In the diagnosis of a given disease it is essential that the physician rest his opinion not upon one or two symptoms, but upon a series of symptoms which when properly put together give him a complete, or nearly complete, picture of the malady. It is as futile for a physician to attempt to base a diagnosis upon one symptom as for an architect to attempt to determine the appearance of a house by seeing one of the stones which has been removed from its walls.

PRACTICAL DIAGNOSIS

THE USE OF

SYMPTOMS AND PHYSICAL SIGNS IN THE DIAGNOSIS OF DISEASE

BY

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SIXTH EDITION, REVISED AND ENLARGED

ILLUSTRATED WITH 203 ENGRAVINGS AND 16 PLATES

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THIS BOOK

IS

DEDICATED TO MY FRIEND AND COLLEAGUE

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PREFACE TO SIXTH EDITION

WHEN the physician is at the bedside, or in his consulting room, he develops the diagnosis of the ailment of his patient by an analysis of the symptoms of which he hears, which he sees, and which he discovers on making a thorough physical examination of the organs of the body. Lastly, he calls to his aid the methods of examination which are carried out in the laboratory.

At the present day there is a great tendency on the part of practitioners who can command laboratory assistance to ignore the measures which had to be relied upon before laboratories were developed, with the result that the various methods of physical diagnosis involving refinement in the senses of touch and hearing and in observation are in danger of becoming lost arts. On the other hand, practitioners who do not live near well-equipped laboratories, or do not possess them, are sometimes careless in the matter of resorting to methods of laboratory investigation.

This book is designed to deal chiefly with the diagnosis of disease by means of the symptoms presented by the patient, and takes up laboratory methods only in those cases in which they are essential to arriving at correct results, as, for example, in the examination of the blood and urine. In other words, the object of this volume is to place before the physician and student the subject of medical diagnosis as it is met at the bedside. To accomplish this the symptoms used in diagnosis are discussed first, and their application to determine the character of the disease follows. Thus, instead of describing locomotor ataxia or myelitis, there will be found in the chapter on the Feet and Legs a discussion of the various forms of and causes of paraplegia, so that a physician who is consulted by a paraplegic patient can in a few moments find the various causes of this condition and the differential diagnosis between each. So, too, in the chapter on the Tongue, its appearance in disease, both local and remote, is discussed. In other words, this book is written upon the only plan which can be followed in practice, namely, the upbuilding of a diagnosis by grouping the symptoms.

In the ordinary treatises the reverse of this plan is followed, compelling the physician to make a supposititious diagnosis and then to turn to his reference book and compare the article dealing with the supposed disease, when if the description fails to coincide with the symptoms of his case he must make another guess and read another article. In this book, however, the discovery of any marked symptom will lead directly to the diagnosis. Thus, if the patient is vomiting, in the chapter on Vomiting will be found its various causes and its diagnostic significance, and the differentiation of each form of this affection from another. The present edition contains a large number of illustrations taken from actual cases.

Basing his efforts upon the experience which he has had in both didactic and clinical teaching of large classes of students during the last twenty-three years, the author hopes that the work may in some degree lighten the labors of the general practitioner and student.

The author wishes to express his appreciation of the cordial reception given to this work in its earlier editions, and to bespeak for it an equally favorable acceptance in its present form, the more so as it appears simultaneously with the twelfth edition of his *Text-book of Practical Therapeutics*, to which it has always been a companion volume.

H. A. H.

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PRACTICAL DIAGNOSIS.

INTRODUCTION.

GENERAL DIAGNOSTIC CONSIDERATIONS.

A CLEAR understanding by the physician of the value of the symptoms of disease which he sees and of those described by the patient is of vital importance for the purpose of diagnosis and treatment, and one of the advantages of older physicians over their younger brethren is the ability which they have gained through long training to grasp the essential details of a case almost at their first glance at the patient. Much of this ability is unconsciously possessed because it is gained by a gradual process, yet it is none the less valuable, and its possession often impresses the patient with the insight which his physician has into his case. At first it is impossible for the novice to cast aside the minor symptoms, which the patient emphasizes as his major ones, and to perceive clearly that one or two facts that have been belittled in the narration of the story of the illness are in reality the stalk about which everything else in the case must be made to cluster.

While the physician should train his eye to take note of all variations from the normal which are shown by the patient, he must be careful not to permit himself to depend for his diagnosis on these signs alone nor upon those described by the sick man. On the contrary, he should carefully train his senses of touch, hearing, sight, and discover the so-called physical signs which can be elicited only by auscultation, percussion, palpation, and mensuration. The objective symptoms seen by the physician, the subjective symptoms described by the patient, and the so-called physical signs elicited by the methods just named, are to be joined together in forming a diagnostic view of the case. Unless this combination is made, a faulty diagnosis will often be reached. In many instances chemical and microscopic examinations of the blood, the urine, the feces, and even of the tissues themselves, may be needful to determine in a given case what the ailment may be.

Again, it is never to be forgotten that negative signs are as important as positive signs in many cases. The absence of a heart mur-

mur in a case of failing circulation is not a sign that the heart valves are healthy, but that the heart is too feeble to make a murmur, and the absence of rales or clearly heard breath sounds in a lung may be due to engorgement without exudate instead of indicating that the lung is not diseased.

The Art of Observing the Patient.—Let us suppose the patient before the physician is one who has been able to walk into the office or dispensary. The attentive physician can at once gather much information about the case from the clothing, the gait, the build, the voice, the expression, and the manner. The thin man, with a peaked face and provided with an unusually warm overcoat, and still further wrapped up with a muffler almost to his eyes, is in all probability a sufferer from some pulmonary or throat difficulty, while the heavily built, phlegmatic individual, with a large head and well-filled paunch, is much more apt to suffer from gastro-intestinal or biliary catarrhs. Such a person will probably be one who habitually wears his coat open on the coldest days. Again, chronic drunkards, or persons whose mental powers are failing, often are exceedingly careless about their clothing, buttoning the coat or the trousers with the wrong buttons, and keeping the garments dirty and spotted. Some cases of diabetes have first been discovered by the white spots on the trousers, as the result of the patient having allowed a few drops of urine to fall on the cloth, where they have dried. Old men who have incontinence of urine often wear trousers which are stained in front, and they often have an ammoniacal odor about them from this cause.

The various forms of gait which indicate actual disease will be found discussed in the chapter on the legs and feet, but it may be mentioned in passing that, in addition to these changes, which are dependent upon actual disease of the legs or the nervous system supplying them, the bearing and stride of a patient will often give a clear idea of his general tone. The neurasthenic patient walks feebly or with a step that might be called ataxic, while the strong, hearty man of good physique strides along with a gait quite different from this, or from that of an individual who is delicate and feeble.

Similarly, the patient's build betokens disease or health. The thin, tall, and hollow-chested person is recognized as a fair mark for the tubercle bacillus, and the heavy, closely knit, phlegmatic man as one who may suffer from hepatic or circulatory disorders. Again, the bearing of a person possessing a highly organized nervous system shows itself in the constant activity of his mind and body. No part is quiet for more than a moment, and drugs are more apt to produce extraordinary symptoms as the result of idiosyncrasies in this type of patient than in any other.

The breath of the patient may call the physician's attention to some important facts in connection with the case. If it is sweet or vinous in odor, this may be due to diabetes mellitus; or if it be ammoniacal or urinous, uremia may be present. The previous use of bismuth may give it a garlic-like smell, due to the contaminating tellurium and various aromatic or volatile drugs, such as turpentine or copaiba, may be eliminated in the breath. In cases of advanced bronchiectasis the breath is often foul, and is insupportably so in gangrene of the lung or when an empyema has ruptured into a bronchus. It is similarly offensive in gangrenous stomatitis, and often very disagreeable in tonsillitis. In diphtheria it has a peculiarly sickening and sweetish odor. When the odor is not dependent on these causes it may be due to ozena, or chronic atrophic nasal catarrh, to the presence of decaying food between the teeth or secretions in the crypts of the tonsils. Moderate fetor is usually due to disordered digestion and constipation.

When the physician has gathered as much information as possible as to the age and general condition of his patient, by a careful scrutiny of his face and extremities, of which scrutiny, however, the patient should be unconscious, he should ask him to tell what brings him for advice, and, as a rule, this will be the opportunity the sufferer seeks to pour out the story of his ailment as he sees or feels it. Often the story will seem wearisome, and, to the educated mind of the physician, wandering or unnecessary; but to the patient every word seems of the greatest importance, and to show any lack of interest may give the impression of carelessness, or it may interrupt the story just as a most important symptom is about to be described. Even if the patient is unable to convey in words a very clear idea of his condition, the manner in which his story is told, the character of his speech, and the expression of his face while speaking may give useful information as to his ailment or general state.

If, instead of the patient being an office or dispensary case, he is one who is being visited at home, the fact that the patient meets the physician in one of the living-rooms rather than in a bed-room indicates either that there is little immediate danger in the case, or, at least, that the difficulty is not acute, but chronic in type, as some slowly progressing form of pulmonary, cardiac, or renal disease. Of course, there are exceptions to this rule, as in the case of a patient who, having caught a heavy cold, is remaining indoors, but not in bed, for prudence' sake. Or, again, if on seeing the patient we find him sitting in a chair only partly dressed and propped up with pillows, or instead leaning forward upon the back of a chair placed in front of him, we know that he is the subject most probably of an acute or chronic heart disease, most likely an acute exacerbation of the latter. A glance at the face of such a patient, revealing a trembling

nostril, blue lips, or an anxious facies, will aid still further in directing attention to the heart or lungs, and the hands if examined will appear relaxed and livid or darkened in hue, indicating capillary stasis and deficient oxidation in the blood. In other cases, however, the patient found sitting propped up with pillows may be a convalescent from some long illness; but if so, the general atmosphere of the patient is better, and the surroundings are apt to be more tidy.

If we find the patient in bed, he may be lying abnormally quiet as the result of faintness or acute nausea, or, perhaps, from partial or complete coma due to cerebral or renal disease, or from the effects of some drug; or, again, he may be rolling about the bed from the pain of acute bellyache, or be keeping his legs and body very still while his hands and head are ever on the move to prevent anything from suddenly approaching or touching his abdominal wall, as in peritonitis. The striking difference between the activity of the head and the fixation of the lower part of the body, in peritonitis, is notable. Sometimes, however, anxious restlessness indicates acute internal or external hemorrhage; but here the movements are minute though active, and the patient does not expend so much strength as he does when suffering from pain.

In acute articular rheumatism the patient lies, if many of his joints are involved, in a peculiarly helpless position, as if he were glued fast to the bed, although the active movement of the eyeballs as they follow persons moving about the room gives quite a different aspect from that of a patient immovable from stupor.

Usually a patient who is lying on his side turns on his back as the physician or nurse approaches, in order to face his visitor; but if he persistently remain on the side without moving, except partly to turn his head, we may suspect that in that posture he is most comfortable, and that the position is assumed for its comfort or to relieve pain or dyspnea, which are suffered from when another attitude is taken. Thus, in acute pleurisy the patient lies with the affected side uppermost, because it is too sore to permit him to touch it to the bed; whereas if the stage of effusion has arrived, he lies on the affected side, in order to give the side which is healthy free play in compensatory respiratory movements, and to remove the pressure of the effusion from the healthy lung. If the patient lying in this posture is not suffering from pleurisy, his position may be due to an effort to relieve the discomfort caused by an enlarged liver. The fact that the patient lies constantly on the back is also a characteristic of grave and advanced disease in some instances. Very ill persons almost never lie on the side, and the fact that a desperately ill patient of yesterday is found lying on the side today is encouraging.

Persons with severe heart disease, emphysema, or chronic bronchitis are rarely, if ever, able to lie prone in bed, and have to be more or

less propped up with bed-rests and pillows. Large growths in the abdominal cavity producing pressure on the diaphragm also necessitate this semiprone posture, and double pleural effusions, or pulmonary consolidation or edema, require the upright or half-reclining attitude in order that the upper parts of the lung may be used to advantage.

In asthma the patient frequently is found sitting up in bed with the arms reaching back of the hips as props to fix the chest and to hold the body erect. So too in advanced cardiac and renal disease the patient will often take a seat in a chair and rest the head and hands on the back of another chair placed in front of him, both eating and sleeping in this attitude.

Again, if the patient wakes when spoken to and then drops off to sleep at once, some form of poisoning may be present, as from opium, or the poisoning of advanced hepatic or renal disease may be present.

(For the significance of picking at the bedclothes, see chapter on the Hands and Arms.)

The Art of Questioning the Patient.—We can next pass to a consideration of the objects to be sought in questioning a patient as to the illness from which he is suffering. Often much information can be gained by a well-directed question, and a favorable impression can be made upon the patient by the manner in which it is put and the bearing which it has on his case. Thus, if a man is evidently much emaciated and his clothes fit him loosely, a question in regard to his loss of flesh is very appropriate; but if he is manifestly too stout for comfort such a question will be most unwise. Or, again, if a young married woman comes complaining of constant sickness of the stomach and a fanciful appetite, and the physician directs all his questions to the condition of the stomach without an eye to a slight increase in size about the waist or below it, his professional acumen is in grave danger of being libelled by that same woman, who knows, or soon finds out, that her discomfort is due to pregnancy.

If the woman is unmarried and there is no evidence of gastric disorder on her tongue, it is well to remember what Battey, of Georgia, said in regard to this condition: "Always believe a young unmarried woman with abdominal tumor, of high social position and unimpeachable virtue, if she has been watched over by a platonic and abstemious young cousin of the male persuasion while the mother went out, to be pregnant."

Again, if a married woman of some years tells her physician that she has no children, the physician naturally asks some questions which elicit the fact that she has had frequent miscarriages. He in this way finds out quite as much about probable syphilitic

infection as if the question had been put: "Have you ever had a sore on your privates?" which would embarrass the patient, produce domestic troubles, and probably be lied about if her husband is forced to answer the question.

Again, when asking a woman about the health of the living parents, or the cause of death of the dead, care should be taken not to ask a direct question, as, for example, whether the mother has died of cancer, for the patient may be already greatly worried lest she has that disease. It is better to ask the cause of death, or of the illness she is suffering from. If the story is that the parents died of "bronchitis," the real cause of death was probably tuberculosis of the lungs.

If the patient complains of pain, past or present, the best way in which to discover its true seat is to ask him to place his hand on the affected part, as in this way errors in his description of his anatomy will not be committed, and false impressions will not be conveyed to the physician's mind. Even this direct method of showing the area of pain is not to be absolutely relied upon, for often pains are referred to parts in which there is no disease. Thus, the pain of coxalgia is apt to be felt in the knee and ankle, and in children the pain of pulmonary or cardiac disease is often described by the patient as felt in the abdomen. If the pain has been really abdominal, there will, in many cases, have been diarrhea or free passage of flatus. It is not to be forgotten, on the other hand, that a question which discovers the fact of several movements of the bowels does not prove the presence of true diarrhea, because a purgative may have been taken by the patient.

In asking questions as to constipation the physician must not forget that the opinion of the patient as to what constitutes regularity of bowel movement is of very little value in many instances. A daily movement is not known to many patients, and a movement every few days may be quite sufficient to justify the statement, in their opinion, that no constipation is present.

The young physician, in particular, in asking questions of women patients of the better class, should not hesitate to ask direct questions as to the state of the bowels or of the menstrual function. To hesitate or ask indirect questions about such matters simply produces embarrassment which otherwise would not exist, and intimates that the question is one of doubtful propriety, when in reality it is most important and proper.

The Examination of Children.—If the patient to be examined is a child, it is well for the physician to remember that his presence as a stranger may be a source of alarm, and that the association in the child's mind of sickness and the doctor, and badly tasting medicines, is sufficient to render him a much-to-be-dreaded individual.

Generally it is best, on entering the room where the child is, to pretend to pay no attention to it whatever, but to engage in conversation with the mother or other person, speaking of the case in a way which the child will not understand. Very often this very lack of attention will result in the child forcing the recognition of his presence upon the physician by making the first advances toward friendship, and this is particularly apt to be the case if the child is already spoiled by over-attention by the family and friends. Time should always be given the child to grow accustomed to the peculiarities of the visitor, and if any instrument for diagnosis is to be employed it is best to hold it in the hand as if it were a plaything before attempting to put it into actual use. The tact which the physician must exercise in diverting a sick child is an essential to the successful treatment of children. Some physicians are welcomed to a house by the sick and well as Santa Claus would be, and others, devoid of the trait of amusing children, are fled from as if they were dragons.

During the time that the physician is allowing the child to get accustomed to his presence he should be gaining much useful information about the case by observing the movements and expressions of the child; its color, size, nutrition, breathing; the shape and size of its head; the condition of its lips, whether moist or dry, red, livid, or pale; and, if the child is speaking, the tone of its voice, or, if crying, the character of its cry. It is needless to state that a child may cry from fright, from pain, anger, or hunger. Constant screaming is, however, nearly always due to the pain of earache or hunger, for abdominal colic is usually intermittent. If there be pain in the ear, the hand will often be rubbed over the affected side of the head, and the child will not be pacified by the offer of the breast. If the child coughs, and then begins to cry, pneumonia or pleurisy may be present; or in other cases the pain is so great that the child is cryless. A sharp, piercing shriek indicates the pain of earache or of meningitis in many cases.

If a crying child be placed at the breast, which it takes with avidity only to drop the nipple in a moment with a cry of pain or anger, one of several conditions is present: either the child has stomatitis or the breast is empty; or, again, if it seizes the breast and then lets go with a gasp, it may have coryza or syphilitic snuffles, which prevents it from breathing through the nose while sucking. Similar signs may be present in any other condition producing shortness of breath.

If a child over four months of age cries and sheds no tears in the course of an illness, this is an unfavorable sign.

It is important to notice whether there is languor or a tendency to play. A healthy infant, when awake and well fed, is always

kicking and cooing and moving its arms about, and has a happy expression on its face; whereas if any cerebral trouble is present, it often has an anxious frown, or its hands are placed on the side of its head or rubbed over the vertex.

In a perfectly healthy child which is sleeping, the respiration should be practically inaudible, and it is a good practice to note the regularity of the breathing in all patients while they are asleep, as it is then unaffected by voluntary effort. In children a sighing breathing, or one disturbed in rhythm, often indicates a disturbed digestion or fever.

The physician should always, by careful questioning of the nurse or mother, find out how long the illness has lasted, the manner in which it began, the fact as to whether a similar attack has occurred before in this or other children of the family, and the state of the temper, appetite, bowels, and urine of the patient, for an irritable temper in a child means ill health, as does also a poor appetite, constipation, diarrhea, or abnormal urine.

The expression of the face, shape of the head, and similar noteworthy points in the diagnosis of the case will be more thoroughly discussed in the chapter devoted to these parts. (See chapter on the Face.)

When it comes to a close examination of the child, great care must be exercised. The character and rapidity of the respirations are best studied at a distance before excitement has disturbed them, and the best time for listening to a young child's chest is when it is held over the shoulder of the mother, as if she were carrying it for a walk, or, if the child can be taken in the physician's arms, its buttocks should rest on one hand, while the front of its chest rests against the other. In this way the physician can listen to the back of the chest without difficulty, keeping the child amused by walking up and down the room while it is in his hands.

If it is not possible by any bribe to cause the child to protrude the tongue for examination, the physician will often be able to see this organ when the mouth is widely opened in crying.

In taking a child's pulse it is best to take it while it is asleep, if possible, as the excitement of the physician's visit or the crying on awakening will greatly increase the pulse rate.

CHAPTER I.

THE FACE AND HEAD.

The expression and color of the face—Facial deformity—Facial paralysis, unilateral and bilateral—Ptosis—Facial spasm—The shape of the head—The movements and position of the head and neck.

So much can be learned by the physician from the expression and general appearance of a patient's face and the carriage and shape of his head that a careful inspection of these parts should always be made. For this reason, in the consulting-room and at the bedside, the physician should always arrange his chair in such a way that the light falls upon the face of his patient, while his own is in the shadow, and this is of importance not only because the facial expression of the patient can thus be well seen, but also because it prevents the patient from making a too close scrutiny of the physician's face with the object of detecting encouragement, lack of sympathy, or alarm.

THE FACE.

The Expression.—The expression is produced by the formation of creases, or alterations in the contour of the skin and subcutaneous tissues by trophic and muscular action, and these changes are in time brought about and perhaps made permanent by the mental tendencies and habits of the patient, his temperament, his intellectual development, his exposure to outdoor or indoor influences, and, finally, and these are very important, by pathological processes which may be going on somewhere in his body. The temper of the man also affects his expression, particularly as he approaches middle life, and he looks amiable, capable of sudden anger, or sullen, as the case may be.

The intellectual face is easily recognized. Sometimes it is deeply thoughtful and placid, at others eager or keenly alive to the surroundings or the conversation, and it separates the man descended from several generations of men who have lived as thinkers from him whose ancestors have been but recently wage-earners by physical labor, involving only ordinary human intelligence.

The skin of the face and the expression about the eyes of one who has been exposed for years to the weather are so characteristic

as to need no description, while the face of the clerk, whose life is almost entirely spent indoors, is pale and wan.

Fulness of the lips, particularly of the lower lip, is supposed to be present in persons of strong sexual appetite, and often indicates a phlegmatic temperament, whereas the thin, mobile lip is typical of the high-strung, nervous individual.

The expression of the lips as a whole is also to be regarded in connection with the expression of smiling. The risus sardonicus of strychnine poisoning or tetanus is quite characteristic, and the simple smile of hysteria is equally notorious.

Similarly, the face of a person who uses alcohol to excess is generally flushed, heavy, and more or less expressionless. The eyelids are redder than normal, and the skin is apt to be puffy and unhealthy looking. Women at the menstrual period, or when suffering from menstrual disorders, often have dark areas under the eyes, and pigmentation of the eyelids is often seen very early in pregnancy. In women, and sometimes in men, excessive fatigue and loss of sleep cause marked infra-orbital discoloration. A puffiness under the eyes, most noticeable in the morning, may indicate renal lesions or the excessive use of arsenic; or if it is unilateral it probably depends upon some local inflammation of the eye or rarely upon disease of one of the cephalic sinuses. So, too, an ecchymotic spot under the eye may be due to a bruise, to some one of the forms of purpura, or to scurvy.

The color of the face is discussed in the chapter on the Skin, but it is not out of place to note at this point the pallor of the face in fright, faintness from hemorrhage (acute or chronic), that due to lack of proper food, and the peculiar pallor of chlorosis. In the latter disease the faint yellowish-green tinge of the skin in some parts of the face, which still retains its plumpness, is quite typical. A parchment-like skin stretched over the face so that it appears as if stretched and dried over the under structures is seen in some young persons suffering from syphilis, and in some cases of hepatic cirrhosis.

The color of the face may be rendered gray or bluish by the ingestion of overdoses of the coal-tar products, such as acetanilid, antipyrine, and phenacetin, and it is curious that this effect is best seen when the patient is viewed at a little distance.

(For the significance of facial cyanosis, see chapter on the Skin.)

In view of the extraordinary variations seen in the expression of the face in the healthy it is not surprising that this part of the body should give the physician, when studying disease, so much useful information. It is an interesting fact, too, and one not unworthy of note, that the true facial expression of a disease is rarely aped by a malingerer, and in all diseases is unrecognized by the patient even

though he sees himself several times daily in a looking-glass. Thus it is by no means uncommon to see a person who is suffering from the onset of some sudden and grave disease bearing upon his face what we call "an expression of anxiety," when he himself as yet has no conception of the gravity of his illness. This expression is very characteristic of serious illness, and, though difficult to describe, when recognized becomes quite valuable as a diagnostic factor, particularly as it rarely, if ever, is exaggerated by the patient who bears it. It is seen most markedly in cases of severe acute croupous pneumonia or in peritonitis, or after severe injuries.

When persons have had continuous pain for a long time, as in patients who have growths of a malignant character or other organic disease, the expression of the face, naturally gentle, often becomes hard and stony, or if the pain be in the head, the expression is not only that of pain, but of profound mental depression. In cases of visceral carcinoma the face becomes thin, its skin yellow and straw-colored, and oftentimes greasy and thick, and there is often a marked expression of anxiety. On the other hand, the patient sometimes has a dogged expression on his face as if he had been told of the true cause of his illness, and was rebelling against the inevitable progress of the disease.

In the case of children, much information can be gained as to the state of the system by the facial expression, particularly while the child sleeps. If it is asleep and healthy and well, the eyelids are closed, the lips are never so slightly parted, the nostrils are practically immobile, and the general expression is very peaceful. If, on the other hand, the eyelids of a sleeping child are slightly parted so as to show the whites of the eyes, there is probably present some digestive or nervous disturbance, perhaps accompanied by moderate pain. If in the course of an illness the eyelids remain far enough apart to result in glazing of the conjunctiva from dryness, this is a sign of grave import. Again, twitching of the eyelids often indicates nervous irritation or the early stages of the convulsive state, and it is not uncommon for an expression to pass over the face of a child who, while sleeping, is suffering from pain, which begins as a smile and ends with a drawing-in of the corners of the mouth, an expression somewhat like that seen on the face of a waking child when it seems to be in doubt as to whether to laugh or to cry. Whether asleep or awake a child in pain, if not crying, has a pinched look about its nose and mouth, and sometimes some idea of the seat of the pain may be gained by the part of the face which is drawn. When pain is in the head, the forehead is apt to be wrinkled into a frown; if the nose is pinched and drawn, it is said to show that the pain is in the chest; and if the upper lip is raised, pain is probably felt in the belly.

Aside from these symptomatic manifestations, however, we find in the face of a child several evidences of important diathetic tendencies, or even hereditary diseases. Thus we see the light flaxen-haired, slimly built child with a refined, *spirituelle* face and transparent skin, whose temporal veins can be easily traced and whose expression is often thoughtful and deep. Such a child often comes of tuberculous parents, and is frequently a victim of tuberculosis, in one of its rapid forms, as it approaches puberty. Or, again, the child is "stocky" and cheesy looking, apparently solid and sturdy, but its features are heavy or perhaps even coarse, while its neck is thick and short. Such a child is often a victim of tuberculous bone or lymphatic disease. In other instances a square projecting forehead with faulty bone development elsewhere indicates rickets, or an immense, bulging forehead with a wizened, puny face beneath shows hydrocephalic tendencies. Sometimes a broadness of the bridge of the nose or marked flatness of it indicates congenital syphilis. Such a child is often much wasted, its features pinched, and its lips thin, while the flattened nasal bridge is bluish, and its face is often that of a little old man, shrivelled and wrinkled. Mucous patches at the corners of the mouth or around the anus are often found in such cases, and, if found, confirm the diagnosis of infantile syphilis. Finally, in respect to facial expression in childhood, attention must be called to the "fish mouth," vacuous, and "nose-pinched" expression of those children who are "mouth-breathers" from nasal obstruction (Fig. 1). Great immobility of the lips and cheeks may be due to mucous patches or other ulcerations of the buccal mucous membrane; and if high fever is present, the presence of herpetic blisters about the lips points to the possible presence of croupous pneumonia in the child or adult.

The face of a patient with acute fever is apt to be red and flushed, and the eyes bright; and if the disease be distinctly infectious, as in some cases of pneumonia, tuberculosis, and acute articular rheumatism, it may be covered with sweat.

In adults the facial expression of many diseases is even more characteristic than it is in children. Thus, we see in acute pulmonary phthisis the widely opened eye, the hunted expression, the quivering nostrils, the red flush over the malar bones, the wasting and dryness of the hair and skin, and the eager or in other cases apathetic glance of the eye.

In severe croupous pneumonia the flushed face, with a deeper red on one cheek than the other, the anxious expression, and the dilated nostril are noteworthy; and in the dyspnea of heart disease the dilated nostril and constant opening of the mouth, as if seeking for air, with the facial pallor or cyanosis, are characteristic. Often, too, in chronic cardiac or pulmonary disease producing slight diffi-

culty in respiration, the patient's lips are seen to be slightly parted and dry, and may appear somewhat cyanotic. In children suffering from lesions of the mitral valve of the heart it is very common for some blurring or indistinctness of the features to be present.

One of the most characteristic facial expressions that we meet with is that of typhoid fever or fevers of a typhoid type. The face is dull and expressionless; the teeth are covered with sordes, which become brown and blackish by exposure or by discoloration from medicines and foods; the lips are often moved in a low muttering delirium; and the whole appearance is that of apathy. Even when

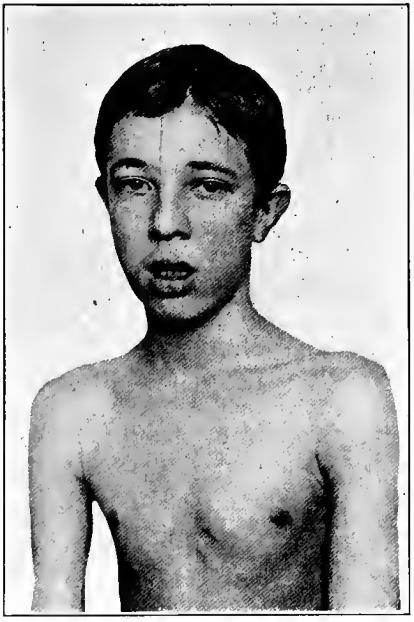


FIG. 1.—Mouth breather, from obstruction of the pharynx; open mouth; vacant expression; pinched nostrils; dull eyes; drooping eyelids; round shoulders.

spoken to, the face of a patient suffering from enteric fever rarely lights up in response to the greeting.

