it remains a threat. It is widely available and easily produced. It is not the ideal agent of choice for an aerosol attack, but it is a major concern as a food and water contaminant. With the increasing number of biological threats, hoaxes, and "how to" Internet resources available, this threat has the potential to become reality. Therefore, the emergency physician must be familiar with its characteristics. Treatment is supportive, and no antidote or vaccine is available.

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CBRNE - Smallpox

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Synonyms, Key Words, and Related Terms

smallpox, variola, variola major, variola minor

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Introduction

Background

Smallpox (variola) represents both the zenith and nadir of human achievement. It is the only disease eradicated through a concerted and extensive effort that transcended political and ideologic boundaries. Because of these efforts, not one documented naturally occurring case of this once high-mortality infection has occurred since October 26, 1977. (The last case was an unvaccinated hospital cook in Somalia.) Smallpox officially was declared eradicated by the World Health Organization (WHO) in

1980. It also represents one of the most devastating potential biological weapons ever conceived.

For centuries, smallpox affected political and social agendas. Epidemics plagued Europe and Asia until Edward Jenner developed a vaccine in 1796; he subcutaneously inoculated patients with the milder cowpox virus. The viral illness incidence of infection in Europe steadily declined from that point onward.

In the Americas, smallpox decimated the native population, who never had been exposed to variola, when it followed closely behind the European explorers in the 1600s. The British forces at Fort Pitt (Pittsburgh, PA) purposefully gave smallpox-contaminated blankets and goods to Native Americans during the French and Indian Wars in an attempt to weaken resistance to colonial expansion. Whether due to this or through natural spread, the subsequent epidemic carried a mortality rate of 50% among native tribes.

Farr first accurately predicted variola infection rates in the 1830s. Once the disease and its method of spread were understood better, smallpox vaccination became mandatory in developed countries in the early 1900s. The development of the vaccinia virus and its subsequent vaccine enabled aggressive immunization by the WHO, which led to variola eradication in 1977.

The variola virus no longer exists outside of a few laboratories around the world. The official virus repositories are at the Centers for Disease Control and Prevention (CDC) in Atlanta, GA, and the Institute of Viral Preparations in Moscow, Russia. Viral stocks also exist at the Russian State Research Center of Virology and Biotechnology in Koltsovo. Multiple dates for destruction of the remaining viral stocks have been proposed by the WHO Committee on Orthopoxvirus Infections, only to be pushed back under pressure from various factions, including the US government.

Various sources from the Soviet Union allege that the military had and currently still pursues an active biological warfare program. For instance, the Russian government confirmed a suspected outbreak from an accidental release of aerosolized anthrax near a military microbiology laboratory in 1992. In 1980, the Soviet Union commenced large-scale production of the smallpox virus and genetic recombination of more virulent strains. Since the fall of the Soviet Union, concern exists that this expertise may be employed in other countries. The extent of smallpox stockpiles in other countries is unknown but may be significant since the collapse of the Soviet Union.

Variola, prior to eradication, carried a mortality rate of 30% in unvaccinated persons. Researchers estimate that vaccinated individuals retain immunity for approximately 10 years, although the duration never has been evaluated fully. Vaccination of the general population in the US ceased after 1980, and vaccination in military personnel was discontinued in 1989. Therefore, the current populace in the US is considered immuno-naïve to the variola virus. Forty-two percent of the US population is younger than 30 years and never was vaccinated. The ease of production and aerosolization of the virus is well documented. Researchers estimate that only 10-100 virus particles are needed for infection; thus, smallpox is a potential biological weapon of staggering lethality.

Pathophysiology

Variola is a member of the Orthopoxvirus genus, of which cowpox, monkeypox, orf, and molluscum contagiosum also are members. Poxviruses are the largest animal viruses visible with a light microscope and are larger than some bacteria. They have the largest genome, comprised of 200 kilobase double-stranded DNA enclosed in a double membrane layer. Poxviruses are the only viruses that can replicate in cell cytoplasm without the need of a nucleus.

The virus is acquired from inhalation, although virus particles can remain viable on fomites (clothing, bedding, surfaces) for approximately 1 week. The virus initially replicates in respiratory tract epithelial cells. From there, a massive asymptomatic viremia ensues, resulting in focal infection of the skin, intestines, lungs, kidneys, and brain. The multiplication in the skin epithelial cells first leads to a rash, progressing into pustules approximately 14 days after inoculation. A cell-mediated immune response is responsible for pustule formation, as immunocompromised rabbits do not produce these characteristic lesions. Patients who survive an initial infection often have severely deformed skin from the pustules and subsequent granulation tissue formation.

Frequency

- **Internationally:** Since the last wild documented case in 1977, only 2 deaths from smallpox have been reported (1978 in Birmingham, England), one from a laboratory worker who infected her mother and the second from a photographer with an office next to the lab space where the accidental exposure to the virus occurred.

Mortality/Morbidity

Variola major, or smallpox, has a mortality of 30%. Variola minor, or alastrim, is a milder form of the virus, carrying a mortality rate of 1%. Four types of variola presentations exist: classic, hemorrhagic, malignant, and modified. Classic smallpox was believed to be the most communicable disease. Approximately 30% of susceptible contacts became infected.

- Pregnant women have a heightened morbidity to variola. In one study prior to virus eradication, morbidity was 27% in vaccinated patients and 61% in unvaccinated patients versus a nonpregnant control morbidity of 6% (vaccinated) and 35% (unvaccinated).
- The hemorrhagic variety of variola also carried a higher mortality and led to death more quickly. Patients often died before the pustular lesions formed, but this variety is recognizable by the hemorrhagic lesions that erupt in the mucosal and cutaneous membranes. Comprehensive studies documenting almost 7000 cases of variola found 200 patients had this form of the disease; 192 died. Pregnant women are more likely to contract this version. Biological warfare design probably would employ this variety for an optimal number of deaths per unit of agent dispersed.
- Prior to eradication, the malignant or flat form of variola affected 6% of the population and

evolved more slowly than the classic presentation. Lesions were not pustular; instead they consisted of a flattened macule, often described as feeling velvety. The mortality rate for this form approaches 100%.

- The modified variety of smallpox essentially consists of those previously vaccinated with some intact immune response. In a vaccinated population, this version would constitute approximately 15%.

Race

No racial predilection exists.

Sex

With the exception of pregnant patients, males and females are infected in equal proportions.

Age

No age predilection exists. In unvaccinated people, the distribution of illness mirrors that of the age distribution of the population. However, in India prior to eradication, 70% of infections were in children younger than 14 years.

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Clinical

History

- Incubation periods for the major types of variola infection range from 7-17 days.
 - An asymptomatic viremia occurs 72-96 hours after infection.
 - At the end of the incubation period, a second viremia results in the onset of clinical symptoms such as fever, myalgias, headache, rigors, and, particularly, backache.
 - Rigors and vomiting are present in more than one half of patients.
 - Delirium occurs in 15% of the infected population.
 - This prodrome lasts 2-4 days, and during this time, viremia is present and patients are most infectious.
- Virus titers in saliva are highest the first week of infection, but infectivity can last up to 3 weeks (until the scabs fall off). Live virus can be cultured from scabs.
- Early in the course of the disease, the rash and macules easily can be mistaken for varicella, given the coincidence of fever and myalgias. The macules give way to papules, and finally, the

characteristic pustules form, although this can take up to 2 weeks from exposure. The distribution and character of these lesions are the sine qua non of variola. The contents of these lesions contain a high viral load and are infectious.

Physical

- In 10% of patients, a fleeting erythematous exanthem can be seen in fair-skinned patients before the typical cutaneous manifestations occur.
- Lesions occur first in the oral mucosa, spreading to the face, then to the forearms and hands, and finally to the lower limbs and trunk. This is in distinction to the rash from varicella, which progresses from the limbs centrally.
 - Lesions favor ventral surfaces and progress through stages of macule, papule, vesicle, papules (often umbilicated, like molluscum contagiosum), and crusts. Unlike in varicella, where lesions in different stages are present, the exanthem of variola is synchronous, with numerous monomorphic lesions.
 - The rash settles centrifugally, sparing the axillae, palms, soles, and antecubital areas. Crusts detach after 2-4 weeks, leaving depressed, hypopigmented scars.
 - Lesions are concentrated on the hands, face, feet, and calves.

Causes

- The variola virus is the only known cause of smallpox. The disease affects only humans, and no animal or arthropod vectors or carriers exist.
- Only two laboratories in the world are known to house smallpox virus: the CDC in Atlanta, GA, and the Russian State Research Center of Virology and Biotechnology in Koltsovo.

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Differentials

Erythema Multiforme

Other Problems to be Considered

Acute leukemia

Monkeypox (endemic in some areas of Africa)

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Workup

Lab Studies

- Perform a viral swab of the pharynx on patients in whom smallpox is suggested or swab a freshly opened pustule, if available. Otherwise, use a scalpel to open a lesion and obtain a culture.
 - Isolation of the variola virus should be undertaken only in a laboratory with Biosafety Level 4 (BSL-4) capabilities. The only laboratories in the US with these capabilities at this time are at the CDC in Atlanta, GA, and the US Army Medical Research Institute of Infectious Diseases (USAMRIID) in Ft Detrick, MD.
 - Send them in a Vacutainer tube with the rubber stopper taped. Double seal it and inform the receiving lab and courier of the potential biohazard. Prior to collection of samples or shipment, CDC or USAMRIID should be consulted directly, as should local public health authorities. In addition to individual state laws concerning highly infectious agents, specific federal laws apply to the shipping of such pathogens across state lines.
 - Once in a lab, viral cultures, polymerase chain reaction (PCR), and/or enzyme-linked immunoabsorbent assay may be undertaken.
 - The prior criterion standard was the chorioallantoic egg membrane culture.

Imaging Studies

- No imaging studies assist in making the diagnosis of variola infection.

Other Tests

- PCR may be used to make definitive laboratory diagnosis.

Procedures

- Include a lumbar puncture in the workup of a hemorrhagic variola to exclude meningococemia.

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Treatment

Prehospital Care

No prehospital care is indicated except to stabilize the patient. Strict blood, body fluid, and droplet protection are required for all personnel involved with treating or transporting patients with known or suspected smallpox. All emergency medical services (EMS) personnel exposed to the patient require quarantine and vaccination.

Emergency Department Care

In the emergency department, containment of the diseases is the single most important intervention in patients in whom variola infection is suggested.

- Immediate contact and droplet isolation of the patient is required.
- The patient and contacts up to 17 days prior to illness (including the treating physician and nursing staff) should remain in isolation until a definite diagnosis is made. Presently, this requires sending a viral culture to the CDC in Atlanta, GA.
- Additionally, notify the local health authorities immediately.
- The most likely scenario of a variola outbreak is from a terrorist attack. Given the highly infective nature of the organism (not taking into account a genetically altered virus), researchers estimate that 1 infected patient subsequently can infect 20 new contacts during the infectious stage of the illness.
- The presentation of a clinically apparent case implies that a larger population probably has been infected.
- Because of the medicolegal and social implications of quarantine and isolation for a minimum of 17 days, coordinated involvement on the federal, state, and local levels is mandatory. In practicality, strict quarantine of a large segment of the population is probably not possible.

Consultations

Consult the infectious disease service early since it may help determine the diagnosis. As mentioned previously, contact the state, federal, and local health authorities.

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Medication

Once the disease is manifested fully, medical treatment for variola is supportive care. Vaccinations and

postexposure interventions are the mainstays of treatment.

Vaccine

The vaccinia (smallpox) vaccine and vaccinia immune globulin (VIG) is available only through the CDC and state health agencies. The calf lymph vaccine is the only one still available, although a replacement vaccinia vaccine produced from cell cultures is under development. In the US alone, enough vaccine (NY Board of Health virus grown on scarified calves) is available to vaccinate 6-7 million individuals. The executive branch of the federal government, via the CDC, which holds the vaccine in cold storage, ultimately decides who gets vaccinated. The state health departments also have access to limited local stock. Additionally, the WHO has 500,000 doses in storage. The current worldwide supply is approximately 50 million doses.

Currently, no large-scale producers exist of either vaccine or immune globulin, and an estimated 36 months is required to actually begin production. Negotiations are currently underway with Great Britain to produce an additional 40 million doses, and some studies on the existing American stockpiles indicate that the vaccine would be effective in dilutions of 1:10. Still, a portion of the existing stockpiles of the New York strain is now known to be ineffective.

Those exposed to variola (meaning all household or other face-to-face contacts after onset of fever) within a few days were found to experience attenuated illness if vaccinated within 4 days. Researchers estimate that of the previously vaccinated population, only approximately 20% still have effective immunity. Those who were not revaccinated within 3 years (eg, lab workers) should be vaccinated again.

| | |
|-------------------|---|
| Drug Name | Calf lymph vaccine- Stimulates a preemptive immune response to the virus. |
| Adult Dose | SC inoculation with a 2-pronged needle (preset CDC dosage) |
| Pediatric Dose | Administer as in adults |
| Contraindications | Given the high mortality of smallpox infection, consider the following contraindications on a case-by-case basis: immunosuppressed (chemotherapy or HIV) patients, patients with eczema, pregnant patients, or patients who are in close contact with any of the above should not receive the vaccine |
| Interactions | None reported |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Postvaccination encephalitis occurred relatively rarely prior to discontinuation of vaccination; in the US and Europe, the occurrence rate ranged between 2.3-2.9 cases per million; encephalitis carried a 25% mortality rate; revaccination carries a risk of death of 1 case per 10 million; other postvaccination complications (eg, progressive vaccinia, eczema vaccinatum, generalized vaccinia, inadvertent inoculation of mucous membranes) also were rare |

Blood Products

Immune globulins bind to the virus particle, stimulate an immune response, and offer transient protection while the host immune system develops antibodies.

| | |
|-------------------|--|
| Drug Name | Immune globulin (IVIG; Gammagard, Sandoglobulin, Gamimune)- Can be administered within 3 d of exposure but is best if given within 24 h; may be necessary to administer VIG in adverse reactions to vaccination; as production of VIG ceased in 1970s, its efficacy (because of its age) is under question; in possession of the CDC. |
| Adult Dose | 0.6 mL/kg IM for exposed individuals |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; IgA deficiency; anti-IgE/IgG antibodies |
| Interactions | Increases toxicity of live virus vaccine (MMR); do not administer within 3 mo of vaccine |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Check serum IgA before IVIG (use an IgA-depleted product, eg, Gammagard S/D); infusions may increase serum viscosity and thromboembolic events; infusions may increase risk of migraine attacks, aseptic meningitis (10%), urticaria, pruritus, or petechiae (2-5 d postinfusion to 30 d) Increases risk of renal tubular necrosis in elderly patients and in patients with diabetes, volume depletion, and preexisting kidney disease; lab result changes associated with infusions include elevated antiviral or antibacterial antibody titers for 1 mo, 6-fold increase in ESR for 2-3 wk, and apparent hyponatremia |

Antivirals

In vitro studies demonstrated cidofovir to inhibit poxvirus replication and cell lysis.

| | |
|-------------------|--|
| Drug Name | Cidofovir (Vistide)- A nucleoside analog DNA polymerase inhibitor; if administered within 48 h of exposure, may attenuate or avoid infection; adefovir, cidofovir, and ribavirin are under investigation for smallpox. Ribavirin as an aerosol treatment for pediatric respiratory syncytial virus is under investigation. |
| Adult Dose | 5 mg/kg IV over 1 h |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity; coadministration with other nephrotoxic agents; serum creatinine >1.5 mg/dL; CrCl <55 mL/min; urine protein >100 mg/dL |

| | |
|--------------|---|
| Interactions | Coadministration of aminoglycosides, amphotericin B, IV pentamidine, and foscarnet may increase nephrotoxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Complications include renal toxicity, neutropenia, fever, anemia, headache, hair loss, uveitis and/or iritis, and abdominal pain; monitor neutrophil counts; IV prehydration with NS and coadministration of probenecid can minimize nephrotoxicity; monitor serum creatinine and urine protein 48 h prior to treatment (adjust dose accordingly) |

| | |
|-------------------|--|
| Drug Name | Ribavirin (Virazole)- Used for respiratory syncytial virus infection in children and in combination with interferon for treatment of hepatitis C. |
| Adult Dose | <75 kg: 400 mg PO am followed by 600 mg pm >75 kg: 600 PO bid Dose interferon alpha 2b at 3 million U SC 3 times/wk when used in conjunction with above |
| Pediatric Dose | Administer as in adults; aerosol route is also available, although not evaluated for variola infections |
| Contraindications | Relative contraindications: Anemia, cardiovascular disease, cardiac disease |
| Interactions | Zidovudine effects are decreased when administered concurrently with ribavirin |
| Pregnancy | X - Contraindicated in pregnancy |
| Precautions | Rare adverse effects include cardiac arrest, hypotension, headache, and respiratory depression; zidovudine effects are decreased when administered concurrently with ribavirin |

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Follow-up

Further Inpatient Care

- Supportive care is the primary intervention for a clinically evident infection. This includes hydration therapy for fluid loss through fever and skin barrier breakdown. Antibiotics may be needed for secondary skin infections. Maintain respiratory and contact isolation 17 days or until the scabs fall off.

Further Outpatient Care

- Once an outpatient, plastic surgery consultation may be necessary for skin disfiguration.

in/Out Patient Meds

- No medications are required other than those previously mentioned (see [Medication](#)).

Transfer

- Make any transfer with full respiratory and contact isolation.

Deterrence/Prevention

- In a variola outbreak, the high rate of spread can be reduced by identification of the disease (a high index of suspicion is needed) and rapid containment.
- The most likely scenario of a variola outbreak is from a terrorist attack.
- Given the highly infective nature of the organism (not taking into account a genetically altered virus), researchers estimate that 1 infected patient subsequently can infect 20 new contacts during the infectious stage of the illness.

Complications

- High morbidity and mortality complications that can be reduced are secondary skin infections and dehydration.

Prognosis

- Smallpox is one of the most communicable of infectious diseases. Studies have shown that approximately 30% of susceptible contacts became infected.
- In general, variola has a mortality of 30% in the unvaccinated population.
- Pregnant women have a heightened morbidity to variola. Morbidity is 27% in vaccinated patients and 61% in unvaccinated patients versus a nonpregnant control morbidity of 6% (vaccinated) and 35% (unvaccinated).

Patient Education

- Heightened awareness of the manifestations of this disease may help reduce the population exposed in an outbreak through early diagnosis and preventive medicine and/or public health initiatives.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to notify the local, state, or federal health authorities or discharging a patient with this disease back into the general population is the gravest potential error.
 - Involvement of the state and local authorities gives the physician the needed support to quarantine individuals and their contacts in the event of an outbreak.
 - This quarantine also involves the physician, nursing staff, and EMS personnel.
 - The presentation of a clinically apparent case implies that a larger population has been infected. Because of the medicolegal and social implications of isolation and quarantine, coordinated involvement on the federal, state, and local levels is mandatory.

Special Concerns

- Because of a relatively immature immune system, the pediatric population is particularly susceptible to death from variola, although children in a native population have the highest mortality rate.
 - When treating smallpox, give special care to patients with HIV, pregnant women, and patients with eczema, leukemia or malignancy requiring chemotherapy, and hereditary immune disorders, all of whom should receive VIG instead of the vaccination.
-

Pictures



Picture 1: Characteristic skin lesion of variola on the arms and legs of an adolescent. Photo used with permission from the World Health Organization (WHO).

Picture type: Photo



Picture 2: Small child with pustular lesions of variola. Photo used with permission of the World Health Organization (WHO).

Picture type: Photo



Picture 3: Infant with advanced lesions of variola. Photo used with permission of the World Health Organization (WHO).

Picture type: Photo



Picture 4: Unvaccinated infant with centrifugally distributed umbilicated pustules on day 3 of ordinary form of variola major strains of smallpox (Reprinted with permission from Fenner F, Henderson DA, Arita I, et al: Smallpox and its eradication. Geneva, Switzerland: World Health Organization; 1988: 10-14, 35-36; photographs by Arita).

Picture type: Photo



Picture 5: Unvaccinated infant with centrifugally distributed umbilicated pustules on day 5 of ordinary form of variola major strains of smallpox (Reprinted with permission from Fenner F, Henderson DA, Arita I, et al: Smallpox and its eradication. Geneva, Switzerland: World Health Organization; 1988: 10-14, 35-36; photographs by Arita).

Picture type: Photo



Picture 6: Unvaccinated infant with centrifugally distributed umbilicated pustules on day 7 of ordinary form of variola major strains of smallpox (Reprinted with permission from Fenner F, Henderson DA, Arita I, et al: Smallpox and its eradication. Geneva, Switzerland: World Health Organization; 1988: 10-14, 35-36; photographs by Arita).

Picture type: Photo



Picture 7: Ordinary form of variola minor strain of smallpox (alastrim) in an unvaccinated woman 12 days after onset of skin lesions. The facial lesions are sparser and evolved more rapidly than the extremity lesions (Reprinted with permission from Fenner F, Henderson DA, Arita I, et al: Smallpox and its eradication. Geneva, Switzerland: World Health Organization; 1988: 10-14, 35-36; photographs by Arita).

Picture type: Photo



Picture 8: Ordinary form of variola minor strain of smallpox (alastrim) in an unvaccinated woman 12 days after onset of skin lesions. The facial lesions are sparser and evolved more rapidly than the extremity lesions (Reprinted with permission from Fenner F, Henderson DA, Arita I, et al: Smallpox and its eradication. Geneva, Switzerland: World Health Organization; 1988: 10-14, 35-36; photographs by Arita).

Picture type: Photo



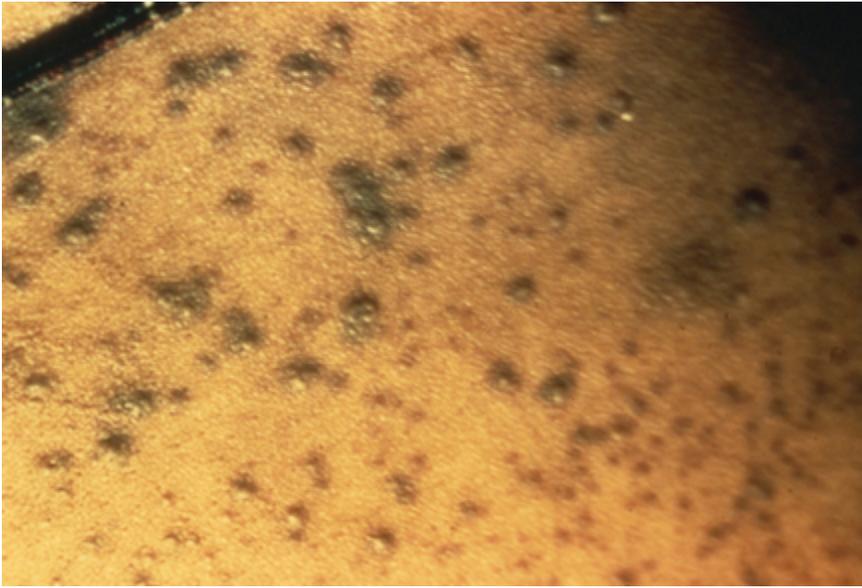
Picture 9: Ordinary form of variola minor strain of smallpox (alastrim) in an unvaccinated woman 12 days after onset of skin lesions. The facial lesions are sparser and evolved more rapidly than the extremity lesions (Reprinted with permission from Fenner F, Henderson DA, Arita I, et al: Smallpox and its eradication. Geneva, Switzerland: World Health Organization; 1988: 10-14, 35-36; photographs by Arita).

Picture type: Photo



Picture 10: Adult with variola major with hundreds of pustular lesions distributed centrifugally (Fitzsimmons Army Medical Center slide file).

Picture type: Photo



Picture 11: Hemorrhagic-type variola major lesions. Death usually ensued before typical pustules developed (Reprinted with permission from Herrlich A, Mayr A, Munz E, et al: Die pocken; Erreger, Epidemiologic und klinisches Bild. 2nd ed. Stuttgart, Germany: Thieme; 1967. In: Fenner F, Henderson DA, Arita I, et al: Smallpox and its eradication. Geneva, Switzerland: World Health Organization; 1988: 10-14, 35-36).

Picture type: Photo



Picture 12: Boy with monkeypox in Democratic Republic of the Congo in 1996. Note the centrifugal distribution as was typical of smallpox (Courtesy of William Clemm).

Picture type: Photo



Picture 13: Boy with monkeypox in Democratic Republic of the Congo in 1996. Note synchronicity of lesions as was typical of smallpox (Courtesy of William Clemm).

Picture type: Photo

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Synonyms, Key Words, and Related Terms

SEB

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Introduction

Background

Toxins are poisons produced by living organisms. Staphylococcal enterotoxin B (SEB) is classified as an exotoxin, since it is excreted by an organism, in this case the *Staphylococcus aureus* bacterium. *Staphylococcus* species thrive and produce toxins in unrefrigerated meats, dairy, and bakery products. SEB normally exerts its effect on the intestines and therefore is termed an enterotoxin. Not all toxins cause a lethal outcome, but they may result in significant morbidity.

SEB is the toxin that most commonly causes classic food poisoning. It also has been demonstrated to cause a nonmenstrual toxic shock syndrome (TSS). SEB has been studied as a potential biological agent of war, since it easily can be aerosolized, is very stable, and can cause widespread systemic damage,

multiorgan system failure, and even shock and death when inhaled at very high dosages. However, SEB is classified as an incapacitating agent because in most cases aerosol exposure results not in death, but in a temporary though profoundly incapacitating illness lasting as long as 2 weeks. Clearly this would be devastating on the battlefield during times of war.

Source

SEB is 1 of 7 enterotoxins produced by certain strains of the coagulase-positive *S aureus* bacteria. Staphylococci are gram-positive cocci that form clumps. *S aureus* colonize the nasal passages and axillae.

Structure

SEB consists of 239 amino acid residues and has a molecular weight of 28 kd. It is 1 of the 6 least antigenically distinct enterotoxin proteins that have been identified (A, B, C, D, E, G).

Properties

SEB is a relatively stable compound that is easily soluble in water. It is very resistant to temperature fluctuations and can withstand boiling for several minutes. In the freeze-dried state, SEB can be stored for more than a year. For aerosol exposures the effective dose, or ED50 (dose capable of incapacitating 50% of the exposed human population), is 0.0004 mcg/kg, and the lethal dose, or LD50, is 0.02 mcg/kg.

Mechanism of toxicity

Many of the effects of SEB (aerosol exposure) are mediated by the stimulation of T lymphocytes by the host's immune system. The toxin is noted to bind directly to the major histocompatibility complex class II proteins on target cells, subsequently stimulating the proliferation of large numbers of T lymphocytes. SEB commonly is referred to as a "bacterial superantigen" because it is an extremely potent activator of T cells, stimulating the production and secretion of various cytokines. The released cytokines most likely mediate many of the toxic effects of SEB. In contrast, ingestion of SEB produces profound gastrointestinal (GI) symptoms including anorexia, nausea, vomiting, and diarrhea, which are believed to be mediated through the release of histamine and leukotrienes from mast cells.

Pathophysiology

After SEB is produced and excreted in improperly refrigerated, stored, and handled foodstuffs, ingestion of SEB causes food poisoning. The incubation period is 1-8 hours (rarely up to 18 hours). Classic symptoms are an abrupt onset of intense nausea, vomiting, cramping abdominal pain, and diarrhea, which incapacitate the patient. Most cases are self-limited and resolve in 8-24 hours.

The onset of symptoms after inhaling SEB may vary from 1-6 hours. Sudden onset of headache, fever, myalgia, nonproductive cough, chills, shortness of breath, and retrosternal pain can be caused by SEB at low doses via inhalation. Fever may last 2-5 days, and cough may persist for up to 4 weeks.

Higher exposure to SEB may lead to septic shock and death.

Frequency

- **In the US:** The actual incidence is unknown; many cases are so mild that patients do not seek treatment. Additionally, diagnoses in the emergency department are usually empiric, and a number of other diseases may mimic SEB-induced gastroenteritis.

Mortality/Morbidity

The disease, while potentially debilitating for short durations, is rarely fatal with adequate hydration.

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Clinical

History

In many cases of enterotoxin-induced gastroenteritis, the history makes the diagnosis.

- Usually symptoms start within several hours of ingestion of potentially contaminated foods, beginning with significant nausea, vomiting, and intestinal cramping, followed by urgency and profuse watery nonbloody diarrhea. Symptoms normally resolve within 12-24 hours. Multiple family members or patrons of the same eating establishment may be affected.
- In inhalation of aerosolized SEB, the clinician is presented with numerous patients of all ages within a short period of time, most likely within 1-6 hours of an exposure at a common location. Because of the difficulties in obtaining large quantities of the toxin, this location most likely would be an enclosed space, such as a gymnasium, arena, or office building.

Physical

Physical examination in SEB intoxication may be unremarkable, but most likely the patient presents with complaints of acute onset and either appears in significant abdominal pain or acutely short of breath. Symptoms of SEB intoxication are abrupt and self-limiting.

- Gastrointestinal exposure
 - If the route of entry of the toxin is GI, patients may appear dehydrated and, depending on the severity of nausea, may complain of acute abdominal cramping and diarrhea.
 - Physical examination may reveal hypotension, tachycardia, hyperperistalsis, and diffuse nonlocalizing abdominal pain. Any stool or diarrhea is hemoglobin negative, barring other pathology.

Causes

After toxin is produced in improperly refrigerated, stored, and handled foodstuffs, ingestion of SEB causes food poisoning.

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Differentials

Bowel Obstruction, Large
Bowel Obstruction, Small
CBRNE - Cyanides, Cyanogen Chloride
CBRNE - Cyanides, Hydrogen
CBRNE - Lung-Damaging Agents, Chlorine
CBRNE - Lung-Damaging Agents, Chloropicrin
CBRNE - Lung-Damaging Agents, Diphosgene
CBRNE - Lung-Damaging Agents, Phosgene
CBRNE - Nerve Agents, Binary: GB2, VX2
CBRNE - Nerve Agents, G-series: Tabun, Sarin, Soman
CBRNE - Nerve Agents, V-series: Ve, Vg, Vm, Vx
CBRNE - Ricin
CBRNE - Vesicants, Mustard: Hd, Hn1-3, H
CBRNE - Vomiting Agents: Dm, Da, Dc
Cholecystitis and Biliary Colic
Gastritis and Peptic Ulcer Disease
Gastroenteritis
Giardiasis
Pancreatitis
Pericarditis and Cardiac Tamponade
Pneumothorax, Iatrogenic, Spontaneous and Pneumomediastinum
Respiratory Distress Syndrome, Adult
Scorpion Envenomations
Shock, Hypovolemic

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Workup

Lab Studies

- Although SEB can be confirmed through enzyme-linked immunosorbent assays of tissue or body fluids, diagnosis is largely clinical and by epidemiologic assays of tissue or body fluids.
- Neutrophilic leukocytosis and an elevated erythrocyte sedimentation rate may be observed in SEB intoxication.
- Toxins may be identified in nasal swabs from persons exposed to respiratory aerosol for at least 12-24 hours, offering an avenue of early diagnosis in the battlefield.

Imaging Studies

- Radiographs of the chest appear normal unless the patient has had significant aerosolized exposure, in which case pulmonary edema or an ARDS presentation is evident.
- Routine abdominal films for SEB gastroenteritis are not necessary but may exhibit significant intestinal gas with no free air.

Other Tests

- Other tests are dictated by the patient's physiologic condition or progress of the disease syndrome.

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Treatment

Prehospital Care

Treatment is supportive.

- In the event of dehydration, vigorous administration of intravenous fluids is indicated.
- For patients exposed through the pulmonary system, supportive treatment with humidified oxygen and steroids for pain control is all that is usually necessary, although significant exposure may

dictate intubation and assisted ventilation with high oxygen concentrations.

- The efficacy of steroids in SEB-induced pulmonary edema or ARDS has not been demonstrated.

Emergency Department Care

Treatment is limited to supportive care, with special attention to elimination of hypotension and hypoxia and pain control as needed.

- Mechanical ventilation may be required in severe cases.
- Antibiotics have not demonstrated efficacy in SEB intoxication, nor have steroids been shown to be effective in SEB-induced pulmonary edema.

Consultations

Consultations are dictated by the patient's physiologic condition.

- If a terrorist attack using SEB is suspected, expeditiously inform local law enforcement personnel, including the local Federal Bureau of Investigation.
- In the event that a cluster of patients present with similar symptoms, either of pulmonary or GI origin, notify local public health officials to begin epidemiologic investigation.

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Follow-up

Further Inpatient Care

- Disposition is dictated by the patient's condition after appropriate observation in the ED. In general, patients who are asymptomatic at rest with no shortness of breath, tolerable chest discomfort, and no progression of symptoms may be discharged home with appropriate follow-up instructions, including a caution to avoid any exertion for at least 24 hours.
- Patients with continued vomiting who are unable to maintain their own hydration or those with significant dehydration require admission to at least an observation unit.

Deterrence/Prevention

- Food-borne SEB can be prevented by proper storage of dairy products and proper storage and preparation of meat products.

Prognosis

- Prognosis with appropriate treatment is excellent in both food-borne and inhalation SEB. Some respiratory symptoms, including nonproductive cough, may persist for up to 4 weeks.

Patient Education

- Dairy products must be stored properly, and meat products must be stored and prepared properly.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to recognize SEB intoxication may lead to a nonmenstrual toxic shock syndrome. In all probability, very young and elderly patients are more susceptible.
- Failure to report sources of SEB (eg, restaurant) may result in epidemic spread of infection.

Special Concerns

- Prophylaxis: No human vaccine against SEB is available. Use protective masks if the use of SEB in biological warfare is threatened.
 - SEB in biological warfare
 - Since SEB causes symptoms when inhaled at low doses, it can render up to 80% or more of exposed personnel clinically ill and unable to perform their duties for up to 1-2 weeks.
 - The US military once referred to SEB by the code name PG, and the toxin was part of the US stockpile prior to its destruction in 1972.
-

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CBRNE - T-2 Mycotoxins

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Synonyms, Key Words, and Related Terms

trichothecene mycotoxin, toxic alimentary aleukia, ATA, yellow rain

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Introduction

Background

Mycotoxins are naturally occurring substances produced by fungi as a secondary metabolite that typically affords the organism survival benefit (eg, penicillin). Many of these toxins are pathogenic to animals and humans. An estimated more than 300 mycotoxins are produced by some 350 species of fungi. The T-2 mycotoxin, which is classified as a trichothecene mycotoxin, is elaborated from the

fusarial species of fungus. According to the current declassified literature, the T-2 mycotoxin is the only mycotoxin known to have been used as a biological weapon. The trichothecene mycotoxins are low molecular weight compounds (250-500 d, averaging 466 d) that are nonvolatile, relatively insoluble in water, and highly soluble in ethanol, methanol, and propylene glycol. The toxin is highly heat stable and resistant to UV light destabilization (2 important factors when considering an agent as a biological warfare agent).

In laboratory rats, the LD50 (dose to cause 50% lethality) is 4 mg/kg when ingested. The LD50 for dermal exposure is reportedly 2-12 mg/kg. In mice, the LD50 for aerosol exposure is 1.2 mg/kg. The trichothecene class of toxins is considered among the most potent naturally occurring toxic substances.

Most information regarding the effects of T-2 mycotoxin on humans has been collected from many incidents of accidental ingestion of moldy wheat or corn. One such incident involved the Orenburg district of Russia during World War II. Most men in the village were fighting in the war, leaving the wheat crop unharvested, which resulted in the crop remaining in the fields over the winter. It was harvested in the spring and consumed, causing the clinical syndrome alimentary toxic aleukia (ATA), with varied reports of 10-60% mortality. Some hypothesize that T-2 mycotoxin may have been the operative agent in the "plague" of Athens in 430 BC. Additional information about the clinical effects of T-2 mycotoxin has been demonstrated in the laboratory using human cell cultures and animal models.

Reports exist of T-2 mycotoxin used as a biological warfare agent. The first suspected use was in the country of Laos during the Vietnam War. The report of "yellow rain" in remote sections of jungle in Laos (1975-81), which resulted in more than 6300 deaths, has been viewed as use of T-2 mycotoxin as a biological weapon. Evidence regarding the use of the toxin in Laos remains hotly debated. Other reported uses of T-2 mycotoxin as a biological weapon concern Kampuchea (1979-81) and Afghanistan (1979-81).

More recently, it has been suggested that T-2 mycotoxin was disseminated near a US military camp in Saudi Arabia during the Desert Storm campaign. An Iraqi missile may have detonated over or near the camp. Some of the troops in the area reported immediate symptoms that could be consistent with dermal mycotoxin exposure. Evidence surrounding this incident is questionable, and the government has not verified any evidence consistent with the use of this agent. Some sources state that exposure to T-2 mycotoxins may be the cause of Gulf War syndrome.

Qualities important to producing an effective chemical or biological weapon are its ease of manufacture, ease of weaponization, durability of the organism or toxin in storage form, ease of dispersal, and chemical stability when exposed to heat and UV radiation. Other factors include ease of concealment and ability to directly obtain the agent or organism that produces the agent. In the early half of the century, biotoxins were investigated as military weapons. These types of weapons fell into disfavor primarily because of problems with weaponization of the biotoxin material. The US closed its biotoxin program in the 1960s. Interest was rekindled in the 1970s with improvements in gene technology and biotechnology. Although the US has no current offensive biological weapons capability, these agents are less expensive

than nuclear and chemical weapons and therefore appeal to smaller countries or terrorist organizations.

Because of limitations in the manufacture of sufficient quantities, biotoxins are not optimal agents for mass dispersal. This agent is better suited as a small-group assassination tool, since a small amount can be dispersed effectively in enclosed areas. As an assassination tool, T-2 mycotoxins can be used as a food or water-borne poison. The T-2 mycotoxin is the only biologically active toxin effective through dermal exposure and respiratory and gastrointestinal (GI) portals. The route of entry and dose dictate the clinical course. Tissues involved in high cellular turnover (eg, GI and respiratory epithelium, bone marrow cellular elements) are most susceptible to the toxin.

Pathophysiology

The pathophysiology of T-2 mycotoxin is multifactorial. It causes DNA breaks, chromosomal abnormalities, and inhibition of protein synthesis. Inhibition of protein synthesis seems to be the primary cause of symptoms in intoxicated patients. Conflicting reports of the mechanism involving the inhibition of protein synthesis exist. One theory relates it to the toxin's affinity for the 60S ribosomal subunit, therefore inhibiting protein synthesis at the initial step. Another theory involves the inactivation of peptidyl transferase, which inhibits the terminal step of protein synthesis. The mechanism of action on DNA is not clear but is believed to be related indirectly to the cessation of protein synthesis.

Frequency

- **In the US:** The only epidemiologic information available is from the ingestion of contaminated foodstuff. No well-documented epidemiologic information is available for exposure to T-2 mycotoxin as a result of bioweapon deployment.

Mortality/Morbidity

No human mortality or morbidity data are reported for T-2 mycotoxin use as a bioweapon. Information regarding mortality from ingestion of contaminated food is quite varied, with 10-60% mortality reported in Russia's Orenburg district. Mortality figures from the Kampuchea and Afghanistan uses of mycotoxin as a bioweapon do not report mortality rates, only total number of deaths. Not knowing the number of exposed individuals as related to the number of fatalities makes the calculation of mortality rates impossible.

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Clinical

History

- Patients with cutaneous symptoms may report seeing clouds of a yellow colored smoke or aerosol, but blue and green aerosols also have been reported.
- Patients may report "yellow droplets" on clothing.
- Immediate skin pain and burning on exposed surfaces is described. Eye pain and burning also should be reported.
- Ingested toxin probably has no taste, since no documentation supports a foul odor or taste in previous epidemics of toxin ingestion. This is further supported by the fact that many individuals become ill when exposed to contaminated food without any suspicion of having ingested tainted food.
- The most common symptoms occurring with most exposures include burning skin (or oral) pain and redness or rash, vomiting, diarrhea, dyspnea, and bleeding.

Physical

- Symptom onset usually is observed within 2-4 hours, although significant exposure can cause immediate onset of symptoms.
- The clinical syndrome of ATA represents a more chronic form of exposure to T-2 mycotoxin, and in some ways the syndrome mimics that of radiation sickness. It occurs in 4 stages.
 - The first stage, which may be seen in the emergency department, refers to the acute injury of exposed cells and tissues. This phase is addressed in greater depth below. The symptoms observed in this phase depend on the form of exposure.
 - The second stage, which typically occurs weeks after the exposure, represents bone marrow suppression secondary to the antimitotic effects of the toxin and includes significant leukopenia, granulocytopenia, and thrombocytopenia. Patients may feel well during this phase.
 - The third stage is considered the hemorrhagic stage. In this phase, the patient experiences petechial hemorrhages, especially of the mucosal areas of the nasopharynx and oropharynx. Bleeding encountered in this phase can be fatal. Related airway edema can be present, making airway compromise a complication of this phase. The patient also is at risk for systemic infection secondary to the compromised immune system.
 - The fourth stage of illness typically is referred to as the recovery phase, when all necrotic lesions heal and the bone marrow recovers, replacing essential blood elements.
- Head, eyes, ears, nose, and throat
 - Nasal and oral cavities are painfully erythematous.
 - If the agent is inhaled through the nose, the patient experiences nasal pruritus, pain, and rhinorrhea. Sneezing, coughing, and epistaxis also are noted.
 - If the agent is ingested, nasal symptoms may be sparse with more intense symptoms involving the oral cavity, throat, and esophagus. This manifests as a complaint of severe throat pain and blood-tinged saliva or sputum.
 - Eye exposure results in immediate symptoms of eye pain and tearing. A sensation of a

foreign body and visual blurring may be present. Scleral induration is noted.

- **Gastrointestinal**
 - GI symptoms also are similar to radiation syndrome.
 - Vomiting is the most common symptom. The patient also may complain of significant crampy abdominal pain.
 - Watery or bloody diarrhea typically is reported with ingestion of toxin.
 - Anorexia also is a typical symptom of both ingested and absorbed intoxication.
- **Systemic:** Severe intoxication can result in early systemic effects including prostration, dizziness, and ataxia. Also noted in systemic intoxication are tachycardia, hypothermia, and vascular collapse.

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Differentials

CBRNE - Biological Warfare Agents

CBRNE - Ricin

CBRNE - Staphylococcal Enterotoxin B

Other Problems to be Considered

Vesicant (mustard and lewisite) exposure

Onset of pain may mimic T-2 mycotoxin exposure.

To differentiate lewisite from T-2 mycotoxin exposure, test the skin and clothing for the arsenic component of lewisite.

Onset of dermal symptoms (blistering, pain) from mustard exposure typically is delayed.

Staphylococcal enterotoxin B

Staphylococcal enterotoxin B may cause respiratory and GI symptoms but lacks skin and oral burn symptoms.