Equally, if not more, characteristic is the facial expression of acute peritonitis. The upper lip is drawn up in such a way as to show the teeth, and the expression of anxiety and nervous unrest is well developed. Similarly in abdominal pain due to other causes than peritonitis there is often a twitching of the muscles of the lip and about the eye which is quite typical. This twitch is said by Fothergill to be peculiar to pain below the diaphragm, and he is

also responsible for the statement that it is best seen in the face of the parturient woman in the second stage of labor.

The facial expression of hysteria may be apathetic, or it is that of devotion, rage, or grief, and these expressions are fixed if the patient be cataleptic. If she is not cataleptic, not infrequently one expression may succeed the other, or in their place there comes that curious smile or vacuous expression of the face which is so characteristic. It should be remembered, however, that this vacant fatuous look may occur in women suffering from the early stages of disseminated sclerosis and in children with chorea. Then we have the elated facial expression of general paralysis of the insane, the excited look of acute mania, the beaten, weary, careworn look or apathetic glance of nervous exhaustion, and the hopeless expression of melancholia.

The face of paralysis agitans, sometimes called the "Parkinsonian visage," is distressed and pathetic, and yet somewhat intense. (See chapter on the Hands and Arms, that part on Tremor.)

A pale, puffy face, generally looking worn and weary, may be seen in cases of chronic or subacute renal disease. In children there is often in this condition a peculiar transparent or pearly look in the lower eyelid, so that it seems somewhat pellucid. Great swelling or edema of the face is seen in erysipelas, dropsy (Fig. 2), and simple inflammatory swelling. (See chapter on the Skin.) In trichiniasis the eyelids are often swollen early in the disease, and then recover their normal appearance only to become swollen again later in the malady.

The facies of exhausting disease about to produce death is very characteristic, and is seen frequently in cholera and in tuberculosis of the lungs or any state associated with profound collapse, such as internal hemorrhage. It is accompanied by pallor, cold extremities, and difficult breathing. This is called the "Hippocratic face," and is peculiar in the sinking-in of the temples where the jaw muscles are inserted; the eyes are sunken, and around them are great hollows, so that the infra- and supra-orbital ridges become greatly accentuated. The eyelids are slightly parted, the cornea somewhat glazed; the nose pinched, its skin drawn; and the lower jaw somewhat dropped. Such a facial expression, if typical, is a sure forerunner of dissolution.

When the face bears a sleepy, listless expression, the forehead being devoid of wrinkles, and there are present faulty movements of the lips, which cannot be approximated, as in whistling, and at the same time the patient is unable to close the eyes entirely, although the lids droop, the physician should think of the possibility of these being the early symptoms of what has been called the "faciohumeroscapular" type of muscular atrophy. (Landouzy and Déjèrine.) The

disease, as its name implies, speedily involves the scapulæ and arms after affecting the face, and exophthalmos is often present. This form of muscular atrophy lacks the fibrillary twitchings seen in spinal progressive muscular atrophy, and there are no changes in electrical excitability, except that owing to the loss of muscle fiber the reaction is feeble. The facts that more than one member of the family is affected and that the disease is of long duration, added to these signs, render the diagnosis easy. It is a rare disease.

An appearance of the face almost identical with that just described is seen in Friedreich's ataxia, and is often one of the earlier manifestations of the disease; but the presence in Friedreich's ataxia of

FIG. 2.—Face of a patient with general anasarca due to chronic parenchymatous nephritis. (From a patient in the author's wards, Jefferson Medical College Hospital.)

the ataxic gait, the jerky articulation, nystagmus, loss of knee-jerks, and absence of muscular atrophy separate it from the Landouzy-Déjèrine type of muscular atrophy just described as faciohumeroscapular atrophy. (See Ataxia in chapter on the Feet and Legs.)

The facial expression of cretinism is exceedingly characteristic. The nose is broad and flat, the eyelids are swollen, the lips greatly thickened, and the enlarged tongue lolls out of the mouth, from which saliva constantly dribbles, while the waxy skin and subnormal temperature of the body, with a poor circulation, slow respiration, and mental hebetude, complete the symptom group. There

is nearly always in well-developed cases marked lumbar lordosis (Fig. 3).

The facial expression of myxedema is heavy and listless, as a rule. (See chapter on the Skin.)

In certain forms of leprosy the face often becomes leonine, or lion-like in appearance.

Facial Deformity.—Facial asymmetry is sometimes seen as a congenital defect, and curiously enough is often developed in children who suffer from congenital wryneck. This is not to be confused with that extraordinary affection called facial hemiatrophy, which

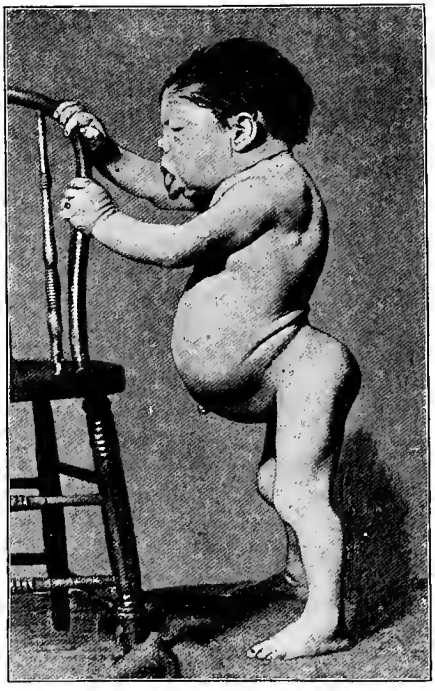


FIG. 3.—A cretin. (Dercum.)

usually begins in childhood in one spot, and slowly proceeds until one side of the face, sharply outlined from the other, becomes wasted in its skin, muscles, bones, color, and hair. Even the eye may be sunken and shrunken. Rarely this wasting extends over the whole of one side of the body, and still more rarely is bilateral.

Sometimes in facial hemiatrophy the wasting is accompanied by painful twitchings, which increase with mental excitement. More rarely there is decrease in the acuity of taste and hearing on the affected side, while myosis, sweating, or excessive dryness of the

skin may also be found on this side. Such symptoms as the last show involvement of the sympathetic nerve fibers. The changes are probably due to disease of the fifth (trifacial) nerve.

As to whether circumscribed scleroderma (morphea) and facial hemiatrophy are identical—that is, whether the first is a well-developed form of the latter—is not decided. Hyde apparently regards them as identical. (See chapter on the Skin, Scleroderma.)

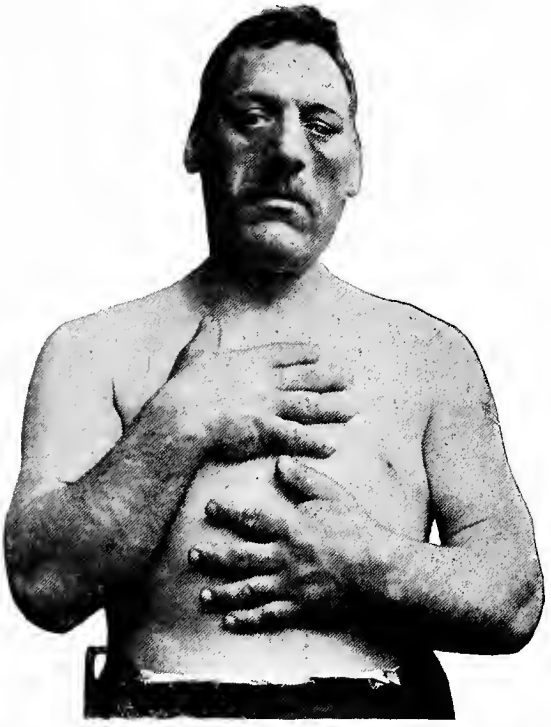


FIG. 4.—Acromegaly, showing the large face and hands. (Dercum.)

Even more rare than facial hemiatrophy is facial hemihypertrophy, one side remaining normal in size and the other becoming gigantic.

The massive face of a person suffering from acromegaly is very characteristic (Fig. 4). The face has a full-moon broadness in myxedema. The enlargement of the bony parts of the skeleton, the kyphosis, and the comparative muscular feebleness of acromegaly aid in the diagnosis of that disease, for in myxedema there is no true bony enlargement. The face in osteitis deformans is shaped like a triangle with the base upward, and the shafts of the long

bones become weakened, and their surfaces roughened from periosteal deposits. (See chapter on the Hands and Arms.)

Unilateral Facial Paralysis.—Very notable changes in the face are produced by paralysis, the palsy being, as a rule, unilateral and depending upon central or peripheral nerve lesions for its cause. Smiling, when unilateral paralysis is present, results in the drawing back of only one corner of the mouth (on the well side), and whistling or the pronunciation of labial sounds is difficult or impossible. The cheek of the paralyzed side is often puffed out with each expiration, but the wrinkling of the skin is on the side of the face which is not paralyzed, owing to contraction of the muscles which are unopposed.

(For a description of the general anatomy and physiology of the nervous tracts involved in paralysis of the face and elsewhere, see chapter on Hemiplegia.)

Unilateral paralysis is, as already stated, the form of facial paralysis most commonly seen, and is generally due to injury of the *facial nerve trunk*. The lesion producing the paralysis may, however, be peripheral—that is, in the nerve itself—or central, that is, in the pons or the cerebral cortex. The former variety is the most common, provided the paralysis is purely facial, and it is usually due to inflammation of the nerve sheath as it passes through the stylomastoid foramen, the loss of function being due to pressure on the axis cylinders owing to the presence of swelling in so limited a canal. Such an attack will generally be found associated with a history of exposure to cold or injury by a blow, or with that of middle-ear disease with caries of the petrous portion of the temporal bone following otitis, which inflammatory process causes pressure on the nerve. It is not necessary for the otitis to be suppurative or for caries to exist in all cases, for it seems probable that by the extension of inflammation along the chorda tympani such a paralysis may result. If the disease be in the petrous portion of the temporal bone, in addition to paralysis of the muscles of expression there will also be loss of taste in the anterior part of the tongue due to involvement of the chorda tympani. The mouth is dry, owing to a lack of saliva, the salivary gland being paralyzed, and there may be deafness from paralysis of the stapedius muscle. Still more rarely facial paralysis results from swelling of the parotid gland or from a tumor in its neighborhood, and it may occur as the result of pressure by growths at the base of the brain, syphilitic or otherwise, from fracture of the base of the skull involving the petrous portion of the temporal bone, and very rarely, when the disease occurs in the newborn, from hemorrhage from the cerebellum during birth, or from pressure of forceps. (See below.) Finally, paralysis due to a peripheral lesion of the nerve may result from neuritis, and from primary hemorrhage into

the nerve sheath or into the stylomastoid canal. Facial paralysis may also arise from locomotor ataxia, the lesion being in the pons, and from hysteria. All these forms are very rare, comparatively speaking. The cerebral or medullary lesions which produce unilateral facial paralysis usually result from hemorrhage and tumor.

The determination that facial paralysis is due to a *peripheral neuritis* or pressure may be impossible at the first visit of the patient, if this visit is made, as it usually is, within a few hours of the onset of the malady; but the peripheral form separates itself from facial paralysis of cerebral origin in the course of ten days or two weeks, for, if the nerve is inflamed or pressed upon in the foramen, the muscles of the face speedily undergo degeneration, because they are cut off from their trophic centres.

In the *cerebral form,* on the other hand, the trophic changes do not occur, and the reactions of degeneration fail to appear, because trophic impulses can still reach the facial nerve trunk and the muscles which it supplies. In other words, electrical response in the paralyzed side remains normal in centric lesions and is lost in peripheral lesions. The only other conditions in which there can be developed the reaction of degeneration and the lesion not be in the nerve trunk or foramen is when there is a tumor at the base of the brain involving the facial fibers below the facial nucleus or destroying the nucleus itself.

Very rarely in cerebral facial paralysis is the loss of power as complete as it is in the peripheral form. Again, in cerebral facial paralysis the eye on the paralyzed side can usually be closed and the forehead wrinkled, whereas in the peripheral form it cannot. Why this should be so is not clear, unless it is that in the muscles used commonly in pairs, as in those of the forehead, there is an adequate nerve supply through direct non-decussating tracts which innervate the muscles. When facial paralysis has associated with it none of the signs of peripheral wasting, and none of the remote causes of hemorrhage, embolism or thrombosis, such as result from impaired bloodvessels or a diseased heart, and when the paralysis comes on gradually (though it may be sudden from surrounding inflammation), the condition is probably due to cerebral tumor. This diagnosis is confirmed by the gradual spread of the paralysis to other parts, as the arm and then the leg on the same side of the body, and by the development, often before each additional spread of the paralysis, of a convulsion.

The facial paralysis resulting from *tumor at the base* of the brain differs from that due to cerebral tumor or hemorrhage by the fact, already stated, that the reaction of degeneration quickly develops in the paralyzed part; that the parts supplied by the upper branch of the facial are often quite as much para-

lyzed as are those supplied by the lower branch, which is rare in the cerebral lesion; and there will commonly be found other evidences of a growth which, in a region so densely filled with important centres, speedily affects other functions. Thus in association with this form of facial paralysis there will nearly always be found paralysis of the oculomotor nerve, causing ptosis, a moderately dilated pupil, and external squint, and there may also be paralysis of the abducens or sixth nerve, which causes internal squint by paralysis of the external rectus muscle. (See Ptosis.) The optic nerve may show choked disk, and there may be disturbance of vision. (See chapter on the Eye.) If the tumor is large, or is so placed as to involve the facial fibers for both sides as well as those of the oculomotor, abducens, and optic nerves on both sides, all these symptoms become, of course, bilateral.

Facial palsy associated with deafness may indicate *cerebellar tumor*, the diagnosis of this cause being decided by the other cerebellar symptoms, such as the peculiar gait. (See chapter on Feet and Legs.) Such growths are not uncommon in children.

Sometimes very shortly after birth the child is seen to have a facial paralysis resulting from *pressure by the forceps*, which have slipped and injured the facial nerve, or have caused an extravasation of blood into the neighborhood of the parotid gland, thereby causing pressure on the nerve. The prognosis is usually favorable if due to such causes; but if the forceps have caused facial palsy by producing a cerebral hemorrhage, the outlook is bad.

The possibility of facial paralysis being due to *hysteria* should not be forgotten. The loss of power under these conditions may be unilateral or bilateral, generally the former. Its association with the symptoms of hysteria described in the chapter on the skin, and elsewhere in this book, will aid in making the diagnosis.

There yet remain to be considered several forms of facial paralysis unilateral in character yet associated with paralysis elsewhere. These are as follows:

Unilateral facial paralysis very rarely occurs in association with monoplegia in *acute anterior poliomyelitis*. So seldom does it occur in this connection that it has been denied an existence. Often it is but temporary, while the monoplegia of the arm is permanent. It occurs more commonly with the disease in adults than in children.

Facial paralysis with arm paralysis of the same side, followed in a short time by paralysis of the leg of the opposite side, is quite a characteristic symptom of *syphilitic arteritis* at the base of the brain.

Crossed paralysis—that is, paralysis of the face on one side, and of the arm and leg on the other—is due to a *lesion in the pons* above the decussation of the pyramids and below that of the facial fibers

(Fig. 5). Thus it is seen in this figure, on the left side, third inscription, that the lesion in the pons cuts off the motor fibers in the place indicated, thereby causing the distribution of the paralysis just named. (See also chapters on Hemiplegia and on the Arms and Hands.)

Sometimes the muscles supplied by the facial nerve escape paralysis, but those of the jaw—namely, the masseters and temporals—

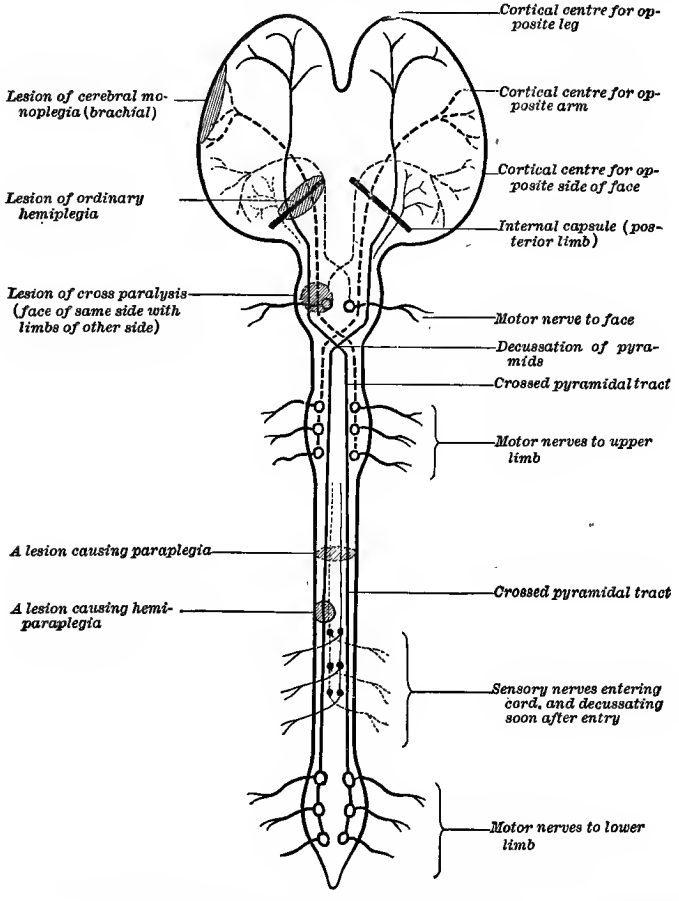


FIG. 5.—Diagram to show the general arrangement of the motor tract and the effect of lesions at various points. (Ormerod.)

become paralyzed either bilaterally or more commonly unilaterally. This is a rare affection, and depends upon paralysis of the inferior maxillary branch of the trifacial nerve. This may be due to pressure produced by growths or inflammatory processes at the *base*

of the skull. It may also occur as the result of hemorrhage into the medulla, or from *progressive bulbar paralysis.*

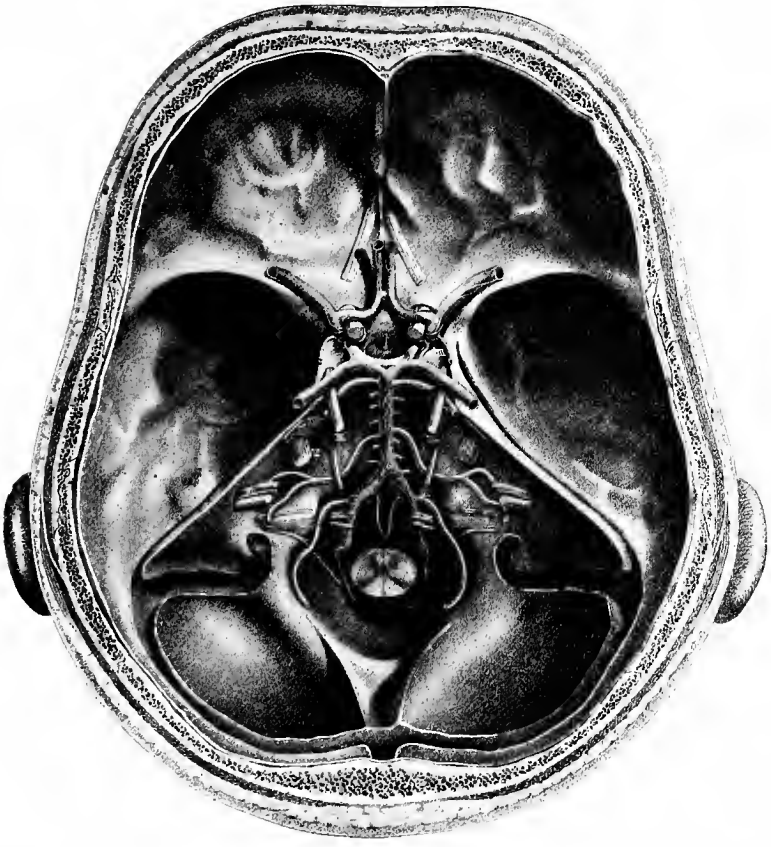
Ptosis.—In connection with the subject of facial paralysis that of ptosis, or drooping of the upper eyelid, must be considered (Fig. 7). It depends upon loss of function of the oculomotor nerve or its nucleus. Ptosis is a symptom of the greatest importance, first, because it is so readily recognized; second, because it is a source of great annoyance and alarm to the patient; and, third, and more important, it often aids us greatly in localizing lesions.

The presence of this symptom should call to the physician's mind the various causes which produce it. In the first place it sometimes occurs as a *congenital defect,* and in such a case the history of the patient renders the diagnosis easy.

If a congenital defect is not responsible for the ptosis it must depend upon disease affecting the *oculomotor nerve itself or its nucleus.* If the disease be sufficient to cause entire loss of function, we find, in addition to ptosis, that there is paralysis of all the external muscles of the eye except the superior oblique and external rectus, and in addition there will be a moderately dilated pupil, which will not contract, and paralysis of the ciliary muscle—that is, loss of accommodation. The eye can be moved outward by the action of the external rectus, and a little downward and inward by the superior oblique. Diplopia is present, and a little exophthalmos may be present owing to the action of the superior oblique, which presses on the ball. If the lesion be in the oculomotor nucleus, the near position of the nuclei of the fourth and sixth nerves will probably cause them to be affected also, thereby causing a general ophthalmoplegia (Fig. 6). If the lesion is not nuclear it is due to disease in the nerve itself, as already pointed out. If this is the case the lesion is probably due to pressure in the *cavernus sinus* or to periostitis of the bones forming the sphenoidal fissure through which the nerve passes. (See Plate I.) Sometimes, however, the paralysis of the nerve may be only partial, so that the external muscles of the eyeball escape, and only ptosis and a dilated pupil are present. Very rarely ptosis results from a *cerebral hemorrhage,* without the other signs of oculomotor paralysis being present. That is to say, the branch of the oculomotor which supplies the levator palpebræ is affected, while the branches supplying the external and internal ocular muscles escape.

If there is a history of a cerebral attack resembling a mild apoplexy, and a unilateral ptosis is present, the lesion is probably in the *cortical centre* for the oculomotor nerve in the angular gyrus just below the interparietal fissure. The lesion is, of course, upon the side of the cortex opposite the ptosis. Such a case is very rare.

PLATE I.



Base of Skull, showing the Oculomotor Nerves (III) Passing through the Sphenoidal Foramen. (Modified from Arnold.)

The other cranial nerves are also numbered, and it is easy to see how an inflammatory exudate at the base might involve many of them.

III. The oculomotor nerves. IV. The pathetic nerves. V. The trifacial nerves. VI. Abducens. VII. The facial nerves.

A fourth cause of ptosis is due to an affection of the *sympathetic nerve*, and is sometimes called pseudoptosis. There are associated symptoms of vascular dilatation, with redness and swelling of the skin of the affected side, elevation of temperature in that part, contraction of the pupil on the affected side, and apparent shrinkage of the eye into the orbit. This form of ptosis results from the paralysis of the unstriped muscular fibers of Müller which exist in the orbital fascia, for as these muscular fibers aid in holding open the lid their

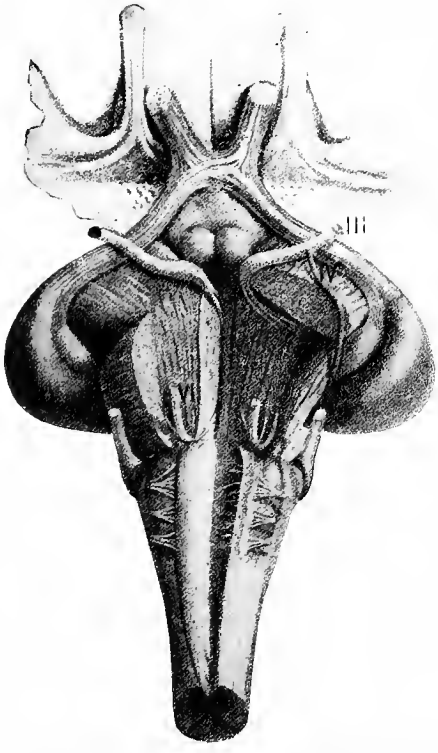


FIG. 6.—Showing the nearness of origin of the oculomotor (3), pathetic (4), and abducens (6). The roots of these nerves are shown by an incision which has divided the pons. III. The third nerve, arising from several roots. IV. The fourth nerve. VI. The sixth nerve, arising from three roots. (Modified from Arnold.)

paralysis results in partial ptosis. Nothnagel asserts that such symptoms occur with lesions in the corpus striatum.

A fifth cause of ptosis is reflex irritation usually through the fifth nerve. This is probably due to an inhibition of the oculomotor centre. It is usually only transient.

Sixthly, it is not uncommon in cases of *nervous syphilis* for so-called alternate ptosis to develop (Fig. 7). First one eye is affected by ptosis and then the other just as the first begins to improve or recover.

Ptosis has been known to complicate tetanus, probably as the result of reflex irritation of the fifth nerve.

Ptosis, either unilateral or bilateral, may arise from hysteria and *idiopathic muscular atrophy*. If from *hysteria* the diagnosis can be made from the age, sex, and history of the patient, from the presence of hysterical sensory changes described in the chapter on the Skin, and from the fact that there is a tendency to spasm of the orbicularis muscle when the patient is made to look up. This contraction of the orbicularis proves that there is no true paralysis of the levators. If the ptosis is bilateral and hysterical the head is tipped back when the patient is told to look up.

FIG. 7.—Ptosis in a case of alternate hemiplegia of syphilitic origin. (Dercum.)

Single or double ptosis is by no means a rare symptom of *locomotor ataxia*, and is often associated with other evidences of oculomotor palsy. Sometimes diplopia due to this disease is the first symptom complained of, and the patient may state that the diplopia comes and goes.

Bilateral ptosis, like unilateral ptosis, may arise from *tuberculous* or *syphilitic changes* about the base of the brain owing to pressure on the cranial nerves (see Plate I), or it may be congenital, or if transient be caused by poisoning by gelsemium or conium. It is also seen in slight degree in feeble, overworked women, particularly in the early morning on awakening.

Again, it is not very rare to see slight drooping of both lids in all the members of a family, in which case the condition is usually most

marked in the women, and is to some extent combated by the frontal muscles, which, in contracting, make the patient frown and draw up the eyebrows. (See chapter on the Eye.)

Ptosis, with hemiplegia of the face and limbs, on the opposite side of the body, associated it may be with hemianesthesia, is due to a lesion in the *crus cerebri*, provided the two sets of paralysis occur simultaneously, otherwise they may be due to two separate lesions. (Hughlings Jackson.)

A very rare condition, of which there are but 27 cases on record according to Darquire, is *recurrent paralysis* of the oculomotor nerve on one side. The attack begins with violent pain on one side of the head, nausea, and vomiting, and these symptoms are followed by ptosis, external strabismus, mydriasis, paralysis of accommodation, and crossed diplopia. It is seen most frequently in women, but may date from as early a period of life as eleven months. The attacks may last for a few weeks, and recur often or only after a lapse of many years.

As already stated, a form of alternate ptosis sometimes also develops in syphilitic persons.

Bilateral Facial Paralysis.—Bilateral facial paralysis is a rare condition, and when it occurs can only be due to a bilateral lesion in the cerebrum, to acute bulbar paralysis, to progressive bulbar paralysis, to a lesion in the pons just where the facial fibers decussate, to bilateral disease of the pons owing to disease of the basilar artery, syphilis at the base of the brain producing a tumor or inflammatory thickening, very rarely to bilateral inflammation of the stylomastoid foramina, resulting from cold or double otitis, from toxic multiple neuritis, but not from that toxic neuritis due to alcohol. Very rarely bilateral facial paralysis results from multiple neuritis in its diphtheritic form.

The development of bilateral facial paralysis due to a double cerebral cortical lesion never occurs without evidences of paralysis elsewhere in the body, such as monoplegia or hemiplegia.

The bilateral paralysis of the facial nerve in *acute bulbar paralysis* is characterized by the limitation of the paralysis, as a rule, to the neighborhood of the lips, by dysphagia, lingual paralysis, affected speech, paralysis of the ocular muscles, and a rapid pulse. This disease is very rare, and depends for its existence upon an acute inflammation or myelitis of the medulla oblongata.