Ricin intoxication

All symptoms for ricin intoxication are similar to T-2 mycotoxin with the exception of the painful topical symptoms, which are not observed in ricin intoxication.

Other considerations

Many of the symptoms of T-2 mycotoxin exposure are radiomimetic; consider radiation sickness. Nausea and vomiting have a large differential diagnosis. Examine patients with these symptoms without any suggestion of toxin exposure for other more common causes of these symptoms. The patient who may be more suggestive of a toxin exposure is a high-profile individual (ie, government official) who is vomiting with oral or cutaneous symptoms.

Patients presenting long after the initial exposure who now may be manifesting symptoms of the second stage of ATA may need to be investigated for a malignant process causing bone marrow replacement. Skin and mucosal erythema, blistering, ulceration, and necrosis can be the manifestation of many systemic diseases, which need to be considered.

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Workup

Lab Studies

- Perform immediate postexposure labs to assess for other disease conditions in the differential diagnosis.
- When considering T-2 mycotoxin exposure as the cause and to verify contamination, collect nasal, throat, or respiratory secretions and send for mass spectrometric evaluation.
- Send serum or tissue samples for toxic detection for patients who are 1-5 days postexposure.
- Assess the urine of patients who are more than 6 days postexposure for toxin metabolites.
- Observing the absolute lymphocyte count over time may differentiate those individuals destined to develop bone marrow suppression.

Procedures

- Warning: This is a dermally active toxin that is transmissible in the healthcare setting. Do not approach the patient without adequate protective gear.
- Decontamination is as follows:
 - Remove all of the patient's clothing and clean and scrub the entire skin surface with soap and water. Washing the contaminated area of the skin within 6 hours postexposure can remove 80-98% of the toxin and has been demonstrated to prevent skin lesions and death in experimental animals.
 - Contain clothing to avoid contamination of the health care environment.

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Treatment

Prehospital Care

- Warning: This is a dermally active toxin that is transmissible in the healthcare setting. Do not approach the patient without adequate protective gear.
- Use hazardous materials teams in patient rescue and decontamination.
- Decontamination is of paramount importance to avoid cross-contamination. Remove all clothing and wash the patient in soap and water.
- After decontamination is complete, attend to airway, breathing, and circulation as immediately as possible without risking contamination of medical staff.
 - While one member of the team is caring for problems involving the airway, breathing, and circulation, another should be concerned primarily with patient decontamination.
 - Remove all clothing and clean and scrub the patient's entire skin surface with soap and water. Washing the contaminated area of the skin within 6 hours postexposure can remove 80-98% of the toxin and has been demonstrated to prevent skin lesions and death in experimental animals.
 - Contain clothing to avoid contamination of the health care environment.
- If the patient complains of eye pain or tearing, irrigate the eyes with copious amounts of water.
- No specific antidote exists for this toxin. General supportive measures are indicated.

Emergency Department Care

- Warning: This is a dermally active toxin that is transmissible in the healthcare setting. Do not approach the patient without adequate protective gear.
- Never assume that a patient has been decontaminated in the prehospital setting. Reassess the patient's decontamination status. If the degree of prehospital decontamination is uncertain, rewash the patient to ensure the safety of staff and facility.
 - While one properly protected team member is caring for problems involving the airway, breathing, and circulation, another should be concerned primarily with patient decontamination.
 - Remove all clothing and clean and scrub the patient's entire skin surface with soap and water. Washing the contaminated area of the skin within 6 hours postexposure can remove 80-98% of the toxin and has been demonstrated to prevent skin lesions and death in experimental animals.
 - Contain clothing to avoid contamination of the health care environment.
- No specific antidote is available for the T-2 mycotoxin. Provide supportive measures addressing

respiratory and cardiovascular status as necessary.

- Administer activated charcoal if toxin ingestion is a possibility.
- If the patient complains of eye pain or tearing, irrigate the eyes with copious amounts of water.

Consultations

- Required consultants are dictated by the disease course. Pulmonary consultation may be required for severe dyspnea or hemoptysis. A hematologist may be consulted for patients presenting with severe pancytopenia.
- Contact the local poison control center for additional clinical guidance. Some larger cities' poison control centers may have specific guidelines to follow concerning weapons of mass destruction.
- Consult the Federal Bureau of Investigation in situations of suspected nuclear, biological, or chemical weapons.

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Medication

Administer activated charcoal to patients exposed via the ingestion route. Some sources advocate the use of activated charcoal for the victim of an aerosol source, with the rationale that toxin adherent to the nasal and oral mucosa may be ingested. This theory is questionable since the amount of active toxin probably is not enough to cause significant GI contamination.

Although not proven clinically, theoretical use exists for administering colony-stimulating factors to patients presenting with bone marrow suppression.

Antidotes, Adsorbent

Used to neutralize toxins.

| | |
|------------|---|
| Drug Name | Activated charcoal (Liqui-Char, Super-Char, Insta-Char, Actidose)- Believed to adsorb ingested toxin, thereby preventing absorption and removing toxin from the GI tract, preventing further cellular damage. |
| Adult Dose | 1 g/kg PO/NG; repeat dose of 20-50 g q2-6h may be used |

| | |
|-------------------|---|
| Pediatric Dose | <1 year: 1 g/kg PO 1-12 years: 25-50 g PO Adolescents: 25-100 g PO Repeat doses in children not established; half initial dose recommended |
| Contraindications | Documented hypersensitivity; unprotected airway; GI tract perforation; in patients with high aspiration risk |
| Interactions | May inactivate ipecac syrup if used concomitantly; effectiveness of other medications decreases with coadministration; do not mix charcoal with sherbet, milk, or ice cream (decreases adsorptive properties of activated charcoal) |
| Pregnancy | A - Safe in pregnancy |
| Precautions | Aspiration; secure airway (intubation) in patients with an aspiration risk; accomplish gastric emptying before administering charcoal |

Granulocyte-Stimulating Factors

Used to correct severe neutropenia.

| | |
|-------------------|--|
| Drug Name | Filgrastim (Neupogen)- Granulocyte colony-stimulating factor that activates and stimulates production, maturation, migration, and cytotoxicity of neutrophils. Although not demonstrated or indicated for use in T-2 mycotoxin exposure, may be theoretical use for granulocyte-stimulating factors for patients presenting with severe neutropenia; in this setting conduct use with hematology consultation. |
| Adult Dose | Chemotherapy-induced neutropenia: 5 mcg/kg/d IV/SC qd for 2 wk until ANC reaches 10,000/cm ³ |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Do not use in conjunction with antineoplastic agents, since these agents have specificity for rapidly developing cells, which filgrastim stimulates |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Risk of developing myelodysplastic syndrome or acute myeloid leukemia in certain patients; leukocytosis; possible tumor growth |

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Follow-up

Further Inpatient Care

- If a T-2 mycotoxin exposure is suggested, admission to the hospital is indicated, considering the inability to quantify the exposure and the unpredictable course the patient may take over the ensuing hours to days. Further inpatient care should be supportive. Monitoring of lymphocyte counts may be helpful in indicating the progression of immune compromise.

in/Out Patient Meds

- Although not proven clinically, a theoretical use exists for administering colony-stimulating factors to patients presenting with bone marrow suppression.

Complications

- Airway compromise may be observed when the disease process includes significant airway edema or hemorrhage.

Prognosis

- Prognosis is difficult to assess, since the amount of toxin in previous human ingestions has not been documented. Death from actual toxin ingestion is much less of a concern than the sequelae of immune compromise and successive infection. This is supported by the documented history of the ingestion version of the disease (ATA). No current literature predicts the outcome of T-2 mycotoxin poisoning.

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Miscellaneous

Medical/Legal Pitfalls

- Currently, discussion regarding the medicolegal issues involving physician reporting of biological, radiological, or chemical exposures to law enforcement agencies is ongoing. No legal guidance is available regarding this issue. Legal team consultation may be recommended prior to an actual incident.

Special Concerns

- If it is believed that a patient may have been exposed to any type of nuclear, biological, or chemical weapon of mass destruction, notify the local office of federal law enforcement authorities (eg, Federal Bureau of Investigation) immediately.
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CBRNE - Urticants, Phosgene Oxime

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Synonyms, Key Words, and Related Terms

CX, dichloroformoxime

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Introduction

Background

Phosgene oxime (CX) is an urticant or nettle agent that causes a corrosive type of skin and tissue injury. While often grouped with the vesicant chemical warfare agents, it is not a true vesicant, since it does not cause blisters. Both vapor and liquid CX cause immediate tissue damage upon contact. CX is a solid at temperatures below 95°F, but the vapor pressure of the solid is high enough to produce symptoms. Although both Germany and Russia developed CX before World War II, no uses of the agent on the battlefield are known. CX is of military interest because it penetrates garments and rubber much more quickly than other chemical agents and it produces a rapid onset of severe and prolonged effects.

Pathophysiology

The mechanism of toxicity for CX is uncertain. It primarily affects the skin, eyes, respiratory system, and gastrointestinal tract. The agent seems to cause its greatest systemic effects in the first capillary bed encountered. For example, skin exposure or intravenous (IV) injection of CX causes pulmonary edema, while injection into the portal vein produces hepatic necrosis but not pulmonary edema.

Mortality/Morbidity

Morbidity and mortality for exposures to CX are dose dependent. The estimated LCt50 (concentration-time product capable of killing 50% of exposures) for CX vapor is 1500-2000 mg·min/m³. The LD50 (lethal dose for 50% of exposures) for skin exposures is estimated at 25 mg/kg. Skin and mucous membrane irritation can begin within 12 seconds of a vapor exposure of 0.2 mg·min/m³. Unbearable pain and irritation occur within 1 minute of vapor exposure to 3 mg·min/m³.

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Clinical

History

- Important historic features of a potential toxic chemical exposure include the following:
 - Estimated time of occurrence
 - Duration and circumstances of exposure
 - Onset and time course of symptoms
 - Odor and/or color of gases or vapors
 - Protective clothing worn (if any)
 - Effects on surroundings (eg, other human or animal casualties)
- Some casualties may experience a peppery or pungent odor during their initial CX vapor exposure, but this typically is lost quickly because of accommodation.

Physical

- Skin: Blanching surrounded by an erythematous ring can be observed within 30 seconds of exposure. A wheal develops on exposed skin within 30 minutes. The original blanched area acquires a brown pigmentation by 24 hours. An eschar forms in the pigmented area by 1 week and sloughs after approximately 3 weeks.
- Eyes: Eye examination typically demonstrates conjunctivitis, lacrimation, lid edema, and blepharospasm after even minute exposures. More severe exposures can result in keratitis, iritis,

corneal perforation, and blindness.

- **Respiratory:** Irritation of the mucous membranes may be observed on examination of the oropharynx and nose. Evidence of pulmonary edema, including rales and wheezes, may be noted on auscultation. Pulmonary thromboses are prominent features of severe CX exposure.
- **Gastrointestinal:** Some animal data suggest that CX may cause hemorrhagic inflammatory changes in the GI tract.

Causes

Exposures to CX result from its deliberate use as a chemical warfare agent. Since this chemical has no useful industrial applications, accidental exposures are extremely unlikely.

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Differentials

Burns, Chemical

CBRNE - Chemical Warfare Agents

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Workup

Lab Studies

- Consider ordering an arterial blood gas to obtain a baseline measure in patients with significant respiratory symptoms after exposure.

Imaging Studies

- Order a chest x-ray to examine for evidence of pulmonary edema in patients with respiratory symptoms after exposure.

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Treatment

Prehospital Care

- The key aspects of prehospital care are removal of casualties from the source of exposure and rapid decontamination with copious amounts of water to remove any agent that has not yet reacted with tissue.
- Administer oxygen to patients with significant respiratory distress. Endotracheal intubation and ventilatory support may be required for patients with severe airway exposures or progressive pulmonary symptoms.
- Administer sufficient doses of systemic analgesics as soon as possible.

Emergency Department Care

Emergency department care is a continuation of prehospital care and is supportive in nature. No antidotes exist for CX exposure. Verify complete decontamination to ensure that no medical personnel become casualties.

- Airway and/or pulmonary
 - Be alert to the possible need for airway management in patients with severe exposure.
 - Administer oxygen to patients with significant respiratory symptoms.
 - Provide supportive care for noncardiogenic pulmonary edema as required.
- Eyes
 - Apply topical antibiotics to reduce risk of infection and adhesions.
 - Topical anticholinergics may reduce the risk of future synechiae formation.

Consultations

- Consult ophthalmology to provide close follow-up care for significant ocular exposures.
- Consult plastic surgery for severe dermal damage.

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Medication

No medications are specific to the treatment of CX exposure. Use analgesics and topical antibiotics as preferred by the emergency department physician.

Parenteral Analgesics

CX injuries are extremely painful and require liberal use of parenteral analgesics. No recommendations for specific parenteral analgesics are available. Select a medication (eg, morphine, meperidine) that is readily available and provides adequate pain relief to the patient.

| | |
|-------------------|--|
| Drug Name | Morphine sulfate (Duramorph, Astramorph, MS Contin)- An alkaloid of opium and a commonly used systemic narcotic analgesic; a good first choice parenteral medication that may be titrated to patient needs. |
| Adult Dose | 2-10 mg/70 kg IV over 4-5 min initially; titrate additional doses to effect |
| Pediatric Dose | 0.1-0.2 mg/kg, not to exceed 15 mg IV q4h |
| Contraindications | Documented hypersensitivity; acute bronchial asthma; upper airway obstruction |
| Interactions | Depressant effects are potentiated by other CNS depressants (eg, alcohol, sedatives, antihistamines, psychotropic drugs) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Closely monitor patients for respiratory depression; morphine-associated pupillary changes may obscure existence, extent, and course of intracranial pathology in patients with head injuries; monitor patients with known seizure disorders for morphine-associated seizure activity; serum half-life may be prolonged in patients with hepatic and renal disease; may precipitate acute respiratory failure in patients with chronic pulmonary disease; may cause urinary retention in patients with disorders of the urinary system; may result in biliary colic in patients with disorders of the biliary system; caution in neonates (monitor respiratory depression) |

| | |
|-------------------|---|
| Drug Name | Meperidine (Demerol)- Narcotic analgesic with multiple actions qualitatively similar to those of morphine, typically administered in conjunction with promethazine. |
| Adult Dose | 50-150 mg slow IV (diluted) or IM/SC q3-4h prn plus promethazine 25 mg IV/IM |
| Pediatric Dose | >2 years: 1-1.8 mg/kg slow IV or IM/SC q3-4h prn plus promethazine 0.25-0.5 mg/kg mg IV/IM |
| Contraindications | Documented hypersensitivity; concurrent use of MAOIs or prior use within past 14 d |
| Interactions | Depressant effects are potentiated by other CNS depressants; concurrent use may result in respiratory depression, hypotension, and profound sedation or coma |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|--|
| Precautions | Not a particularly good analgesic choice for repetitive dosing (seizures have been reported after normeperidine accumulation, which is more likely with quantities of analgesics required to manage CX exposure); may increase intracranial pressure and respiratory depression in patients with head injuries; pupillary changes may mask presence, extent, and course of intracranial pathology; inadvertent arterial injection results in vascular spasm with subsequent distal ischemia; rapid IV injection increases risk of respiratory depression, severe hypotension, and circulatory collapse; may result in acute respiratory failure in patients with chronic pulmonary disease; may result in severe hypotension in patients whose ability to maintain blood pressure is compromised by depleted blood volume or concurrent use of anesthetic agents |
|-------------|--|

Topical Antibiotic Ointments

Indicated for treatment of CX injury of skin and eyes; no specific ointments are recommended; select an available broad-spectrum ophthalmic or skin preparation (eg, bacitracin, Ilotycin).

| | |
|-------------------|---|
| Drug Name | Bacitracin (AK-Tracin, Baciguent)- Broad-spectrum antibiotic topical ointment that is a good first choice for superficial wound care. |
| Adult Dose | Apply to affected area qd/tid |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | None reported |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Prolonged use may result in overgrowth of nonsusceptible organisms |

| | |
|-------------------|---|
| Drug Name | Erythromycin ophthalmic ointment (Ilotycin, E-mycin)- Broad-spectrum macrolide antibiotic indicated in treatment or prevention of superficial ocular infections. |
| Adult Dose | 1 cm applied to affected eye; not to exceed 6 times/d |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; viral, mycobacterial, or fungal infections of eye; patients using steroid combinations after uncomplicated removal of a foreign body from cornea should avoid using this product |
| Interactions | None reported |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |

| | |
|-------------|--|
| Precautions | Do not use topical antibiotics to treat ocular infections that may become systemic; prolonged or repeated antibiotic therapy may result in bacterial or fungal overgrowth of nonsusceptible organisms and may lead to secondary infection (take appropriate measures if superinfection occurs) |
|-------------|--|

Oral Analgesics

Patients may be switched from parenteral analgesics to an oral form once their injuries have improved sufficiently to tolerate alternative pain control measures; no specific recommendations are available; use a readily available product (eg, Percocet, Tylenol with codeine) that provides adequate pain relief and is well tolerated by the patient.

| | |
|-------------------|---|
| Drug Name | Oxycodone/acetaminophen (Percocet)- Semisynthetic opioid analgesic with multiple actions similar to those of morphine; acetaminophen is a nonopiate, nonsalicylate analgesic and antipyretic. |
| Adult Dose | 1-2 tab q6h prn; not to exceed 4 g/d acetaminophen |
| Pediatric Dose | Not recommended |
| Contraindications | Documented hypersensitivity |
| Interactions | Increased CNS depression may be observed with concomitant use of other opioids, general anesthetics, phenothiazines, sedative-hypnotics, and alcohol; concurrent use of MAOIs or TCAs may increase effect of either antidepressant or oxycodone; concurrent use of anticholinergics may result in paralytic ileus |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Respiratory depression may be enhanced in patients with head injuries; pupillary changes may mask presence, extent, and course of intracranial pathology; administer with caution to certain patients (eg, elderly or debilitated patients; those with severe impairment of hepatic or renal function, hypothyroidism, Addison disease, prostatic hypertrophy, urethral stricture); may cause inability to operate a motor vehicle or dangerous machinery |

| | |
|------------|--|
| Drug Name | Acetaminophen/codeine (Tylenol with codeine)- Combines analgesic effects of a centrally acting opium-derived alkaloid (codeine) and a peripherally acting nonopioid analgesic (acetaminophen). |
| Adult Dose | 1-2 tab Tylenol #3 (30 mg codeine phosphate plus 300 mg acetaminophen) or Tylenol #4 (60 mg codeine phosphate plus 300 mg acetaminophen) q4-6h prn, not to exceed 360 mg codeine and 4 g acetaminophen in 24 h |

| | |
|-------------------|--|
| Pediatric Dose | Tylenol with codeine elixir: 0.5-1 mg/kg/dose based on codeine q4-6h; 10-15 mg/kg/dose based on acetaminophen content; not to exceed 75 mg/kg/d of acetaminophen <3 years: Not established 3-6 years: 5 mL (1 tsp) PO qid prn 7-12 years: 10 mL (2 tsp) PO qid prn |
| Contraindications | Documented hypersensitivity |
| Interactions | Increased CNS depression may be observed in concomitant use with other narcotic analgesics, antipsychotics, antianxiety agents, or other CNS depressants, including alcohol; concurrent use of anticholinergics may result in paralytic ileus |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Respiratory depression may be enhanced in patients with head injuries; pupillary changes may mask presence, extent, and course of intracranial pathology; administer with caution to certain patients (eg, elderly or debilitated patients; those with severe impairment of renal or hepatic function, hypothyroidism, Addison disease, prostatic hypertrophy, urethral stricture) |

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Follow-up

Further Inpatient Care

- Pain associated with CX exposure typically remains severe for several days. Consider admission for pain control. Admit any patients demonstrating significant respiratory symptoms for observation and supportive care.

Further Outpatient Care

- Patients may be treated on an outpatient basis once respiratory symptoms have resolved and nonparenteral analgesics are adequate for pain control.
- Instruct the patient on appropriate wound care techniques and provide close follow-up care to the patient to ensure adequate healing.
- Ophthalmology follow-up care to ensure resolution of ocular injuries also is important.

in/Out Patient Meds

- Inpatient medications include parenteral analgesics (eg, morphine, meperidine), broad-spectrum

ophthalmic antibiotic ointments for eye injuries, and broad-spectrum skin antibiotic ointments for skin burns.

- Outpatient medications include oral analgesics (eg, codeine, oxycodone) if continued pain management is required after discharge and continued antibiotic ointments for eye and skin injuries until full healing has occurred.

Transfer

- Transfer to a higher medical center may be required for severe pulmonary CX injuries if the initial hospital is unable to provide the necessary intensive care support. Secure the airway and initiate ventilatory support prior to transfer.

Complications

- Potential complications include scarring, wound infections, loss of vision, and death from severe respiratory injury.

Prognosis

- Prognosis generally is good for minimal exposures. Severe and early respiratory distress portends a poor prognosis.

Patient Education

- Educate outpatients about the signs and symptoms of wound infection for which they immediately should seek further medical care.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to provide rapid, early decontamination
 - Failure to provide early airway support measures in patients with severe pulmonary exposures
 - Failure to provide adequate pain management to patients
-

Pictures



Picture 1: Anteroposterior portable chest radiograph in a male patient who developed phosgene-induced adult respiratory distress syndrome. Notice the bilateral infiltrates and ground glass appearance.

Picture type: X-RAY

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CBRNE - Venezuelan Equine Encephalitis

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Synonyms, Key Words, and Related Terms

VEE

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Introduction

Background

Venezuelan equine encephalitis (VEE) is an acute viral disease characterized by fever and one or more of the following: chills, headache, back pain, myalgias, prostration, nausea, and vomiting. The disease may progress to encephalitis.

Clinical symptoms are similar to those of many other zoonotic infections that cause fever and headache (eg, St Louis encephalitis, Japanese encephalitis, West Nile encephalitis, dengue). Unlike these infections, which are caused by a Flavivirus, VEE is caused by an enveloped single-stranded RNA virus of the Togaviridae family.

Several members of the Alphavirus genus possess characteristics that make them well suited for weaponization, which was recognized in the 1930s and 1940s. Alphaviruses can be produced in large quantities, are relatively stable, and can be delivered effectively via the aerosol route. Producing a highly effective vaccine also has proven to be technically difficult.

Even today VEE remains a potentially potent biological weapon. If it were deployed efficiently it could incapacitate thousands of people for a week or more and cause untold psychological stress among millions. The virus that causes VEE also is quite susceptible to genetic manipulation. This has proven useful in the laboratory in the development of more effective vaccines; however, it also could be exploited to produce more effective biological weapons.

History of disease

VEE, first recognized in the 1930s, has been responsible for numerous outbreaks of febrile illnesses and encephalitis involving thousands of humans and hundreds of thousands of equines, primarily in tropical America. VEE viruses are transmitted among equines and rodents by a variety of mosquito species.

Human and animal infections have occurred in equatorial South and Central America, including Colombia, Panama, Peru, Brazil, Venezuela, French Guiana, Guyana, and Surinam. Mortality rates vary in horses but reportedly are as high as 80%.

The impact caused by VEE epidemics is best illustrated by examining large Venezuelan and Colombian epidemics that occurred in 1962-63, 1967, and 1995. In the 3 epidemics combined, more than 300,000 humans were infected, with more than 4% experiencing severe neurologic symptoms and more than 2000 deaths (<1%) reported. Clinically apparent but less severe neurologic manifestations occurred in additional patients.

Symptomatic manifestations of disease are variable. The incidence among humans during epidemics has been as high as 300 in 1000 persons per month, especially among children younger than 15 years. In some epidemics, more than 50% of persons residing in rural villages have become ill, while during other outbreaks, the incidence has been less, although minimally infected persons may not have been reported.

Initial diagnosis may be difficult; therefore, a high index of suspicion is required in examining a symptomatic patient with a history of travel to an endemic area or an area experiencing an active epidemic.

Pathophysiology

VEE is caused by a virus of the Togaviridae family, Alphavirus genus. Other alphaviruses cause diseases similar to VEE, including eastern equine encephalitis and western equine encephalitis. Togaviruses also cause human diseases with different clinical manifestations and outcome, including familiar diseases such as rubella and rarer diseases such as chikungunya fever and Sindbis virus.

Zoonotic transmission

VEE has a zoonotic reservoir in bats, birds, rodents, equines (horses, donkeys, mules), and certain tropical jungle mammals. Rodents and other small animals are the most important amplifiers in endemic preservation of the virus in tropical forests, swamps, and marshlands. Horses are the most important amplifier hosts in large epidemic outbreaks.

Humans and horses are infected by a wide variety of mosquito vectors, including *Culex*, *Mansonia*, *Psorophora*, and *Aedes* species. Humans acquire infection as an incidental dead-end infection of the normal animal-mosquito-animal cycle in nature. Blood viral loads of infected patients may exceed the threshold level required to infect mosquitoes, and human-mosquito-human transmission has been suspected in some epidemics.

Other potential modes of transmission

Can person-to-person transmission of VEE occur? VEE virus has been isolated from throat washings of patients. Furthermore, aerosol transmission of the virus has occurred as a result of laboratory accidents or lack of laboratory precautions. An analysis of laboratory incidents suggests that the aerosol form of VEE is highly infectious, making VEE a potential biowarfare agent. This could be especially worrisome if strains are altered genetically to increase pathogenicity.

The Centers for Disease Control and Prevention (CDC) extensively analyzed the 1995 VEE outbreak in northwest Colombia and reported a 5% secondary household attack rate. Whether these secondary attacks were from bites by mosquitos infected from animals or humans was unclear. At the present time, direct human-to-human transmission is not proven scientifically but is suspected.

Virus action

The virus gains access to human tissue after a bite by an infected mosquito. VEE is lipid and glycoprotein-enveloped and contains RNA of approximately 12 kilo base pairs in length. It first enters lymphatic and bone marrow cells by receptor-mediated endocytosis. After replication and release, it infects other cells and results in febrile illness. In a subset of patients, the virus gains entrance into the CNS, leading to acute encephalitis.

An initial immune response with immunoglobulin M (IgM) occurs specific to viral surface components, followed by neutralizing antibody and other immune defenses against the virus infection. Serologic studies of populations exposed during epidemics have demonstrated a high seroconversion rate, with most persons experiencing only flulike symptoms or no symptoms at all.

Frequency

- **In the US:** VEE is rare in the US. A major epidemic in horses occurred in Texas in the past, but less than 100 laboratory-confirmed cases in humans are documented. Data from international outbreaks suggest that many more infections occurred that were subclinical or mild. Unless a large-scale epidemic in US horses occurs, VEE observed in US EDs will have been acquired abroad or due to an intentional release of the pathogen.
- **Internationally:** Endemic areas: The incidence in endemic subtropical and tropical areas has not been determined clearly, since isolated cases resulting from rodent-mosquito-human transmission may remain undocumented.

Epidemic areas: Epidemics that occur every few years have demonstrated that equines are highly susceptible to severe disease and respond poorly. Mosquito-exposed humans in these areas are also at risk, and most are believed to become infected; however, most human infection is subclinical or mild.

Mortality/Morbidity

- Overall mortality from epidemics is 0.5-1%. In patients who develop encephalitis, mortality is in the range of 20%.
- Encephalitis is clinically diagnosed in 2-4% of adults and 3-5% of children.

Sex

Gender is nonspecific; however, many ranch workers in at-risk areas are male, thus increasing their risk.

Age

Data from epidemics demonstrate that children have the highest risk of acquiring moderate or severe forms of the infection.

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Clinical

History

Symptoms range from mild to severe.

- Patients give a history of exposure to mosquitoes in an area endemic for VEE. A patient who has

been exposed in an endemic area may present to a US ED with VEE, since the incubation period ranges from 1 day to 1 week.

- Suspect VEE in anyone with a febrile illness who recently has traveled in rural areas of central or tropical South America. Consider it in the differential whenever considering the diagnosis of dengue in persons coming from those areas.
- Mild disease: Persons with the mild form of illness may describe only minimal flulike symptoms of low-grade fever, myalgias, or headache.
- Moderate disease: Typical symptoms of moderate disease found on history include fever, intermittent chills, myalgias, back pain, headache, photophobia, vomiting, and/or hypesthesia. Less common complaints include sore throat.
- Severe disease: Patients with the severe form present with acute-onset high fever (39-40°C), severe myalgias, severe back pain, headache, photophobia, vomiting, weakness, prostration, and confusion.
- Biological warfare: Suspect biological warfare or terrorism when large numbers of patients present with VEE from a nonendemic region.

Physical

The physical examination results in nonspecific findings of an acute febrile illness. In patients with advanced disease in which encephalitis has developed, findings may include nuchal rigidity and ataxia.

- Fever
 - May be low grade in patients with mild cases
 - In patients with moderate-to-severe cases, may be 38-40°C
- Nuchal rigidity in patients developing encephalitis
- Altered mental status in moderate and/or severe infections
- Coma in patients with progressed encephalitis

Causes

VEE is caused by exposure to mosquitos infected with VEE in endemic or epidemic areas or by intentional release of a VEE biological weapon.

- Endemic-acquired infections: These are the most difficult infections to diagnose. In many mosquito-infested areas of central and tropical South America, rodents dwelling in the jungle, swamp, marsh, or forest harbor VEE. Because many other infectious agents from these areas also cause febrile illnesses, VEE is not always considered early in the patient's course.
- Epidemic-acquired infections: These infections usually are well publicized, with public health and veterinary officials containing the infection in a high-visibility manner. Patients with fever originating from these areas may present with worries about VEE and may request that the disease be excluded specifically.

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Differentials

Dengue Fever
Encephalitis
Malaria
Meningitis
Yellow Fever

Other Problems to be Considered

St Louis encephalitis

West Nile fever

Japanese encephalitis

Western equine encephalitis

Eastern equine encephalitis

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Workup

Lab Studies

- Draw standard laboratory tests for patients that present to the ED with a febrile illness, including CBC, electrolytes, LFTs, urinalysis, and other tests as indicated by history and physical examination. Most lab studies from patients infected with VEE are nonspecific for febrile illnesses. However, CBC and LFTs may demonstrate the following:
 - CBC - Lymphopenia and thrombocytopenia
 - LFTs - Elevated lactate dehydrogenase (LDH) and aspartate aminotransferase (AST)

Imaging Studies

- A chest x-ray may help exclude other infectious causes of fever such as pneumonia or tuberculosis.
- MRI may be useful in diagnosing encephalitis.

Other Tests

- Serology: Send serum to a laboratory with the capacity to measure IgM and immunoglobulin G (IgG) using enzyme-linked immunosorbent assay (ELISA) tests.

Procedures

- Lumbar puncture: Cerebrospinal fluid may demonstrate elevated lymphocytes in patients with encephalitis. Cerebrospinal fluid sent to appropriate laboratories can undergo further testing to identify VEE.

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Treatment

Prehospital Care

No specific treatment other than supportive care is available.

Emergency Department Care

Treatment is symptomatic and in the ED most likely involves correcting fluid deficiencies.

Consultations

Contact an infectious disease specialist if VEE is suggested. In addition, involve the local county health department.

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Medication

No specific medications are approved for treatment of VEE. In vitro laboratory studies suggest that ribavirin and other nucleoside analogues may be future agents, but they are not used clinically.

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Follow-up

Further Inpatient Care

- Admit patients with possible VEE and observe them for progression to encephalitis.

Transfer

- In hospitals that do not have infectious disease specialists on staff, consider transferring the patient to a tertiary referral medical center.

Deterrence/Prevention

- Endemic areas: Protection from mosquito bites is important. Visitors to endemic areas should take appropriate precautions to avoid mosquito bites, including proper clothing, insect repellent, and mosquito nets.
- Epidemic areas: Persons other than medical teams that are part of a response organized or approved by the local or national governments should avoid visiting areas experiencing ongoing epidemics.
- Vaccines: A live attenuated virus can be used to vaccinate equines. A formalin-inactivated virus has been used to vaccinate human laboratory workers at risk, but this vaccine is not available to the general public.
- Biological warfare or terrorism: Health care provider must maintain a high index of suspicion for this diagnosis.

Complications

- Admit patients with suggested VEE and observe them for progression to encephalitis. In patients who develop encephalitis, mortality is in the range of 20%. Encephalitis is clinically diagnosed in 2-4% of adults and 3-5% of children.

Prognosis

- Recovery from the illness occurs in 99% of adults and 97-98% of children.

Patient Education

- Educate patients on [Deterrence/Prevention](#).

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Miscellaneous

Medical/Legal Pitfalls

- Preexposure: Failure to warn patients who plan to travel to endemic areas about VEE may place the physician at a small risk.
 - Postexposure: Failure to diagnose VEE may place the physician at a medicolegal risk. However, since no treatment is available and direct human-to-human transmission is debatable, this risk is small. The most common error in the US may occur from misdiagnosing VEE as influenza in a patient returning from an endemic area.
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CBRNE - Vesicants, Mustard: Hd, Hn1-3, H

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Introduction

Background

Mustard agents are vesicants (blister agents) used in warfare to produce casualties, degrade fighting efficiency, and force opposing troops to wear full protective equipment. Mustard agents include nitrogen mustards (HN-1, HN-2, HN-3), sulfur mustards (H, HD, HT), and mustard-lewisite (HL). Mustard agents are oily liquids ranging from colorless (in pure state) to pale yellow to dark brown, depending on the type and purity. They have a faint odor of mustard, onion, garlic, or horseradish, but because of olfactory fatigue, do not rely on odor for detection. Volatility varies with the particular compound. Mustard agents are only slightly soluble in water and may persist for long periods. HN-1 is more volatile and less persistent than HD, but it is only one fifth as potent a vesicant to the skin. HN-3 is less volatile and more persistent than HD and has equal vesicant effects.

Mustard agents rapidly penetrate clothing and skin. Chemical protective mask with charcoal filters, chemical protective overgarments with charcoal, and butyl rubber chemical protective gloves and boots afford full protection against mustard agents.

More than 2 dozen nations may have the capability to manufacture offensive chemical weapons. Mustard agents are simple to manufacture and therefore can be a first choice for countries or terrorists who decide to have a capacity for chemical warfare agents. Mustard agents may be delivered by artillery shell, mortar shell, rockets, bombs, or aircraft spray.

Mustard agents constitute both a vapor and a liquid threat. Mustard agents cause tissue damage within several minutes of contact. No immediate symptomatic or local reaction occurs to mustard vapor or liquid. Decontamination must be performed immediately after contact to prevent injury. A latent period occurs, ranging from 4-12 hours after mild exposure and 1-3 hours after severe exposure, prior to the onset of symptoms. More than 80% of mustard casualties are from vapor exposure, but more severe injuries are caused after contact with liquid mustard agents.

Mustards first were produced in 1822, but their harmful effects were not discovered until 1860. On July 12, 1917, the Germans delivered artillery shells containing HD on a World War I battlefield near Ypres, Belgium. More than 20,000 casualties resulted from this first use of mustard as a chemical warfare agent. Subsequently, mustard agents accounted for 80% of chemical casualties in World War I. Among 6980 cases of mustard burns during World War I, the location of the lesions were as follows: eyes, 86%; respiratory, 75%; scrotum, 42%; face, 27%; anus, 24%; legs, 11%; buttocks, 10%; hands, 4%; and feet, 1.5%. Fewer than 5% of casualties from mustard who reached medical treatment died. Mustard injuries were slow to heal and necessitated an average convalescent period of more than 6 weeks.

Italy allegedly used mustard against Abyssinia in the 1930s. Japan allegedly used mustard agents against the Chinese from 1937-1944.

Nitrogen mustard agents were synthesized in the late 1930s. Mechlorethamine (HN-2) became the prototypical mustard agent used as a cancer chemotherapeutic agent. Germans and Americans started the military production of nitrogen mustard agents in 1941 and 1943, respectively. They have not been used on the battlefield.

Toward the end of World War II, a German air attack on the Italian port of Bari struck a US ship loaded with mustard agent munitions. Large amounts of mustard agents were released to the atmosphere and into the harbor water. Many soldiers and sailors were exposed to the mustard-contaminated water. Of 617 US mustard casualties, 83 died.

During the Yemen War of 1963-1967, Egypt reportedly used mustard bombs against the royalist troops in North Yemen.

During the Iran-Iraq war from 1979-1988, approximately 5000 Iranian soldiers were reported killed by Iraqi chemical agents, 10-20% by mustard agents. Additionally, 40,000-50,000 individuals were injured.

After the February 1991 cease-fire ending the Persian Gulf War, United Nations inspection teams discovered mustard agents at Al Muthanna, Iraq.

In Sweden, recurring incidents of mustard agent exposures involve fisherman who encounter discarded chemical weapons that were dumped in the waters off the coast after World War II.

The US stockpile of mustard chemical warfare agents currently is undergoing destruction.

Pathophysiology

Mustard agents are lipophilic and are absorbed readily across intact skin and mucous membranes. The rapid penetration is enhanced by moisture, heat, and thin skin. Approximately 10% of the mustard dose binds to the skin as reacted (fixed) mustard, and the remaining 90% is distributed in the circulation as unreacted (free) mustard to almost all organs and tissues. Because of dilutional effects, systemic effects are observed only at high doses. Mustard is eliminated from the body in the urine as a by-product of alkylation.

No single mechanism or clear understanding exists for the biological damage caused by mustard agents. The toxic effects of mustards depend on their rapid covalent binding to a large number of biological molecules and in the formation of a reactive cyclic ethylene sulfonium ion. Mustard agent molecules contain 2 reactive binding groups. Mustards can bind to nucleophiles such as nitrogen in the base components of nucleic acids and sulfur in SH-groups in proteins and peptides. Mustards can destroy a large number of cellular substances by alkylation.

Mustards also bind to cellular glutathione, a small peptide that is a major free radical scavenger. Glutathione depletion leads to inactivation of enzymes, loss of calcium homeostasis, lipid peroxidation, cellular membrane breakdown, and cell death.

Mortality/Morbidity

The concentration-time product capable of killing 50% of exposures (LCt50) of mustard vapor is 1500 mg·min/m³, and the lethal dose to 50% of exposures (LD50) of liquid mustard on the skin is 100 mg/kg.

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Clinical

Physical

- The eyes are most sensitive and vulnerable to mustard. Ocular effects precede cutaneous

manifestations and occur at lower concentrations. HN causes more severe and earlier ocular lesions than HD.

- Conjunctivitis follows an exposure time of approximately 1 hour to a concentration barely perceptible by odor that does not affect the skin or respiratory mucosa significantly.
- In World War I, 75% of eye exposures were classified as mild conjunctivitis.
- After mild exposure, a latent period of 4-12 hours is followed by lacrimation, a sensation of grit in the eyes, conjunctival injection, and edema (palpebral and bulbar). Recovery requires 1-2 weeks.
- After heavy exposure, eye signs and symptoms appear after 1-3 hours, and severe lesions may appear. Blepharospasm is common.
- A steamy haziness of the cornea or an orange-peel roughening of the cornea may occur. Spotty hemorrhagic discolorations of the iris may be observed. Temporary blindness is common, but permanent blindness is rare.
- Mild corneal involvement demonstrates corneal erosions with fluorescein staining. Superficial corneal scarring and vascularization or iritis may occur.
- With severe corneal involvement, dense corneal opacification with deep ulceration and vascularization occurs. Local necrosis of the cornea may rupture the globe. Panophthalmitis may occur and result in eye loss if appropriate therapy is not instituted.
- Recovery from the ocular effects, especially with corneal involvement, may take months.
- A longer latent period (18-24 h) may occur before the onset of respiratory symptoms. In patients with eye symptoms, expect the development of respiratory effects. Inhalation of mustard vapors may damage the laryngeal and tracheobronchial mucosa. Single exposure to a low concentration of vapor does not cause significant injury. Repeated or chronic exposures may lead to the development of pulmonary fibrosis, chronic bronchitis, and bronchiectasis.
 - Respiratory effects develop slowly and reach maximal severity in several days. Early symptoms begin with hoarseness and loss of voice. This is followed by a cough, which subsequently becomes productive. Fever, dyspnea, rhonchi, and wet crackles may develop. Mild symptoms last 1-2 weeks. Recovery is slow, and coughing may persist for 1 or more months.
 - Moderate acute exposure leads to mucous membrane hyperemia, edema, and necrosis. Profuse, thin, mucopurulent rhinorrhea occurs; sinusitis may develop later. Mucosal findings range from small discrete ulcerations to extensive sloughing.
 - Pharyngitis usually appears 1-3 days after inhaling mustard vapors and may occur with nasal involvement in mouth breathers. The palate, uvula, tonsils, and pharynx are hyperemic and edematous. Multiple whitish ulcerations appear, varying in size according to severity of exposure. Laryngeal involvement resembles that of the pharynx. Edema and necrosis may lead to airway obstruction. Hoarseness, which almost always is present, may last 3-6 weeks or longer.
 - Severe inhalation exposures lead to a diphtherialike pseudomembrane, which may form a cast of the tracheobronchial tree. Mechanical obstruction from pseudomembrane formation and laryngospasm may cause death in the first 24 hours. Mild patchy pulmonary edema and focal atelectasis occur. Chemical pneumonitis may appear after the first 24 hours. Hemorrhagic pulmonary edema is not common and occurs only with severe damage.

Suppurative bacterial bronchitis and bronchopneumonia are frequent complications; the latter is responsible for almost all deaths from vapor exposure.

- In World War I, early mortality occurred in slightly more than 2% of US troops exposed to mustard and was caused almost entirely by pulmonary complications. Approximately 10% of the Iranian casualties treated in western European hospitals during the Iran-Iraq War developed progressive stenosis of the tracheobronchial tree.
- Moderate-to-severe liquid cutaneous, inhalational, or GI exposures cause systemic symptoms of nausea, vomiting, fever, malaise, and prostration.
 - The most severe patients also may present with CNS symptoms (eg, CNS depression) and parasympathetic effects such as bradycardia and other dysrhythmias.
 - Hemoconcentration and hypovolemic shock may occur due to fluid shifts and losses or GI hemorrhaging.
 - With systemic absorption of near lethal doses, hematopoietic and lymphatic tissue injuries occur, resulting in leukopenia, thrombocytopenia, and anemia. The thymus, spleen, and lymph nodes may involute rapidly. The development of shock, thrombocytopenia, leukopenia, and hemorrhagic diathesis are grave prognostic signs. Bone marrow failure is the most frequent cause of late deaths from mustard exposure.