When due to *progressive bulbar paralysis* (glossolabiopharyngeal paralysis) the paralysis is confined chiefly to the lips, and is associated with alterations in the tongue (see chapter on the Tongue) and speech, with tremor of the tongue and stiffness of the lips. The mouth stands half-open, the lower lip is pendulous, and the patient's

expression is that of a person about to burst into tears. The symptoms of glossolabiopharyngeal paralysis may, however, be exactly reproduced by *diphtheritic paralysis*, with this difference in prognosis: the first type always die and the second type usually get well.

In making a diagnosis of bulbar paralysis it should be remembered that another condition exists in rare instances in which no definite pathological changes can be found in the nuclei in the medulla oblongata, and yet many of the symptoms manifested by the patient are identical with those of glossolabiopharyngeal paralysis (true bulbar paralysis). This condition has been called "*asthenic bulbar paralysis*," and in it we find, as early symptoms, that the muscles of swallowing and of speech become easily tired on exertion, showing failure of the nuclei of the fifth nerve; that defects in articulation and speech are developed, indicating disorder of the nuclei of the ninth and tenth nerves; and clumsy movements of the tongue are present, which is a sign that the nuclei of the hypoglossal and twelfth pair are involved. These symptoms are practically identical with those of true bulbar paralysis. What are the symptoms which by their presence in the true disease and their absence in asthenic bulbar paralysis aid us in separating the two affections? The answer to this question is that the drooling of saliva, the atrophy of the tongue, lips, and extremities, the fibrillary twitching of the affected muscles, and the loss of electrical irritability in these muscles, all of which symptoms belong to true degenerative bulbar paralysis, are not to be found in the so-called asthenic form. There is, however, in the latter disease a condition rarely found in the degenerative form, namely, paralysis of the oculomotor, the lower facial, and the inferior division of the fifth or trifacial nerve, causing dilated pupils, diplopia (which, however, is not accompanied by strabismus), and ptosis (from the oculomotor failure), facial paralysis about the mouth (from facial nerve failure), and loss of expression about the eyebrows and forehead (due to facial and trifacial failure). Whether the diagnosis be true degenerative bulbar paralysis or the asthenic form just discussed, in both the prognosis is most unfavorable. Indeed, the asthenic form is often the more rapidly fatal of the two. In the latter the nuclei in the pons are probably always involved, but, as already stated, no pathological changes have been demonstrated in any of these nervous centres.

A very rare affection is oculofacial paralysis, which is congenital or develops in childhood, and is chronic. There are present paralysis of the facial muscles and ptosis.

Facial Spasm.—Spasm of the facial muscles may result from functional and organic disease, and occurs far more frequently in women than in men. The cause of the functional forms we do not

understand, as they occur in neuropathic persons and about the climacteric period. Rarely the spasm arises from reflex irritation through the trifacial nerve, resulting from a decayed tooth or a cause in the eye or in the skin. *Habit spasm* arises from an acquired trick. The organic causes are many. Thus there may be an irritative lesion of the facial nerve trunk or one in the cortical centre for the face, a tumor pressing on the nerve at its point of origin, or an aneurysm of the vertebral artery. The spasm may be confined to one side or distributed over both sides, and may be clonic or tonic in type. Sometimes it occurs only on attempted movement; in other cases it is constant. The clonic form is the more common.

Spasm of the face is seen in chorea, convulsive tic, blepharofacial spasm, in tetanus, meningitis, and epilepsy. When due to *chorea* it nearly always is clonic or twitching, as it is also in convulsive tic and habit spasm, but in tetanus, meningitis, and epilepsy it is generally rigid or tonic. In chorea the spasm is most marked about the corner of the mouth and the eyebrow or eyelids. The movements of *convulsive tic* are exceedingly sudden, darting across the face and involving all the muscles supplied by the facial nerve. As a rule this affection is unilateral. These spasmodic movements of convulsive tic may be almost constant or appear in paroxysms, and rarely the muscles of the jaw, the neck, and tongue are affected. The disease depends upon a disorder of the facial nerve, or its centres, which is not understood. The prognosis is bad so far as cure is concerned. Spasm of the levator palpebræ superioris muscle is sometimes seen as a symptom of exophthalmic goitre. It is called "Abadie's sign."

In blepharofacial spasm there are paroxysmal spastic contractions of the orbicularis palpebrarum and other facial muscles. The spasm often tightly closes the lids. Generally in children there is also photophobia with the spasm of the eyelids, which is often tonic in character and generally bilateral. This condition has associated with it what have been called "Graefe's spots,"[1] namely, the presence of spots near the supra-orbital foramen or over the vertebræ, which when pressed on cause sudden relaxation of the spasm. These should always be sought for, as they aid in giving relief to the patient.

Spasmodic movements about the eyes such as have just been described are sometimes paralleled by what is called *nictitating* or clonic spasm, which is probably due to some undiscovered cause of reflex irritation.

[1] This term should not be confused with the more common term "Graefe's sign," used to indicate the condition in exophthalmic goitre, in which the lids fail to follow the eyeballs when the patient looks down.

The development of facial spasmodic twitching accompanied by a sudden burst of explosive speech, repeating the last word heard or said by the patient in conversation (called echolalia), or the sudden bursting out with some blasphemous or filthy word (called coprolalia), is sometimes seen in neurotic adults or children, and is often associated with perversion of moral sense. It is called by Gilles de la Tourette "Maladies des tic convulsifs," but this is an unfortunate term, because it is apt to be confused with ordinary convulsive tic of children or adults. (See Electric Chorea and Myoclonus Multiplex in the chapter on the Hands and Arms.)

In *tetanus* the muscles of the jaw, the masseters and temporals, are first involved in the tonic contractions, and these are followed by rigidity of the muscles of the neck and body. Often the *risus sardonicus* is marked from the first, and the face soon looks like that of an old man owing to the muscular contractions.

In *meningitis* the characteristic symptoms which label the malady render facial spasm a comparatively unimportant symptom, and in *epilepsy* the convulsive seizure soon makes easy the diagnosis of the cause of the facial spasm unless the epilepsy is limited in its character, when the history of the presence of an aura, or of unconsciousness, or biting of the tongue may be discovered.

Spasm or contractures of the muscles of the face sometimes follow facial paralysis as recovery begins, and the contractures involve the formerly paralyzed muscles, whereas in paralysis in the limbs the contractures generally take place in the muscles which are not paralyzed. Sometimes these contractures in the face are permanent, and are due to incomplete restoration of the functions of the muscles affected.

Care should be taken to remember that not very uncommonly contractures in the muscles of the face result from hysteria, and that they are often on the side opposite the facial paralysis if the latter exists.

Active spasm of the muscles of the face may follow exposure to cold, and it sometimes follows the paralysis due to this cause, or, in other words, is a sequence of Bell's palsy.

THE HEAD AND NECK.

In examining the head we look for variations from the normal in its shape, its fontanelles if the patient is a young child, the position in which it is held, and its movements as governed by the cervical muscles. Of the last I shall speak first, although they have been mentioned under the heading of wryneck. The head is moved

abnormally in nodding spasm, in chorea, and in tetanus and strychnine poisoning. It is also thrown backward and forward or from side to side in epilepsy, and in hysteria or in the convulsive seizures occurring in young children.

Nodding Spasm.—Nodding spasm of the head, depending upon somewhat rhythmical contractions of the sternomastoid and trapezius muscles, is sometimes seen in half-fed or rickety children. It also occurs in hysterical women, and in men who are not hysterical. The nodding may be slow and infrequent, only coming on with excitement, or it may be practically constant. It always becomes worse when the patient is examined, and may be so rapid and forcible as to seem almost severe enough to shake the head off the shoulders. Often the muscles involved will be found very rigid.

If the spasmodic movement be not rhythmical, as it usually is in nodding spasm, and yet be more or less constant though irregular, the cause is probably chorea minor if it is present in a child, or it may belong to the irregular movements of adults classed under the various forms of tic or choreiform spasm. (See chapters on the Hands and Arms and on Convulsions and Spasms.)

Wryneck consists in a drawing of the head to one side by spasm of the sternomastoid muscle, and at the same time the head may be tilted backward or forward according to the accessory muscles which may be involved in the spasm.

Sometimes a tonic spasm of the sternomastoid muscle, produced by exposure to cold or due to a distinct nervous lesion, causes the head to be drawn down over the shoulder, or bilateral spasm of this muscle causes fixation of the head. If the cause be exposure, with resulting myositis, the history of exposure, combined with that of a sudden onset, will permit a correct diagnosis and a favorable prognosis, it being remembered, however, if the patient is a female, that hysterical spasm may be the cause. If hysteria is the cause, the history of the patient, the presence of alteration in her color fields and the other signs of hysteria can probably be elicited. (See chapters on the Eye and on the Skin.) On the other hand, if the contraction has come on gradually, after some injury or in association with some nervous affection elsewhere, it is probable that a true nervous lesion underlies the disorder.

If it is a tonic spasm the involved muscle is on the side toward which the head is drawn, and the muscle on the opposite side is seen to be prominent owing to its being stretched by its opponent. The chin is, moreover, directed upward and away from the affected side. Rarely the trapezius is the only muscle involved, in which case the head is drawn backward and toward the diseased side, or, if the sternomastoid and trapezius muscles are both involved the head is

tilted laterally and backward until the patient looks up in the air. Pain in the muscles only occurs from fatigue. This tonic spasm affecting the muscles which support the head can be separated from that occurring in tetanus by the fact that in tetanus there is a general diffusion of the spasm to other muscles, although in that form of tetanus called "head or cephalic tetanus" the diagnosis is more difficult.

Cephalic tetanus usually has the following diagnostic points: there is a history of infection, the character of the onset is sudden, there are trismus, difficult swallowing, respiratory disturbance, and facial paralysis with rare involvement of the ocular muscles. The spasm in cephalic tetanus is also often increased by movement or by the attempt to take food. Strychnine poisoning is also to be thought of, but the limited character of the convulsion excludes that condition. Should the muscles be affected by a clonic spasm the head is jerked about instead of remaining fixed.

Retraction of the head in children is an indication in many cases of serious brain disease, and commonly arises from a basal meningitis. It is to be remembered that some of these cases recover, though such a result is rare. Again, we should not forget that *caries of the cervical vertebræ* may cause this position, or that tender and enlarged glands in the neck may produce such a result. Sometimes, too, it occurs after falls without there being any other indication of meningeal irritation. Rarely in neurotic babies retraction of the head, as a temporary symptom, accompanies attacks of indigestion. Similarly in adults suffering from *cerebrospinal fever* the head is often held in a retracted posture.

The *posture* of the head may also aid us in diagnosis when no spasm of its governing muscles exists. Thus, chronic deafness in one ear may cause the patient to hold one side of his head further forward than the other, in order to catch the sounds he seeks with the good ear, and pronounced strabismus may cause a child to so carry its head as to improve its sight and avoid diplopia.

Persons suffering from great mental depression with a tendency to melancholia often sit for hours with the head bowed forward with the chin resting on the chest.

The changes from the normal in the *shape of the head* are to a certain extent considered in that part of this chapter dealing with the symmetry and appearance of the face, but there still remain to be discussed the changes in the shape of the head as a whole. These occur in acromegaly, osteitis deformans, and in hydrocephalus, microcephalus, rickets, idiocy, myxedema, and cretinism.

The head of *hydrocephalus* is greatly enlarged above the level of the ears, and this causes the face, already having a tendency to faulty

development, to look small and wizened. The eyes seem somewhat bulging, the orbital plates are oblique, and the back of the head is flattened. Sometimes in true hydrocephalus the fontanelle remains pulsating for a long period. Again, in true hydrocephalus choked disk is sometimes manifested quite early. (See Chvostek's and Trousseau's Signs.) In *microcephalus*, on the other hand, the head is small and often narrow. Technically, the term microcephalus is applied to idiots whose heads are less than seventeen inches in circumference. Nearly always the head of an idiot is abnormally formed.

The *cretinoid* head is large, heavy, and massive.

When a young child has unusually prominent parietal and frontal bones, which seem bulging, and there is a general resemblance in the shape of the skull to that of hydrocephalus, we suspect the presence of *rickets*. As a rule, the forehead is broad and high, the top of the head flat, and the shape of the head more round than in the genuine disease. Sometimes in such a child we find, in addition to these changes from the normal, spots of thinned bone in the occipital and parietal regions. These may be also somewhat softened, and this condition, called "craniotabes," is usually a sign of rickets which exists in association with infantile syphilis. Rickets is seen nearly twice as often in boys as in girls, and there is usually to be found deficient development of the bones everywhere, particularly in the ribs and legs. (For the rachitic rosary, see chapter on the Thorax.)

The condition of the *fontanelles* in young children is of importance in diagnosis. In the healthy child all the fontanelles save the anterior fontanelle close during the early weeks after birth, but the latter opening does not close entirely until the infant is about one year and a half old. During the first few months this fontanelle closes very slowly indeed, but after this time has elapsed its edges become rapidly approximated. The presence of other fontanelles in a child's skull after it is several months old indicates rickets, syphilis, hydrocephalus, or some intracranial growth producing pressure on the cranial bones, preventing their approximation. Generally, however, these minor fontanelles are not found open but closed, and the condition of the anterior fontanelle is the guide in diagnosis. In severe cases of rickets the anterior fontanelle remains open until the third or fourth year, and should the rachitic tendency be developed early in life the edges of the fontanelle may not only fail to be approximated, but may actually recede from each other. Sometimes if the edges of the fontanelle are found to be softer than usual the diagnosis of rickets can be so confirmed. If syphilis be the cause of the deficient bone development, evidences of this disease

in mucous patches about the mouth and anus may be found or a history of heredity adduced; while if the condition be hydrocephalus the fontanelle will be markedly bulging.

If the skin over the *fontanelle be found to be bulging temporarily* to a slight extent, the cause probably lies in some acute disease with fever, producing cerebral congestion; whereas, if permanent, and if the general dimensions of the skull are not increased, an intracranial growth may be the cause, or a cerebral hemorrhage, a purulent meningitis, or some cystic formation may be present, or sometimes a thrombosis of a cerebral sinus produces hydrocephalus and bulging. In other cases thrombosis causes sinking-in of the fontanelle. This difference in the tension of skin over the fontanelle aids us in separating the meningeal symptoms of pneumonia from those of true meningitis, for in the true form the scalp is tense and in pneumonia it is often retracted.

Marked *sinking in or collapse of the fontanelle* always indicates a grave condition arising from some disease which seriously weakens the heart and general circulatory system, particularly marasmus and cholera infantum. The other symptoms associated with this state are usually a sunken appearance of the eyes, slight duskiness of the face, a cool skin, and a rapid, feeble pulse. The patient is almost comatose, and there may be slight convulsive seizures. Such a condition has been called the "hydrocephaloid state," and has been confused with symptoms of cerebral effusion arising from tuberculous meningitis.

If there be marked diarrhea present the following table of Symes will serve to clear the diagnosis:

Hydrocephaloid State from Diarrhea.	Cerebral Effusion (as in Tuberculous Meningitis).
Diarrhea.	Constipation.
No ocular paralysis.	Ocular paralysis and squint.
No rise of temperature.	Slight feverishness.
No headache.	Headache (if old enough to complain).
No tension or bulging of fontanelle.	Bulging fontanelle.
No rigidity and	Rigidity and retraction of head in many cases.
No retraction of head.	

Sometimes in rachitic babies auscultation of the fontanelle will reveal a murmur, hemic in origin. This murmur can, however, occasionally be heard when no such disturbance of nutrition exists.

Excessive sweating of the head, producing a wet pillow, is often an indication of rickets when it occurs in a child.

A *swelling in the neck* in the median line, or on both sides of the median line, anteriorly, is probably due to *goitre*. If it is associated with cardiac palpitation and distress, exophthalmos, tremor, ner-

vousness, and depression of spirits, it is called *exophthalmic goitre* (Fig. 8). If these symptoms are absent, the condition is simply one of overgrowth of the thyroid gland.

Aside from swelling of the glands of the neck due to syphilis, Hodgkin's disease (see chapter on Fever), struma, and tuberculosis, there may be *enlargement of the parotid gland* on one or both sides, just in front of the ears and extending under the angle of the jaw. This swelling may be due to the specific inflammation involving

FIG. 8.—Exophthalmic goitre. (Meltzer.)

these glands, known as mumps, or be due to other infections, such as typhoid, typhus, and pyemic fever. If the latter be the cause, suppuration usually ensues. Rarely enlargement of the parotid glands follows trauma or disease of the abdominal viscera or pelvic organs. Sometimes the enlargement is chronic after the acute inflammation is passed by.

(For the movements of the head in epilepsy and hysteria, see chapter on Convulsions.)

CHAPTER II.

THE HANDS AND ARMS.

The general appearance of the hands and arms—The shape of the hands in disease—Spasms of the fingers—Tremors of the hands—Paralysis of the hands and arms.

Inspection of the Hands.—The appearance of the hand and arm often gives us valuable hints in the diagnosis of disease, chiefly by reason of variation in their shape, manner of movement, and general consistency; but as all these conditions vary widely in normal individuals, we can only regard distinct and well-marked alterations from the normal type as indicative of a definite disease. We can, however, often gather general information as to the patient from the hands, particularly as to his occupation; thus we see the smooth, soft hand of the professional man or clerk, the horny hand of the laborer, the blackened nails and skin of the machinist, or the blue-black dottings of the hand of the miner; and Hirt asserts that atrophy of the antithenar eminence often ensues in cabinetmakers, perhaps from the excessive use of the plane. Even when no pathological condition exists we are wont to regard the heavy and somewhat thick and clumsy hand as an evidence of a phlegmatic temperament, and the thin, wiry, dexterous hand as indicative of the nervous temperament. Similarly, we recognize as the hand of the strumous that one in which the fingers are slender between the joints and the joints themselves thick and clumsy, or, again, in persons with tuberculous tendencies, we see a slender, delicate hand, easily compressed and somewhat effeminate in type. Very commonly, too, in children who have developed heart disease in early life the hand becomes square looking, and the fingers are club-shaped through thickening at the tips. A similar clubbing also manifests itself in many cases of emphysema and chronic phthisis in adults, and unilateral clubbing with incurvation of the nails of one hand is sometimes seen in thoracic aneurysm.

From the appearance of the nails we can often gain important information; thus, whenever the color of the blood in the capillaries under the nails is dusky we know that a deficient pulmonary function exists or that the circulation is impaired, it may be from feebleness or from cold. In anemia the nails are often very pale, and Stephen Mackenzie has asserted that if pressure on the tip of the

finger completely empties the capillaries under the nails so that the appearance is pale the red corpuscles are present in only half the usual number.

White spots in the nail may be due to injury of the matrix by picking at the base of the nail, or be due to acute fevers producing trophic changes.

When the nails are *striated* and in longitudinal ridges the patient is often of the gouty diathesis, while transverse ridges may indicate arrest of nail growth through local injury to the matrix or the impairment of the general nutrition as the result of some severe systemic shock, such as a severe surgical operation or prolonged illness. Sometimes these marks result from a severe attack of gout, and Fothergill tells us that it took about seven months for such a mark to grow out of his nails. Ordinarily, this mark will be found about half-way up the nail three months after the attack. In hemiplegia or acute infantile palsy the growth of the nail of the paralyzed part is generally arrested, as can be determined by staining it and watching it from day to day to see if the stained part gradually moves away from the base. When the nails are distorted and thickened the cause may be local injury or peripheral neuritis, or any condition of the nervous system resulting in decided trophic influences, as in that rare condition syringomyelia.

Hypertrophy of the nails so that they are abnormally elongated is usually associated with thickening and the development of great fragility. The nail may even be spirally twisted (onychogyrophosis), or, if very wide, may cut into the skin and produce paronychia. These conditions may result from skin lesions, such as eczema or lichen ruber, at or near the matrix, or be due to syphilis, and Vogl asserts that mere thickening may arise from severe fevers. They may also be seen in cases of Raynaud's disease, or in sclerodactyle, and in cases of pulmonary osteo-arthropathy.

Atrophy of the nails may apparently arise from causes identical with those which produce hypertrophy, and Kaposi has seen the nails soft and membrane-like, with abscesses under them, from psoriasis of the fingers.

A diagnostic indication given by the fingers is seen in *dactylitis due to syphilis*. Similar deformity, often multiple, also occurs in scrofulosis or tuberculosis. In other cases this is replaced by an eruption on the skin of the hand characteristic of syphilis. Another indication is seen in the ulcers at the bases of the finger nails, with ecchymotic spots on the skin, produced by the chloral habit; and still another is the sores seen at the bases of the finger nails in persons who handle irritating drugs, such as elaterium. (See chapter on the Skin.)

Congested veins on the hand may indicate obstruction to the

venous circulation of the arm, or general lack of vascular tone and a feeble heart.

When the hand is *cold and clammy* the condition may be due to bromidrosis, or a local disturbance in innervation of the sweat glands. It is often seen in cases of so-called spinal irritation and nervous exhaustion. Excessive sweating of the hand is also often seen in cases of progressive muscular atrophy.

There are two sets of movements associated with the muscles of the wrist and hand which possess grave prognostic and diagnostic importance in the presence of exhausting fevers. The first of these is *twitching of the muscles of the forearm* (subsultus tendinum). It indicates severe and dangerous disease. The second is picking at the bedclothes. The grave import of this dangerous symptom, "*picking up the bedclothes,*" or carphologia, was recognized by Shakespeare in his description of the death of Falstaff: "After I saw him fumble with the sheets, and play with flowers, and smile upon his fingers' ends, I knew that there was but one way; for his nose was as sharp as a pen." Hippocrates has well emphasized the gravity of this symptom, for he says: "In acute fevers, in peripneumonias, in pleuritis, and in headaches the hands are moved to and fro about the face, seeking in the void, as if gathering bits of straw, picking at the coverings, or detaching objects from the walls of the room, constituting so many signs of a fatal termination."

The fingers are often *distorted* and twisted out of their normal position from the trophic changes which take place in gout and arthritis deformans (rheumatoid arthritis). In gout the deformity invades the small joints in particular, and in many instances appears most marked in the forefinger. Fixation and deformity of the fingers occur through the deposit of urate of sodium in large amounts about the joints in their tendons and sheaths, so that the fingers are as in splints. The knobs of urate of sodium appear as hard, white masses, and, if very superficial, as glistening masses, the surfaces of which often break down and allow the escape of material looking like powdered wet chalk. The joint surfaces themselves are not primarily much altered, but secondarily grave changes occur in them.

Very commonly in *gout* the only joints of the hand which are involved are the first joints of the fingers, a knob developing on either side of the knuckle (Fig. 10). The little finger in gout is often bent at an acute angle at the middle knuckle, so that it is held in an awkward hooked position. This is most commonly seen in women, while in men it is common to see forced flexion of the first phalanx of the middle finger into the palm of the hand, even when very little if any deposit of urates has taken place. This drawing down of the fingers is considered by Paget to be pathognomonic

of gout, although the patient will claim that it is due to the use of a cane, a hammer, or other extraneous cause.

Distortion of the hand with drawing of the finger or fingers into the palm may be due to *Dupuytren's contraction,* which results from burns or other injury to the palmar fascia.

While the history of the patient, the localization of the manifestations of the disease, and its character render a differential diagnosis between the hand of gout and that of arthritis deformans a possibility, it should not be forgotten that the deformities of gout may take every position assumed by those of arthritis deformans.

In *arthritis deformans* the distortion of the hand may be far more marked than in gout, for here there is not a splint-like deposit about the joint, but in its stead the development of exostoses on the edges of the articular surfaces, which at once lock and disjoint the fingers,

FIG. 9.—Arthritis deformans in a woman of twenty-one years. Characteristic distortion of ring finger on left hand and seal-fin hand on right side. (From the author's wards.)

while at the same time the opposite side of the joint may be partially absorbed, so that dislocation is still more readily produced. As a result there is sometimes developed what is called the "seal-fin hand" (also seen in cases of gout), a hand in which the digits are deflected chiefly toward the ulna, through the action of the extensor muscles, which are supplied with nerves which are reflexly irritated by the condition of the joints, and thereby cause spasm (Charcot) (Fig. 9).

The term Heberden's nodes is applied by some to the exostoses at the sides of the phalangeal joints met with in rheumatoid arthritis or arthritis deformans; by others, as by Duckworth, to somewhat similar gouty deposits. These are sometimes called "Haygarth's nodosities."

Chronic rheumatism may produce gradual changes in the shape of the hand chiefly through disuse and the alterations which it causes in the capsules and ligaments. The chief alteration is immobility or stiffness. Some persons believe that when the hand wastes it does so not from disuse, but through reflex nervous influences. Chronic rheumatism rarely, if ever, occurs in the hands alone, but when it does the joints are often swollen and somewhat tender, but never as hard as in gout.

The finger-joints are not commonly involved in *acute articular rheumatism*, certainly very rarely as the only manifestation of the disease. The inflammatory process is more apt to be about the

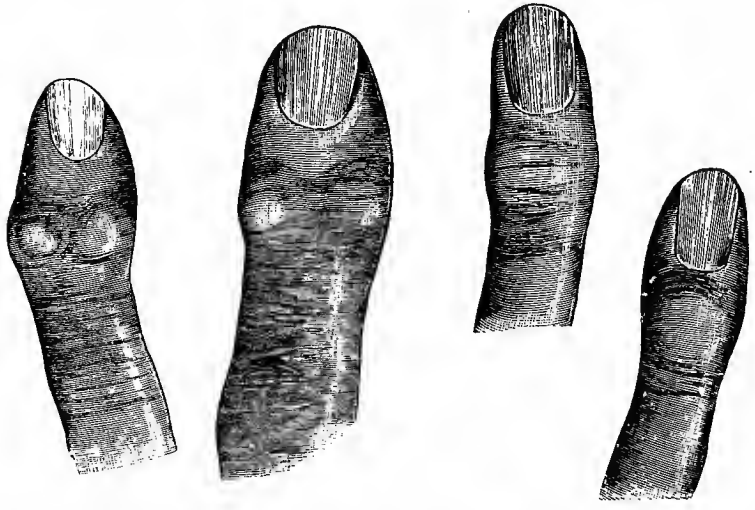


FIG. 10.

Heberden's gouty nodes. Illustrating common forms of terminal phalangeal deflection. Forefinger and little finger of a woman aged seventy years. "Crab's-eye" cysts over the joints are also depicted. (Duckworth.)

Nodular swellings (Herberden's nodes) due to gouty arthritis on the forefinger and little finger of a woman aged fifty years. (Duckworth.)

ball of the thumb, or in the wrist and carpal joint. The hand is seen under these circumstances as a clumsy, swollen mass, puffy, and exquisitely tender and hot. Sometimes it is quite red at the joints, but otherwise quite pallid, particularly in the puffy, edematous area on the back of the hand. The presence of intense local inflammation, the history of sudden onset, and the intense pain on movement readily separate acute rheumatism from chronic gout and arthritis deformans, and leave it to be separated from sprain, septic arthritis, and deep-seated inflammation of the hand proper. The first is excluded by the history, the second by the history and general lack

of evidence of gonorrhea or sepsis or purpura, and the third by the lack of accompanying general systemic disturbance and the absence of a history of traumatism or infection. In this connection it should not be forgotten that synovitis of the joints of the hands, wrists, and elbows sometimes occurs during the fall of temperature in scarlet fever, and is often not associated with any rise of temperature as a result of its development. The condition is sudden in onset and usually rapid in its course. The same state may exist in the joints of the lower limbs, but Marsden found it in the hands and wrists in 72 instances out of 100 cases, and only 25 times in the larger joints out of 100 cases. The condition usually appears, however, in rheumatic children and those with a rheumatic heredity, and is generally relieved by salicylates, so it is not a pure septic arthritis.

The nervous disturbances which change the appearance of the hands are very numerous.

Angioneurotic edema is not peculiar to the hand, although frequently involving this part of the body. It consists of a swelling varying in size from a dime to a silver dollar, which is not edematous in the sense that it can be pitted on pressure. This swelling, which may be multiple, red in color, or pale and waxy in appearance, lasts but a few hours or days, disappears, and often speedily returns.

Somewhat allied to angioneurotic edema is that condition of the hand (or toes) characterized by a white and waxy or slate color of the fingers, associated with coldness, swelling, and mottling of the skin, termed *Raynaud's disease.* Often this is a passing condition, but in its severe forms there is finally developed dry gangrene in the fingers involved. The conditions of the hand resembling it, from which it must be separated, are senile gangrene, in which the advanced age of the patient and the presence of diseased and thickened bloodvessels will enable us to decide on the latter as the cause; frost bite, in which the history of exposure will be of value, although exposure to cold often precipitates an attack of Raynaud's disease; ergotism, which can be discovered by the history of the patient having for a long time taken food which may have contained bad rye; leprosy, which will probably be seen more marked in other parts, and in the patches of which can be found the leprous bacillus; and alcoholic neuritis, of which we shall speak later. (See chapter on the Skin).