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Differentials

Bronchitis

Burns, Chemical

Burns, Ocular

Burns, Thermal

CBRNE - Arsenicals, Arsine

CBRNE - Chemical Decontamination

CBRNE - Chemical Detection Equipment

CBRNE - Chemical Warfare Agents

CBRNE - Chemical Warfare Mass Casualty Management

CBRNE - Evaluation Of A Chemical Warfare Victim

CBRNE - Incendiary Agents, Magnesium and Thermite

CBRNE - Incendiary Agents, Napalm

CBRNE - Incendiary Agents, White Phosphorus

CBRNE - Irritants: Cs, Cn, Cnc, Ca, Cr, Cnb, PS

CBRNE - Lung-Damaging Agents, Chlorine

CBRNE - Lung-Damaging Agents, Phosgene

CBRNE - Lung-Damaging Agents, Toxic Smokes: Nox, Hc, Rp, Fs, Fm, Sgf2, Teflon

CBRNE - Personal Protective Equipment

CBRNE - Urticants, Phosgene Oxime

CBRNE - Vesicants, Organic Arsenicals: L, ED, MD, PD, HL

Conjunctivitis

Hazmat

Pharyngitis

Pneumonia, Bacterial

Sinusitis

Toxicity, Arsenic

Toxicity, Phosgene

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Workup

Lab Studies

- No hospital laboratory test exists to identify or quantify mustard exposures, since mustard is biotransformed and bound to tissues within minutes of adsorption.
- Obtain a complete blood count, serum electrolytes, and coagulation studies; observe these periodically in all patients except those with isolated mild ocular or cutaneous involvement.
- Leukocytosis occurs during the first day. After large systemic adsorption, leukopenia may begin on day 3-5. A leukocyte count of 500 or less is an unfavorable prognostic sign.

Imaging Studies

- Chest radiographs: Mustards may cause a chemical pneumonitis. Secondary infection may lead to lobular or lobar consolidation. Radiographic appearance follows the characteristics of the type of secondary pneumonia.

Other Tests

- The US military has the capability of detecting mustard agents in the environment with the use of the M256A1, M272 water testing kit, miniature chemical agent monitor (MINICAMS), individual chemical agent detector (ICAD), M18A2, M21 remote sensing alarm, M90, M93A1 Fox, Bubbler, chemical agent monitor (CAM), depot area air monitoring system (DAAMS), and M8 or M9 chemical detection paper.

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Treatment

Prehospital Care

- Patients contaminated with mustard agents endanger unprotected health care providers. Decontaminate patients exposed to mustard agents before transport and entry into medical treatment facilities to prevent vapor accumulation. Providers attending contaminated patients should have protective masks, butyl rubber gloves, and chemical protective overgarments.
- Unless carried out within 1-2 minutes, decontamination of victims exposed to mustard agents does not prevent subsequent blistering. After that brief window, decontamination still should be carried out to prevent secondary contamination.
 - The first step is to cut away all of the victim's clothing. Also cut away and discard mustard-contaminated hair.
 - Exposed skin and scalp can be decontaminated using the military M291 or M258A1 skin decontamination kits. Alternately, use 0.5% aqueous chlorine solution to thoroughly wash the skin and hair. Wash off the decontamination solutions within 3-4 minutes with soap and water. If the victim already has erythematous skin, decontaminating the skin with just soap and water is recommended.

Emergency Department Care

- No specific treatment or antidote can reverse or prevent the cellular effects of mustard agents.
- Apply steroid ointments and antibiotic ointments and relegate their further use to an ophthalmologist. Ophthalmic ointments containing boric acid 5% provide lubrication.
 - Do not patch the eyes and do not allow the eyelids to stick together. Sterile petroleum jelly can be used to lubricate and prevent sealing of the eyelids.
 - Use cycloplegic eye drops (atropine or homatropine) 3 times a day in patients with severe blepharospasm and photophobia for pain and to prevent future synechiae formation. Keep patients in a darkened room.
 - Systemic narcotic analgesics are recommended for pain control. Do not use topical ophthalmic anesthetics.
 - Hospitalization seldom is required for mild eye exposures; early and prolonged hospitalization with ophthalmologist consultation is required for moderate and severe cases.
- Débride ruptured vesicles or bullae. Cleanse the underlying skin with sterile saline. Small areas of involvement can be dressed with petroleum gauze. Facial lesions are best covered with bacitracin ointment and left open.
 - Applying a 1/8-inch thick layer of mafenide acetate or silver sulfadiazine burn cream may treat larger areas of involvement best. Clean and redress these larger wounds twice a day. Multiple or large areas of vesication are cleansed easily with whirlpool bathing.

- Culture wounds that become infected similar to thermal burns and administer appropriate parenteral antibiotics.
- Avoid overhydration, since fluid losses generally are less than with thermal burns.
- Liberal uses of narcotic analgesics are warranted to treat painful skin lesions.
- Treatment of systemic toxicity from mustard is supportive. Atropine sulfate (0.4-0.8 mg SC) may be used in reducing GI hyperactivity. General discomfort, restlessness, and pain may be treated with sedative and/or narcotic analgesics. Adequate nutrition and fluid and electrolyte replacement are mandatory for patients with severe poisonings who have vomiting, diarrhea, leukopenia, hemoconcentration, and shock. Patients with severe leukopenia require isolation and may require appropriate antibiotics.

Consultations

- Seek ophthalmologic consultation as soon as possible when eye involvement is present. Admit patients with corneal findings to the hospital.
- Involve plastic surgeons in the care of those with cutaneous injuries admitted to the hospital.
- Consult hematology and/or oncology specialists for patients with aplastic anemia, which is much more common after HN exposure.

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Medication

The goals of pharmacotherapy are to neutralize toxicity, reduce morbidity, and prevent complications.

Cycloplegics and Mydriatics

Instillation of long-acting cycloplegic agents can relax any ciliary muscle spasm that can cause a deep aching pain and photophobia.

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| Drug Name | Homatropine ophthalmic drops 2-5% (AK-Homatropine, Isopto-Homatropine)- Contains homatropine hydrobromide, which blocks action of certain parasympathetic nerves and cholinergic drugs; used in ophthalmology for mydriatic and cycloplegic effects; peripheral effects are much weaker than those of atropine; preferred to atropine for diagnostic purposes because its action is more rapid, less prolonged, and is controlled readily by physostigmine; effect is exerted in 15-30 min and passes off in 12-24 h; usually does not produce complete paralysis of accommodation in children. |
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| Adult Dose | 1-2 gtt q3-4h |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; narrow-angle glaucoma |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Regular ophthalmologic examination is required, since possibility of adverse effects on corneal permeability and danger of disruption of corneal epithelium with prolonged or repeated usage of benzalkonium chlorideâ€preserved preparations cannot be excluded; caution when using over an extended period in patients with extensive ocular surface disease; caution in elderly patients (increased intraocular pressure may be present); toxic anticholinergic systemic adverse effects are possible, but incidence is rare when used sparingly (more common in children, especially infants); following administration, compressing lacrimal sac by digital pressure for 1-3 min minimizes systemic absorption; hypersensitivity is not uncommon and appears as conjunctivitis; systemic reactions have followed absorption of anticholinergics from eye drops, particularly in children; mydriatics and cycloplegics may increase intraocular pressure (caution in elderly patients and others in whom an increase may be encountered); tonometric examination prior to drop instillation is advisable |

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| Drug Name | Atropine ophthalmic drops 1% (Isopto, Atropair, Atropisol)- For use as long-acting mydriatic and cycloplegic; most potent ophthalmic parasympatholytic available; by paralyzing sphincter pupillae muscle, helps dilate pupil; also paralyzes ciliary muscle; effect lasts 7-10 d; also indicated to decrease GI motility. |
| Adult Dose | 1 gtt bid |
| Pediatric Dose | 1 gtt bid or apply 1% ointment qd/bid |
| Contraindications | Documented hypersensitivity; narrow-angle glaucoma |
| Interactions | Coadministration with other anticholinergics has additive effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

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| Precautions | Hypersensitivity is not uncommon and occurs as conjunctivitis; systemic toxicity may be produced by instillation of anticholinergic ophthalmic solution, particularly in infants; systemic absorption may be minimized by compressing lacrimal sac for 1-2 min following instillation; may increase intraocular pressure (caution in elderly patients and others in whom an increase may be encountered); tonometric examination prior to drop instillation is advisable; overdosage may cause systemic effects such as ataxia, incoherent speech, restlessness, hallucinations, disorientation, failure to recognize people, and tachycardia; psychotic reactions and behavioral disturbances have been encountered in children; physostigmine salicylate (1-2 mg IV/IM/SC) controls central and peripheral effects; excitement may be controlled by small doses of a short-acting barbiturate such as thiopentone sodium 100 mg |
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GI Antispasmodic/Antimotility Agents

Thought to work centrally by suppressing conduction in vestibular cerebellar pathways. They may have an inhibitory effect on the parasympathetic nervous system.

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| Drug Name | Atropine sulfate injectable (Atropair, Isopto, Atropisol)- Acts at parasympathetic sites in smooth muscle and decreases GI motility. Dosage may require reduction in elderly patients due to possible occurrence of cardiovascular and CNS adverse effects. |
| Adult Dose | 0.3-1.2 mg IV/IM/SC q4-6h if needed |
| Pediatric Dose | Not typically recommended (as antimotility agent) in children |
| Contraindications | Documented hypersensitivity; narrow-angle glaucoma; concomitant acute MI and/or ischemia; thyrotoxicosis; tachycardia; coronary heart disease; congestive heart failure; cardiac arrhythmias; hypertension |
| Interactions | Levodopa, phenothiazine, and agents with cholinergic mechanisms decrease atropine anticholinergic effects; thiazides and amantadine increase atropine anticholinergic effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Overdosage may cause systemic effects such as ataxia, incoherent speech, restlessness, hallucinations, disorientation, failure to recognize people, and tachycardia; Down syndrome or in children with brain damage, because they may demonstrate a hyperreactive response to atropine; psychotic reactions and behavioral disturbances have been encountered in children; physostigmine salicylate (1-2 mg IV/IM/SC) controls central and peripheral effects; excitement may be controlled by small doses of a short-acting barbiturate such as thiopentone sodium 100 mg |

Analgesics

Pain control is essential to quality patient care. Analgesics ensure patient comfort, promote pulmonary toilet, and have sedating properties, which are beneficial for patients who have sustained burns.

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| Drug Name | Morphine sulfate (Duramorph, Astramorph, MS Contin)- DOC for narcotic analgesia because of its reliable and predictable effects, safety profile, and ease of reversibility with naloxone; morphine sulfate administered IV may be dosed in a number of ways and commonly is titrated until desired effect is obtained. |
| Adult Dose | Initial dose: 0.1-0.2 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h Relatively hypovolemic patients: Start with 2 mg IV/IM/SC; reassess hemodynamic effects of dose |
| Pediatric Dose | Neonates and infants <6 months: 0.05-0.1 mg/kg/dose IV/IM/SC q3-4h prn Children: 0.1-0.2 mg/kg/dose IV/IM/SC q3-4h prn |
| Contraindications | Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult |
| Interactions | Phenothiazines may antagonize analgesic effects; TCAs, MAOIs, and other CNS depressants may potentiate adverse effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Avoid in respiratory depression, nausea, emesis, constipation, and urinary retention; caution in atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

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| Drug Name | Meperidine (Demerol)- Narcotic analgesic with multiple actions similar to those of morphine; may produce less constipation, smooth muscle spasm, and depression of cough reflex than similar analgesic doses of morphine. |
| Adult Dose | 25-75 mg PO/IV/IM/SC q3-4h prn |
| Pediatric Dose | 1-1.8 mg/kg (0.5-0.8 mg/lb) PO/IV/IM/SC q3-4h prn; not to exceed adult dose |
| Contraindications | Documented hypersensitivity; concurrent MAOIs; upper airway obstruction or significant respiratory depression; during labor when delivery of premature infant anticipated |
| Interactions | Cimetidine and protease inhibitors may increase toxicity; hydantoins may decrease effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

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| Precautions | Caution in patients with head injuries, since may increase respiratory depression and CSF pressure (use only if absolutely necessary); caution postoperatively and with history of pulmonary disease (suppresses cough reflex); substantially increased dose levels due to tolerance may aggravate or cause seizures even if no history of convulsive disorders; monitor closely for meperidine-induced seizure activity if seizure history |
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| Drug Name | Hydrocodone bitartrate and acetaminophen (Vicodin ES)- Drug combination indicated for relief of moderate to severe pain. |
| Adult Dose | 1-2 tab or cap q4-6h prn for pain |
| Pediatric Dose | <12 years: 10-15 mg/kg/dose acetaminophen q4-6h prn; not to exceed 2.6 g/d of acetaminophen or 5 mg of hydrocodone bitartrate/dose >12 years: 650 mg acetaminophen q4h; not to exceed 5 doses/d acetaminophen or 10 mg of hydrocodone bitartrate/dose |
| Contraindications | Documented hypersensitivity; elevated intracranial pressure |
| Interactions | Phenothiazines may decrease analgesic effects; CNS depressants or TCAs may increase toxicity |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Tablets contain metabisulfite, which may cause hypersensitivity; caution in patients dependent on opiates, since this substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction |

Bronchodilators

Primary action is to decrease muscle tone in both small and large airways in the lungs, thus increasing airflow and ventilation. This category includes beta-adrenergic, methylxanthine, and anticholinergic medications.

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| Drug Name | Albuterol (Proventil, Ventolin)- Bronchodilator in reversible airway obstruction due to asthma; relaxes bronchial smooth muscle by action on beta 2-receptors with little effect on heart rate. |
| Adult Dose | 7.5 mg INH over 60-90 min divided tid; dilute 2.5 mg in 3 mL of saline or use premixed nebulers |
| Pediatric Dose | 0.15 mg/kg INH q20min for 3 doses |
| Contraindications | Documented hypersensitivity |

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| Interactions | Beta-adrenergic blockers antagonize effects; inhaled ipratropium may increase duration of bronchodilatation; cardiovascular effects may increase with MAOIs, inhaled anesthetics, TCAs, or sympathomimetic agents |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hyperthyroidism, diabetes mellitus, sensitivity to sympathomimetic amines, coronary insufficiency, and hypertension; excessive use may result in tolerance; adverse reactions may occur more frequently in children aged 2-5 y |

Antibiotics

Topical and ophthalmic antibiotics routinely are used for dermal and ocular burns, respectively. Injured tissues lose many of their protective mechanisms and are at increased risk of infection.

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| Drug Name | Silver sulfadiazine (Silvadene)- Used topically for dermal burns and useful in prevention of infections from second-degree or third-degree burns; has bactericidal activity against many gram-positive and gram-negative bacteria, including yeast. |
| Adult Dose | Apply to a thickness of 1/16th inch qd/bid; continually cover burned area; remove all previous medication before applying each new dose |
| Pediatric Dose | <2 months: Not recommended (may exacerbate bilirubin toxicity) >2 months: Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Reduces effectiveness of proteolytic enzymes |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Patients with G-6-PD deficiency and renal insufficiency |

Toxoids

Used to induce active immunity against tetanus in selected patients.

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| Drug Name | Tetanus toxoid- Immunizing agents of choice for most adults and children >7 y are tetanus and diphtheria toxoids. Necessary to administer booster doses to maintain tetanus immunity throughout life. Pregnant patients should receive only tetanus toxoid, not a diphtheria antigen-containing product. In children and adults, may administer into deltoid or midlateral thigh muscles. In infants, preferred site of administration is mid thigh laterally. |
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| Adult Dose | Primary immunization: 0.5 mL IM; give 2 injections 4-8 wk apart; third dose 6-12 mo after second injection Booster dose: 0.5 mL q10y |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; history of any type of neurologic symptoms or signs following administration of this product; FDA recommends that elective tetanus immunization be deferred during any outbreak of poliomyelitis because tetanus toxoid injections are an important cause of provocative poliomyelitis |
| Interactions | Patients receiving immunosuppressants, including corticosteroids or radiation therapy, may remain susceptible despite immunization because of poor immune response; cimetidine may enhance or augment delayed-hypersensitivity responses to skin-test antigens; avoid concurrent use with systemic chloramphenicol since it may impair amnestic response to tetanus toxoid; concurrent use of tetanus immune globulin may delay development of active immunity by several days (interaction is nevertheless clinically insignificant and does not preclude concurrent use) |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Do not use to treat actual tetanus infections or for immediate prophylaxis of unimmunized individuals (use instead tetanus antitoxin, preferably human tetanus immune globulin); diminished antibody response to active immunization may be seen in patients receiving immunosuppressive therapy; better to defer primary diphtheria immunization until immunosuppressive therapy discontinued; routine immunization of symptomatic and asymptomatic HIV-infected persons is recommended |

Antitussives

Indicated for control of excessive cough.

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| Drug Name | Guaifenesin and codeine (Robitussin AC, Guaiatuss AC, Mytussin AC, Brontex liq)- Treats minor cough resulting from bronchial and throat irritation. |
| Adult Dose | 5-10 mL PO q4-8h, not to exceed 60 mL/24h |
| Pediatric Dose | 1-1.5 mg/kg codeine/d PO divided qid |
| Contraindications | Documented hypersensitivity |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in renal impairment |

Corticosteroid Creams

Indicated for inflammation of skin.

| | |
|-------------------|---|
| Drug Name | Hydrocortisone 1% (Cortaid, Dermacort, Westcort)- Adrenocorticosteroid derivative suitable for application to skin or external mucous membranes. Has mineralocorticoid and glucocorticoid effects resulting in anti-inflammatory activity. |
| Adult Dose | Apply sparingly to affected areas bid/qid |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; viral, fungal, and bacterial skin infections |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Prolonged use, applying over large surface areas, application of potent steroids, and occlusive dressings may increase systemic absorption of corticosteroids and may cause Cushing syndrome, reversible HPA suppression, hyperglycemia, and glycosuria |

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Follow-up

Further Inpatient Care

- Patients with moderate-to-severe cutaneous effects are best managed in a hospital burn unit. The period of recuperation is much longer than that for thermal burns.
- Those with severe leukopenia require infection isolation.
- Patients with significant pulmonary involvement usually require ICU admission.

Further Outpatient Care

- For 12 hours prior to discharge, observe patients who are exposed to mustard and who are initially asymptomatic.

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Miscellaneous

Medical/Legal Pitfalls

- Failure to make the correct diagnosis

Special Concerns

- Mustard is classified as a mutagen and a carcinogen, but no association between a single exposure and cancer has been proven. Repeated symptomatic exposure over a period of years is established as a casual factor in an increased incidence of upper airway cancers.
-

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CBRNE - Vesicants, Organic Arsenicals: L, Ed, MD, Pd, HI

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Synonyms, Key Words, and Related Terms

chemical warfare, vesicant, blistering agents, lewisite, L, ethyldichloroarsine, ED, methyldichloroarsine, MD, phenyldichloroarsine, PD

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Introduction

Background

Vesicants are a class of chemical weapons named for their ability to cause vesicular skin lesions. The 4 organic arsenicals are lewisite (L), methyldichloroarsine (MD), phenyldichloroarsine (PD), and ethyldichloroarsine (ED). These agents, together with the mustard agents and phosgene oxime, make up

the vesicant class. Although not as well known as the mustards, the organic arsenicals are a group of potent vesicants that medical planners should not overlook.

Interest in organic arsenicals dates back to the mid-19th century. While investigating possible new fumigants, European chemists discovered that chloroarsines (ie, arsenic-chloride compound in which 1 of the chlorine atoms is replaced by an organic radical) tended to be destructive both to insects and to human tissue. With the start of World War I, both sides employed chemists to create chemical warfare (CW) agents. The trench warfare stalemate created a tactical need for a chemical weapon that was both short acting (eg, nonpersistent, volatile) and lethal. To fill this need, German chemists delivered the first weaponized organic arsenical, MD. Two additional organic arsenicals, PD and ED, soon augmented MD. While MD, PD, and ED were being deployed on the battlefields of Europe in 1917 and 1918, a team of American researchers, lead by Captain Lewis of the US Army Medical Corps, was working on the fourth and final organic arsenical. Lewisite, as it was named, never was deployed in World War

I.

Although mustard vesicants have been used in numerous regional wars since 1918, organic arsenical weapons have had limited use. L may have been used by Italy against Ethiopia in 1935 and again by Japan in China from 1937-1944.

Today, arsenicals still are considered a threat, not so much from large nation states but from smaller, less developed nations and/or by terrorist organizations. The relative ease of production coupled with their effectiveness against an unprotected population make organic arsenicals a continued threat in the 21st century.

Pathophysiology

The exact mechanism of biological activity and toxicity of the organic arsenicals is unknown. DNA alkylation and/or inhibition of glutathione-scavenging pathways are 2 postulated mechanisms of action. What is certain is that a blistering reaction occurs on any tissue that an arsenical contacts, whether it is skin, eye, or pulmonary tissue. The onset of symptoms after arsenical exposure occurs in seconds as compared to 4-8 hours for mustard exposure. Either a liquid or vapor (ie, gaseous form of a substance at temperatures below boiling point) can cause toxicity. The organic arsenicals tend to have high volatility at room temperature and thus pose a significant vapor threat to exposed personnel.

Animal data and limited human trials demonstrated that organic arsenicals readily penetrate the skin. Within seconds of contact, the chemical fixes itself to the epidermis and dermis. Pain is immediate. Since the agent penetrates deeper, destruction of subcutaneous tissue results. Protease digestion of anchoring filament at the epidermal-dermal junction occurs. The separation of dermis from epidermis together with capillary leakage causes fluid-filled vesicles.

Vapor contact with the conjunctiva may be the victims' first symptom. Severe conjunctival irritation and

blepharospasm result upon eye contact. More severe exposure can cause loosening of corneal epithelial cells and swelling and edema of the cornea.

The respiratory tract's mucosa and submucosa are susceptible to vapor exposure. Mucosal damage starts in the nose and descends down the respiratory mucosa in a dose-dependent fashion. Immediate pain, lacrimation, and irritation accompany the damage. Damaged respiratory mucosa slough off, filling the airways with debris. Damage to the lung parenchyma causes the secretion of blood and mucous that, with the pseudomembranes, can cause asphyxiation. In animal studies, large doses of L caused this "dry land drowning" within 10 minutes.

The gastrointestinal tract also is susceptible. PD vapor in particular produces a phenyl radical that causes vomiting. Vomiting usually develops within 1-2 minutes after exposure to PD.

The immediate onset of symptoms following exposure makes severe or systemic toxicity to organic arsenical unlikely. However, if a victim does not have protective gear or cannot move out of a contaminated area, prolonged contact may lead to multiorgan involvement. Blood-borne arsenicals can trigger increased permeability of capillaries throughout the body. Leakage of proteins and plasma then can cause third space fluid shifts, hypovolemia, and shock. Intravascular hemolysis of erythrocytes with subsequent hemolytic anemia may result.

Frequency

- **In the US:** Organic arsenical weapons never have been used within the US. Other sources of arsenic poisoning are common, and arsenic ingestion is the most common cause of acute metal poisoning in the US. Inorganic arsenic is found in insecticides, rodenticides, and herbicides and is used in mining and smelting industries.
- **Internationally:** Any nation or terrorist group that has access to a basic pesticide production facility can produce these agents with relative ease. The former Soviet Union is known to have combined sulfur mustard (H) and L into a binary weapon known as HL.

Mortality/Morbidity

Although vesicants have a relatively low mortality rate when compared to other CW agents, survivors usually require prolonged care and rehabilitation. These requirements placed a tremendous burden on the medical infrastructure during World War I.

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Clinical

History

Victims of an L attack may remember observing puddles of a brown liquid or of smelling an odor similar to geranium. MD and ED reportedly smell like rotting fruit. Almost instantaneous pain and irritation of the skin, eyes, and nasal pharynx follow exposure. The patient usually relates a history of trying to remove himself or herself from the noxious stimuli.

- The skin burns and itches. A history of erythema followed by the appearance of vesicles may be obtained.
- Eye pain is severe and is accompanied by blepharospasm and/or photophobia.
- Extreme irritation of the nasal mucosa and upper airway induces coughing and sneezing. Coughing may become productive, and shortness of breath may appear as the irritation of the airway mucosa progresses.
- PD often precipitates severe vomiting within 1-2 minutes after exposure.

Physical

Physical signs of organic arsenical exposure are similar to those of mustard agents. The major difference is the time of onset of signs. Organic arsenicals cause immediate signs, whereas signs of mustard exposure appear after a latent period of several hours.

- An erythematous rash appears within 15-30 minutes. This is followed by the development of fluid-filled vesicles.
 - Vesicles initially are filled with clear fluid and may coalesce to form large bullae.
 - In more severe exposures, the vesicular fluid may take on a yellow and then red color, and a central area of necrosis may form.
 - An L skin lesion has more actual tissue destruction (but less surrounding erythema) than a mustard lesion. Compared to distilled mustard, L is gram-for-gram more toxic. The LD50 (lethal dose for 50% of the population) of L is 2.8 g on the skin.
- Vapor damage to the upper respiratory mucosa causes epistaxis, massive rhinorrhea, and lacrimation.
 - Laryngitis and dysphonia changes result from exposure and can lead to laryngospasm.
 - With larger vapor exposure, destruction of the bronchiolar mucosa and submucosa causes pseudomembrane formation and obstruction.

Causes

Organic arsenical exposure can be caused by military CW attack or potentially from terrorist incident.

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Differentials

CBRNE - Irritants: Cs, Cn, Cnc, Ca, Cr, Cnb, PS

CBRNE - Lung-Damaging Agents, Phosgene

CBRNE - Vesicants, Mustard: Hd, Hn1-3, H

Other Problems to be Considered

Exposure to plants (eg, poison ivy) or adverse drug reactions can cause the same skin changes as those found in organic arsenical exposure. Mustard gas causes the same signs and symptoms; however, a latent period of several hours occurs between exposure and symptoms. The eye and respiratory irritation produced by organic arsenical vapor is similar to that of the choking agents (eg, phosgene) or riot control agents (eg, CS).

Maintain a high index of suspicion to make the diagnosis of organic arsenical exposure. Diagnosis in solitary cases is difficult.

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Workup

Lab Studies

- The US military and the North Atlantic Treaty Organization (NATO) have developed several field tests to detect various CW agents. The field tests that reliably detect L and the other organic arsenicals include the M256A1, individual chemical agent detector (ICAD), miniature chemical agent monitor (MINICAMS), M18A2, M21, M90, M93A1 Fox, chemical agent monitor (CAM), and depot area air monitoring system (DAAMS). Less sophisticated means of detection are M8 detection paper (turns red with L) and M9 paper (turns color when exposed to arsenicals).
- With exception of urinary arsenic excretion, no specific tests exist for organic arsenical exposure. Leukocytosis and other nonspecific markers of tissue destruction may appear.
- Culture damaged skin routinely to stave off opportunistic skin infections.
- Also perform sputum Gram stain and culture if the respiratory system is affected.

Imaging Studies

- Pneumonia commonly follows pulmonary damage in 3-5 days. Obtain a chest x-ray as indicated.

Procedures

- Massive tissue damage to the respiratory mucosa can cause acute airway compromise from laryngospasm and/or necrotic debris. Emergent endotracheal intubation may be required. Obtain a bronchoscopy consultation if pseudomembrane formation is suggested.

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Treatment

Prehospital Care

- The top 2 priorities are protecting the caregiver and removing the offending agent from the casualty as quickly as possible. Then assess airway, breathing, and circulation (ABCs) as usual.
 - All medical personnel who may come into contact with vesicant vapor or liquid should wear protective gear.
 - The activated charcoal in the chemical protective mask adequately adsorbs these agents.
 - Protective boots, gloves, pants, and jacket (eg, mission-oriented protective posture [MOPP] gear) protect the skin; however, organic arsenicals attack rubber and can cause it to break down with prolonged exposure. This is especially true of L.

Emergency Department Care

- As with any chemical disaster, the emergency department's disaster plan should have a system in place to efficiently triage contaminated patients.
 - After assessing for life-threatening conditions, additionally decontaminate patients in the triage area as indicated.
 - Once in the emergency department, reassess ABCs as usual.
 - Assessment of volume status is a must. Patients who have been in hot protective gear are predisposed to volume depletion and hyperthermia. Correction of fluid and/or electrolyte abnormalities is essential in these patients.
 - Once a victim has been undressed and fully decontaminated, no danger to the caretakers remains.
- Denude fluid-filled vesicles larger than 2 cm and irrigate them with sterile saline. Apply topical antibiotics such as silver sulfadiazine. Intense pain and itching may require systemic analgesics and antipruritics.
- Upper respiratory symptoms can be alleviated with humidified oxygen and cough suppressants. Reserve antibiotics for patients with pulmonary damage who develop fever. Specific antimicrobial therapy then is based on Gram stain and cultures.

- Eyes may be irrigated with normal saline, followed by application of topical antibiotics. Petroleum jelly can be applied to the edges of the lids to prevent them from sticking together.

Consultations

- Consultation can be made to various departments (eg, dermatology, ophthalmology) as needed.
- A 24-hour hotline regarding CW agents is available at 800-424-8802.
- Help also can be obtained by calling the US Army Medical Research Institute of Chemical Defense at 410-436-3628.

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Medication

In addition to the medications mentioned in the preceding section, the organic arsenicals have an antidote in the form of British antilewisite agent (BAL, dimercaprol). BAL is a chelating agent that was developed at the end of World War I specifically to treat L casualties.

Chelating Agents

As a chelating agent, BAL binds to the arsenic moiety, thereby preventing or reversing its binding to tissue enzymes. The BAL-arsenic moiety then is excreted renally. Dimercaprol currently is used as a chelating agent for heavy metals such as arsenic, gold, and mercury.

| | |
|-------------------|--|
| Drug Name | Dimercaprol (BAL in oil)- Packaged in 3-mL ampules with 100 mg/mL. Formerly supplied as an ophthalmic and dermatologic ointment during World War II; these preparations are no longer available. |
| Adult Dose | 4-5 mg/kg (approximately 1 mL/50 lb) IM, not to exceed 4 mL; repeat q4h for a total of 4 doses; in severe exposure, additional doses can be administered as 2 mg/kg qd for 3-4 d |
| Pediatric Dose | Administer as in adults |
| Contraindications | In pregnant women, only use in life-threatening toxicities |
| Interactions | None reported |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|---|
| Precautions | High incidence of adverse effects; 4 mg/kg dose is estimated to have a 14% chance of adverse reaction; reactions can include nausea, vomiting, burning sensation of mouth and eyes, lacrimation, rhinorrhea, salivation, burning of the extremities, dental pain, chest pain, anxiety, agitation, hypertension, and tachycardia; adverse effects usually peak 10-30 min after administration and usually subside within 1 h; treatment for BAL overdose is supportive |
|-------------|---|

Analgesics

Pain is common and can be severe.

| | |
|-------------------|---|
| Drug Name | Morphine sulphate (Duramorph, Astramorph, MS Contin)- Mechanism of action is via the opiate receptors. |
| Adult Dose | Starting dose: 0.1 mg/kg IV/IM/SC Maintenance dose: 5-20 mg/70 kg IV/IM/SC q4h |
| Pediatric Dose | Administer as in adults using weight-based dosing |
| Contraindications | Documented hypersensitivity; respiratory depression |
| Interactions | Phenothiazines may antagonize analgesic effects of opiate agonists; TCAs, MAOIs, and other CNS depressants may potentiate adverse effects of morphine |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in hypotension, respiratory depression, nausea, emesis, constipation, urinary retention, atrial flutter, and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate |

Antipruritic Agents

Cutaneous exposure can produce severe pruritus.

| | |
|-------------------|--|
| Drug Name | Diphenhydramine (Benlyn, Benadryl)- For symptomatic relief of symptoms caused by release of histamine in allergic reactions. |
| Adult Dose | 25-50 mg PO qid 10-50 mg IV, not to exceed 400 mg/d |
| Pediatric Dose | 5 mg/kg/d PO/IV divided qid |
| Contraindications | Documented hypersensitivity; neonates |
| Interactions | Potentiates effect of CNS depressants; due to alcohol content, do not give syrup dosage form to patient taking medications that can cause disulfiramlike reactions |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |

| | |
|-------------|--|
| Precautions | May exacerbate angle-closure glaucoma, hyperthyroidism, peptic ulcer, or urinary tract obstruction; xerostomia may occur |
|-------------|--|

Antibiotics

Cutaneous damage caused by vesicants can leave the victim susceptible to bacterial infection.

| | |
|-------------------|---|
| Drug Name | Silver sulfadiazine 1% (Silvadene)- Useful in prevention of infections. Has bactericidal activity against many gram-positive and gram-negative bacteria, including yeast. |
| Adult Dose | Apply 1/16-inch thin film of ointment to cleansed and debrided burn wound bid; reapply if removed accidentally |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity; neonates <2 mo and late pregnancy |
| Interactions | Effect of proteolytic enzymes is reduced when used concomitantly with this product; leukopenia increased with concomitant cimetidine |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | G-6-PD deficiency and impaired renal or hepatic function; not recommended in nursing mothers |

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Follow-up

Further Inpatient Care

- Victims of severe toxicity may require prolonged care for dermatologic, ophthalmic, and pulmonary sequelae.

Further Outpatient Care

- Patients with only minor skin and eye lesions can be discharged with outpatient follow-up care.

Complications

- Permanent discoloration can occur at blister sites, especially with PD.

- Patients with mild conjunctival irritation can be expected to fully recover in 1-2 weeks. Patients with moderate conjunctival irritation with mild corneal damage can expect recovery in 4-6 weeks, but severe corneal damage may be irreversible, especially in PD exposure.
- Victims who suffered mild-to-moderate pulmonary exposure can expect full recovery in 1-4 weeks. Severe vapor exposure can lead to permanent damage to the respiratory mucosa. This can lead to an increased risk of future infection and/or neoplasia. Secondary bacterial pneumonia is most common 3-6 days postexposure.
- Victims with severe exposure to an organic arsenical can experience long-term complications in a number of organ systems, including neurologic, endocrine, and thermoregulatory disorders. The exact mechanism of these effects is unknown.

Prognosis

- Prognosis is good in all but the most severe cases.

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Miscellaneous

Medical/Legal Pitfalls

- The use of BAL as an antidote for organic arsenical toxicity is sound medicolegal practice.
-

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CBRNE - Viral Hemorrhagic Fevers

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Synonyms, Key Words, and Related Terms

VHF

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Introduction

Background

Viral hemorrhagic fevers (VHFs) are a group of febrile illnesses caused by RNA viruses from several viral families. These highly infectious viruses lead to a potentially lethal disease syndrome characterized by fever, malaise, vomiting, mucosal and gastrointestinal (GI) bleeding, edema, and hypotension. The 4 viral families known to cause VHF disease in humans include the Arenaviridae, Bunyaviridae, Filoviridae, and Flaviviridae. General characteristics of these viral families can be found in the table

below.

Table 1. Viral Families Causing Viral Hemorrhagic Fever

| Virus Family | Disease (Virus) | Natural Distribution | Usual Source of Human Infection | Incubation (Days) |
|---------------------|--|--------------------------------|---------------------------------|---------------------------------------|
| Arenaviridae | | | | |
| Arenavirus | Lassa fever | Africa | Rodent | 5-16 |
| | Argentine HF (Junin) | South America | Rodent | 7-14 |
| | Bolivian HF (Machupo) | South America | Rodent | 9-15 |
| | Brazilian HF (Sabia) | South America | Rodent | 7-14 |
| | Venezuelan HF (Guanarito) | South America | Rodent | 7-14 |
| Bunyaviridae | | | | |
| Phlebovirus | Rift Valley fever | Africa | Mosquito | 2-5 |
| Nairovirus | Crimean-Congo HF | Europe, Asia, Africa | Tick | 3-12 |
| Hantavirus | Hemorrhagic fever with renal syndrome, hantavirus pulmonary syndrome | Asia, Europe, worldwide | Rodent | 9-35 |
| Filoviridae | | | | |
| Filovirus | Marburg and Ebola | Africa | Unknown | 3-16 |
| Flaviviridae | | | | |
| Flavivirus | Yellow fever | Tropical Africa, South America | Mosquito | 3-6 |
| | Dengue HF | Asia, Americas, Africa | Mosquito | Unknown for dengue HF, 3-5 for dengue |

Arenaviridae

Arenaviridae are spread to humans by rodent contact and include Lassa virus in Africa and several rare South American hemorrhagic fevers such as Machupo, Junin, Guanarito, and Sabia. Lassa virus is the most clinically significant of the Arenaviridae, accounting for serious morbidity and mortality in West Africa.

Lassa fever first appeared in Lassa, Nigeria, in 1969. It has been found in all countries of West Africa and

is a significant public health problem in endemic areas. In populations studied, Lassa fever accounts for 5-14% of hospitalized febrile illnesses. Its natural reservoir is a small rodent whose virus-containing excreta is the source of transmission.

Bunyaviridae

This group includes Rift Valley fever (RVF) virus, Crimean-Congo hemorrhagic fever (CCHF) virus, and several hantaviruses. The RVF and CCHF viruses are both arthropod-borne viruses. RVF virus, an important African pathogen, is transmitted to humans and livestock by mosquitos and by the slaughter of infected livestock. CCHF virus is carried by ticks and causes a fulminant, highly pathogenic form of VHF notable for aerosol transmission of infective particles. Outbreaks of CCHF have occurred in Africa, Asia, and Europe.

Many hantaviruses are spread worldwide, causing 2 major syndromes: hemorrhagic fever with renal syndrome (HFRS) and hantavirus pulmonary syndrome (HPS). They are divided into Old World hantaviruses (such as the prototypical Hantaan virus of Korea), which generally cause HFRS, and New World hantaviruses, causing HPS. Rodents carry both types. A previously undiscovered Hantavirus, Sin Nombre virus, was the cause of an outbreak of highly lethal HPS in the southwestern US in 1993.

Filoviridae

The most notorious of the VHF viruses, including Ebola and Marburg viruses, belong to the Filoviridae family. Ebola virus first was described in 1976 after outbreaks of a febrile, rapidly fatal hemorrhagic illness were reported along the Ebola River in Zaire (now the Democratic Republic of the Congo) and Sudan. Sporadic outbreaks have continued since that time, usually in isolated areas of central Africa. An outbreak in Kikwit, Zaire, in 1995 led to 317 confirmed cases, with an 81% mortality rate. Two thirds of the patients were among health care workers caring for infected individuals. A recent outbreak in Uganda has claimed over 150 lives, with similar demographics. Ebola has 4 distinct subtypes: Ebola-Zaire, Ebola-Sudan, Ebola-Ivory Coast, and Ebola-Reston, a form that causes illness only in nonhuman primates. The natural reservoir of Ebola virus remains unknown.

Marburg virus, named after the German town where it first was reported in 1967, is another highly pathogenic member of the Filoviridae family that is traced to central Africa. As in Ebola, the natural host for the virus is unknown. Marburg virus was contracted by a traveler to central Africa in 1987 and has been endemic since 1998 in Durba, Democratic Republic of the Congo, and in persons exposed in gold mines.

Flaviviridae

Yellow fever and dengue fever are the most well known diseases caused by flaviviruses. Both are mosquito-borne; yellow fever is found in tropical Africa and South America, and dengue fever is found in Asia, Africa, and the Americas. They are notable for their significant effect on prior military campaigns

and their continued presence throughout endemic areas.

Pathophysiology

The primary defect in patients with VHF is that of increased vascular permeability. Hemorrhagic fever viruses have an affinity for the vascular system, leading initially to signs such as flushing, conjunctival injection, and petechial hemorrhages, usually associated with fever and myalgias. Later, frank mucous membrane hemorrhage may occur, with accompanying hypotension, shock, and circulatory collapse. The relative severity of the clinical presentation may vary depending on the virus in question, amount, and route of exposure.

In acute disease, patients are extremely viremic, and messenger ribonucleic acid (mRNA) evidence of multiple cytokine activation exists. In vitro studies reveal these cytokines lead to shock and increased vascular permeability, the basic pathophysiologic processes most often seen in VHF infection. Another prominent pathologic feature is pronounced macrophage involvement. Inadequate or delayed immune response to these novel viral antigens may lead to rapid development of overwhelming viremia. Extensive infection and necrosis of affected organs also are described. Hemorrhagic complications are multifactorial and are related to hepatic damage, consumptive coagulopathy, and primary marrow injury to megakaryocytes. Aerosol transmission of some VHF infections is reported among nonhuman primates and likely is a mode of transmission in patients with severe infection.

Multisystem organ failure affecting the hematopoietic, neurologic, and pulmonary systems often accompanies the vascular involvement. Hepatic involvement varies with the infecting organism and is at times seen with Ebola, Marburg, RVF, CCHF, and yellow fever. Renal failure with oliguria is a prominent feature of HFRS seen in Hantavirus infection and may be seen in other VHFs as intravascular volume depletion becomes more pronounced. Bleeding complications are particularly prominent with Ebola, Marburg, CCHF, and the South American arenaviruses.

Frequency

- **In the US:** Cases of VHF in the US are extremely rare and usually are found in patients who recently have visited endemic areas or among those with potential occupational exposure to hemorrhagic fever viruses. Lassa fever has been reported in the US in travelers from West Africa and last was reported in the US in 1989. In 1994, a virologist working with Sabia, a Brazilian HF virus, accidentally contracted the disease. No human cases of Ebola or Marburg virus disease have been reported in the US. In 1989, an outbreak of hemorrhagic fever among imported macaque monkeys in Reston, Virginia, led to the discovery of Ebola-Reston, a variant of Ebola virus that originated in the Philippines and does not cause disease in humans.
- **Internationally:** Lassa fever is responsible for an estimated 100,000-300,000 infections per year, with 5,000 deaths. Cases have been reported throughout West Africa, particularly in Nigeria, Sierra Leone, Guinea, and Liberia. Other arenaviruses are responsible for sporadic VHF outbreaks throughout South America.

RVF and CCHF are responsible for intermittent epidemics in Africa (for RVF) and in areas of Africa, Asia, and Europe (for CCHF). HFRS due to Hantavirus infection continues to be an ongoing health concern, particularly in Asia, affecting up to 200,000 patients annually.

Ebola virus appears sporadically in endemic areas of the former Zaire and Sudan. Ebola virus also has been reported in Gabon, the Ivory Coast, and Uganda. Outbreaks appear to propagate in hospital settings, often involving health care providers. Marburg has been identified in humans only 5 times: 4 times in Africa and once in Germany and Yugoslavia.

Yellow fever continues to be a serious problem in tropical areas of South America and Africa, where vaccination is not widespread. The World Health Organization estimates that approximately 200,000 cases per year occur in Africa. Dengue HF is endemic in Southeast Asia, and more than 1 million cases occur annually.

Mortality/Morbidity

Case fatality rates of patients with VHF vary from less than 10% (eg, in dengue HF) to approximately 90%, as has been reported in patients with Ebola-Zaire. The most recent outbreak of Ebola-Sudan in Uganda had a 50% case fatality rate.