In that state known as *Morvan's disease,* or "pain anesthesia with whitlow," there is a slowly progressive loss of power in the hand, with atrophy and ulcers about the bases of the nails. Sometimes the terminal phalanges undergo necrosis, and enlargement of the fingers, through swelling, may be very marked. It is probable that this condition represents two separate lesions, namely, neuritis and syringomyelia, and it is an exceedingly rare disease.

Swelling of the hand, followed in some months by rupture of the skin, may, in a person from the tropics, mean *mycetoma,* which is, however, seen more commonly in the lower extremity as "Madura foot."

In addition to these trophic changes in the hand we have the so-called "spade-like" hand seen in myxedema, acromegaly, and the pulmonary osteo-arthropathy of Marie. In *myxedema* the deformity depends upon the alterations in the subcutaneous tissues, rather than on changes in the bones, so that the hand is swollen or boggy looking, but does not pit on pressure as in true edema. In *acromegaly* the enlargement is chiefly osseous, as it is also in *pulmonary osteo-arthropathy,* the formation being on a gigantic scale. In the latter disease, however, the hands and feet are alone affected, and the enlargement is not symmetrical. Further, this condition is nearly always associated with changes in the lungs, such as emphysema, tumors, and old bronchial troubles. The hands are not only greatly enlarged, but deformed, so that a side view of the finger tips reminds one of the shape of a parrot's beak, the nail being turned over the end of the finger. This is particularly well marked in the thumb.

The differentiation of pulmonary osteo-arthropathy from acromegaly is to be found in the fact that in the first-named disease there are no changes in the face, the skin, lips, or orbital ridges. Neither is there spinal kyphosis in the cervical region, although it may be present lower down. Again, in pulmonary osteo-arthropathy the long bones of the upper extremities are greatly enlarged in their epiphyses, while in acromegaly they are not so locally enlarged.

Alterations in the contour of the hand are, however, far more frequently produced by atrophic processes than by those which result in hypertrophy. They arise in cases of paralysis not only from wasting of the muscular tissues, so that hollows or sunken places occur, but also from the distortions caused by the contractions of healthy muscles, which, having no opposition as in health, speedily draw the bones of the hand into abnormal positions. In other cases the diseased muscular fibers may be spasmodically contracted, overcoming the resistance of the healthy muscles.

The *wasting of the hand* seen in old age, particularly in women, and in advanced phthisis, diabetes mellitus, and other conditions in which the tissues of the body in general lose their plumpness, is so universally distributed that a diagnosis of wasting from old age is not difficult. On the other hand, the wasting due to nervous lesions is generally not universal, but limited to single muscles or groups of muscles, the remaining portion of the hand having its normal appearance or being only indirectly influenced.

Under the name of "*clawhand,*" or "*main-en-griffe,*" we find

a deformity of the hand which is in itself very characteristic, although indicative of several causes which all operate in an identical manner. The back of the hand loses its normal convexity and becomes somewhat concave, the tendons on the extensor surface stand out in ridges, the proximal phalanges are drawn backward toward the wrist, while the second and third phalanges are drawn toward the palm of the hand (Fig. 11). Sometimes, however, the tips of the fingers are drawn toward the back of the hand. This deformity results from atrophy and paralysis of the interossei muscles and lumbricales, which are supplied by the median and ulnar nerves. The extensor communis digitorum and flexor digitorum produce a dorsal flexion of the first phalanges and a complete palmar flexion of the second and third phalanges. A certain amount of immobility is also caused by the fact that flexion of the hand is impossible in the fingers and almost lost at the wrist.

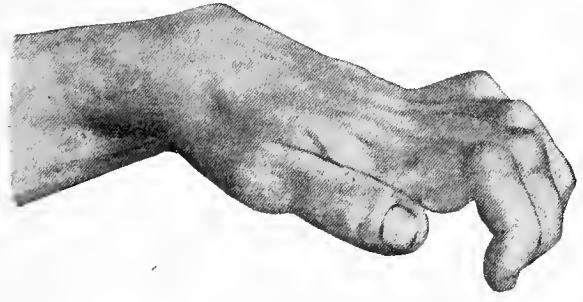


FIG. 11.—Clawhand. (Gray.)

The clawhand having been recognized, it remains to be decided what are its causes. It may be due to disease of the peripheral nerves (the ulnar and median), of the cells in the spinal cord, and of the cells in the cerebral cortex in the hand area.

Taking up for consideration paralysis of the median and ulnar nerves as a cause of clawhand, we find that the most common cause is a *neuritis* produced by some mechanical injury resulting from an accident, or from the following of some occupation in which, for example, the artisan presses his elbow constantly on some hard surface. The deformity may be, therefore, either unilateral or bilateral (generally the former), and there will be evidences of local injury, or a history which will indicate that the lesion is peripheral. Further than this, there will nearly always be found, in ulnar and median injury, sensory as well as motor paralysis; and Hirt asserts the remarkable fact that the clawhand may develop in cases in which sensory disturbances are the only evidence of median and ulnar difficulty—in other words, before motility is lost through paralysis.

(See chapter on the Skin, Anesthesia of the Skin.) Toxic neuritis very rarely, if ever, causes clawhand, as the musculospiral nerve is more commonly affected in this condition and the extensors become paralyzed.

There are several spinal causes of clawhand, the most important of them being *progressive muscular atrophy,* that disease in which there are atrophy and abnormal change in the anterior horns of the gray matter of the spinal cord, particularly in the cervical region (Fig. 12). It will be remembered, too, that the anterior nerve roots and motor nerves become involved in this process. As a result of

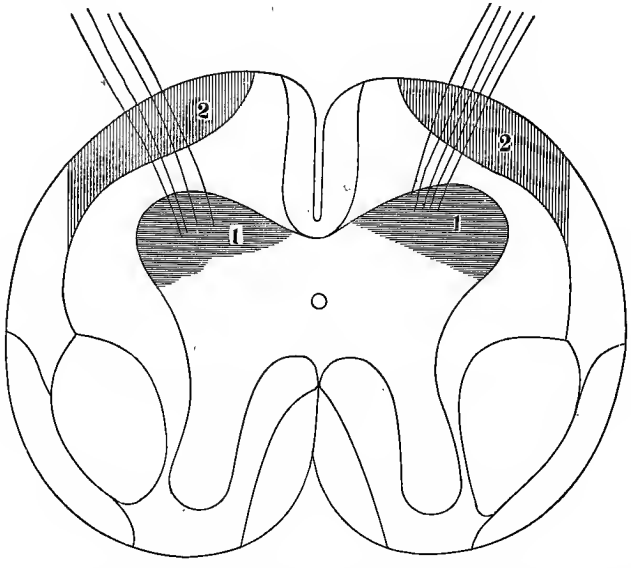


FIG. 12.—Areas of spinal cord involved in progressive muscular atrophy. The areas involved are the anterior horns of gray matter chiefly (shading heavy) (1) and the anterior lateral tracts (2) and anterior root zones. The anterior nerve roots which arise from the anterior horns also atrophy, and the atrophy extends all the way to the nerve plates in the muscles—along the nerves.

these changes, we have developed loss of power in the hand and arm followed by the development of a clawhand from wasting of the same muscles, as already described, the disease process being generally bilateral, but affecting the right hand and arm more than the left, as a rule. As progressive muscular atrophy often makes its first manifestation in these muscles, the hand affords much diagnostic information in suspected cases, and if the patient with this disease be watched as he unbuttons his coat, it will be found that he does not use his thumb and first finger, but pushes the buttons

or the edge of the buttonholes with the back of his fingers. The additional symptoms are pain or paresthesia in the affected parts prior to the wasting, and the spread of the paralysis, as its name indicates, from muscle to muscle (Fig. 13). Thus, beginning in the ball of the thumb it passes to the interossei and thence up the forearm and arm. Sometimes, however, the forearm muscles escape, and the shoulder muscles are attacked secondarily. Very rarely are the shoulder muscles first affected. Soon after this the dorsal muscles fail and lordosis begins, or the head falls forward on the chest. Finally, the respiratory muscles are attacked. The irritability of the muscles is increased, so that they contract if tapped, and fibrillary tremors constantly affect them in many instances. No vasomotor changes take place in the affected part, but, finally, the reactions of degeneration develop. The disease may last for many years.

Sometimes in *chronic poliomyelitis* in the adult a deformity somewhat like that of clawhand may exist, but this is a very rare con-

FIG. 13.—Hand and forearm in chronic spinal muscular atrophy, showing especially wasting of thenar and hypothenar eminences. (Dercum.)

dition, comparatively speaking, and is separated with difficulty from the clawhand of peripheral neuritis of a general and severe type. As the result of the *acute poliomyelitis of infancy*, we may also have the hand distorted by contractures, such as forced extension in paralysis of the flexors, forced flexion in the paralysis of the extensors, and clawhand in paralysis of the interossei, but in most cases of this disease the foot is the part involved in the disorder. In progressive muscular atrophy the atrophy often precedes the paralysis, whereas in poliomyelitis the paralysis precedes the atrophy, so that in the former the reaction of degeneration develops late, and in the latter develops early.

A somewhat claw-shaped hand is also sometimes seen in that very rare condition called Morvan's disease, but it has not the characteristic appearance of *main-en-griffe*, there being a slow symmetrical wasting of the muscles with a drawing of the fingers into flexion.

There are also analgesia and *painless whitlows*. It usually occurs in young and middle-aged males. Morvan's disease of the fingers, as already stated, may arise from a syringomyelia and neuritis, or neuritis alone.

Another spinal lesion producing great alterations in the appearance of the hand and arm, through wasting of the thenar and antithenar and interossei and the muscles of the arm, is *amyotrophic lateral sclerosis*. Here again the hand often shows the first manifestations of the disease in the loss of power of which the patient complains. The early symptoms of amyotrophic lateral sclerosis may closely resemble those of progressive muscular atrophy in the loss of power in the thumb muscles, but in this disease the reflexes are markedly increased in the affected muscles, whereas in progressive muscular atrophy they are lost, although fibrillary muscular twitchings may be caused by tapping. Again, the patient is usually manifesting some of the symptoms of lateral sclerosis when he comes before the physician, such as weariness, stiffness, and loss of power in the legs. (See chapter on the Legs, Paraplegia.) There are also exaggerated knee-jerks and ankle clonus, and wrist-jerk is marked.

FIG. 14.—Right hemiplegia, with contractures and retarded growth of arm. Onset of disease at eight years of age, following typhomalarial fever. (Sachs.)

Wasting of the muscles of the hand, causing distortion, may also be due to *syringomyelia*, but generally there will be, with this, loss of power and disturbance of sensation, such an anesthesia. Often in syringomyelia there will be developed an arthropathy of the arms such as is seen in the legs in tabes.

Wasting of the hand, with flexion and rigidity and sometimes contractures, is seen rarely in *advanced paralysis agitans* in place of the characteristic tremor.

In the "*cerebral palsy of children*," sometimes called "spastic infantile hemiplegia," the hand may be flexed on the forearm, and the forearm on the arm, the thumb drawn into the palm of the hand and the fingers flexed as in Fig. 14. These deformities are not necessarily confined to one arm alone, but are sometimes bilateral. A peculiarity of these cases is that the muscles waste very slightly, and do not develop the reactions of degeneration, so that the case sepa-

rates itself from poliomyelitis. The fingers in the cerebral palsy of children can often be placed in curious positions with ease, and, if the limb be suddenly flexed, a lock-like sensation will be imparted to the physician's hand. Convulsive seizures of an epileptiform type are very frequent in cases of cerebral palsy in children. Cohn asserts that there are on record eight cases in which intention tremor has taken the place of the spastic rigidity just described, and he reports a ninth. Similar lesions may follow infantile cerebral hemorrhage, thrombosis, or embolism.

Again, in persons who have had *apoplexy* it is not uncommon as time goes on for the temporary spasm seen in the muscles of the hand and arm to be replaced by permanent contractions resulting in deformity. These contractions, if they occur early, are an evidence of irritation of the pyramidal tract or the fibers just behind the knee of the internal capsule, and are of serious import, as they indicate the extension of marked inflammatory processes. When they come on later they show that a degenerative process is descending the pyramidal tracts. Wasting finally comes on. (For further discussion of the significance of paralysis in the arm and hand, see succeeding pages and chapter on Hemiplegia.)

A very important point always to be remembered in examining contractures of the hand and arm, or of the lower limbs, is the fact that they often are due to *hysteria*, in which case the history is that they set in suddenly, and they are generally accompanied by other hysterical manifestations, which can be discovered if sought for. As a rule, the muscles do not waste or develop degenerative reactions, but rarely such wasting may occur. Care must be taken in giving a prognosis for cases of hysterical contracture, since organic lesions sometimes supervene. Charcot states that if the contractures persist when the patient is under anesthesia, and the muscles are atrophied, organic disease exists. It is important to remember this, for these contractions may be practically permanent when once induced, and, as injuries may produce either a true organic or a false hysterical contracture, much medicolegal interest centres about this differential diagnosis. Closely allied to these cases are those of hysterical contracture, in which after grasping an object the patient cannot let go until the muscles are stroked. Putting an Esmarch bandage on such a forearm will usually produce the spasm.

When in the course of an acute illness in a child the fingers are drawn down into the palm of the hand, with the tips touching the palm and the thumb turned in beneath them, with its tip pressing the palm, the patient may have *meningeal congestion* or inflammation, or hydrocephalus, and a general convulsion may be imminent.

When the fingers are bent toward the palm, but the tips extended and the thumb turned in ("the accoucheur's hand"), the position

is typical of *tetany*, but in this condition the rest of the body will give evidence of involvement. The nervous irritability in this condition is greatly increased, and pressure on a large bloodvessel or nerve trunk will often produce the spasm. Curiously enough, gastric dilatation or thyroid wasting will often be found with tetany. In other cases it appears to be due to profound debility, as after prolonged nursing. (See Tetany in chapter on Convulsions and General Spasms.) Care must be taken to separate the so-called carpopedal spasm of rickety, hydrocephaloid children rom true tetany, in which the body is usually involved, and from spastic paralysis due to infantile cerebral palsy.

Spastic rigidity of the arms is often one of the earliest signs of chronic hydrocephalus, even before the skull begins to enlarge, and convulsions may be present from time to time. In congenital spastic rigidity due to sclerosis or defective development of the cortex cerebri the spastic condition is usually confined to the legs. (See chapter on the Legs and Feet.)

Spasm of the fingers of a rigid type on attempting to make certain movements is also seen as the result of excessive use of the part involved, and occurs in seamstresses, cigarette rollers, cigar rollers, typewriters (rarely), telegraphers, milkers (rarely), persons who use a pen to excess, and in piano, flute, clarionet, and violin players, or in persons engaged in any occupation requiring constant and comparatively minute and well coördinated effort. It seems to be more common in men than in women by a large proportion (39 to 4). Sometimes paralysis, tremor, or vasomotor disturbances take the place of occupation spasm.

The spasm resulting from occupation must be separated from that sometimes seen in the hand in posthemiplegic chorea, progressive muscular atrophy, the various forms of toxic peripheral neuritis, and that due to irritative cerebral foci, such as tumors of the brain. The history nearly always clears up the diagnosis. Spasm of the muscles of the hand and arm, rhythmical or otherwise, may also be due to hysteria, and may resemble when due to this cause, true tetany (not tetanus).

The position of the hand may be very various. Thus, the hand may drop edgewise from the radius toward the ulna in cases of rheumatoid arthritis, from paralysis of the extensors on the radial side of the forearm, resulting from neuritis or acute infantile poliomyelitis, while marked *wrist-drop* may occur from paralysis of the extensors in chronic lead poisoning, or in any form of neuritis, toxic or otherwise, involving the nerve supply of these muscles (musculospiral nerve). Wrist-drop may also be developed by pressure upon the musculospiral nerve, as in crutch palsy. If the wrist-drop is bilateral, it may be due to toxic neuritis; but if

unilateral, it is probably, but not positively, due to pressure paralysis from sleeping with the head resting on that arm, or from pressure by a crutch, or from some similar pressure capable of injuring the nerve. Very rarely unilateral wrist-drop is seen in lead poisoning. When lead is the cause, the supinator longus usually escapes, as does also the short extensor of the thumb, so that the forearm can be flexed and the thumb extended. Pain is rarely present in pressure or lead wrist-drop, but is present in wrist-drop due to alcoholic and other forms of toxic neuritis. Often, too, in these cases the flexors are considerably involved. (See part of this chapter on Brachial Monoplegia.)

Choreic movements of the hands and arms in children are seen chiefly as a manifestation of chorea minor. They are usually met with in rheumatic and neurotic children, and heart murmurs are generally to be heard in these cases. The first evidences of spasm may be developed in the hand, and be limited to that member in rare cases, and the hand often drops things that are placed in it. The hand itself is rarely involved alone, and the muscles of the arm toss the entire arm and hand with a fidgety, jerking movement which is very characteristic. A form of chorea minor, usually limited to the arm, is called *paralytic chorea.* It comes on suddenly, and is characterized by loss of power with a few feeble twitches. It affects only children. The same term, "paralytic chorea," is also applied to a condition sometimes seen after an apoplectic stroke, choreic movements taking place as degenerative changes in the muscles are developed. Sometimes choreic movements come on in the latter half of life, often preceded by emotional disturbances. These movements are not true chorea. They are often called senile chorea.

In some cases of adult chorea the patient tends to become maniacal, particularly toward night. Such cases usually occur in women, and the prognosis as to life is bad. There is often in these cases great mental hebetude.

Several other affections which somewhat resemble true chorea are sometimes met with, but all of them lack, with one exception, the peculiarity of its movements. One of these is what has been called habit chorea, or, more correctly, habit spasm, in which condition the patient acquires a nervous trick of jerking a muscle or a set of muscles. Unlike true chorea, it is more frequently seen in adults than children. Its limitation, as a rule, to a single set of muscles and the history of the case usually separate it from chorea minor, and it is to be recalled that the movements consist in sudden twitchings rather than jerking, irregular muscular movements.

In *paramyoclonus multiplex* the disease, as the name implies, usually involves symmetrical parts, the contractions of the muscles appear in paroxysms, and the muscles involved are usually the biceps,

deltoid, and triceps in the arms, and the quadriceps femoris and calf muscles of the lower limbs. Myoclonus multiplex is a disease of adult life, and chorea is usually seen in childhood. Sometimes the muscles in myoclonus are exceedingly irritable.

Under the name of *electric chorea*, or "Dubini's disease," Dubini described a disease, affecting both sexes and all ages, in which sudden shock-like contractions of the muscles take place, as if they were being stimulated by a slowly interrupted faradic current. The disease usually begins in the upper extremities, and gradually involves the rest of the body, and progressively passes to a fatal issue. This is a very rare disease, and the sudden contraction of the muscles in tonic spasm separates it from chorea.

Still another form of electric chorea is that of Bergeron, which is probably identical with what has been called hysterical chorea. Here, again, the shock-like muscular contractions are manifested chiefly about the shoulders. The patient is usually a female, and has the stigmata, sensory and otherwise, of hysteria. (See chapters on the Skin, Eye, and Feet and Legs.)

Again, the physician may meet, exceedingly rarely (almost never in the United States or England), with a condition called *convulsive tic* or palmus, which has also been called "the jumpers," in which the movements are not in the slightest degree like true chorea, but are sudden muscular movements, usually imitative of the act of some other person or animal. This is often associated with echolalia —that is, repeated or echoed speech—or coprolalia or filthy speech.

Finally, another very rare disease is that known as Huntingdon's or *hereditary chorea*, a condition in which the twitching usually begins in the face and extends to the arms and legs. This ailment is hereditary, rarely begins before thirty years of age, is accompanied by progressive mental deterioration, a tendency to melancholia, and may last ten or twenty years.

Mercurial poisoning producing tremor may cause so coarse a movement in advanced cases that the case may be thought choreic. (For a description of tremors, see latter part of this chapter.)

In "*Thomsen's disease*" the hand is placed in tonic spasm as soon as voluntary movement is attempted. Closely resembling Thomsen's disease, or myotonia congenita, is what is called *paramyotonia congenita*, which exists in three forms: first, a patient suffering from paralysis agitans on attempting to move is seized with rigidity of the muscles, which holds him fixed; second, a patient is suffering from ataxia and muscular weakness, and is seized with an attack of muscular rigidity; and, third, a patient may have the muscular fixation occurring just as it does in Thomsen's disease, save that it is produced by cold or exposure, and not by intention movement, and may last for hours. (See also Athetosis.)

Tremors of the Hand and Arm.—The movements of the hand should always be carefully watched in cases of suspected nervous disease. The most common alteration from the normal will be found to be tremor, which may indicate paralysis agitans, disseminated sclerosis, general paresis, chronic mercurial, plumbic, or alcoholic poisoning, hysteria, senility, and Graves' disease. Sometimes a tremor may be found in naturally nervous women who are drinkers of tea to excess.

In *paralysis agitans* the whole hand is involved, and generally both hands are equally affected. The tremor is rhythmical and fine or minute in character at first, but later may be quite coarse. It is a slow tremor of about five vibrations per second, which is more or less constant, and worse when attention is called to it, but it is not greatly increased, and, perhaps, is even decreased, by a voluntary act, such as an attempt to raise a glass of water. Very rarely, however, the reverse holds true, and the tremor is increased by voluntary effort. The fingers are generally semi-extended and the thumb is adducted, so that it constantly rubs the index finger with its pulp, as if it were attempting to rub off the skin of that member. Frequently there are pain and aching of the extensor muscles of the forearm and wrist from the constant exertion. (See chapter on the Feet and Legs, the part on Gait.)

The tremors of *disseminated sclerosis* are also slow, but coarse in character. They are not constant, but are developed upon intentional movement, and have a greater amplitude than those of Parkinson's disease (paralysis agitans). Indeed, they may be so coarse as to be choreic in type, or even ataxic. Often threading a needle will be possible for a person with this disease, because it is a short act, while lifting a glass of water will be impossible. The symptoms of disseminated sclerosis are well summarized in the following table drawn up by Charcot.

I. Spinal Symptoms:		
	Positive	Tremor on voluntary movements of the extremities—"intention tremor" (arms and head; more rarely of legs).
		Titubation.
		Paresis (spasmodic) of the extremities.
		Contracture, with exaggeration of the reflexes—spastic rigidity.
	Negative	No sensory symptoms, or only very slight disturbance.
		Vesical disturbance none or very slight.
II. Cerebral Symptoms:		Dysarthria—slowness of speech; scanning of words.
		Nystagmus—blank expression.
		Attacks of vertigo—spasmodic myosis.
		Transitory amblyopia—white atrophy of the papillæ.
		Diplopia—associated paralysis of ocular muscles.
		Mental enfeeblement.
		Apoplectiform and epileptiform attacks.
		Difficulty in deglutition.

III. Abnormal or Unusual Symptoms:

Trophic	Muscular atrophies (amyotrophies), bedsores.
Tabetic	Lightning pains.
	Romberg symptom.
	Anæsthetic areas.
	Vesical and rectal paresis.
	Gastric crises.

Frequent remission of all the symptoms is characteristic of the malady.

It is not to be expected that all these symptoms will be found in one case. But many of them will occur. Charcot taught that tremor involving the head indicated disseminated sclerosis, and excluded paralysis agitans; but cases of head tremor in the latter disease do occur. (See chapter on the Feet and Legs, part on Gait.)

The tremor of *mercurial, plumbic, and alcoholic poisoning* resembles that of paralysis agitans, save that it is more rapid, reaching nine or ten vibrations per second, and in the case of alcoholic tremor is decreased by a large drink of liquor, while those due to lead and mercury may be relieved in a short time by potassium iodide. Further than this, the tremor of alcoholism is generally worse in the morning.

A point of some importance in plumbic neuritis producing tremor and wrist-drop is the fact that painful sensations are rarely present; in arsenical neuritis, on the other hand, they are often the most prominent symptoms, even preceding the motor disturbance. In mercurial neuritis, on the other hand, tremor precedes all evidence of loss of power, and, finally, may become so coarse as to resemble chorea.

The tremor of *general paresis* is also rapid, eight or nine per second, and is a very fine tremor, which may be felt only when the arm is extended and the finger rested on the hand of the physician. In other words, the tremor of the hand in general paresis is generally not a predominant symptom, but is elicited when the muscles are put upon a strain. In regard to the fineness of the tremor of general paresis, it should be remembered that it closely resembles that of *Basedow's* or *Graves' disease* (exophthalmic goitre, eight or nine per second), since the tremor of this condition is not only equally fine, but generally unseen except when the arm is extended and tips of the fingers rested upon the fingers of the doctor. This tremor has been called the "railroad-bridge tremor," because of its fineness and vibratory character. The individual fingers do not separately tremble in Graves' disease.

In *posthemiplegic tremor* the trouble is unilateral, there is a history of cerebral disease, and paralysis is present.

Tremor of a very marked character may be due to *hysteria*, and arises most frequently in those who have been exposed to shocks or accidents. The tremors may occur constantly or only with inten-

tion movements, or be increased in amplitude, but not in rhythm on movement. The latter form is known as the "type Rendu," and has a rhythm of seven to nine per second, while the slower hysterical tremor may be four or five per second.

Beyond the state of tremor should be recalled the movements of *chorea*, which may be limited to one arm or hand, and which in their milder forms may be confused with the pronounced movements produced by effort in disseminated sclerosis. The latter are often very arhythmical, and so the choreic movement the more closely resembles them; but those of sclerosis are purposive, while those of chorea are not, since the movement contemplated in chorea is opposed by a contradictory contraction.

General Movements of the Hand and Arms.—Aside from the movements of tremor, careful notes should be made of the movements of the hand as a whole, of the coördination of its fingers and of the arm governing it. Thus, trembling contractions of the extensor tendons (subsultus tendinum) are a sign of grave and advanced forms of typhoid fever, and picking at the bedclothes (carphologia) is of still graver import. (See beginning of this chapter.) Inability to write, to play musical instruments requiring the use of the fingers, or to sew, may indicate the rare form of locomotor ataxia involving the upper extremities, so that if the patient is asked to close his eyes and feed himself the fork or spoon misses his mouth through lack of coördination, although loss of power may not be present.

Sometimes in *locomotor ataxia* as the disease becomes advanced paroxysmal twitching of the fingers may come on, or involuntary movements of the fingers occur in association with voluntary movements elsewhere.

In locomotor and Friedreich's ataxia also the movements of the hand are often lacking in coördination. The hand may be advanced past the object which the patient desires to grasp, or else falls short of it. On endeavoring to pick up an object the fingers are spread over it like a widespread claw. Generally these ataxic symptoms will be more marked in the other parts of the body and be bilateral, but Ormerod has reported an instance in which only one hand (the left) was involved. This faulty movement of the hand may, however, be due to the fact that the ocular muscles are affected, and the "erroneous projection" due to this cause leads the patient to pass the hand beyond the object reached for. Overdoses of strychnine sometimes cause this symptom of "erroneous projection."

When fibrillary twitchings of the muscles occur and tapping the muscles produces idiopathic muscular contraction, progressive muscular atrophy may be present or profound asthenia.

Sometimes, as the result of infantile cerebral paralysis or from lesions developing in later life, the muscles of the hand are affected

by a slow, constant movement, so that the fingers assume curious, constrained, and unusual postures, being moved into extreme or forced extension, flexion or pronation, or supination. This condition is called *athetosis*, and is separable from chorea in that the movements are slower and limited to the fingers and wrists, the arm escaping.

Very rarely athetoid movements of the fingers occur in advanced spinal tabes (locomotor ataxia), probably as the result of a related lesion, and not from tabes itself.

In this connection mention should be made of "mirror writing," a curious condition in which the patient writes from right to left instead of left to right. It occurs in some cases of mental feebleness, hereditary or acquired, and rarely in hysteria. "Mirror writing" may also be present in cases of cerebral paralysis.

Paralysis of One Arm, or Brachial Monoplegia.—Absolute loss of power in one hand and arm without the necessary development of subsequent deformity results from cerebral or peripheral lesions, as a rule, being rarely spinal in origin, and is called brachial monoplegia. The causes of this loss of power when its origin is cerebral may be various. Thus, the lesion may be cortical or subcortical; that is, in the surface of the brain or in the internal capsule, or between the cortex and the capsule in the corona radiata. As a rule, however, monoplegia is cortical in origin, for below the cortex the motor fibers run so closely together that only a very small lesion can involve one without involving all, and so producing a hemiplegia. These cortical lesions when they do occur are generally, but not always, associated with a convulsive seizure in the paralyzed limb, and Seguin has called this convulsion the "signal symptom" indicating a cortical lesion. Brachial monoplegia not due to hysteria or neuritis, preceded and accompanied by a convulsion and loss of consciousness, and lacking in signs of involvement of lower nervous centres, is, therefore, cortical, and is generally due to the formation of a clot in the hand and arm centre resulting from injury or from the ordinary vascular causes of apoplexy. In other cases it is due to cerebral embolism or thrombosis, or to the growth of some neoplasm, specific or otherwise, or to a localized meningitis.