Complications from VHF infection include retinitis, orchitis, hepatitis, transverse myelitis, and uveitis. In patients who recover from Lassa fever infection, deafness is the most common complication. Spontaneous abortion also is common. Renal insufficiency is associated with HFRS infection.

Race

No racial predilection has been identified, although cases have originated in African areas.

Sex

No predilection for either sex has been identified.

Age

VHF affects all ages according to exposure and local demographics.

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Clinical

History

- Obtain a detailed travel history, paying particular attention to recent travel to tropical or rural areas, such as Central or South America (yellow fever, arenaviruses), West Africa (Lassa fever), or to endemic portions of Central Africa (Ebola, Marburg, RVF, CCHF). Ask about contact with potential arthropod or rodent reservoirs.
 - Since the natural reservoir for Ebola and Marburg viruses is unknown, contact with infected monkeys or humans is not a prerequisite for transmission of infection.
 - Direct contact with rodents infected with hemorrhagic fever viruses (eg, arenaviruses, hantaviruses) is not necessary for transmission of infection, since aerosolized excreta may transmit infection.
- Because of their extreme pathogenicity and potential for transmission by fine particle aerosol, VHF viruses are considered potential biological warfare agents. In addition, Dr Ken Alibek, the former Deputy Director of the once massive Soviet bioweapons program, Biopreparat, claims Soviet scientists successfully had produced a stable Marburg virus biological weapon that could be delivered as an aerosol.
 - Large numbers of military personnel with VHF symptoms suggest such an attack.
 - An outbreak of VHF in a nonendemic area also suggests a biological warfare attack.
- The initial symptoms correspond to development of viremia and include the following:
 - High fever
 - Headache
 - Fatigue
 - Abdominal pain
 - Myalgias
 - Prostration

Physical

Depending on the progress of the disease, patients with VHF initially may present with minimal signs, suggesting a more benign viral syndrome. Maintain a high index of suspicion. As the disease progresses, more classic findings are present as follows:

- Fever
- Pharyngitis
- Conjunctival injection
- Nondependent edema
- Petechial or ecchymotic rash
- GI bleeding
- Hypotension and/or shock

- Most hemorrhagic fevers, except Rift Valley Fever, can produce a variety of cutaneous findings that are principally caused by vascular instability and bleeding abnormalities. Such findings include flushing, petechiae, purpura, ecchymoses, and edema.
- The Old World arenavirus causing Lassa fever results in the greatest amount of edema of any of the hemorrhagic fever viruses. Additionally, no bleeding abnormalities are present. The New World arenaviruses (Junin, Machupo, Sabia, and Guanarito) cause less edema and variable amounts of petechiae, purpura, ecchymoses, palatal hyperemia, and mucosal hemorrhage.
- The greatly feared Filoviruses (Marburg and Ebola) exhibit fairly characteristic exanths that are best seen in fair-skinned patients. Soft palatal hyperemia accompanies the flu-like prodrome and is followed between days 5 and 7 by a nonpruritic, centripetal, pinhead-sized papular, erythematous exanthem. Within 24 hours, this can develop into large and coalescent, well-demarcated, sometimes hemorrhagic macules and papules. In severe cases, hemorrhage exudes from mucous membranes, venipuncture sites, and body orifices.
- Dengue virus causes a characteristic erythematous exanthem with striking islands of sparing.

Causes

- See [Table 1](#).

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Differentials

Disseminated Intravascular Coagulation

Hemolytic Uremic Syndrome

Leptospirosis

Malaria

Salmonella Infection

Systemic Lupus Erythematosus

Thrombocytopenic Purpura

Other Problems to be Considered

Typhoid fever

Shigellosis

Meningococemia

Rickettsial infections

Acute leukemia

Idiopathic or thrombotic thrombocytopenic purpura

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Workup

Lab Studies

- Because of risks associated with handling infectious materials, perform the minimum necessary laboratory testing for diagnostic evaluation and patient care.
- A complete blood count often indicates leukopenia and thrombocytopenia. These findings may not be present in Lassa fever.
- Elevated hepatic transaminases are observed in VHF and are predictive of high mortality in Lassa fever infection.
- Prothrombin time, activated partial thromboplastin time, international normalized ratio, and clotting times are prolonged.
- A disseminated intravascular coagulation profile including fibrinogen level, fibrin degradation products, and platelet count may be useful.

Other Tests

- Most patients are viremic at the time of presentation (Hantavirus is an exception). Specific viral diagnosis can be made using serologic tests, including enzyme-linked immunosorbent assay (ELISA) and polymerase chain reaction. Difficult cases may require tissue cultures.
 - Because of the need for specialized microbiologic containment and handling of these viruses, initiate contact with the Centers for Disease Control and Prevention (CDC; Atlanta, GA) as soon as possible and prior to transport of specimens for virus-specific diagnosis. Specific state and federal statutes govern the shipment of highly infectious disease agents.
 - The CDC and the US Army Medical Research Institute for Infectious Diseases (USAMRIID; Frederick, MD) are the only 2 Biosafety Level 4 (BSL-4) laboratory facilities in the US with such diagnostic facilities.

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Treatment

Prehospital Care

Supportive care is based on the patient's physiologic condition. Because most patients requiring prehospital evaluation and transport are in the early stages of the disease, universal precautions should be adequate. In patients with respiratory symptoms (eg, cough, rhinitis), use face shields and high-efficiency particulate air (HEPA) filter masks.

Emergency Department Care

- Fluid resuscitation and supportive care are the mainstays of emergency department therapy. Intravenous crystalloids, oxygen, and cardiac monitoring are the most appropriate initial steps in the treatment of patients in whom VHF is suggested.
- Administer blood and blood products as clinically indicated.
- Avoid intramuscular injections and the use of aspirin or other anticoagulants.
- Minimize invasive procedures because of the risk associated with viral transmission from sharp objects.
- Place patients in a private room.
 - A negative pressure room is not necessary during early stages of the disease but may be necessary if patients have prominent cough, vomiting, diarrhea, or hemorrhage.
 - Prevent nonessential staff and visitors from entering the room.
- Persons coming within 3 feet of the patient should wear face shields or surgical masks with eye protection (including side shields). Use HEPA filter masks if patients have prominent respiratory, GI, or hemorrhagic symptoms.
- If large amounts of blood or other body fluids are present in the environment, use leg and shoe coverings.
- Before exiting the room, discard all used protective barriers and clean shoes with a hospital disinfectant or solution of household bleach. If possible, use an anteroom for putting on and removing protective barriers and for storing supplies.

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Medication

No specific antiviral therapy is available for Ebola or Marburg virus infection. The use of convalescent serum (ie, sera from patients who have survived infection) is suggested as a possible therapy. Late during

the 1995 Kikwit, Zaire, outbreak, 8 Ebola patients received blood transfusions from Ebola survivors. Of these, 7 survived. However, no clear evidence exists that links their survival directly to this therapy.

Lassa fever and HFRS due to Hantavirus infection have been treated effectively with IV and oral ribavirin. Because of this, ribavirin has been recommended as a potential treatment for other arenaviruses and bunyaviruses. Treatment is most effective when given early in the clinical course. Ribavirin also is recommended for postexposure prophylaxis.

Development of a Lassa virus vaccine is underway at CDC. Yellow fever vaccine is readily available and is both safe and effective. Argentine HF (Junin) vaccine is also effective and may protect against Bolivian HF as well. RVF and Hantaan (HFRS) vaccines are also available. Recently, a vaccine demonstrated effectiveness against Ebola virus in nonhuman primates. No human vaccine is available.

Antivirals

The goals in the use of antivirals are to shorten the clinical course, prevent complications, prevent the development of latency and/or subsequent recurrences, decrease transmission, and eliminate established latency.

| | |
|-------------------|--|
| Drug Name | Ribavirin (Virazole)- Nucleoside analog with antiviral activity; may significantly reduce mortality in Lassa fever and Hantavirus infection if treatment begun within 6 d of onset. |
| Adult Dose | Loading dose: 30 mg/kg IV, followed by 15 mg/kg q6h for 4 d and 7.5 mg/kg q8h for 6 d |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | Decreases zidovudine effects |
| Pregnancy | X - Contraindicated in pregnancy |
| Precautions | Mild hemolysis and suppression of erythropoiesis reported after ribavirin therapy (both are reversible); closely monitor patients with COPD and asthma for deterioration of respiratory function |

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Follow-up

Further Inpatient Care

- Hospitalize patients with suspected or confirmed VHF infection because of the significant risk for nosocomial spread of the infection.
- Notification of local and state public health departments and CDC may provide resources for further epidemiologic investigation into the source of the infection.
- Appropriate barrier precautions should remain in place throughout the hospital course because of the highly pathogenic nature of VHF infection and because various causes of VHF often are clinically indistinguishable.

Deterrence/Prevention

- As the natural reservoirs for Ebola and Marburg virus infection remain unknown, no specific prevention measures are established.
- Efforts are underway in West Africa to educate people in high-risk areas about ways to decrease rodent populations, thereby reducing transmission of Lassa fever.
- Strict barrier precautions in the treatment of patients with known or suspected VHF infection reduce nosocomial transmission.

Complications

- Complications from VHF infection include retinitis, orchitis, encephalitis, hepatitis, transverse myelitis, and uveitis.
- In patients who recover from Lassa fever infection, deafness is the most common complication. Spontaneous abortion also is common.
- Renal insufficiency is associated with HFRS infection.

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Miscellaneous

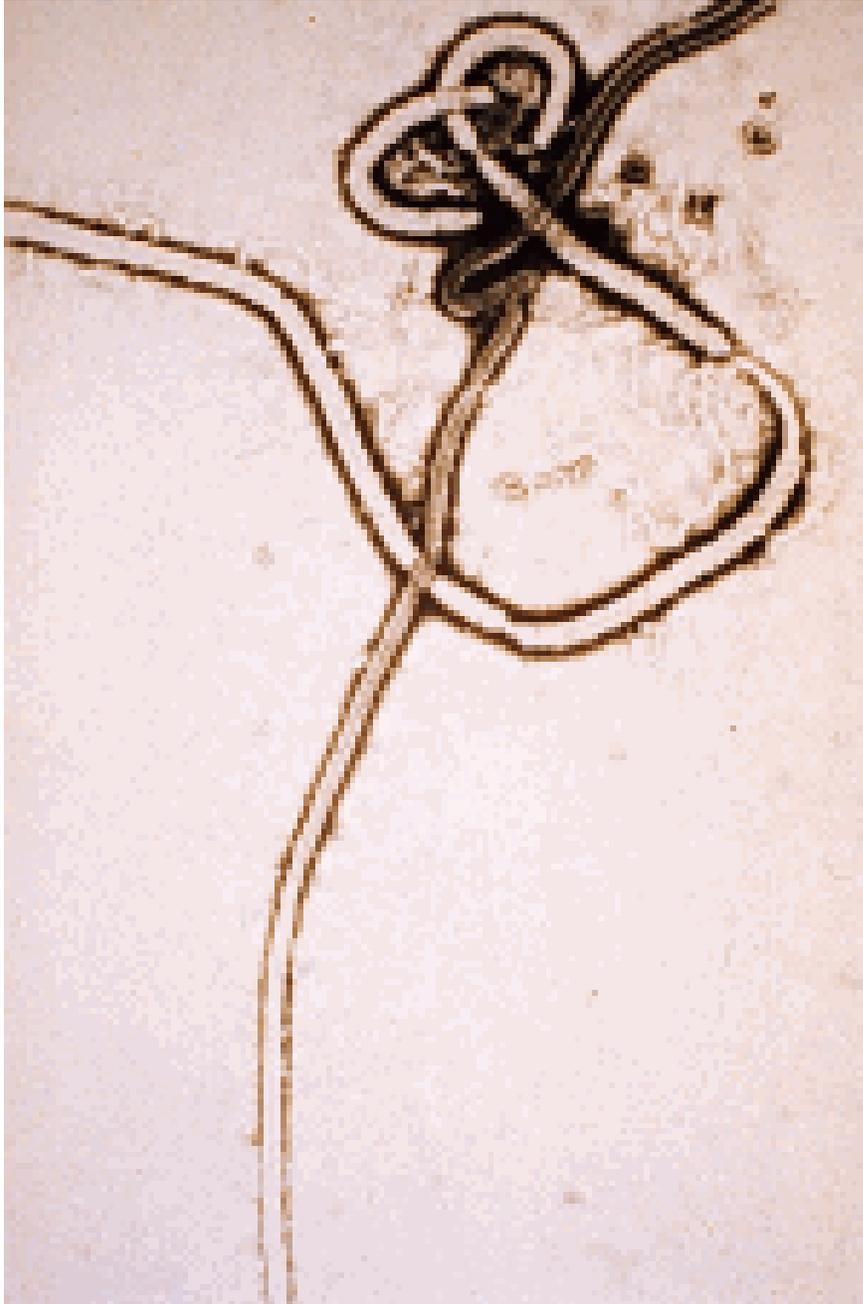
Medical/Legal Pitfalls

- Failure to admit patients for supportive therapy and definitive diagnosis
- Failure to maintain appropriate isolation precautions or failure to prevent unnecessary patient-staff contacts
- Failure to exclude other possible diagnoses that may require specific therapies

Special Concerns

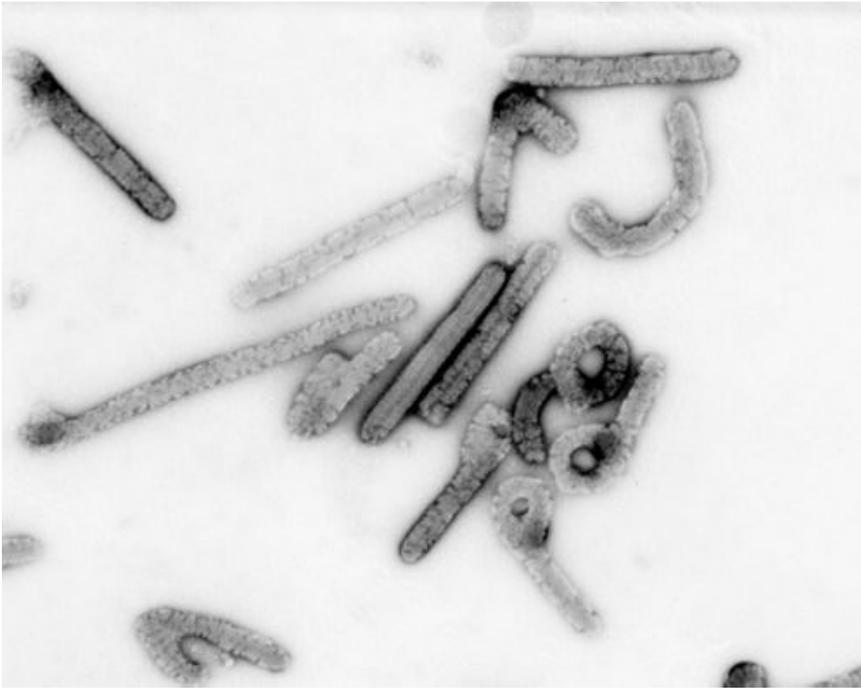
- CDC Fact Sheets on VHF are available online at <http://www.cdc.gov/ncidod/dvrd/spb/mnpages/factmenu.htm>.

Pictures



Picture 1: Ebola virus (Electron micrograph courtesy of the Centers for Disease Control and Prevention.)

Picture type: Photo



Picture 2: Marburg virus (Negative stain image courtesy of the Centers for Disease Control and Prevention.)

Picture type: Photo



Picture 3: Mastomys rodent, natural host of Lassa virus (Image courtesy of the Centers for Disease Control and Prevention.)

Picture type: Photo



Picture 4: Site of Lassa Fever outbreak in Liberia, 1988. (Courtesy of Peter B Jahrling, PhD)

Picture type: Photo





Picture 5: Old World Arenavirus - Lassa Fever. Patient with Lassa fever during an outbreak in Liberia in 1988. Note the periorbital edema and conjunctival hemorrhage. Lassa fever patients typically exhibit significant edema without hemorrhagic manifestations (Reprinted with permission from Atlas of Infectious Disease Volume VII: External manifestations of systemic infections. Mandell GL, Fekety R, editors. Viral hemorrhagic fevers. Chapter 10. Peters CJ, Zaki SR, Rollin PE. Churchill-Livinstone. Philadelphia. 1997. Figure 10-26B, page 10.15). Contact Current Medicine, 400 Market Street South, Suite 700, Philadelphia, PA 19106, 215-574-2266.

Picture type: Photo



Picture 6: The fertile Pampas of Argentina, site of Argentine Hemorrhagic Fever due to Junin virus. (Courtesy of Peter B Jahrling, PhD)

Picture type: Photo



Picture 7: New World Arenavirus - Junin. (a) Conjunctival hemorrhage in patient with Argentine Hemorrhagic Fever caused by Junin virus. (Photo Courtesy of Peter B. Jahrling PhD) Contact Current Medicine, 400 Market Street South, Suite 700, Philadelphia, PA 19106, 215-574-2266.

Picture type: Photo



Picture 8: New World Arenavirus - Junin. Gingival bleeding in a patient with Argentine Hemorrhagic Fever (Reprinted with permission from Atlas of Infectious Diseases Volume VII: External manifestations

of system infections. Mandell GL, Fekety R, editors. Viral hemorrhagic fevers. Chapter 10. Peters CJ, Zaki SR, Rollin PE. Churchill-Livingstone. Philadelphia. 1997. Figure 10-28, Page 10.14. Photo courtesy of D. Enria, MD). Contact Current Medicine, 400 Market Street South, Suite 700, Philadelphia, PA 19106, 215-574-2266.

Picture type: Photo



Picture 9: Site of Sabia virus infections in Brazil. (Courtesy of Peter B Jahrling, PhD)

Picture type: Photo



Picture 10: New World Arenavirus - Machupo. Oral mucosal hyperemia and hemorrhage in a patient with Bolivian Hemorrhagic Fever caused by Machupo virus (Courtesy of C.J. Peters, MD).

Picture type: Photo



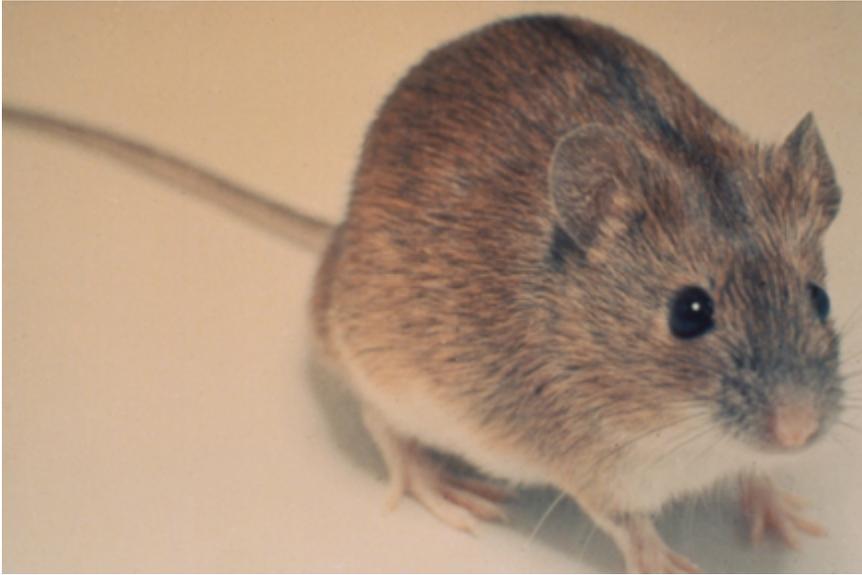
Picture 11: Bunyavirus infection - CCHF Virus. Ecchymoses encompassing left upper extremity one week after onset of CCHF. Ecchymoses are often accompanied by hemorrhage in other locations: epistaxis, puncture sites, Hematemesis, melena, and hematuria. Reprinted from Jahrling PB. Viral Hemorrhagic Fevers. Chapter 29 In: Sidell Fr, Takafuji ET, Franz DR, eds. Medical Aspects of Chemical and Biological Warfare. In: Zajtcuk R, Bellamy RF, eds. Textbook of Military Medicine. Washington, DC: US Department of the Army, Office of the Surgeon General, and Borden Institute; 1997: 595. Photo courtesy Robert Swaneopoel, PhD, DTVM, MRCVS, National Institute of Virology, Sandringham, South Africa.) Government publication, no permission needed, just give credit. See example on fax enclosed from Borden Institute.

Picture type: Photo



Picture 12: Bunyavirus Infection - CCHF Virus. Ecchymoses complicating CCHF (Reprinted with permission from Atlas of Infectious Diseases Colume VIII: External manifestations of systemic infections. Mandell GL, Fekety R, editors. Viral hemorrhagic fevers. Chapter 10. Peters CJ, Zaki SR, Rollin PE, Churchill-Livingstone. Philadelphia. 1997. Figure 10-27, Page 10.14. Photo courtesy of D.I.H. Simpson, MD). Contact Current Medicine, 400 Market Street South, Suite 700, Philadelphia, PA 19106, 215-574-2266.

Picture type: Photo



Picture 13: Apodemus asgrarius, the vector of Korean Hemorrhagic Fever caused by a hantavirus. (Courtesy of David McClain, MD).