The probability of the lesion being an embolism or thrombosis is decreased by the recollection of the fact that the cortex is so well supplied by vessels from the pia mater that paralysis of a centre from lack of blood supply from such a cause is rare, unless the lesion is subcortical, or, in other words, not deep enough to involve fibers from other centres as they approach each other, and yet sufficiently deep to prevent the tissues from partaking of the nutrient blood supply from the pia mater as just mentioned. Aside from the discovery of a condition of the internal organs, such as cardiac

valvular disease or sepsis, which might cause embolism, the diagnosis between paralysis from hemorrhage and embolism is practically impossible, and this is also true of the paralysis due to thrombosis, except that in cases of thrombosis we often find the presence of general endarteritis or an infectious disease, and the paralysis of thrombosis may be slow and gradual in its onset. If the paralysis rapidly spreads, the lesion is probably due to a hemorrhage.

The history of there having been some sudden cause for an increase in arterial tension, as by muscular effort, and the presence of atheromatous vessels aid us in deciding as to the probability of the lesion being due to a hemorrhage, and the sudden onset, coupled with the symptoms named, makes the diagnosis clear in a certain proportion of cases.

Neoplasms or tumors of the brain producing monoplegia are gradual in their development, accompanied generally by headache, by changes in the optic disks, and sometimes by mental disturbances or pressure symptoms. A specific history pointing to the formation of a syphilitic tumor is of value in the diagnosis. (See chapter on Headache.)

If brachial monoplegia results from a lesion in the internal capsule, the lesion must be very limited, or, in other words, only large enough to cut off the hand and arm fibers. Tumors and lesions from traumatisms in this area are very rare, and hemorrhages, which frequently cause paralysis by affecting this area, are generally profuse enough to cause hemiplegia—that is, injury of the motor fibers supplying the leg muscles as well. Sometimes, however, a sudden inflammatory process is set up in the tissues surrounding a tumor, and this may precipitate sudden paralysis.

Although the onset of a monoplegia due to cortical, subcortical, or capsular causes is sudden, the reactions of degeneration do not come on for a long period of time in such cases, because the muscles in the paralyzed area are still connected with the trophic centres in the cord, and this affords us a valuable point in differential diagnosis.

Sometimes a suddenly developed monoplegia affecting the arm comes on as a manifestation of hysteria, and follows the type of true cerebral hemorrhage so closely as to almost defy diagnosis. This condition may be accompanied by hysterical edema, the hand becoming puffy and swollen. The presence of a neurotic temperament and other hysterical signs, coupled with the prompt development of contractures, and the fact that the muscles do not rapidly waste, point to the cause of the loss of power in some cases, and this is emphasized if the presence of hysterical anesthesia of the skin can be discovered. Further, if the hand is affected, Patrick asserts that in making an attempt to grasp an object the thumb and forefinger are chiefly used; but if the object is placed suddenly in the

ulnar part of the hand, the remaining fingers can grasp it easily. (See chapter on the Skin for additional hysterical symptoms.)

Brachial monoplegia is very often the result of injury to the brachial plexus or to some of its important branches. The symptoms consist in heaviness or numbness of the arm with more or less loss of power. The motions of the arm which are particularly affected are usually abduction and elevation, which movements depend upon the circumflex nerve. If the power of extending the arm is lost, the loss depends upon paralysis of the musculospiral, which supplies the triceps; whereas if the power to flex the forearm is lost, there is paralysis of the musculocutaneous, which is the supply of the brachialis anticus and biceps. If the supinator longus is involved, the musculospiral is also affected.

When brachial monoplegia depends for its existence upon primary brachial neuritis there is pain in the wrist and hand at first, or on the scapula and in the axilla, thence radiating down the arm. This pain is constant and dull, and now and then excruciating, and is made worse by movement, even when the loss of power is comparatively slight. Sometimes, on the other hand, when the neuritis is septic in origin, it may start in the ulnar nerve and gradually extend up to the plexus. In still other cases brachial monoplegia may depend upon fracture or dislocation of the head of the humerus, and in such a case the paralytic symptoms are apt to be very well developed. The musculospiral nerve is often paralyzed by fracture of the humerus, and this results in paralysis of the muscles of the back of the arm and forearm and back of the hand, and loss of sensation in the skin covering these parts.

In all cases of brachial monoplegia due to peripheral lesions, as in severe neuritis, we find that atrophy of the muscles comes on very rapidly owing to the cutting off of the muscles from their trophic centres in the spinal cord.

Sometimes in locomotor ataxia the peripheral nerves seem quite as much involved as the spinal cord, and symptoms precisely like the paralysis of a toxic neuritis develop. Thus, Strümpell has reported cases of musculospiral paralysis from this cause, and Remak and Hirt record cases in which the median nerve has been affected, so that not only loss of power but wasting of the muscles has resulted. This is particularly the case if the muscles are much used in daily pursuits. The ulnar nerve may also be affected. Such cases are separated from pseudotabes, due to neuritis, by the pupillary reflexes and other pathognomonic ataxic symptoms. (See chapter on the Feet and Legs and that on the Eye.) Widespread muscular atrophy of the arm sometimes takes place in locomotor ataxia as a result of a coincident neuritis.

There are still to be considered two comparatively rare forms of

brachial monoplegia of the plexus type, namely, that due to pressure of growths in the neck or axilla, and brachial paralysis of the upper arm type, sometimes called Erb's paralysis. This latter form occurs from paralysis of the fifth and sixth cervical nerves or their roots. In adults this commonly results from blows or heavy weights striking on the shoulder, and in infants from pulling on the neck in difficult labor. As already said, it is an upper arm palsy, and is due to the loss of nerve supply to the deltoid, biceps, brachialis anticus, and supinator longus and brevis, and the supra- and infraspinatus muscles. The adult form is often associated with anesthesia and is persistent. In infants it is often temporary, and sensory symptoms are commonly absent.

When the lower arm is paralyzed as the result of trouble in the brachial plexus, the lesion is in the nerves arising from the seventh and eighth cervical and first dorsal roots, and the muscles affected are the triceps, the flexors of the wrist and fingers, the pronators of the hand, the extensors of the fingers, and the muscles of the hand. The arm can still be raised by the deltoid and the forearm flexed on the arm by the biceps.

When there is wasting with paralysis of the thenar, hypothenar, and interossei muscles, not due to progressive muscular atrophy, with anesthesia in the arm and forearm in the part supplied by the ulnar nerve, and in addition myosis on the side of the lesion, with sluggish pupil, retraction of the eyeball, and partial closure of the lids, there is probably a lesion of the first dorsal root of the brachial plexus and the communicating branch of the second dorsal. The cause may be neuritis or pressure by a tumor. This form is sometimes called "Klumpke's paralysis."

(For a description of the areas involved in the spinal cord, which cause loss of power in the arms and legs, see chapter on Feet and Legs, part on Paraplegia, and tables of localization in that chapter, also plates in chapter on Skin.)

The presence of bilateral brachial monoplegia should always make the physician suspicious of lead poisoning or crutch paralysis.

Apparent brachial monoplegia, in reality a syphilitic pseudo-palsy, has been described particularly by Parrot. A child apparently perfectly well, and but a few weeks old, suddenly loses the power of its arm, so that the member hangs like a flail. No wasting takes place, no degenerative reactions occur, but there may be some pain and crepitation on moving the arm. The cause of these symptoms lies in the fact that there has been a separation of the epiphyses from the shafts of the bones, with consequent helplessness. Sometimes general paralysis of the extremities arises from the extension of the disease to other limbs. The prognosis as to life is bad.

It yet remains for us to discuss the paralysis of several important

groups of the muscles of the arm. If the forearm cannot be flexed, there is loss of power in the biceps and brachialis anticus, and to some extent in the supinator longus; and as the first two muscles are supplied by the musculocutaneous, and the third by the musculospiral, such a failure in flexion shows paralysis of these fibers.

Paralysis of the extensors of the forearm, wrist, and hand, and of extension of the elbow, with wrist-drop in consequence, and flexion of the tips of the fingers, is due to disease affecting the musculospiral nerve, but the fingers can still be partly extended through the action of the interossei and lumbricales, provided the tips are flexed. The back of the hand and wrist become unduly prominent after a short time because of the forced flexion of the hand and rapid wasting of the extensors. In most cases the supinator longus, which supinates the forearm after it is pronated, is paralyzed. When the ability to pronate the forearm is greatly

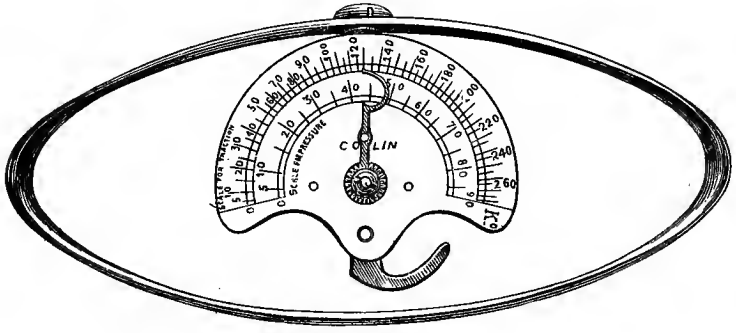


FIG. 15.—Dynamometer.

impaired, and the thumb is extended and abducted, so that it cannot be brought in contact with the tips of the fingers, the trouble is probably paralysis of the median nerve, and this is confirmed if all the phalanges are paralyzed except the first.

If the arm cannot be moved outward, away from the body, there is paralysis of the deltoid supplied by the circumflex nerve. In this connection attention should be called to the loss of power with wasting of the muscles seen after direct blows on the muscle or after injuries to the joint, sometimes called "joint palsies."

Brachial Paresthesia.—Disturbances of sensation in the hand and arm consist in anesthesia, analgesia and numbness, tingling and pain. The area of these sensations depends upon the nerve trunks involved, and to some extent upon the degree of involvement. Thus, if the function of the nerve is merely impaired, the sensation may be that of tingling or pain; if still further impaired, the sen-

FIG. 16.—Testing the elbow, or biceps, jerk.

FIG. 17.—Testing the elbow, or triceps, jerk.

sation may be that of numbness; and if the sensory fibers be totally destroyed or paralyzed, absolute anesthesia and analgesia may be present. (See Anesthesia, chapter on the Skin.)

Physical Methods Employed in Examining the Hand and Arm.

The Dynamometer.—The use of the dynamometer is to determine whether there is a marked difference in the strength of the flexor muscles in either forearm. The dynamometer most commonly used is that of Mathieu, which is an elliptical spring surrounding a semicircular scale over which moves an indicator according to the flattening of the ellipse produced by pressure. (See Fig. 15.)

The Elbow-jerk.—This is produced by striking the tendon of the triceps or the biceps, the forearm being somewhat flexed on the arm, and supported by an assistant or by the physician himself. (See Figs. 16 and 17.) (For methods of testing the various forms of sensibility in a limb, see chapter on the Skin.)

CHAPTER III.

THE FEET AND LEGS.

The general appearance of the feet and legs when clothed—The gait—Spastic paraplegia—Paraplegia without spastic contraction—Crural monoplegia—Deformities of the feet and legs—The joints—Alterations in the nutrition of the feet and legs aside from a change in the muscles.

As the physician sees a patient approaching him, he can often gain information as to the ailment from which the man is suffering by noticing his gait and the appearance of the legs and feet, for, while the gait varies greatly in normal individuals, in some diseases it is so typical that he who runs may read the diagnosis. A glance at the feet revealing one foot more loosely covered than the other, or a slit in the shoe, or a very loose lacing, will point to the presence of some inflammatory or dropsical swelling, which forces the patient to give it room; and if the legs of a man of ordinary build look swollen and fill the trousers tightly, while a glance at his face reveals that it is puffy, rather than one which is obese, dropsy still more widespread is probably the cause.

Gait.—Aside from local injuries causing a lame gait, we find that gout, rheumatism, and sciatica are the common causes of a limping gait, arising from trouble in one leg, and that in such cases there is a pained expression of the face at each movement, which shows the suffering that walking causes. The gait of such a patient is slow and cautious, and he is apt to rest every few steps, bearing his weight at such times chiefly on the well leg, or, by means of his hands, upon chairs or tables that may be near. Aside from the alterations of gait produced by these causes, we see very typical gaits produced by locomotor ataxia, pseudolocomotor ataxia (peripheral neuritis) due to alcoholic or lead poisoning, syphilis, or peripheral neuritis arising from other causes, Friedreich's ataxia, general paresis, chronic myelitis, lateral sclerosis, acute poliomyelitis, pseudomuscular hypertrophy, cerebral infantile palsy, multiple sclerosis, paralysis agitans, cerebellar disease, organic and hysterical hemiplegia, and osteomalacia, and the gaits caused by rickets and other bony defects.

In Locomotor Ataxia the gait is unsteady and waveringly uncertain, resembling that of a blindfolded person who is told that he is approaching some inequality in the floor. The patient continually seems to be feeling for the ground with his feet, and carefully picks his way along a perfectly smooth surface in a labored fashion, using

a cane to help him both in the way of support and of feeling the ground. If he looks up from the ground while walking, he sways suddenly and may fall; and if prevented from returning his eyes to the pavement, almost surely falls if no aid is given him.

The gait of *pseudotabes* is sometimes identical with that just described, is usually associated with a history of alcoholic excess, and is due to multiple neuritis. In a majority of the cases, however, it is distinctive, and has been called the "steppage" gait. The foot is thrown forward and the toe is raised so that the heel first strikes the ground in much the manner adopted when one attempts to step over some obstacle. Sometimes this gait is found in cases of arsenical neuritis and that due to lead, but in alcoholic tabes there are generally mental symptoms associated with this gait, while in lead poisoning the pathognomonic signs of this condition, such as the blue line on the gums and wrist-drop, when combined with the history, clear up the diagnosis. It must not be forgotten, however, that the differential diagnosis of tabes from pseudotabes is sometimes very difficult, and as Dana has well said: "When Déjèrine described as locomotor ataxia a case which now appears to have been one of alcoholic peripheral neurotabes, when Buzzard has diagnosticated as true spinal tabes a case of postdiphtheritic ataxia, when Seligmueller mistakes a case of wall-paper poisoning for one of true spinal tabes, we may easily suppose that errors have been made by many others."

The important symptoms which point to *true locomotor ataxia* are the swaying of the body when the eyes are closed (Romberg's symptom), the loss of knee-jerk (Westphal's sign), the history of gastric, laryngeal, or vesical crises, the presence of numbness in the feet, the slow onset of the disease, and the absence of any history of exposure to the causes of neuritis just named. Additional diagnostic points are the inability of the patient to stop and turn quickly and steadily at the command of the physician.

Fränkel states that in many cases of this disease the sensation of passive motion at the joints is impaired. To determine the presence of this symptom the toe is grasped by the thumb and forefinger, and moved very slowly and gently so as not to disturb the rest of the limb. The patient must be blindfolded, and in ataxia fails to appreciate that passive joint movement has been made. If all these signs are present, and are combined with that important symptom, the Argyll-Robertson pupil, the diagnosis is practically certain.

Grube has, however, reported three cases of diabetes mellitus producing a pseudotabes due to neuritis which had the Argyll-Robertson pupil, and in addition attacks of abdominal pain like the crises of true ataxia.

Another sign of locomotor ataxia is undue relaxation of the muscles, which has been called hypotonus. This is a point much insisted upon by Fränkel. He points out that if a healthy man be placed in a horizontal position on a couch, he cannot raise the leg very high if the knees be kept extended. On the other hand, if he has locomotor ataxia, even with the knee fully extended, he can raise the leg to a sharp angle with the plane of the body, amounting to 60°, 80°, or 100°; whereas in health he cannot raise it to an angle greater than 30° to 50°. This is due to the fact that the semimembranosus and semitendinosus resist the movement in health; whereas in locomotor ataxia they are so relaxed that this resistance does not occur. Again, because of hypotonicity of the quadriceps, it may be possible in such patients to flex the knees to such an extent that the heel can readily touch the buttocks in voluntary motion. So, too, the thighs when the legs are flexed may be so widely abducted that the knees come in contact with the couch, because of relaxation of the abductors.

THE STAGES OF TABES DORSALIS.

Initial Period.	Second Stage.	Final Station.
Inco-ordination, but no change of gait.	Greater inco-ordination, and marked ataxic gait.	Cannot walk because of ataxia.
Numbness of the feet.	More marked anæsthesias.	Extensive anæsthesia.
Shooting-pains in the legs.	Pains worse.	Pains less.
Diminished or lost knee-jerks, one or both.	Lost knee-jerks.	Lost knee-jerks.
Sluggish or lost pupillary reflex to light.	Lost pupillary reflex to light and myosis.	Lost reflex to light, myosis, paralysis of accommodation.
Weakness of sexual function.	Impotence.	Impotence.
Transient diplopia; transient ptosis	Ocular palsies rare, or marked ophthalmoplegia.	Ophthalmoplegia.
Sluggish micturition.	Increased vesical weakness.	Catheterization needed.
Optic atrophy.	Optic atrophy rarely develops.	Blindness.
Trophic changes in the joints.	Trophic changes not so common.	More marked if they began in early stage.
Hemiatrophy of tongue.	Deafness.	Increased.
	Laryngeal and visceral crises.	Not so common.
	Girdle sensation.	Unnoticed.

Reflex action is decreased and the gait altered in locomotor ataxia, because, though the motor tracts are open, the sensory tracts in the nerves, the posterior nerve roots, and the posterior columns of the cord are diseased. (See Fig. 18.) For these reasons the reflex arc is destroyed and the coördination of the muscles lost. The patient cannot tell how to use his muscles unless he can see them and

coördinate them by the aid of the eye. The sensations of formication or numbness are also due to these sensory lesions. (For descriptions of motor and sensory tracts of the spinal cord, see early part of chapter on Hemiplegia and the chapter on the Skin.)

In neuritis causing pseudotabes we have a history of rapid onset of the symptoms, paralysis, and wasting of the muscles, an absence of vesical symptoms and of the Argyll-Robertson pupils.

Sometimes not only the gait, but the entire set of the ordinary symptoms of locomotor ataxia are aped by hysteria so closely that

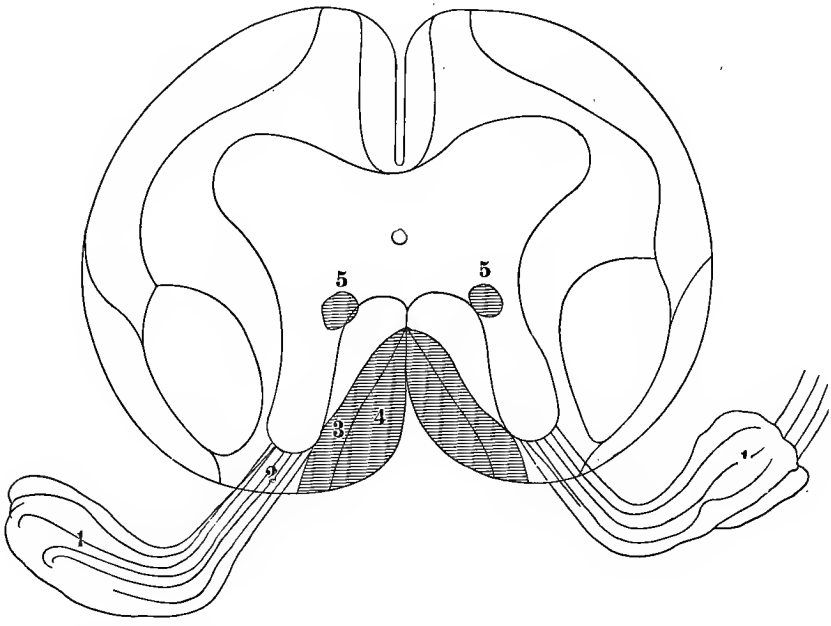


FIG. 18.—Showing the areas of the cord involved in locomotor ataxia. 1. Primary lesion in sensory neurones in posterior ganglia. 2. Sensory nerve roots in which the earliest and most extensive lesions are found. 3 and 4. The shading includes both the column of Goll, the inner, and that of Burdach, the outer. 5. Also Clarke's column in the gray matter. It is to be remembered that the lesions of locomotor ataxia are found in the peripheral nerves as well.

a diagnosis may be almost impossible, but the Argyll-Robertson pupil, the lost knee-jerks, and the optic atrophy will not be present if hysteria be the cause of the symptoms. On the other hand, Romberg's symptom may be marked to an extraordinary degree. The patient who is hysterical, in falling nearly always falls the same way, keeping her frame stiff like a board. (See chapter on Eye for differential ocular symptoms.)

General Paresis.—The feebleness of the limbs, the reflex iridoplegia (Argyll-Robertson pupil), and the ataxic gait sometimes seen as the chief manifestations of general paresis may cause an error in diagnosis in favor of locomotor ataxia, but careful examination will reveal mental feebleness in the paretic case, or at least evidences of delusions, and if the disease is at all advanced there will be a history of the patient having had convulsions or apoplectiform attacks. Sometimes there will be found present in paretic dementia (general paresis) increased knee-jerks and many of the symptoms of ataxic paraplegia, but the associated mental failure and fine intention tremor of the hands decide the diagnosis in favor of paretic dementia.

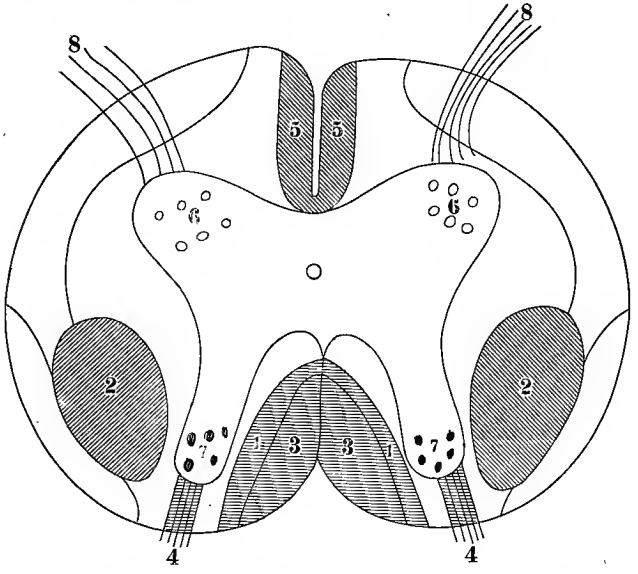


FIG. 19.—Showing the spinal areas chiefly involved in Friedreich's ataxia. The areas are the column of Burdach (1); the lateral pyramidal tracts (2); the columns of Goll (3); the posterior nerve roots (4); the direct pyramidal tracts (5); atrophy of cells in anterior horn (6) and in the posterior horn (7); and the anterior nerve roots (8).

In Friedreich's Ataxia the gait is peculiar. The legs are widely separated and moved in an uncertain, hesitating manner, and if the feet are placed close together and the patient is told to stand still, swaying at once develops. If the eyes be closed, the swaying may greatly increase. The movements of the arms are incoördinated. These symptoms, which to a certain extent simulate true locomotor ataxia, are associated, as a rule, with others which separate the two affections, for in this disease the symptoms often come on in very early life, there is sometimes nystagmus, usually a history of heredity there is a slow and jerky articulation, scoliosis, and talipes equinovarus, but there is no Argyll-Robertson pupil (Fig. 19).

The following table shows the differential points between locomotor ataxia and Friedreich's ataxia:

Locomotor Ataxia.	Friedreich's Ataxia.
Argyll-Robertson pupils.	No Argyll-Robertson pupils.
No nystagmus.	Present late in disease.
Painful crises.	Crises usually absent.
Intellect unimpaired.	Becomes impaired.
Gait ataxic.	Cerebellar ataxic gait.
Speech normal.	Speech halting.
No head tremor.	Head tremor present.

Hereditary Cerebellar Ataxia.—Friedreich's ataxia must be separated from another rare disease in which the gait is ataxic and the disease hereditary, namely, hereditary cerebellar ataxia, in which we have the following symptoms not seen in Friedreich's disease, namely, normal or exaggerated knee-jerks, Argyll-Robertson pupils, and a beginning of the malady after twenty years of age.

Hereditary cerebellar ataxia may also be confused with disseminated sclerosis.

Hereditary Cerebellar Ataxia.	Disseminated Sclerosis.
1. Gait: ataxic, groggy; feet wide apart.	1. (*a*) Spastic paraplegia; feet close together. (*b*) Ataxic, groggy; feet wide apart. (*c*) Ataxic paraplegia (*a* + *b*).
2. *Station: Romberg's symptom absent.*	2. *Romberg symptom may be present.*
3. Arms: ataxy and some intention tremor.	3. Intention tremor; sometimes ataxy.
4. Oscillations and jerky movements of the head and trunk.	4. Oscillations and jerky movements of the head and trunk.
5. Exaggerated contractions of facial muscles during speaking.	5. Twitching in facial muscles during speaking.
6. *Speech: hesitating and abrupt, or simply monotonous.*	6. *Laborious, scanning,* or monotonous speech.
7. Eyes: jerky nystagmus; optic atrophy, contracted field of vision. The external recti muscles may be paretic or paralyzed.	7. Jerky nystagmus; optic atrophy, contracted field of vision; ocular nerve palsies.
8. Myotatic irritability increased, knee-jerks exaggerated, ankle-clonus; contractures and muscular rigidity.	8. Myotatic irritability increased; knee-jerks exaggerated, ankle-clonus; contractures and muscular rigidity.
9. Mental impairment in varying degrees.	9. Mental impairment in varying degrees.
10. *Vertigo sometimes.*	10. *Vertigo common.*
11. *Vesical functions rarely affected.*	11. *Vesical functions more frequently disturbed.*
12. *Apoplectiform seizures do not occur.*	12. *Apoplectiform seizures occur in a small proportion of cases.*
13. *Heredity common.*	13. *Heredity uncommon.*

Disseminated Sclerosis.—The gait in disseminated sclerosis is often markedly spastic and paretic—that is, stiff and feeble—and may in the early stages of the disease closely resemble that of spastic paraplegia due to lateral sclerosis. When the patient attempts to pick up a small object with his fingers there are tremor and oscillation of the hand. Scanning speech and nystagmus develop later on in these cases. It is, however, important to remember that multiple cerebrospinal syphilis may closely simulate multiple, or dissemi-

nated, sclerosis. Sometimes they may be differentiated by the fact that in disseminated sclerosis there is apt to be paresthesia, whereas in syphilis there is more apt to be pain. An important differential symptom is that nystagmus is rare in syphilis, common in disseminated sclerosis, and ocular palsies are common in syphilis, rarely so severe in sclerosis, so that complete oculomotor palsy with ptosis and squint would be more likely syphilitic than sclerotic. (See chapter on the Eye.)

Stieglitz has pointed out that in certain cases of acute disseminated myelitis and encephalomyelitis following the acute infectious diseases, the symptoms of an acute or subacute multiple sclerosis are presented, more especially the intention tremor, the increased reflexes, and the scanning speech. The disease may ultimately

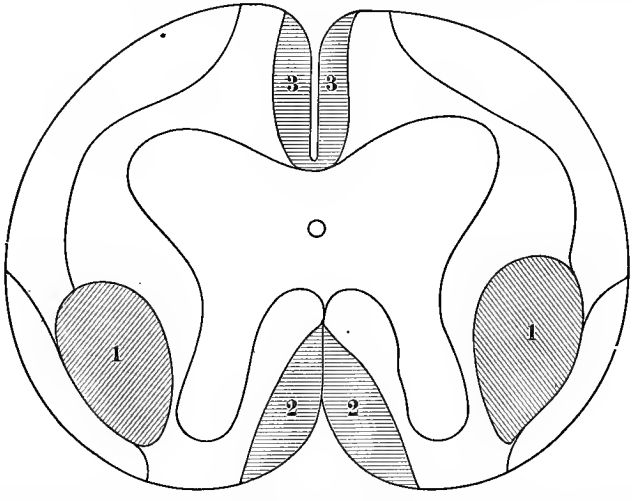


FIG. 20.—Showing areas of spinal cord involved in ataxic paraplegia, which is practically a combination of locomotor ataxia and lateral sclerosis. 1. Lateral or crossed pyramidal tracts. 2. Posterior columns of Goll and Burdach. 3. Direct pyramidal tracts or Türck's columns.

form the basis of a typical chronic insular sclerosis with its recurrent attacks, etc. It may, however—and this is a point of importance—subside after a shorter or longer period and end in recovery.

Myelitis.—In chronic myelitis in the early stages, while motion is still preserved, the gait is typically that of feebleness, and the legs respond slowly to the cerebral desires, being dragged along after the patient, who leans forward, supporting some of his weight on crutches or canes.

Ataxic Paraplegia.—If the lesions of the disease involve the lateral pyramidal tracts to a considerable extent, the gait is somewhat spastic, and if the sensory fibers are also much involved it may be

like that of ataxia. Under these circumstances the attitude and gait of a patient are sometimes a combination of those of lateral spinal sclerosis (spastic paraplegia) and locomotor ataxia. In some instances the spastic symptoms are more marked, in others the signs of locomotor ataxia are more prominent. This condition is called ataxic paraplegia, and in it we find the exaggerated knee-jerks of lateral sclerosis associated with the swaying of the body (Romberg's symptom) of ataxia. Ankle clonus is also present. The crises of locomotor ataxia do not occur, and the Argyll-Robertson pupil is usually not present. (See Fig. 20.)

FIG. 21.—Typical pseudomuscular hypertrophy. Note the scoliosis and enlarged calves of the legs. (Dercum.)

Lateral Sclerosis.—In lateral sclerosis the gait is typically spastic, the legs being rigid from the hip-joint down, and the toe being dragged in a semicircle from behind forward.

Poliomyelitis.—When the gait of a young child is stumbling, or the leg is dragged, or the ankles bend so that locomotion is impossible, the probable diagnosis is that the cause is *acute poliomyelitis*. (See Paralysis of the Leg.)

Pseudomuscular Hypertrophy. — In pseudomuscular hypertrophy there is a peculiar waddling gait, a tendency to stumble, the body is usually bent forward, and there is difficulty in getting up from the floor and on going up and down stairs. The patient in all his movements shows a marked loss of power in the legs with a great apparent increase in the size of the muscles in the legs (Fig. 21).

The gait of pseudomuscular hypertrophy is sometimes closely reproduced in children suffering from severe rickets. The other features of the case which may mislead the physician are that the child, if fat, will have bulging legs, as if the muscles were hypertrophied, and lordosis due to spinal weakness. In the rickety case, however, the knee-jerk is preserved, and in the case of pseudomuscular hypertrophy it is lost.

Infantile Cerebral Paralysis.—The gait of a child suffering from infantile cerebral paralysis is quite characteristic. In the first place it is spastic, and the patient walks on the toes, or in some cases clubfoot develops. The heels are everted and the toes turned inward, the knees being so closely approximated that the clothes may become worn between them from the rubbing. So great is the extension of the legs that the toes are very apt to drag, and, finally, the adduction spasm may be so great that the legs overlap each other as walking is attempted (Fig. 22).

FIG. 22.—Spastic paraplegia; crossed-legged progression. (From a patient of Dercum's in the Jefferson Medical College Hospital.)

FIG. 23.—Side view of a case of paralysis agitans, showing forward inclination of trunk. Tendency to propulsion. (Dercum.)

Paralysis Agitans.—In paralysis agitans the patient's gait is hurried because, from the bent-over position of his body, his centre of gravity is too far forward, and he runs to keep up with it. This is called festination. The gait is also somewhat trotting or toddling (Fig. 23).

Cerebellar Disease.—In cerebellar disease the gait may closely resemble that of a drunken man, and the patient has the greatest difficulty in keeping from sheering off to one side as he walks, swaying, too, from side to side (cerebellar titubation). The middle lobe of the cerebellum is usually affected; but Nothnagel asserts that if these symptoms are associated with paralysis of the oculo-motor nerves and other symptoms of brain tumor there is a growth in the corpora quadrigemina.

Hemiplegia.—In hemiplegia the gait is peculiar in the dragging along of the paralyzed limb by a peculiar outward swing, which soon wears away the sole of the shoe on the inner side near the ball of the foot. It is sometimes called a mowing gait, because the leg sweeps around in a half-circle. Very often the shoulder opposite the paralyzed side is raised in order to tilt the pelvis on the paralyzed side, so as to make circumduction easy.

Hysteria.—The gait of hemiplegia is to be clearly separated from that due to *hysterical paralysis,* for in this condition the leg is dragged after the body without this outward swing. It is dragged along like the broken hind limb of one of the lower animals, or is shoved forward and the well foot drawn after, the reverse of what happens in organic paralysis. The footsteps of the hysterical hemiplegic are, moreover, apt to be careful and mincing. Further, the loss of power is usually left-sided, and associated with characteristic hysterical anesthesia (see chapter on the Skin), and often with areas of hyperesthesia. Again, in the gait of hysterical paralysis the patient is apt to be excessively laborious in her progress, and will exhaust her muscles in her strained movements. An altered gait due to irregularly distributed paralysis of groups of muscles is nearly always hysterical, and sometimes the patient who has hysterical loss of power will suddenly fall through giving way of her knees.

A condition of the gait and station of the patient varying from normal, which occurs most commonly in hysteria, consists in an inability to coördinate the movements of the muscles of locomotion or those used in standing. This is called "astasia abasia." It is in reality a form of ataxia often developing only when the patient attempts to walk. There is no loss of power in the legs, but an inability to use them regularly or with power while walking, although if the patient be made to lie down the movements of the limbs as made in walking can be performed perfectly. The knee-jerks are rarely lost, and in addition the general symptoms of hysteria can nearly always be found. The body often reels to and fro, and occasionally the muscles seem to be somewhat spastic. This symptom generally follows some severe shock, and is most commonly seen in young persons, usually young women.

Osteomalacia.—In osteomalacia there is increasing difficulty of walking, partly due to pain and partly to muscular weakness. The gait is hobbling, tottering, and is made up of short and evidently painful steps, "the pelvis and leg being jerked forward as if in one piece." The kyphotic deformity of the spine, muscular tenderness, and lateral compression of the chest and pelvis, with distortions of the limbs, aid in making the diagnosis.

Rickets.—The gait of rickets is only peculiar when curvature of the limbs or spine destroys the normal posture of the body or interferes with the movements of the limbs, and it is nearly always more or less waddling.

PARAPLEGIA.

Given a case of paraplegia, or paralysis of the lower extremities, What may be its cause? It may arise from a *cerebral lesion*, which is very rare, except in children, when it is common,[1] and if cerebral it must depend upon a lesion on both sides of the cerebral cortex or in each capsule; that is to say, there must be present a lesion in the leg centres on both sides of the cortex or in the fibers going to the legs through the internal capsules. Much more commonly the lesions causing paraplegia are in the spinal cord, very rarely this symptom is due to involvement of the nerve trunks on both sides, after they have left the cord, and sometimes it is caused by hysteria and reflex irritation.

When paraplegia occurs in a young child it is due in a great majority of the cases to caries of the vertebræ, and the pressure so produced does not necessarily depend upon compression by the bones, but by the inflammatory exudate.

The *spinal* lesions giving rise to paraplegia of the lower extremities are numerous, and are perhaps best grouped in the following table of Bramwell:

1. Organic disease . . .	Inflammation of cord	Medullary.
	Softening " "	
	Hemorrhage " "	
	Tumors " "	
	Meningitis " "	Meningeal.
	Meningeal hemorrhage	
	Injuries	
	Tumors	
	Caries of bone	Osseous.
	Tumors of bone	
2 Functional . . .	Hysterical.	
	Reflex.	
	Malarial and anæmic.	
	Dependent on idea.	

[1] Such an occurrence in adults is very rare, but it is quite common in young children, as many as 14 per cent. of the cases of infantile cerebral palsy being paraplegias. (Sachs.)

Cerebral Spastic Paraplegia.—The paraplegia of cerebral infantile paralysis is spastic, and follows difficult labors or injuries to the head of the child before or after birth. Contractures nearly always ensue, and exist chiefly in the adductors of the thighs, so that the attitude is very characteristic (Fig. 24). Epileptic convulsions very often complicate these cases. Often these paraplegias are not manifested for some months, or even longer, after birth. In many cases they are first noticed when the child attempts to walk.

Care must be taken not to confuse the contractures which sometimes develop as the result of the acute anterior poliomyelitis of infancy with the spastic state of the muscles arising from infantile cerebral paralysis. These contractions with their resulting deformity arise in muscles, otherwise healthy, which have been deprived of their natural antagonists. These contractures are not spastic and often do not occur except upon intention movement.

FIG. 24.—Spastic diplegia, congenital, presenting choreiform and athetoid movements. (Dercum.)

Arrested Development.—Cerebral spastic paraplegia in infants also sometimes comes on in cases of so-called arrested development. Such infants present no abnormality for the first few months of life, then cease to develop in mental brightness, fail to recognize the nurse or mother, cease to play, gradually lose their vision, and develop nystagmus. Death usually takes place in one or two years at the latest. Convulsions do not occur in this state, but tremors are often present in the arms. There is no history in such cases of difficult labor or premature delivery.

Amaurotic Family Idiocy.—Closely allied to this state is that known as "amaurotic family idiocy." In this rare condition, only seen so far in the children of Hebrew parents, there is in association with the symptoms just described a pathognomonic ocular lesion, consisting in the appearance of a whitish-gray patch in the region of the macula lutea, which covers an area nearly twice the size of the optic

disk. In both this and the infantile cerebral form of spastic paraplegia the pyramidal tracts are degenerated.

Care should be taken that the spastic paraplegia of rickets is not mistaken for a birth palsy.

Multiple Sclerosis.—A cerebrospinal cause of spastic paraplegia in adults is multiple cerebrospinal sclerosis, in which condition the loss of power amounts to a paresis rather than the absolute paralysis. The presence of intention tremors, exaggerated knee-jerks and ankle clonus, nystagmus, and vertiginous, epileptiform, or apoplectiform seizures, with staccato speech, and local areas of loss of power elsewhere, associated with the spastic paraplegia, renders the diagnosis easy. (See early part of this chapter.)

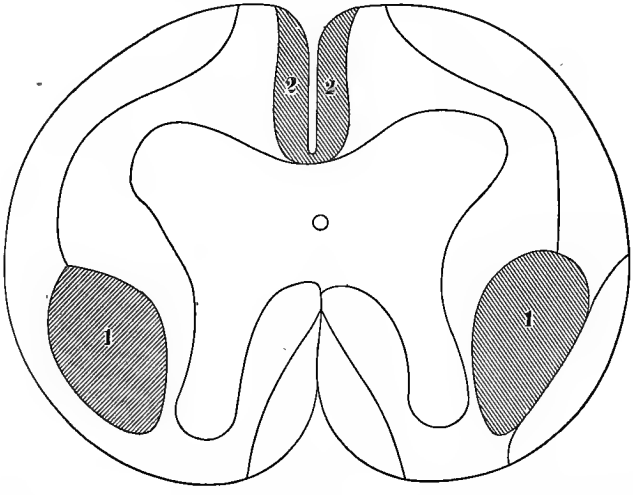


FIG. 25.—Shading shows areas involved in lateral sclerosis: 1, the crossed pyramidal tracts; 2, the direct pyramidal tract of the cervical region, which is affected late in the course of the disease.

Spinal Spastic Paraplegia. Lateral Sclerosis.—In the adult, when there is loss of power in the lower limbs with spastic contraction of the muscles when the patient attempts to move them, so that they become rigid, or if before the stage of rigidity develops the gait is spastic and stiff and the reflexes are greatly exaggerated, the disease is generally lateral spinal sclerosis (Fig. 25). There is also in lateral spinal sclerosis absence of both sensory disorders and rectal and bladder troubles, but sometimes there are present excessively hasty urination and defecation. The reason why the reflexes are increased in lateral sclerosis, and similar ailments associated with spastic paraplegia, is that the inhibitory fibers which descend from Setschenow's reflex inhibitory centre in the medulla oblongata are destroyed in the lateral pyramidal tracts.

In *amyotrophic lateral sclerosis* similar symptoms associated with wasting of the muscles are present in the later stages, but in the early stages the arms are chiefly affected by the wasting and paralysis (Fig. 26). (See chapter on the Hands and Arms.)

Spinal Pachymeningitis.—Spastic paraplegia may also be due to spinal pachymeningitis, and the associated symptoms may so closely resemble those of myelitis that a diagnosis is impossible; but the spastic character of the paraplegia, the early appearance and severity of the pain, and the comparatively slow development of the symptoms in pachymeningitis will aid in separating the two affections, as will also the presence of persistently increased reflexes from the first. Sensory disturbances, aside from pain, are common in myelitis, but rare in this condition. If the inflammatory process becomes wide-

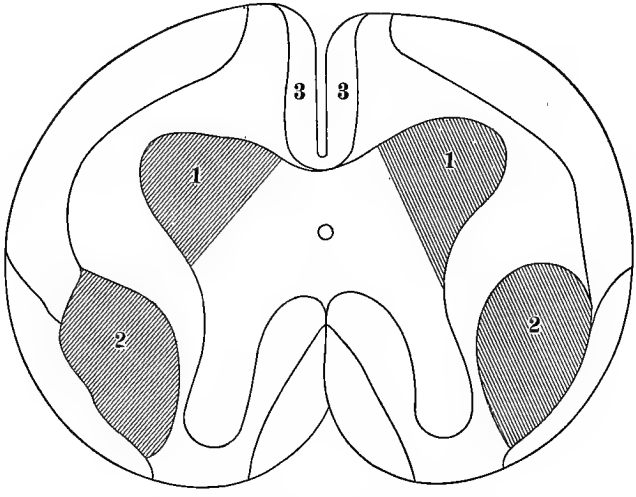


FIG. 26.—Showing areas of spinal cord involved in amyotrophic lateral sclerosis: 1, anterior horns of gray matter containing the trophic cells; 2, crossed pyramidal tracts; 3, the direct pyramidal tract.

spread there may be sensory disorders and trophic sloughs, owing to invasion of the portions of the cord connected with the sensation and nutrition by a secondary myelitis. The development of signs of spinal caries, sepsis, or a psoas abscess in such cases at once shows the condition to be meningeal in origin, and the history of traumatism will point to meningitis rather than myelitis.

Spinal Syphilis.—Spastic paraplegia, greatly increased tendon reflexes, low muscle tension, vesical disorder, and slight sensory disturbances in an adult should make the physician think of spinal syphilis.

Pott's Disease.—Spastic paraplegia in early childhood, when not due to cerebral lesions, as already discussed, is usually due to Pott's disease. The reflexes are exaggerated, the hands are drawn up, and the feet are extended. If the lumbar cord is diseased, the reflexes are lost. Inquiry will perhaps reveal a history that the child has been easily tired before the paralysis came on, and has complained of bellyache, which has really been due to pain along the intercostal nerves from the irritation at their roots. Thus, pain in the region of the navel suggests inflammation at the eighth dorsal vertebra, or at the ensiform cartilage at the fourth or fifth dorsal vertebra. Early in the disease pressure on the spinal cord may increase the reflexes. The area of the cord involved can be determined by the symptoms as detailed on pages 81 and 82. The prognosis is not always unfavorable, as extraordinary recoveries take place.

Hereditary Spastic Paralysis.—Hereditary spastic spinal paralysis of children is to be separated from infantile cerebral paralysis by the absence of a history of injury to the head at birth, and the absence of convulsions and defective mental development, all of which appear in the cerebral form. This absence of convulsions and defective mental power in this form of spastic paraplegia almost certainly separates it from the cerebral infantile type of paralysis. It is to be separated from the spastic paraplegia of lateral sclerosis by the facts that it occurs in early life, and that there is a history of heredity, or of several members of the family being affected by the disease. There are usually rigidities and contractures, but the bladder and rectum escape the paralysis, and there are no trophic changes. The reflexes are increased. The disease is rare.

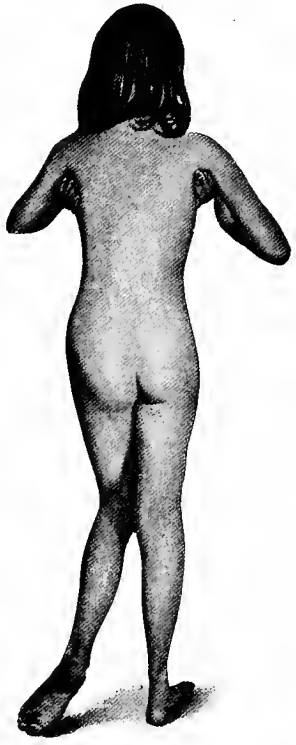


FIG. 27.—Hysterical spasm and contracture, showing attitude in erect position. (Bramwell.)

Hysteria.—An important form of contracture following paralysis or occurring without it, prone to lead to a mistake in diagnosis, is that seen in hysteria (Fig. 27). As a rule, the contractures come on in association with paraplegia. Sometimes, however, they affect the arms or an arm. It is a characteristic of these contractures

due to hysteria that they set in suddenly, and are always accompanied by such hysterical symptoms as borborygmi, ovarian tenderness, and often areas of anesthesia. Weir Mitchell has divided these cases into two forms. The first only involves single parts or limited muscle groups, and, though the contractures may last for years, joint or muscle changes do not occur. In the second class, one limb after another is attacked until all means of locomotion, or even moving the trunk, are lost, and the muscles, joints, and areolar tissue undergo organic changes. The reflexes are lost in such cases in the late stages, and the electrical reaction of the muscles is impaired. The diagnosis is to be reached by the sex, the personal history, the history of the illness, the presence of anesthesia (see chapter on Skin), and hyperesthesias. Usually the contracture comes on suddenly; it is very rigid, and the muscles on both sides of the limb are fixed—that is, the contracture involves antagonistic muscles. Sleep does not always cause a relaxation of hysterical contraction, but ether or chloroform usually does so. (See chapter on the Hands and Arms.)

Transverse Myelitis.—In transverse myelitis there is often in the later stages of the malady spastic paraplegia as a result of the irritability of the spinal centres below the seat of the lesion, which may cause a spastic state of the muscles. In distinction from lateral sclerosis we find in myelitis that there are girdle pains, involvement of the bladder and rectum, and sensory paralysis.

Spastic Monoplegia or Single Contracture.—Spastic monoplegia may arise from several causes. It may develop as a manifestation of progressive muscular atrophy in those rare cases in which the disease begins first in a lower limb, the so-called peroneal, or leg, form of the disease. The extensor muscles of the toes lose their power, the interossei waste, the foot may be flattened or claw shaped, or, in other instances, any one of the forms of clubfoot may develop. If the deformity is bilateral, it is a strong evidence of its being the leg type of progressive muscular atrophy, and that it is not due to acute infantile paralysis. There will probably be a history of heredity in such cases. This state of the foot must be carefully separated from the pes equinus seen as a result of acute infantile spinal paralysis involving the tibialis anticus. The toes are hyperextended, and the foot is very broad when viewed from side to side at the metatarsal joints. It is stated that this sign is considered characteristic of the early development of the disease in families with the heredity. Sometimes in place of this deformity the foot becomes almost parallel with the tibia in excessive extension, with eversion as the result of shortening of the peroneus longus. In other instances the deformities undergo marked changes as the disease progresses, so that they not only grow worse, but are altered in type. In distinction from ordinary progressive muscular atrophy this leg type often has marked disturbance of sensation associated with it. (Dana.) It generally

occurs in males. According to Marie, another form of clawfoot is seen in Friedreich's ataxia, there being associated with it clubfoot.

Progressive muscular atrophy of the peroneal type is a rare disease, which must be separated from multiple neuritis by the pain of the latter affection and the fact that neuritis rarely produces double clubfoot, and, further, that in neuritis there is no history of heredity. From poliomyelitis we separate it by the fact that in this peroneal type of paralysis the onset is more slow and by the fact that there is a loss of the reflexes in severe poliomyelitis, though they are preserved for a long time in the peroneal type. From Friedreich's ataxia it is separated by the fact that in that disease the reflexes are lost, there is a peculiar unsteadiness in walking, and an absence of electrical changes in the muscles.

A shrivelled, undeveloped foot and leg with drawing up and deformity are seen most commonly as the ultimate result of the acute cerebral paralysis of infancy.

Deformity or distortion of the legs may result from the secondary muscular atrophy following upon chronic inflammation in a joint or joints. The muscular wasting under these circumstances may arise from neuritis, which is associated with the arthritis, but its cause is often difficult to discover.

Non-spastic Paraplegia.—Passing from spastic paraplegia we come to those forms of paraplegia lacking this peculiarity. They are quite numerous and important. If the paraplegia comes on suddenly the cause may be hemorrhage into the substance of the cord or into the spinal membranes, or be due to compression or destruction of the cord by injuries of the back, whereby there is laceration of the soft parts or fracture or dislocation of the vertebræ, or it may be due to acute transverse myelitis.

When the paraplegia is slower in onset but not sudden, the spinal causes are acute ascending paralysis or Landry's paralysis, acute central myelitis, and acute transverse myelitis. On the other hand, the slowly oncoming non-spastic paraplegias are due to chronic myelitis, to locomotor ataxia, poliomyelitis, neuritis, and pressure due to disease of the vertebræ or to spinal tumors. Finally, we have what are called reflex and hysterical paraplegias.

Hemorrhage into the Spinal Cord.—Hemorrhage into spinal cord is an exceedingly rare condition unless preceded by grave disease of its tissues. Indeed, the existence of such a condition in man has been denied. The patient, previously in good health, is stricken suddenly to the ground, and there may be almost as much cerebral disturbance as in cerebral apoplexy, but consciousness is generally preserved. The amount of paraplegia may be instantly complete, or not be complete for twenty-four hours. Bed-sores speedily develop, and death ensues from exhaustion or from extension of

the hemorrhage upward to the vital centres. Practically identical symptoms ensue when the hemorrhage takes place between the membranes covering the cord. In both instances the reflexes are lost if the hemorrhage be sufficient to produce total paralysis.

Acute Ascending Myelitis.—If, on the other hand, after a prodromal period of short duration, during which there is some fever, the patient is suddenly attacked with paraplegia, the cause may be the *acute ascending myelitis* of Landry, and the rapid extension to the trunk, the arms, and the respiratory muscles, with the consequent early death of the patient, will confirm the diagnosis. There is usually no involvement of sensation or trophic paralysis, and the sphincters of the bladder and rectum escape the paralysis. Landry's paralysis is very rare. Similar symptoms associated with sensory disturbances are probably due to a polyneuritis.

Diller and Meyer state that the cardinal points for diagnosis of Landry's paralysis are:

1. Flaccid paralysis of the muscles, spreading rapidly from one point over the rest of the body, generally beginning in the legs, but sometimes following the reverse order, as in the French zoölogist Cuvier.
2. Absence of muscular atrophy and of electrical reaction of degeneration.
3. Tendon and superficial reflexes absent.
4. Sensibility not, or only slightly, impaired.
5. Sphincters, as a rule, intact (exceptions rather frequent).

Myelitis.—By far the most common cause of paraplegia is myelitis in one of its forms; but whether the onset be rapid or slow, it must be remembered that the symptoms of myelitis depend, first, upon the level at which the spinal cord is involved, and, second, upon whether the lesion involves the white matter or the gray. If the lesion is an *acute central myelitis* of the gray matter, it usually produces many of the symptoms about to be detailed under acute transverse myelitis, but the onset is malignant and the areas involved are usually widespread. It is attended by fever of a marked type, though the temperature of the paralyzed parts is below normal, and by early evidences of trophic lesions. Multiple arthritis may come on. The bladder and rectum are paralyzed, and, finally, delirium may develop. The prognosis is unfavorable. Acute central myelitis is to be separated from Landry's paralysis by the facts that in it sensation is lost, and there are rectal and vesical paralysis, fever, and rapid trophic changes. From polyneuritis it is separated by the facts that there are no great trophic changes in this form of neuritis, and the rectum and bladder are rarely paralyzed.

The symptoms of *acute transverse myelitis* are capable of being divided into three groups, in the first of which the onset is as sudden

as is that of apoplexy, in the second the symptoms come on quickly, and in the third, more subacutely. In the acute forms, however, the history will be that after a period of numbness, heaviness, and weakness of the legs, with more or less pain in the back, the patient has found it impossible to move his legs, has lost control of his bladder and rectum, or suffers from retention of the urine and feces instead, and at the same time has developed anesthesia of his lower extremities and the girdle sensation, or, if the lesion be situated high up in the cord, tingling in his arms. (See chapter on the Skin.) The reflexes may be abolished at first, and then return in an exaggerated form in the segments of the cord below the area affected. In other cases the reflexes do not return if the lesion is completely transverse. The patient is speedily bedridden, and to these symptoms just detailed is soon added the development of bed-sores and sloughs on dependent parts of the legs or on the buttocks, followed, it may be, by death from exhaustion, although the case may survive for months and even become somewhat better. If improvement takes place, sensation returns in the course of from one to six months, some motion in from six to eighteen months, and, finally, spasms and contractures may result from descending degeneration of the lateral tracts.

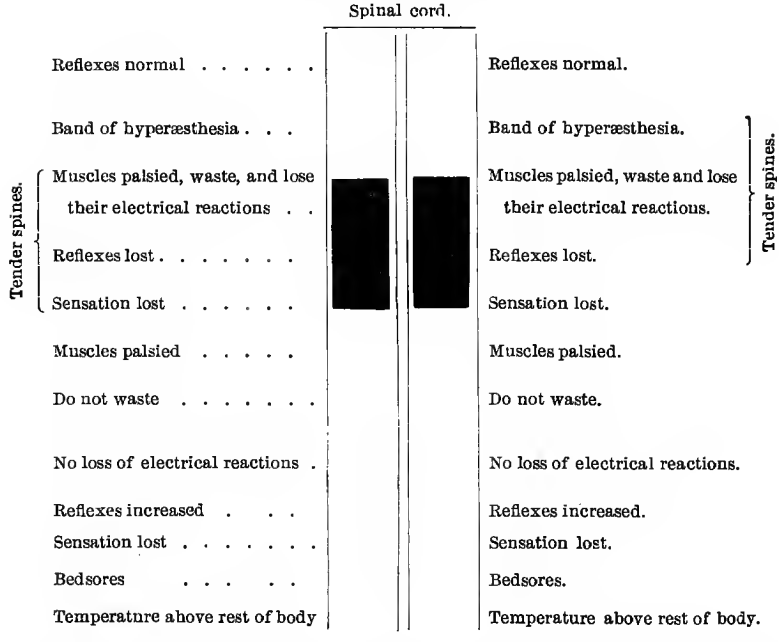
In cases in which paraplegia results from the more *subacute form* of transverse myelitis the symptoms are usually not quite so rapid in their onset as in the type just named. The patient first notices that his bladder and rectum are unduly irritable, and in his limbs there may be subjective sensory disturbances. (See Paresthesia, in chapter on the Skin.) The motor symptoms begin by a feeling of heaviness or inability to quickly move the lower limbs, so that the patient feels tired on slight exertion. Soon these symptoms deepen into absolute anesthesia and motor paralysis, and the girdle sensation on the trunk becomes well developed. (See chapter on the Skin.) The bladder, which at first was irritable, may now be toneless, paralyzed, and retentive or incontinent: retentive if the lesion is above the lumbar cord, and incontinent when the lower part of the lumbar enlargement is diseased. The reflexes at first may be abolished, but very soon some of them return, only those reflexes the centres for which are destroyed by the transverse lesion being abolished; that is, the reflexes recover after the first shock of the attack, and those muscles and tendons having spinal centres below the lesion have their reflexes increased because they are cut off from the inhibiting centres higher up in the cord or medulla. The muscles of the legs, which at the first shock of the onset of the malady were all flaccid and paralyzed, now divide themselves into two classes: those that are connected with the diseased part of the cord, which remain paralyzed, and those which are connected with the lower centres,

which recover some power; but as the lesion is so placed as to cut off all off them from cerebral influences, voluntary motion is lost as completely as if all were deprived of spinal influence. The truly paralyzed muscles waste, but the others which have unimpaired spinal centres do not, except very slowly from disuse. On the contrary, they often become spastically contracted. Other trophic changes, such as bed-sores and bullæ, develop in the skin connected with the diseased focus, but not in the skin connected with centres below the lesion. Anesthesia is present because the lesion prevents the sensory impulse from reaching the brain. (See chapter on the Skin.)

The following diagram from Seymour Taylor's *Index of Medicine* presents the symptoms of a lesion in the spinal cord in transverse myelitis.

SYMPTOMS IN TRANSVERSE MYELITIS.

The darkened portion represents the seat of lesion.



When the entire cord is not evenly involved in the transverse lesion certain groups of muscles partly escape. It is asserted that the extensors escape oftener than the flexors. The height of the paralysis also depends upon the situation of the lesion of the cord, and if high enough to involve the cervical region, and yet not high enough to paralyze the diaphragm and cause death (third or fourth cervical), there may be contraction of the pupil by involvement of the fibers

from the nucleus of the third nerve, which runs down the cord to the last cervical vertebræ before joining the sympathetic. When the legs become spastic late in transverse myelitis the cause is supposed to be a descending degeneration in the pyramidal tracts.

The symptoms of *chronic transverse myelitis* producing paraplegia are practically identical with the more acute form just described, except that they are very slow in their development.