Picture type: Photo



Picture 14: Bunyavirus Infection - Hantaan Virus. Patient with Korean Hemorrhagic Fever caused by Hantaan virus demonstrating typical 'sunburn flush' of cheeks, chin, and base of neck. (Courtesy of John Huggins, PhD)

Picture type: Photo



Picture 15: Bunyavirus Infection - Another patient with Korean Hemorrhagic Fever demonstrating conjunctival hemorrhages, facial petechiae, and 'sunburn flush' of cheeks. (Courtesy of John Huggins, PhD)

Picture type: Photo



Picture 16: Charcoal pit in Kikwit, Zaire (Democratic Republic of the Congo), site of 1995 Ebola fever outbreak. (Courtesy of Peter B Jahrling, PhD)

Picture type: Photo



Picture 17: Filovirus disease - Ebola Fever. Patient with Ebola hemorrhagic fever during 1976 outbreak in Zaire demonstrating palatal petechiae and hemorrhage (Courtesy of Joel Breman)

Picture type: Photo



Picture 18: Patient with morbilliform exanthem of dengue fever. Note islands of sparing characteristic for dengue. (Courtesy Duane Gubler, PhD)

Picture type: Photo



Picture 19: Patient with dengue hemorrhagic fever complicated by ecchymoses (Courtesy of Duane Gubler, PhD)

Picture type: Photo

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CBRNE - Vomiting Agents: Dm, Da, Dc

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Synonyms, Key Words, and Related Terms

vomiting agents, DM, diphenylaminearsine, adamsite, 10-Chloro-5,10-dihydrophenarsazine, DA, diphenylchlorarsine, diphenylarsinous chloride, DC, diphenylcyanoarsine, diphenylarsinous cyanide

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Introduction

Background

The chemical warfare agents diphenylchlorarsine (DA), diphenylcyanoarsine (DC), and diphenylaminearsine (DM, adamsite) belong to a group of chemicals classified as vomiting agents. DA appears as colorless crystals, DC as a white solid, and DM as light yellow-to-green crystals. DA and DM are odorless, and DC reportedly has an odor similar to garlic or bitter almonds. All 3 agents are insoluble in water.

The synthesis of these agents dates back to the early 20th century. In 1915, Wieland, a German chemist, synthesized the agent DM. Three years later, a US chemist, Robert Adams, independently developed this same compound and named it adamsite. Since that time, these agents have been produced for 2 purposes, as riot control agents and as emesis-inducing agents to promote removal of personal protective gear during chemical warfare.

Pathophysiology

Vomiting agents typically are disseminated as aerosols. The primary route of absorption is through the respiratory system. Exposure also can occur by ingestion, dermal absorption, or eye impact.

The effects of the vomiting agents by any route of exposure are slower in onset and longer in duration than typical riot control agents (eg, CS). On initial exposure, vomiting agents are irritants. This irritation is delayed for several minutes after contact. As a result of this delay, less early warning properties are present for those exposed. By the time symptoms of irritation occur and personnel consider donning their protective equipment, significant contamination already may have occurred. Systemic signs and symptoms subsequently follow the initial irritation and consist of headache, nausea, vomiting, diarrhea, abdominal cramps, and mental status changes. Symptoms typically persist for several hours after exposure. Death has been reported with excessive exposure.

Frequency

- **In the US:** The use of vomiting agents within the US against civilians never has been reported. Currently, the US government is funding numerous programs to prepare the nation for potential chemical terrorist attacks against its citizens and military.
- **Internationally:** The use of vomiting agents has been reported during international conflicts. DA first was used by German troops in 1917. DA was not well filtered by the standard issue gas masks at that time. It resulted in nausea and vomiting, causing enemy troops to remove their masks. This rendered those personnel vulnerable to the toxic effects of other agents such as phosgene and chlorine gas. The Germans also produced DC and DM, but limited documentation exists for use of these agents during World War I. Questionable reports exist of vomiting agents used in other countries as riot control agents. No recent use of vomiting agents is documented.

Mortality/Morbidity

DM is the most toxic agent of this group, with an estimated LCt₅₀ of 11,000 mg.min/m³ (eg, an estimated 50% lethality for a group of patients breathing air with a concentration of 11,000 mg/m³ for 1 min). Other factors also are important, such as the exposed patient's preexisting health status and the time from exposure to medical care. The dose at which vomiting reportedly begins for DM is estimated as 370 mg.min/m³.

Race

No published studies demonstrate a significant difference in effects of vomiting agents on various races.

Sex

No published studies demonstrate a significant difference in effects of vomiting agents by gender.

Age

Intuitively, those at the extremes of age would be less tolerant of exposure to these 3 chemical agents. However, no published studies prove this.

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Clinical

History

- A history of exposure to an aerosolized substance that resulted in ophthalmic and pulmonary irritation and then progressed to nausea, vomiting, abdominal cramps, and headache suggests exposure to a vomiting agent.
- In the early phases of an emergency response, the toxin's identification would be unknown and the history misleading and inaccurate.
 - Fear, anxiety, and mistrust are likely to affect victims, emergency responders, bystanders, and the entire community after such an incident.
 - Overwhelming emotions in some patients, rescuers, and hospital staff are likely to cause acute anxiety reactions and mass psychogenic illness. Patients truly suffering from vomiting-agent poisoning and those suffering from mass psychogenic illness would be difficult to separate, because the symptoms are similar. Patients with either condition may complain of nausea, vomiting, diarrhea, headache, tearing, dizziness, chest tightness, and shortness of breath.
 - Since it may be difficult to differentiate mass hysteria from a true vomiting-agent poisoning, treat all patients experiencing symptoms as true toxic emergencies. The potential exists for patients with mass psychogenic illness to overwhelm the entire emergency response system and hinder timely treatment of those with true toxic emergencies.

Physical

The signs and symptoms encountered in a person exposed to a vomiting agent may vary. Factors that determine clinical effects include the amount of the agent encountered and the route of exposure. Depending on these variables, the progression of signs and symptoms can range from mild mucosal irritation to cardiovascular collapse and death. The following list constitutes findings that may be noted on physical examination following exposure to vomiting agents:

- Eye - Conjunctival injection, tearing, and blepharospasm
- Nose - Excessive nasal discharge, sneezing, mucosal injection, and edema
- Throat - Mucosal injection and edema
- Lungs - Excessive cough, wheezing, rhonchi, prolonged expiratory phase, and tachypnea
- Heart - Tachycardia
- Abdomen - Hyperactive bowel sounds, intestinal cramps, emesis, and diarrhea
- Skin - Erythema and edema at the site of dermal contact
- Mental status - Central nervous system depression, syncope, and death (possible with significant exposure)

Causes

Human exposures to vomiting agents rarely have been reported. Potential causes of exposure to these agents are laboratory accidents, terrorist events, or military conflicts.

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Differentials

CBRNE - Chemical Warfare Agents

CBRNE - Incapacitating Agents, 3-quinuclidinyl Benzilate

CBRNE - Incapacitating Agents, Agent 15

CBRNE - Irritants: Cs, Cn, Cnc, Ca, Cr, Cnb, PS

CBRNE - Lung-Damaging Agents, Chlorine

CBRNE - Lung-Damaging Agents, Chloropicrin

CBRNE - Lung-Damaging Agents, Diphosgene

CBRNE - Lung-Damaging Agents, Phosgene

CBRNE - Lung-Damaging Agents, Toxic Smokes: Nox, Hc, Rp, Fs, Fm, Sgf2, Teflon

CBRNE - Nerve Agents, Binary: GB2, VX2

CBRNE - Nerve Agents, G-series: Tabun, Sarin, Soman

CBRNE - Nerve Agents, V-series: Ve, Vg, Vm, Vx

Toxicity, Ammonia

Toxicity, Chlorine Gas

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Workup

Lab Studies

- No rapid tests are available that enable health care providers to definitively determine exposure to vomiting agents. Consider these agents when exposure to an unknown substance inflicts pulmonary and ophthalmic irritation and then progresses to nausea, vomiting, abdominal cramps, and diarrhea.
- Obtain a complete blood count, electrolytes, clotting studies, and renal and liver function tests in any person who potentially was exposed to a chemical warfare agent.
- If a patient is markedly agitated or comatose, obtain a urine myoglobin and/or creatine phosphokinase to exclude rhabdomyolysis.
- If considering chemical warfare agent poisoning in the differential, obtain extra blood and urine samples for subsequent toxicologic testing.

Imaging Studies

- A chest x-ray may need to be obtained to exclude chemical pneumonitis in a patient exposed to vomiting agents who presents with marked pulmonary irritation.
- Vomiting agents rarely may cause altered mental status. If the etiology is uncertain, obtain a head CT scan to exclude other intracranial pathology.

Other Tests

- ECG: Vomiting agents are not reported to cause significant cardiac dysrhythmias. Sinus tachycardia may result from the stress of the event. In symptomatic persons at risk for coronary artery disease or in those with preexisting disease, obtain an ECG to exclude evidence of ischemia. When the causative agent is not identified definitively, obtaining an ECG is a reasonable approach to exclude conductive disturbances induced by other toxins.

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Treatment

Prehospital Care

- A military or terrorist event involving the exposure of military personnel or civilians to vomiting agents would create confusion and panic. Large numbers of potential casualties may overwhelm emergency response teams. Chaos may occur.
- Prehospital care providers must place their personal safety first before the treatment of potentially contaminated patients. With aerosolized exposure, secondary contamination of health care providers is unlikely.
- Perform general supportive measures such as obtaining intravenous (IV) access and administering oxygen to those with signs of respiratory irritation.

Emergency Department Care

The initial care of patients exposed to vomiting agents primarily is supportive. No specific antidotes are available. Focus care on relieving irritant and systemic effects.

- Respiratory irritation
 - These effects typically are transient and resolve soon after exposure ceases. Duration of irritation depends on the dose of agent inhaled and the premorbid status of the patient.
 - Patients with preexisting lung disease (eg, asthma, emphysema) may develop exacerbations of these diseases that are slow to resolve.
 - If a patient has dyspnea with wheezing, albuterol nebulizations may be necessary. Steroids may be considered.
 - In most patients without preexisting lung disease, symptoms abate spontaneously.
- Emesis: Treat patients with repetitive emesis with IV hydration and antiemetics. Numerous antiemetics are available. No specific agent is documented as superior.
- Central nervous system depression
 - Acute mental status changes rarely have been reported. One death after DM exposure is documented, but complete information on this fatality has not been released.
 - If a patient presents in marked respiratory distress with mental status changes, intubation and mechanical ventilation may be necessary.

Consultations

The following consultations may be necessary:

- Intensivist: In the rare event that a patient exposed to vomiting agents presents with acute respiratory distress or acute mental status changes, early consultation with a physician trained in critical care medicine may be necessary.
- Poison control center and/or local health department: Report adverse events caused by toxins to

the local poison control center and health department. This allows coordination of information with other health care facilities and expedites assistance in determining the etiology of the poisoning.

- **Law enforcement:** If the cause of the exposure is unknown or believed to be a terrorist act, contact local and federal law enforcement.
- **Ophthalmologist:** If significant eye exposure has occurred and the patient develops persistent ophthalmologic signs and symptoms or evidence of corneal damage, contact an ophthalmologist.

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Medication

Medical therapy focuses on controlling the emesis induced by the agents. Initial antiemetic therapy may begin with routine doses of drugs commonly used to combat vomiting, such as promethazine, prochlorperazine, or droperidol. High doses of metoclopramide may be administered. If these agents are unsuccessful, 5-HT₃ receptor antagonists may be administered to control nausea and vomiting. This class of drugs is comparatively expensive but well tolerated with few adverse effects. These agents include dolasetron, ondansetron, and granisetron.

Antiemetics

A common effect of DA, DC, and DM is emesis. Consider antiemetics in patients with persistent vomiting.

| | |
|-------------------|---|
| Drug Name | Prochlorperazine (Compazine)- May relieve nausea and vomiting by blocking postsynaptic mesolimbic dopamine receptors through anticholinergic effects and depressing reticular activating system. In addition to antiemetic effects, has advantage of augmenting hypoxic ventilatory response, acting as a respiratory stimulant at high altitude. |
| Adult Dose | 10 mg IV q3-4h prn |
| Pediatric Dose | 0.1 mg/kg IM q3-4h prn |
| Contraindications | Documented hypersensitivity; bone marrow suppression; narrow-angle glaucoma; severe liver or cardiac disease |
| Interactions | Coadministration with other CNS depressants or anticonvulsants may cause additive effects; with epinephrine may cause hypotension |
| Pregnancy | C - Safety for use during pregnancy has not been established. |

| | |
|-------------|---|
| Precautions | Drug-induced Parkinson syndrome or pseudoparkinsonism occurs quite frequently; akathisia is most common extrapyramidal reaction in elderly patients; lowers seizure threshold; caution with history of seizures |
|-------------|---|

| | |
|-------------------|---|
| Drug Name | Promethazine (Phenergan)- For symptomatic treatment of nausea in vestibular dysfunction. Antidopaminergic agent effective in treating emesis. Blocks postsynaptic mesolimbic dopaminergic receptors in brain and reduces stimuli to brainstem reticular system. |
| Adult Dose | 12.5-25 mg IV q4h prn |
| Pediatric Dose | 0.25 mg/kg IM q4h prn |
| Contraindications | Documented hypersensitivity |
| Interactions | May have additive effects when used concurrently with other CNS depressants or anticonvulsants; coadministration with epinephrine may cause hypotension |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in cardiovascular disease, impaired liver function, seizures, sleep apnea, and asthma |

| | |
|-------------------|--|
| Drug Name | Droperidol (Inapsine)- Neuroleptic agent that may reduce emesis by blocking dopamine stimulation of chemoreceptor trigger zone. |
| Adult Dose | 2.5 mg IV q3-4h prn |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity |
| Interactions | May increase toxicity of CNS depressants |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Hypovolemic patients may experience hypotension; may decrease pulmonary arterial pressure; tardive dyskinesia in patients receiving droperidol is 40%; elderly patients may experience high rate of extrapyramidal reactions; life-threatening arrhythmias may occur in patients receiving this medication |

| | |
|----------------|---|
| Drug Name | Metoclopramide (Reglan, Clopra, Maxolon)- Dopamine antagonist that stimulates acetylcholine release in myenteric plexus. Acts centrally on chemoreceptor triggers in floor of fourth ventricle, which provides important antiemetic activity. |
| Adult Dose | 1 mg/kg IV tid prn |
| Pediatric Dose | Administer as in adults |

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|-------------------|--|
| Contraindications | Documented hypersensitivity; pheochromocytoma or GI hemorrhage, obstruction, or perforation; history of seizure disorders |
| Interactions | Anticholinergic agents may antagonize effects of metoclopramide; opiate analgesics may increase metoclopramide toxicity in CNS |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Caution in history of mental illness and Parkinson disease |

5-HT₃ Receptor Antagonists

A more expensive drug category compared to the other available antiemetics noted above. These agents typically are reserved for severe cases of emesis not responsive to the above medications.

| | |
|-------------------|---|
| Drug Name | Ondansetron (Zofran)- Selective 5-HT ₃ receptor antagonist that blocks serotonin both peripherally and centrally. |
| Adult Dose | 0.15 mg/kg IV |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Although potential for cytochrome P-450 inducers (barbiturates, rifampin, carbamazepine, phenytoin) to change half-life and clearance of ondansetron, dosage adjustment usually is not required |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Medication is to be administered for prevention of nausea and vomiting, not for rescue of nausea and vomiting |

| | |
|-------------------|---|
| Drug Name | Dolasetron (Anzemet)- Selective 5-HT ₃ receptor antagonist that blocks serotonin both peripherally and centrally. |
| Adult Dose | 1.8 mg/kg IV, not to exceed 100 mg |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | Although potential for cytochrome P-450 inducers (barbiturates, rifampin, carbamazepine, phenytoin) to change half-life and clearance of ondansetron, dosage adjustment usually is not required |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Medication is to be administered for prevention of nausea and vomiting, not for rescue of nausea and vomiting |

| | |
|-------------------|--|
| Drug Name | Granisetron (Kytril)- At chemoreceptor trigger zone, blocks serotonin peripherally on vagal nerve terminals and centrally. |
| Adult Dose | 10 mcg/kg IV over 5 min |
| Pediatric Dose | Administer as in adults |
| Contraindications | Documented hypersensitivity |
| Interactions | None reported; does not appear to interact with P-450 system; interaction with other medications is not expected |
| Pregnancy | B - Usually safe but benefits must outweigh the risks. |
| Precautions | Caution in liver disease |

Bronchodilators

Acute bronchospasm may result when exposure occurs to aerosolized chemicals. Bronchodilators are administered to attempt to alleviate bronchospasm that causes decreased pulmonary airflow and wheezing.

| | |
|-------------------|--|
| Drug Name | Albuterol (Ventolin, Proventil)- Beta-agonist for bronchospasm refractory to epinephrine. Relaxes bronchial smooth muscle by action on beta2-receptors with little effect on cardiac muscle contractility. |
| Adult Dose | 2.5-5 mg in 2 mL NS nebulized prn bronchospasm |
| Pediatric Dose | 0.10-0.15 mg/kg/dose (not to exceed 5 mg) in 2 mL NS prn bronchospasm |
| Contraindications | Documented hypersensitivity |
| Interactions | Beta-blocking agents may cause blunted effect of this drug; other sympathomimetics may interact to cause potentiated effects |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution administering beta-agonists to patients with known coronary artery disease, since increased heart rate may occur; also may aggravate preexisting diabetes, resulting in hyperglycemia |

Cycloplegics

Eye muscarinic antagonists that cause mydriasis and alleviate ciliary spasm. May alleviate symptoms in patients who develop a chemical conjunctivitis caused by eye exposure.

| | |
|-------------------|--|
| Drug Name | Cyclopentolate (Cyclogyl, AK-Pentolate)- Prevents muscle of ciliary body and sphincter muscle of iris from responding to cholinergic stimulation. Induces mydriasis in 30-60 min and cycloplegia in 25-75 min. |
| Adult Dose | 1 gtt to affected eye |
| Pediatric Dose | Not established |
| Contraindications | Documented hypersensitivity; narrow-angle glaucoma |
| Interactions | Decreases effects of carbachol and cholinesterase inhibitors |
| Pregnancy | C - Safety for use during pregnancy has not been established. |
| Precautions | Caution in patients (eg, elderly) in whom increased intraocular pressure may be present; can cause toxic anticholinergic systemic adverse effects (common in children, especially infants), but incidence is rare when used sparingly; compressing lacrimal sac by digital pressure for 1-3 min following application may minimize systemic absorption |

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Follow-up

Further Inpatient Care

- Inpatient care for patients exposed to vomiting agents is no different than the care discussed in [Emergency Department Care](#).
- Symptomatic patients exposed to these agents should remain in a health care setting until signs and symptoms abate and they are able to take adequate fluid by mouth without repeat emesis. Continued use of IV fluids and antiemetics may be necessary.
- Patients who demonstrate marked bronchospasm may need repeated nebulized albuterol as necessary.

Further Outpatient Care

- Most patients exposed to vomiting agents recover within the first few hours postexposure and demonstrate no further toxicity.
- If marked ocular toxicity occurs and corneal injury is documented, obtain follow-up care with an ophthalmologist to ensure that healing is progressing. Schedule this follow-up visit within 24 hours of discharge.

Transfer

- A health care facility that is unable to adequately provide care for a patient intoxicated with a vomiting agent should consider transfer to a facility that can care for such patients. Health care facilities may be overwhelmed quickly if a large-scale exposure occurs with multiple casualties. Disaster plan implementation and appropriate transfer of patients to less stressed facilities may be necessary.

Complications

- Complications are expected to be rare in persons exposed to vomiting agents if rapid and adequate supportive care is initiated. If significant ocular exposure occurs, corneal chemical burns may develop. In persons with preexisting lung disease, exacerbation of the lung disease may occur. If a patient sustains large exposure, coma may develop with subsequent risk of developing anoxic brain injury and aspiration pneumonia.
 - Corneal chemical burns: A significant exposure to vomiting agents can lead to damage of the cornea. If the patient complains of significant eye discomfort, foreign body sensation, photophobia, or decreased visual acuity, consider eye irrigation. Thoroughly examine the eye and include visual acuity testing. Perform slit lamp examination with fluorescein. If a chemical corneal burn is documented, a cycloplegic may be used to reduce pain; apply topical antibiotic ointment. Arrange follow-up care with an ophthalmologist within 24 hours.
 - Acute bronchospasm: As with many types of chemical inhalation exposures, acute bronchospasm may develop. This is especially true of patients with preexisting lung disease (eg, asthma). If acute bronchospasm occurs leading to respiratory distress, treatment with bronchodilators (eg, albuterol) may be necessary.
 - Anoxic brain injury: If an exposed person becomes comatose and loses his or her ability to maintain ventilatory function, hypoxia may develop, leading to anoxic brain injury. Unless massive levels are encountered, this complication is exceedingly rare after exposure to vomiting agents.
 - Aspiration pneumonia: Inability of exposed patients to maintain their airway may result in aspiration of gastric contents into the lungs.

Prognosis

- The prognosis is good for persons exposed to vomiting agents if they do not develop secondary injuries. Full recovery is expected in most patients.

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Miscellaneous

Medical/Legal Pitfalls

- From a medicolegal standpoint, few pitfalls exist in dealing with patients manifesting signs and symptoms of vomiting agent poisoning. If a physician demonstrates good supportive care, the risk of litigation against the caregiver should be minimal. Call the appropriate authorities if exposure to these agents is believed to have occurred.

Special Concerns

- Currently, no publications address the clinical effects of vomiting agents in special populations. A pregnant female exposed to these agents may be at an increased risk of miscarriage because of the stress of the event. No published studies have been performed on pregnant females, and no study has demonstrated whether these agents are teratogenic. No published studies have demonstrated a difference of clinical effects either on the exposed pediatric or geriatric patient.

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