The Seat of the Lesion.—Having discussed the various forms of myelitis, we have still to study the question of the seat of the lesion. Before doing this, it is to be remembered, in studying the relationship of the spinal cord to the vertebræ, that the so-called segments of the cord in no way correspond with the various segments of the spinal column bearing similar names. Thus the cord extends only to the level of the upper part of the second lumbar vertebra, although the spinal canal reaches much lower than this. There are thirty-one segments of the cord, each of which gives off a pair of spinal nerves. The first segment of the cord is at the foramen magnum and the last at the second lumbar vertebra.

This is well shown in the accompanying figure, modified from one by Gowers (Fig. 28).

Not only is this true, but the nerves do not emerge from the spinal canal where they leave the cord, but at a lower level. In the case of the lumbar and sacral segments of the cord the nerves form a bundle which extends down the remaining part of the spinal canal.

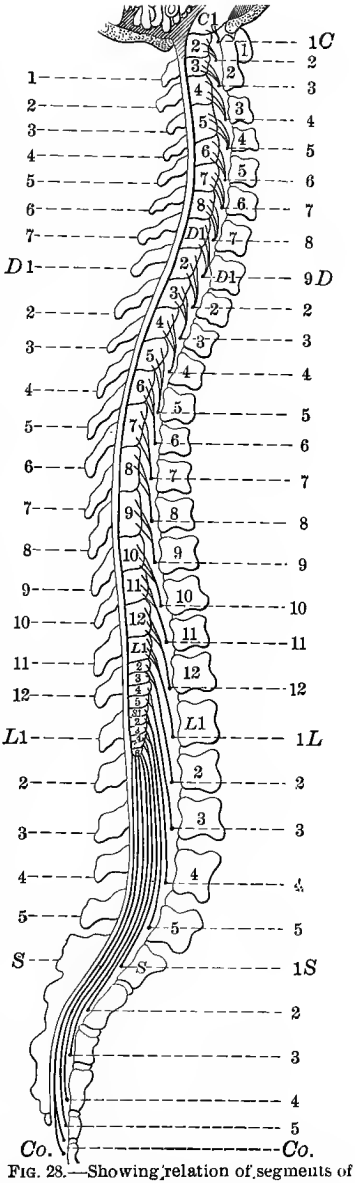


FIG. 28.—Showing relation of segments of cord to vertebræ and relation of spinal nerve roots to the cord and their levels of exit (modified from Gowers). The figures to the left refer to the spines of the vertebræ, the next column of figures to the segments of the cord, the next column to the bodies of the vertebræ, and the last figures to the right to the spinal nerves.

There are two areas in the cord of greater importance than the others, namely, the cervical enlargement, which gives off the nerves to the upper extremities, and the lumbar enlargement, which supplies the lower extremities. The eighth cervical and first dorsal segments of the cord lie opposite the spine of the seventh cervical vertebra and the lumbar-sacral enlargement opposite that of the spine of the tenth dorsal vertebra.

Let us suppose that a patient presents himself with the following condition: There is complete paralysis of his arms and legs, with paralysis of the muscles of the trunk, and total anesthesia of the same areas. The legs are in a state of spastic paralysis, their reflexes

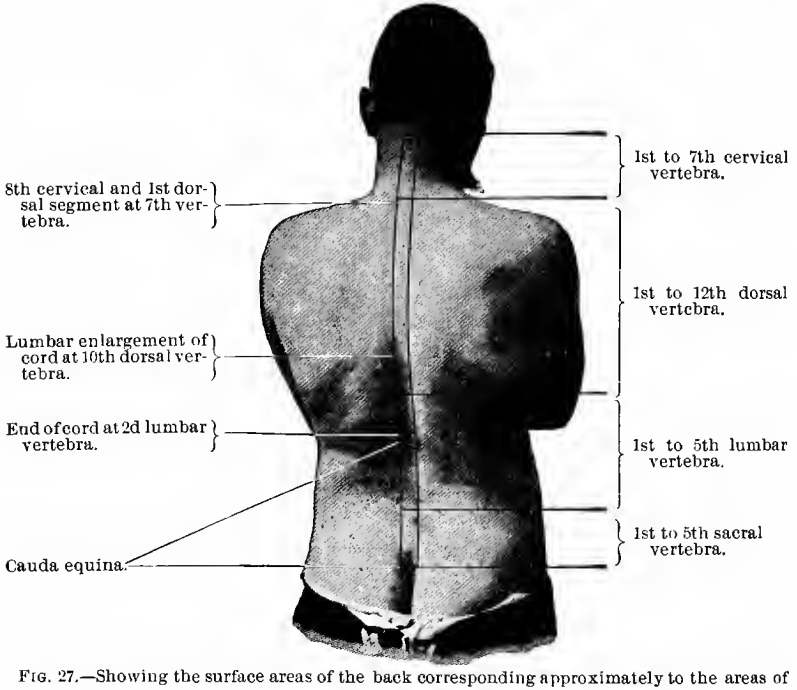


FIG. 27.—Showing the surface areas of the back corresponding approximately to the areas of the spinal cord supplying the trunk and limbs, according to the diagram on the preceding page.

are increased, and their nutrition is unimpaired; although the arms are found relaxed and flaccid, devoid of reflex excitability, and undergoing degenerative atrophy. The bladder and rectum are not retentive. All these symptoms point to a transverse lesion of the spinal cord in the cervical region, probably between the fifth cervical and first dorsal vertebræ.

If, on the other hand, the upper extremities are not affected (except, perhaps, the small muscles of the hand), but there is the same loss of power in the legs, with spastic contraction of the

muscles, and the other symptoms just named are present, combined with degeneration of the muscles of the trunk, the lesion is probably somewhere between the second and twelfth dorsal vertebræ.

Again, if the paralysis of motion and sensation be only in the lower limbs, and there be flaccidity of the muscles (where before we discovered spastic contraction), with muscular degeneration, loss of reflexes, and paralysis of the bladder and rectum, the lesion is between the tenth dorsal and first lumbar vertebra.

Still further, if there be loss of power with degeneration of the small muscles of the feet, and loss of sensation of the outside of the feet and toes, and of the skin about the anus, with preservation of power in the thighs and of the patellar reflex, the lesion is at the end of the cord in the area of the cone.

In this connection the reader should study that part of the chapter on the Skin which deals with anesthesia.

DIFFERENTIAL DIAGNOSIS OF LUMBAR, DORSAL, AND CERVICAL MYELITIS.[1]

	Lumbar myelitis.	Dorsal myelitis.	Cervical myelitis.
Paralysis.	Paraplegia.	1. Dorsal, abdominal, and intercostal muscles, according to height of lesion. 2. Legs.	Neck-muscles, diaphragm, arms, trunk, and legs.
Sensation.	Pains in legs, or girdle-pains around loins; hyperæsthetic zone around loins; anæsthesia of legs, complete or uneven distribution.	Girdle-pain and hyperæsthetic zone between ensiform cartilage and pubes.	Hyperæsthesia and pains in certain nerve-distributions of arms; below this anæsthesia of arms, body, and legs.
Atrophy.	Of legs.	Of dorsal and abdominal (and intercostal muscles not subject to examination) corresponding to height of lesion; sometimes mild and slow of legs.	Atrophy of neck-muscles (rare) or more commonly of arms.
Electrical reaction.	R. D. in atrophied muscles; or in mild cases quantitative diminution.	R. D. in dorsal and abdominal muscles; slight quantitative changes only in legs when wasted.	R. D. in atrophied muscles.
Bladder.	Incontinence from paralysis of sphincter.	Retention, or intermittent incontinence from reflex action; later from overflow. Cystitis common.	Same as in dorsal myelitis.
Bowels.	Incontinence from paralysis of sphincter; disguised by constipation.	Involuntary evacuation from reflex spasm or constipation.	Same as in dorsal myelitis.
Reflexes, superficial,	Lost.	Temporary loss, then rapid increase.	Same as in dorsal myelitis.
Reflexes, deep.	Lost.	Temporary loss, then slow increase.	Same as in dorsal myelitis.
Priapism.	Absent.	Often present.	Often present.

[1] From Prince's article in Dercum's Nervous Diseases,

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD. (According to STARR)

Segment.	Muscles.	Reflex.	Sensation.
II. and III. Cervical.	Sterno-mastoid. Trapezius. Scaleni and neck. Diaphragm.	Hypochondrium. (?) Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex. Neck.
IV. Cervical.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infra spinatus.	Pupil. 4th to 7th cervical. Dilatation of the pupil produced by irritation of the neck.	Neck Upper shoulder. Outer arm.
V. Cervical.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus. Supinator brevis. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular. 5th cervical to 1st dorsal. Irritation of skin over the scapula produces contraction of the scapular muscles. Supinator longus. Tapping its tendon in wrist produces flexion of fore arm.	Back of shoulder and arm. Outer side of arm and forearm, front and back.
VI. Cervical.	Biceps. Brachialis anticus. Pectoralis (clavicular part). Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps. 5th to 6th cervical. Tapping elbow tendon produces extension of forearm Posterior wrist. 6th to 8th cervical. Tapping tendons causes extension of hand.	Outer side of forearm, front and back. Outer half of hand.
VII. Cervical.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Latissimus dorsi. Teres major.	Anterior wrist. 7th to 8th cervical. Tapping anterior tendons causes flexion of wrist. Palmar. 7th cervical to 1st dorsal. Stroking palm causes closure of fingers.	Inner side and back of arm and forearm. Radial half of the hand.
VIII. Cervical.	Flexors of wrist and fingers Intrinsic muscles of hand.		Forearm and hand, inner half.
I. Dorsal.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar eminences.		Forearm, inner half. Ulnar distribution to hand.
II. to XII. Dorsal.	Muscles of back and abdomen. Erectores spinæ.	Epigastric. 4th to 7th dorsal. Tickling mammary region causes retraction of the epigastrium. Abdominal. 7th to 11th dorsal. Stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen, in bands running around and downward corresponding to spinal nerves. Upper gluteal region.
I. Lumbar.	Ilio-psoas. Sartorius. Muscles of abdomen.	Cremasteric. 2d to 3d lumbar. Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum.
II. Lumbar.	Ilio-psoas (sartorius). Flexors of knee (Reinak). Quadriceps femoris.	Patellar tendon. Striking tendon causes extension of leg.	Outer side of thigh.

This subject is still further subdivided and elucidated by the above table and by Fig. 27.

Finally, it is possible for disease of the cauda equina to produce symptoms of a lumbar-sacral lesion, owing to the fact that this bundle is composed of fibers derived from these two areas. The patellar reflex may be preserved, as the lesion is below the reflex arc, and all the fibers may not be involved.

Segment.	Muscles.	Reflex.	Sensation.
III. Lumbar.	Quadriceps femoris. Inner rotators of thigh. Abductors of thigh.		Front and inner side of thigh.
IV. Lumbar.	Abductors of thigh. Adductors of thigh. Flexors of knee (Ferrier). Tibialis anticus.	Gluteal. 4th to 5th lumbar. Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh and leg to ankle. Inner side of foot.
V. Lumbar.	Outward rotators of thigh. Flexors of knee (Ferrier). Flexors of ankle. Extensors of toes.		Back of thigh, back of leg, and outer part of foot.
I. to II. Sacral.	Flexors of ankle. Long flexor of toes. Peronei. Intrinsic muscles of foot.	Plantar. Tickling sole of foot causes flexion of toes and retraction of leg.	Back of thigh. Leg and foot, outer side.
III. to V. Sacral.	Perineal muscles.	Foot reflex. Achilles tendon Overextension of foot causes rapid flexion; ankle clonus. Bladder and rectal centres.	Skin over sacrum. Anus. Perineum. Genitals.

Locomotor Ataxia.—Paraplegia when due to locomotor ataxia is nearly always so surrounded by other typical symptoms of this disease as to render easy its separation from the paraplegia of myelitis, and, further, there is rarely a true loss of power. The stabbing and darting pains of ataxia (see chapter on Pain), the presence of the Argyll-Robertson pupil, the absence of the patellar reflex, and the atrophy of the optic nerve are all characteristic of ataxia, and are absent in myelitis. (See early part of this chapter on Gait.)

Ataxic Paraplegia.—The symptoms of lateral sclerosis and amyotrophic lateral sclerosis have already been discussed under Gait and Spastic Paraplegia, but in the paraplegia called *ataxic paraplegia*, also already discussed, there are in association lateral sclerosis and posterior sclerosis, and for this reason some of the symptoms of both are found to be present. Thus, in addition to loss of power there is a spastic condition of the legs with exaggerated reflexes, absence of the Argyll-Robertson pupil and of crises of pain, but the Romberg symptom, or swaying when the eyes are closed, is present. The condition which most closely resembles ataxic paraplegia is that of tumor of the middle lobe of the cerebellum, but in such cases we have, in addition, headache, vertigo, optic neuritis, titubation, and sometimes vomiting.

Poliomyelitis.—The onset of paraplegia in a young child, preceded by an attack of fever, vomiting, restlessness, and general illness, lasting but a few hours or days, and which may be complicated by convulsions, all point to the cause being *poliomyelitis* of a severe type. The legs are, however, as a rule, only completely paralyzed for a brief period after the attack. Eventually the storm clears off, and only the muscles directly connected with the diseased cells in the cord (anterior cornua) remain paralyzed. There is no loss of sensation, but reflex action is abolished in the paralyzed parts. Far and away the most important point in the diagnosis is the symptom of rapid wasting of the muscles in the paralyzed parts and the rapid development of coldness in these areas, which is due to the destruction of the trophic centres in the spinal cord. (See Fig. 30.)

Paraplegia Resulting from Tumor of the Cord or its membranes only ensues when the growth is so placed as to cut off all the motor tracts supplying both limbs, which is rarely accomplished until after a long history of more or less well-developed motor and sensory failure. The paralysis is developed in the areas supplied by the centres in the cord below or at the level of the growth, and the violent pain nearly always present in cases of tumor points to the diagnosis. Very painful paraplegia, therefore, indicates spinal tumor as its cause. The areas of anesthesia and the muscles involved may also give definite information as to the seat of the growth. (See chapter on the Skin, and Starr's table just quoted.)

Fracture of Vertebræ.—If the paraplegia be due to compression from fracture or dislocation of the vertebræ or to other direct injury the history of the patient and the evidences of external local mischief will decide the diagnosis.

Reflex Paralysis.—Very rarely during the course of severe disease, producing irritation of the bladder, kidney, bowels, or rectum, as in violent cystitis, stone in the kidney, and dysentery, paraplegia comes on, due in some cases to an infectious myelitis, but in others to what is apparently only a reflex paralysis, as it often passes away with the removal of the source of irritation. Even worms in the intestine are said to have produced such a paralysis, and their removal has been followed by cure. Generally sensation in the limbs is unimpaired and the bladder and rectum act normally. Sometimes, however, in the presence of severe renal disease, as renal calculus, there may be all sorts of disturbance of sensation and pain, as well as great motor paralysis, with total loss of reflexes, following an exaggeration of the reflexes. Probably these severe cases are always due to a coincident myelitis rather than to a reflex irritative cause.

Hysteria.—No form of paraplegia presents so many types or represents so many organic diseases as does that due to *hysteria*, for there may be not only great loss of motion, but exaggerated or lost reflexes

relaxation or spastic contraction of the muscles, anesthesia and hyperesthesia, pain or no pain. The very occurrence of such irregular manifestations in a young, neurotic girl, the fact that the anesthetic areas constantly tend to shift their position, and, finally, that the contractures, if present from hysteria, disappear on administering an anesthetic to a stage in which muscular relaxation is produced in the ordinary individual, aid us in making a diagnosis. (See that part of this chapter on Contractures.)

Scurvy.—A pseudoparalysis of the legs with immobility sometimes occurs as a symptom of scorbutus in infancy. The parents notice that the child flinches when it is picked up or handled, and seems as if tender from rheumatism. Often the gums are swollen and bleeding, and purpuric eruptions appear on the skin. The shafts of the bones of the legs or of the arms may be enlarged, and hematuria or bloody stools may appear.

Rickets.—Pseudoparaplegia may also occur in rickety children from faulty muscular and bony development. It is to be separated from the ordinary paraplegias of childhood by the state of the bones, the presence of knee-jerks, and the absence of local wasting or spasm although general spasms, or capropedal spasm, is often seen in rickety children.

Diphtheritic Paralysis.—Not uncommonly a partial paraplegia occurs as a result or sequel of diphtheria. The condition, however, is more ataxic than paraplegic, and Bourges asserts that there is no muscular atrophy such as occurs in true paraplegia due to neuritis, or in that due to some spinal lesions.

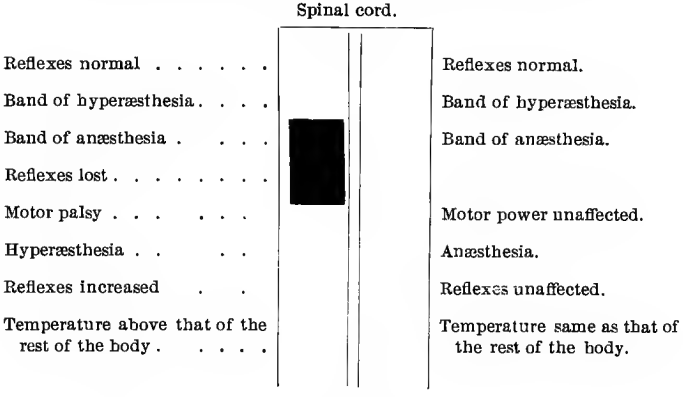
Neuritis.—When neuritis produces sudden paraplegia the symptoms very closely resemble those of acute myelitis. Neuritis may also cause pseudotabes if its onset is slow. The neuritis is always multiple and involves the arms and the body after affecting the legs; there is well-developed anesthesia (see chapter on the Skin), preceded by sensory disturbances and marked muscular and nerve-trunk tenderness; but there is no girdle sensation, as there is in myelitis and tabes. There are often trophic changes in the skin in neuritis (see chapter on the Skin), but no bed-sores as in myelitis. J. M. Da Costa stated that malarial neuritis may cause paraplegia of the lower limbs, but, as a rule, toxic neuritis produces loss of power in the arms. Very rarely paraplegia of the lower extremities results from diabetes mellitus, the lesion being in all probability a multiple neuritis.

Family Periodic Paralysis.—A very rare condition, involving not only the legs, but the entire body, is what is called *family periodic paralysis*, in which flaccid motor paralysis, with loss of electrical reaction and reflex activity, comes on suddenly, lasts for a few hours or days, and is then followed by perfect health. It is always hereditary.

Monoplegia of a Lower Extremity may be due to a cerebral lesion or to spinal or nerve-trunk lesions. The cerebral lesion producing monoplegia in one leg is very rare, and if it occurs, at any age, indicates a lesion in the convolutions at the upper end of the fissure of Rolando, and the continuation of this area in the paracental lobule of the marginal convolution. Unlike the paraplegias of infantile cerebral paralysis, monoplegia of the leg very rarely arises from this cause. If there are no signs of cerebral trouble, the presence of a complete leg monoplegia can mean one of several things, namely, a lesion limited to one side of the cord, as, for example, a hemilateral myelitis, hysterical paralysis, in which there will be irregular anesthesia (see Chapter on the Skin), and the other hysterical signs, or a tumor pressing on the crural nerve in the pelvis, or section of the nerve by injury. Apparent monoplegia may, however, be due to muscular pain or a painful phlebitis producing muscular fixation.

DIAGRAM SHOWING SYMPTOMS IN HEMILATERAL MYELITIS.[1]

(The darkened mass represents the site of the lesion.)



If the condition is due to a lesion on one side of the cord, the symptoms are quite characteristic. There is paralysis of all the muscles of the leg which are supplied by the part of the cord affected or below it. The muscles, the nerve supply of which comes directly from the affected part, eventually waste and undergo degenerative changes. The most typical symptom of this lesion is, however, the crossed character of the sensory paralysis. That is to say, there is loss of sensation in the limb opposite that in which motion is lost, and in the limb in which motion is lost there is hyperesthesia, so that the lightest touch may be very painful. The cause of this is obscure,

[1] From Seymour Taylor's Index of Medicine.

for the studies of Mott have proved that the sensory tracts in the cord do not decussate on entering it, as has been supposed heretofore. There is, however, a symmetrical band of anesthesia round the body at the level of the lesions, and a similar band of hyperesthesia above the lesions. The reflexes of the parts supplied by the diseased area are lost, but those supplied by the area below the lesions are increased as in ordinary myelitis. Very commonly the paralyzed limb is over-warm from vasomotor palsy.

Paralysis of Certain Groups of Muscles or a single muscle in the legs is most commonly due to anterior poliomyelitis or neuritis (Fig. 30). In poliomyelitis the child will be found to have loss of power in certain muscles in one or both legs (see also Paraplegia), so that

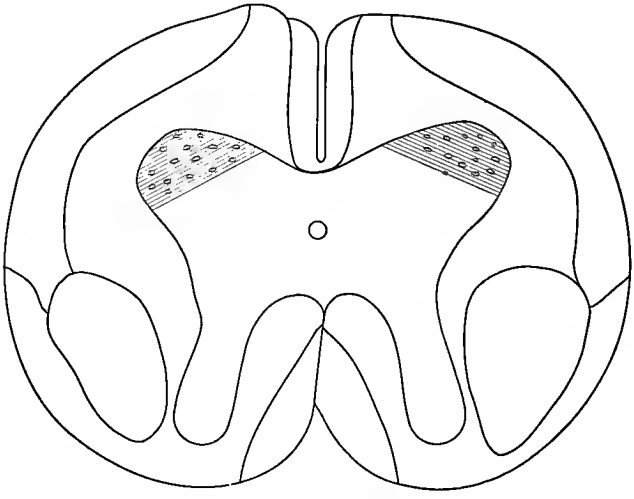


FIG. 30.—Areas involved in acute and chronic poliomyelitis. In children it is sometimes called acute infantile paralysis. The shaded area indicates the cells in anterior cornua of gray matter which are involved.

there is a dragging of the toe, or "foot-drop," the shoe becomes irregularly worn through, being dragged on one edge along the ground, the involved muscles being peculiarly relaxed and flaccid, so that the leg may waddle, to use a crude term. This is sometimes called a "Punchinello leg." There is no tendency to spastic contraction, the reflexes are rapidly lost in the affected part, and the muscles speedily waste and develop the reaction of degeneration When contractures take place they are not spastic, but are due to healthy muscles being unopposed by the diseased ones. The temperature of the paralyzed part is lower than normal. The history in poliomyelitis is that of sudden onset, with fever, vomiting, and restlessness. The two conditions of acute cerebral paralysis and

anterior poliomyelitis are so clearly separated in well-marked cases that no error can be made, particularly if the history of the attack be borne in mind, unless it be in the obscure forms of cerebral infantile palsy in the early stages. In acute infantile paralysis of spinal origin the right lower extremity is most frequently affected, after this, a close second, the left leg. Sometimes muscular atrophy may be masked in young children by the abundance of subcutaneous fat. A point of some importance in examining the reflexes is that presence of knee-jerk should not exclude the diagnosis of poliomyelitis, because the reflex act is only destroyed if the centres which are concerned in this jerk are diseased—that is, if the disease has only affected that part of the cord supplying the foot, a tap on the knee may readily produce a response, whereas if the disease be higher up in the cord the reflex will be lost. The chronic anterior poliomyelitis of adult life presents very similar symptoms to the acute form of infancy, but is a very rare disease.

Care must be taken that *paralysis of the leg resulting from an injury to the peroneal nerve* with resulting neuritis is not mistaken for acute poliomyelitis. The history of an accident, of pain, swelling, and the presence of a bruise aid us in making a diagnosis. If these symptoms occur in an adult, a possible cause is paralysis of the peroneal nerve occurring in the course of tabes. (In connection with this chapter see that on the Significance of Anesthesia of the Skin.)

Deformities of the Feet and Legs.—Much of what has been said in the preceding chapter as to the diseases which produce alterations in the shape of the hand and arm applies equally to the changes from the normal seen in the appearance and movements of the feet and legs. The feet are greatly enlarged symmetrically in *acromegaly* and in *Marie's pulmonary osteo-arthropathy*. In the latter disease the enlargement is particularly noticeable because it is the extremities which are chiefly hypertrophied, whereas in acromegaly there is simultaneous enlargement of the shafts of the long bones. (See chapter on the Hands and Arms.) It is to be remembered that in both acromegaly and pulmonary osteo-arthropathy the enlargement seems to be due to hypertrophy of all the tissues composing the foot, whereas, on the other hand, in myxedema the foot, though enlarged, is puffed and swollen in appearance through *increase* of the subcutaneous tissues alone. Often the foot appears to be a good deal enlarged as the result of deformity, particularly that which consists in partial displacement of the articular surfaces of the metatarsal and phalangeal bones through the wearing of badly fitting shoes, or joint troubles, of which we shall speak later.

Under the name of "*sciopedy*" Power has reported a case of congenital symmetrical enlargement of the anterior part of the foot

not involving the heel. Any enlargement of the legs associated with this condition, he states, *is due to* hypertrophy of the muscles resulting from the effort to lift the feet.

In some cases of locomotor ataxia *flatfoot* from loss of the plantar arch is seen, and various dystrophies of the joints take place as the disease progresses.

The peroneal leg type of progressive muscular atrophy may so closely resemble the so-called Aran-Duchenne type of progressive muscular atrophy as to defy diagnosis, but, as a rule, the latter disease affects the arms first, and sensation is not involved. (See early part of this chapter on the Hands and Arms.)

Wasting of the muscles of the inner surface of the foot affecting the big toe, and those on the outer side involving the movements of the little toe, the interossei and the flexor brevis communis, may occur from the neuritis due to locomotor ataxia, and as the plantar aponeurosis retracts the toes are rendered immovably flexed; in other cases in place of flexion there is strong extension.

The Joints.—The joints of the lower limbs may be swollen from an arthritis arising from many causes, such as septicemia, gonorrheal infection, syphilis, acute articular rheumatism, typhoid fever, tuberculosis, cerebrospinal meningitis, locomotor ataxia, hemiplegia, rheumatoid arthritis (arthritis deformans), acute myelitis, Morvan's disease.

Gonorrheal Rheumatism.—When *arthritis is due to gonorrheal infection it* is generally seen in the knees or ankles, and occurs in men as a rule. It is an infectious arthritis and lasts very persistently, often attacking at the same time joints so rarely involved by rheumatism as the jaw, the vertebral joints, and the sterno-clavicular articulation. According to the late Dr. Howard, of Montreal, it occurs in five forms:

(*a*) Arthralgic, in which there are wandering pains about the joints, without redness or swelling. These persist for a long time.

(*b*) Rheumatic, in which several joints become affected, just as in subacute articular rheumatism. The fever is slight; the local inflammation may fix itself in one joint, but more commonly several become swollen and tender. In this form cerebral and cardiac complications may occur.

(*c*) Acute gonorrheal arthritis, in which a single articulation becomes suddenly involved. The pain is severe, the swelling extensive and due chiefly to peri-articular edema. The general fever is not at all proportionate to the intensity of the local signs. The affection usually resolves, though suppuration occasionally supervenes.

(*d*) Chronic hydrarthrosis. This is usually monarticular, and is particularly apt to involve the knee. It comes on often without

pain, redness, or swelling. Formation of pus is rare. It occurred only twice in 96 cases tabulated by Nolen.

(*e*) Bursal and synovial form. This attacks chiefly the tendons and their sheaths, and the bursæ and the periosteum. The articulations may not be affected. The bursæ of the patella, the olecranon, and the tendo Achillis are most apt to be involved.

Acute Articular Rheumatism.—Acute articular rheumatism in the knee or ankle produces swelling of the joint, redness, heat, exquisite tenderness, immobility from pain, swelling of the surrounding tissues. It does not remain for a long period unchanged in one joint, is a process accompanied by fever, and usually modified by full doses of the salicylates.

Acute Synovitis.—Acute synovitis is generally the result of an injury, is confined to one joint, is often accompanied by far greater effusion into the joint than is seen in rheumatism, and there is no systemic disturbance. Should a single joint be affected by an aberrant attack of acute rheumatism or synovitis not following injury, the physician should never forget the possibility of its being a syphilitic arthritis.

Locomotor Ataxia.—The most marked alterations in the joints are those produced by advanced locomotor ataxia, and are called arthropathies. Often they are associated with spontaneous fractures of the bones. The knees are most commonly involved, then the ankles and hips. A joint or several joints may become suddenly swollen with or without pain, and without apparent cause, until the swelling becomes quite massive. There are then developed osseous hyperplasia and a tendency to dislocation with crepitation on movement, and the ends of the bone become worn away and absorbed. Dislocation and fractures are common, and the bones are atrophied.

Rheumatoid Arthritis.—In rheumatoid arthritis there is a gradual enlargement of the joints from accumulation of fluid, which in turn is absorbed, leaving the articulating surfaces roughened, uneven, and deformed, but there are no deposits of urate of sodium as in gout, the deformities being due to alterations in the articulating surfaces themselves, and the peri-articular development of bone. The disease always remains in the joint originally attacked, although new joints are involved. Pain is often severe, dislocations and fractures are rare, and the small joints are often involved. (See chapter on the Hands and Arms.)

Rheumatoid arthritis when it progresses to an advanced stage causes great deformity by the locking of the joints through the development of osteophytes. By the destruction of the cartilages, wasting of the muscles, and thickening or contraction of the ligaments, it may cause dislocation of all sorts, and false positions. In the great

majority of cases it occurs in women between twenty and thirty years of age, but it may develop in early childhood. Pain is severe in some cases, absent in others. The thighs become flexed upon the abdomen, and the leg on the thigh. The number of joints involved varies greatly, but the involvement is generally symmetrical.

Sometimes this disease, which is generally gradual in its onset, becomes very acute, speedily involving many joints, causing swelling of the synovial sheaths and bursæ, and being accompanied by some febrile movement. The suddenness of its onset, the febrile movement when the onset is sudden, and the pain may cause it to resemble acute articular rheumatism, but the absence of redness in the joints and of the migration of the swelling from one joint to another which is the characteristic of acute articular rheumatism, aids in the differentiation. The arthritis of acute central myelitis is sudden in its onset, generally multiple, and accompanied by the other symptoms of that disease. (See Paraplegia and Anesthesia of the Skin.)

Arthritis of Cerebrospinal Meningitis is a typical infectious arthritis, and the presence of the characteristic signs of the disease renders its cause evident. The joints are many of them affected simultaneously with swelling, pain, and serous or purulent effusions.

Septic Arthritis.—In cases of septic arthritis the joints become swollen and often suppurate, so that the articular surfaces become more or less destroyed. This may occur after infection during the puerperium or in any case of pyemia. Another arthritis, probably infectious, is sometimes seen in epidemic dysentery and in scarlet fever. Rarely immediately after or some months after typhoid fever a hypertrophic osteo-arthritis comes on as a result of a local disease produced by the bacillus of Eberth. This is to be separated from ordinary septic arthritis following typhoid fever.

Acute Osteomyelitis.—The onset of an inflammation in the lower end of the femur or in the upper end of the tibia, producing what, at first glance, seems to be an arthritis and sometimes simultaneously involving other areas near joints, should raise a suspicion of acute osteomyelitis, which is a fatal disease in many cases unless surgical aid comes to the patient. The symptoms consist of boring pain in the part, great tenderness and swelling, and the skin soon breaks down as a purulent and offensive discharge makes its way to the surface.

Closely allied to this is the *acute epiphysitis of infancy*, in which there is suddenly developed a chill followed by great pain and swelling of the joints or their neighborhood. The skin becomes engorged with blood and the joint fills with pus. Care must be taken to separate this condition from rheumatism and the joint swelling sometimes seen after typhoid fever and that form which is due to infantile scurvy.

Subperiosteal Hematoma.—Great swelling of the thigh or leg occurring in a child may be due to subperiosteal hematoma

Möller-Barlow's disease). Aspiration of the swelling will reveal the haracter of its contents, and the fact that the child is usually a ufferer from rickets will aid in the diagnosis.

Gout.—Although gout is capable of causing deformity in the lower xtremities, it has one fact about it which is of practical importance, amely, that it involves the small joints of the foot, while rheumatism ttacks the large joints, such as the knee, by preference. Gout nvolves the feet most commonly, the big toe being the favorite place or gouty manifestation, whereas rheumatism is more frequently seen n the hand, if small joints are affected. Aside from the swelling, red- ess, and exquisite tenderness of gouty joints, all of which symptoms xceed in acuteness, if possible, similar manifestations in acute rheu- natism, there is often an additional and permanent cause of deformity n the chalk stones which are deposited about the joints, and which re never seen in rheumatism. The history of frequently recurring ttacks lasting but a few days, accompanied by enlargement of the eins about the joint and shedding of the skin locally, points, when dded to the symptoms named, to a typical case of gout. In chronic ases it may be almost impossible to determine whether a case be ne of chronic rheumatism or gout unless chalk deposits can be ound. (See chapter on the Hand.)

Sometimes in chronic lead poisoning we have developed what is nown as plumbic gout, owing to the deposition about the joint of rate of lead and sodium.

Dengue.—The onset of a multiple arthritis, with which there are eadache, chills, intense aching in the bones, joints, and muscles, nd a fever rising as high as 106° or 107°, and rarely an erythematous ash, may indicate the presence of dengue. The joints are swollen nd painful, and often both the large and small ones are involved.

In Schönlein's Disease, which is a form of very severe purpura, nultiple arthritis, with great pain, and purpuric eruptions occur nd the presence of the subcutaneous exudate with edema and loughing of the mucous membrane of the mouth adds to the pic- ure. The patient seems very ill, but death rarely follows. Such ases are rare, but the writer saw one in consultation with Dr. Wilson, of Woodbury, New Jersey, in which alarming sloughs of he tonsils and buccal mucous membrane occurred in addition to he arthritis changes. (See chapter on the Skin.)

Very nearly allied to this are the *joint involvements of hemo- philia*, which in their sudden onset and pain closely resemble rheu- natism, particularly as the large joints are commonly involved. The istory of the patient being a bleeder, or of his being related to one, may clear up the diagnosis.

Spontaneous Dislocation of the Hip.—So-called spontaneous dislo- ations of the hip have been recorded during convalescence from

typhoid fever, scarlet fever, and acute rheumatism. The luxation in the latter disease usually follows severe pain, and the ligaments are often found torn from their attachments. In typhoid and scarlet fevers the dislocation occurs insidiously and announces itself by the pain it causes.

Intense Swelling.—Intense swelling of the leg, aside from that due to ordinary edema, may be due to milk leg, which is a condition of swelling of the entire limb, generally limited to one side, and seen during the puerperium or after any one of the infectious fevers, such as typhoid. The joints are not particularly affected. On the contrary, the calf of the leg is the part most affected, it being white, firm, hot, but slightly, if at all, edematous. Pain is excessive, there is entire loss of power in the affected limb, and its temperature is much higher than normal.

If the swelling of the leg is bilateral and pits on pressure, it is practically always the result of anasarca from renal or cardiac disease; but if unilateral, it may be, as just stated, due to thrombosis of the femoral vein. (See chapter on the Skin; Edema.)

Gangrene.—Three very important and serious alterations in the nutrition of the foot remain to be noted, namely, perforating ulcer due to tabes dorsalis, diabetic gangrene and senile gangrene.

Perforating ulcer usually appears in one foot, beginning with the formation of a bleb, which changes to an abscess, which in turn is followed by necrosis of all the tissues of the foot immediately underlying the destroyed skin. With it are associated the signs of ataxia. Sometimes perforating ulcer of the foot occurs during the course of diabetes mellitus, but it is probable in many such cases that locomotor ataxia is associated with diabetes.

In *diabetic gangrene* the toes are nearly always affected in preference to other parts of the body. An analysis of the urine will aid the diagnosis. (See chapter on the Skin.)

In *senile gangrene* the age of the patient, the presence of bad blood-vessels, and the absence of a sufficient cause for gangrene, as, for example, trauma, separate the case from any other condition, while the additional facts that senile gangrene generally affects the inner side of the foot, especially the big toe, and is a dry gangrene, render the diagnosis easy.

Gangrene of the lower extremities sometimes follows the infectious diseases, such as scarlet and typhoid fevers,[1] from thrombosis of the femoral artery. It may also occur in the course of exophthalmic goitre.

More rarely gangrene of the foot and hand follows embolism due to cardiac valvular disease. It is extremely painful, and septic fever may ensue.

[1] See "The Medical Complications and Sequelæ of Typhoid Fever," by the author. Lea Brothers & Co., Philadelphia, 1899.

Mycetoma.—In this connection mention may be made of "Madura foot," or mycetoma, a chronic local disease of tropical climates, and called "fungus foot disease" in India. A small tumor develops on the foot or hand, which, after the lapse of twelve to twenty-four months, bursts and leaves several sinuses from which escape black particles or whitish-red bodies like fish-roe. The disease may spread up the leg. The pale particles in the discharge look like actinomyces.

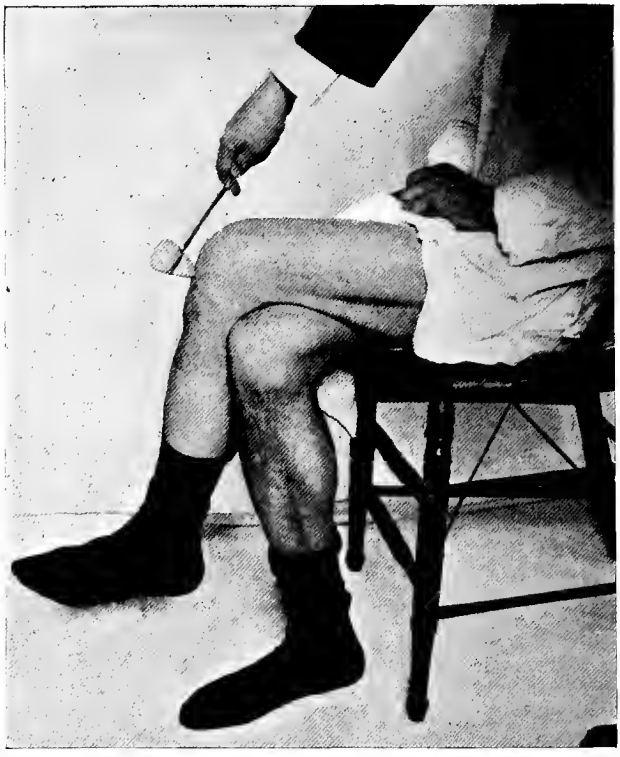


Fig. 31.—Testing the knee-jerk with the percussion hammer.

Ainhum.—Ainhum is a disease peculiar to dark-skinned races, characterized by gradual drying up and separation of the toes (by a constriction), usually the little toe. It has been thought to be related to leprosy, but this is doubtful.

Periosteal Thickening.—Alterations in the appearance of the tibiæ or shins often give us a clear idea of the presence of late syphilis, either because of gummatous swellings in this neighborhood or owing to the development of periosteal thickening and exostoses.

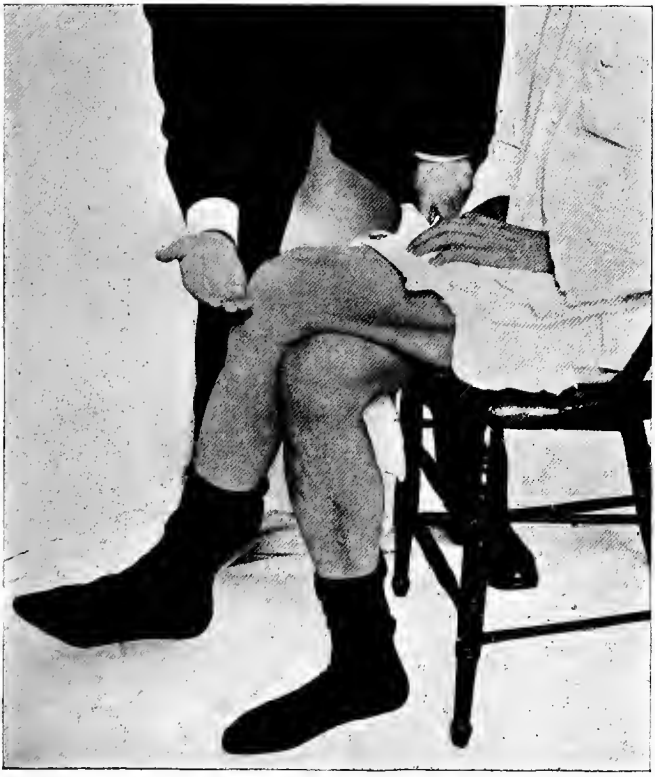


FIG. 32.—Testing the knee-jerk by a blow with the ulnar edge of the hand.

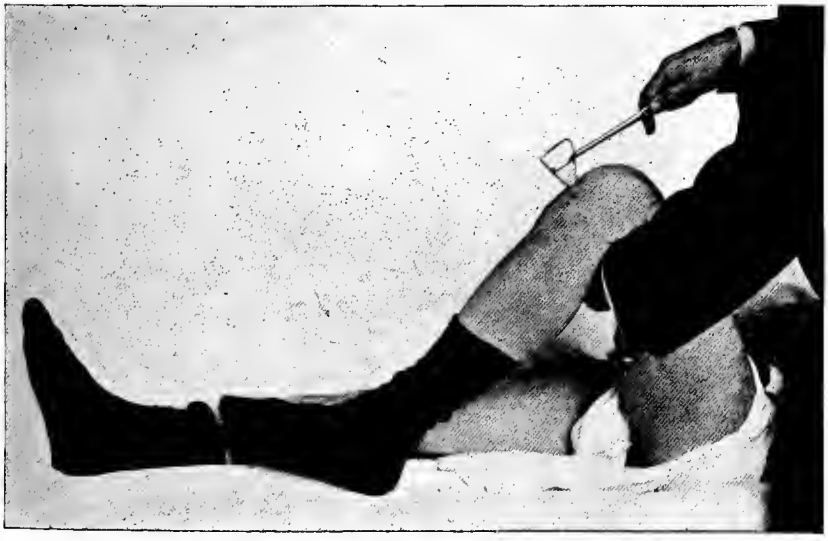


FIG. 33.—Testing the knee-jerk by means of the percussion hammer, the patient being recumbent.

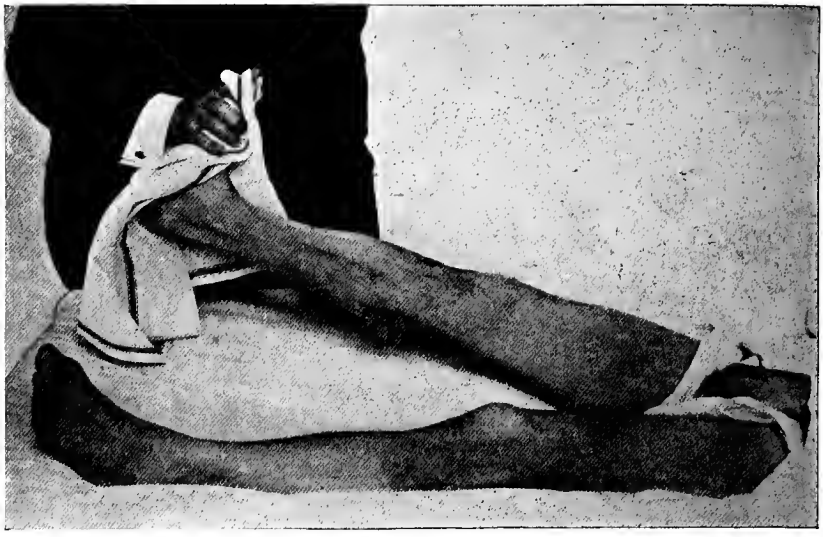


FIG. 34.—Testing ankle clonus with the patient recumbent.

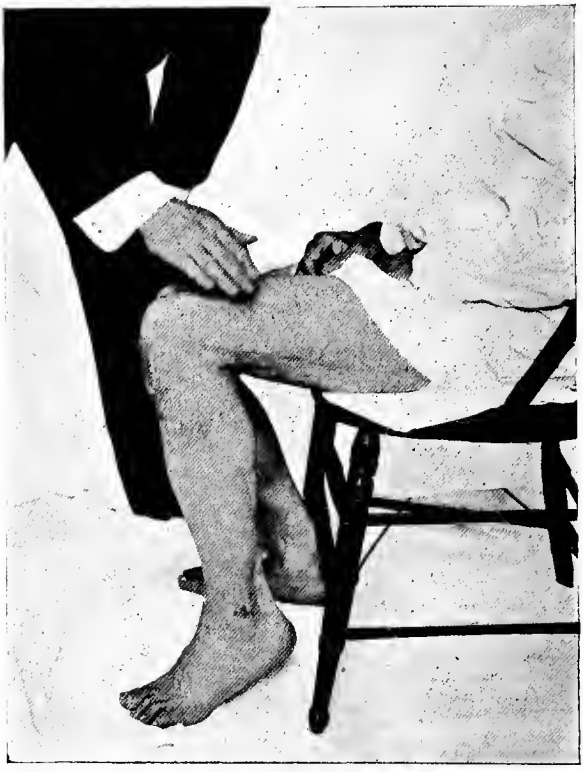


FIG. 35.—Testing ankle clonus when the patient is seated,

Physical Methods Employed in Examining the Feet and Legs. **The Knee-jerk.**—The method of testing the knee-jerk consists in seating the patient on a chair of ordinary height, instructing him to cross his legs in the position which he would occupy if sitting at ease, and then to sharply tap the tendon of the knee-cap, between the patella and its insertion into the tibia, by means of a rubber hammer or with the ulnar edge of the hand. (See Figs. 31 and 32.)

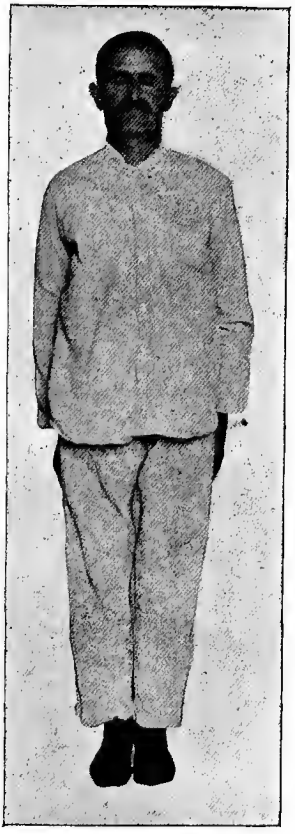


FIG. 36.—Testing the "station." Eyes closed; feet close together.

When the patient is too weak to sit up, the lower limb may be elevated from the bed so that the thigh and leg are at an angle of about 45 degrees, the lower third of the thigh resting on the left hand of the physician in such a way that the leg hangs supine. (See Fig. 33.)

Ankle Clonus.—This test can be made when the patient is seated or recumbent. When recumbent the examiner grasps the leg near

the knee with the left hand in order to support it. He then extends the leg on the thigh with the right hand, grasping the ball of the foot. When the limb is in this position, the leg being well extended on the thigh, the foot is flexed sharply toward the knee and immediately released from pressure. (See Fig. 34.) If clonus is present, the foot will undergo a series of coarse, tremulous movements in extension and flexion.

Another method of testing clonus is to seat the patient in a chair with the leg resting on the ball of the foot on the floor, and then to apply sudden pressure, which is quickly relieved, to the knee, when, if clonus is present, the limb undergoes a series of tremulous up-and-down movements. (See Fig. 35.)

Station.—This is determined by having the patient stand with his feet close together and then directing him to close his eyes, or if he cannot be relied upon to completely close his eyes, to blindfold him. (See Fig. 36.) A normal patient should stand almost perfectly steadily under these circumstances, but in several diseases, notably locomotor ataxia, he will sway very greatly, or even fall.

The Cremasteric Reflex.—This reflex consists in a retraction of the testicle and scrotum upon irritating the skin upon the inner surface of the thighs. For methods of testing the various conditions of sensibility in a limb, see chapter on the Skin.

CHAPTER IV.

HEMIPLEGIA.

HAVING considered the manifestations of disease as seen in the arms and legs in connection with monoplegia and paraplegia, spasm and contracture, we must now study the diagnostic meaning of hemiplegia, or that form of paralysis which involves the arm and leg and head on one side of the body. This form of paralysis, when complete, is always due to a lesion arising above the spinal cord—that is, in the brain, and is due to lesions in the lower tracts of the brain or in its cortex. The character of the paralysis, the association of other symptoms with it, and the history of the patient and of his illness will render a diagnosis easy as to the approximate site of the lesion in most cases. The most common causes are hemorrhage into the cerebral tissues from a ruptured bloodvessel, or embolism or thrombosis of some vessel supplying important areas. Still other causes of hemiplegia are brain tumors, meningeal hemorrhage, degenerative processes, and hysteria.

Before we enter into consideration of the various symptoms resulting from central nervous lesions it is well to stop for a moment for the purpose of clearly understanding the anatomy and physiology of the parts involved, in order that we can properly study the results of lesions in the nerve centres or nervous tracts.

It is not necessary to remind the reader that the brain is divided into three areas, the frontal area being concerned with intellection, the middle area with motion, and the posterior area with sensation and special sense. These areas are again divided into subareas, each of which governs or is connected with several functions, and still further subdivisions exist, in which reside the centres governing small areas, as, for example, a single muscle or group of muscles. (See Fig. 37.) Disease of any part of the brain surface, therefore, modifies more or less the function of that part and the part of the body tributary to it. Beneath the surface, through the so-called white matter, various fibers pass, which carry to or from the centres in the cerebral cortex the impulses connected with their function, and these fibers approximate one another more and more closely in the lower part of the brain until they form a bundle (the corona radiata). Thus we see in Fig. 37 how the fibers arising from the middle area of the cortex cerebri pass down through the

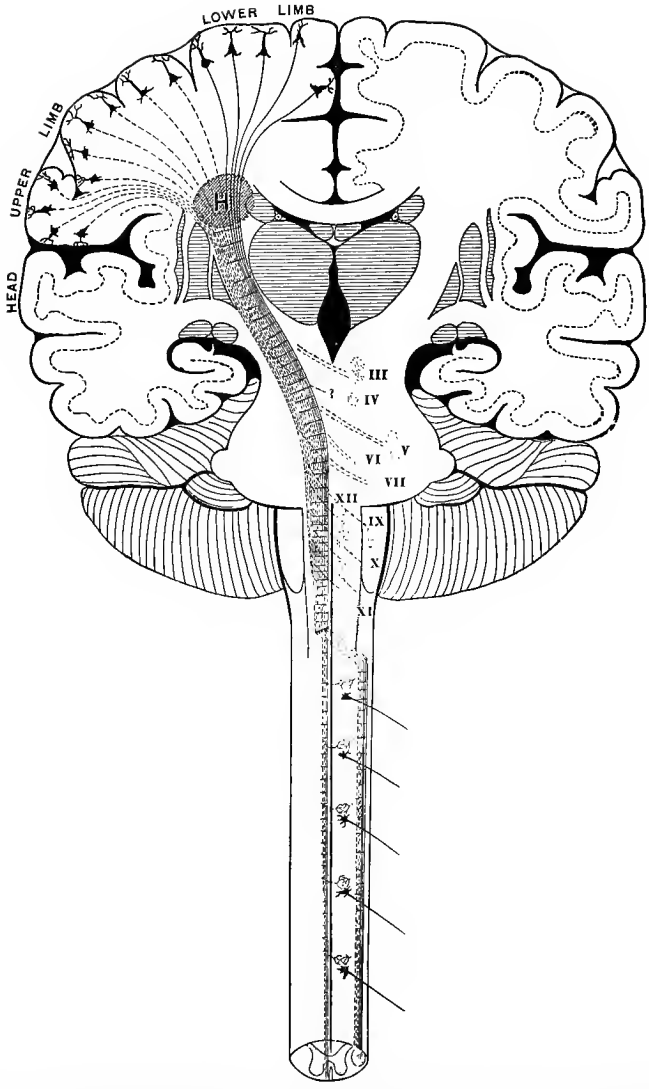


FIG. 37.—Diagram showing the fibers from the cortex forming the corona radiata, which after they are approximated pass into the internal capsule. It also shows the decussation of the pyramid of the left side, which passes to the right side of the spinal cord, and the direct or uncrossed tract (Türck's column). Finally it also shows the secondary degeneration which occurs after cerebral hemorrhage or softening, and which follows the course of the motor tracts into the spinal cord. *H.* Site of lesion. The continuous lines are fibers going to the legs, the dotted are those going to the arms and motor cranial nerves. (Modified from Van Gehuchten.)

lenticular nucleus into the knee or angle of what is called the internal capsule. These fibers are arranged in such a way that those arising from the lower part of the cortex, as in the face center, lie nearest the knee of the capsule, and those highest, farthest from

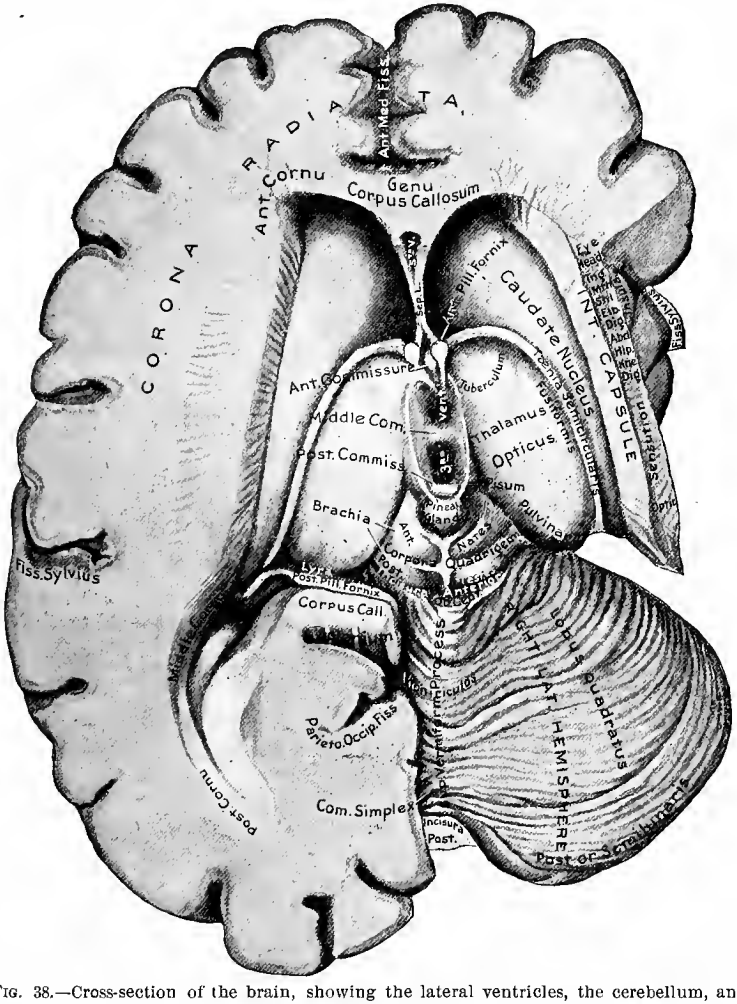


FIG. 38.—Cross-section of the brain, showing the lateral ventricles, the cerebellum, and, most important, the cross-section of the motor fibers in the internal capsule. (Modified from Fuller.)

this point (Fig. 38). After the motor fibers have passed through the internal capsule they pass into the crus cerebri of that side, which (the crus cerebri) connects the hemisphere of the same side with the cerebellum behind it, and the pons and medulla below it. The crura

cerebri are two thick, cylindrical bundles of white matter which emerge from the anterior border of the pons (see Plate II), diverge as they pass upward and outward to enter the under part of each hemisphere, as if stretching out to receive the motor fibers from the internal capsule. From the crura cerebri the motor fibers pass downward

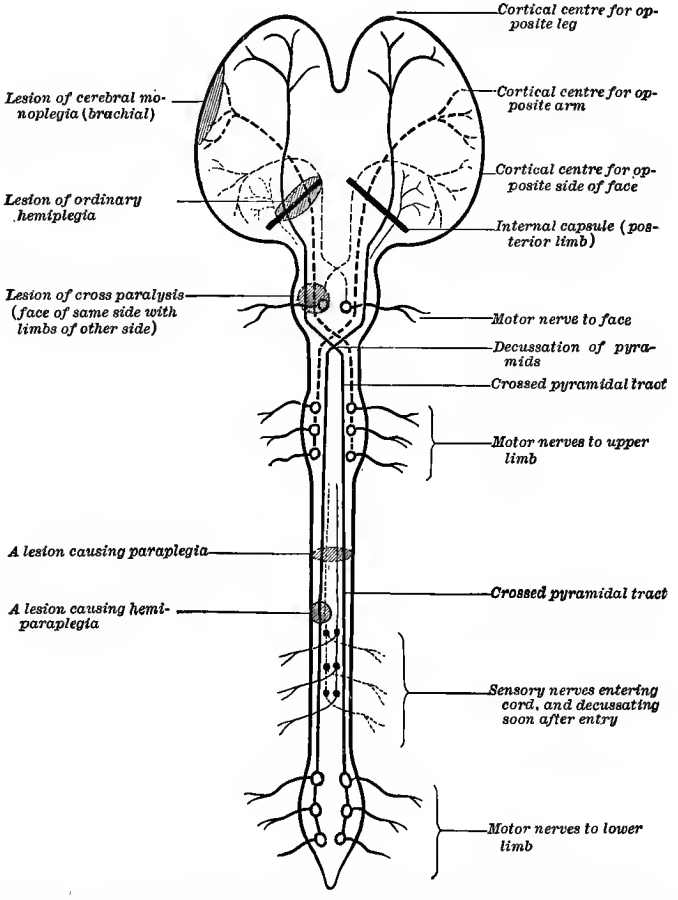
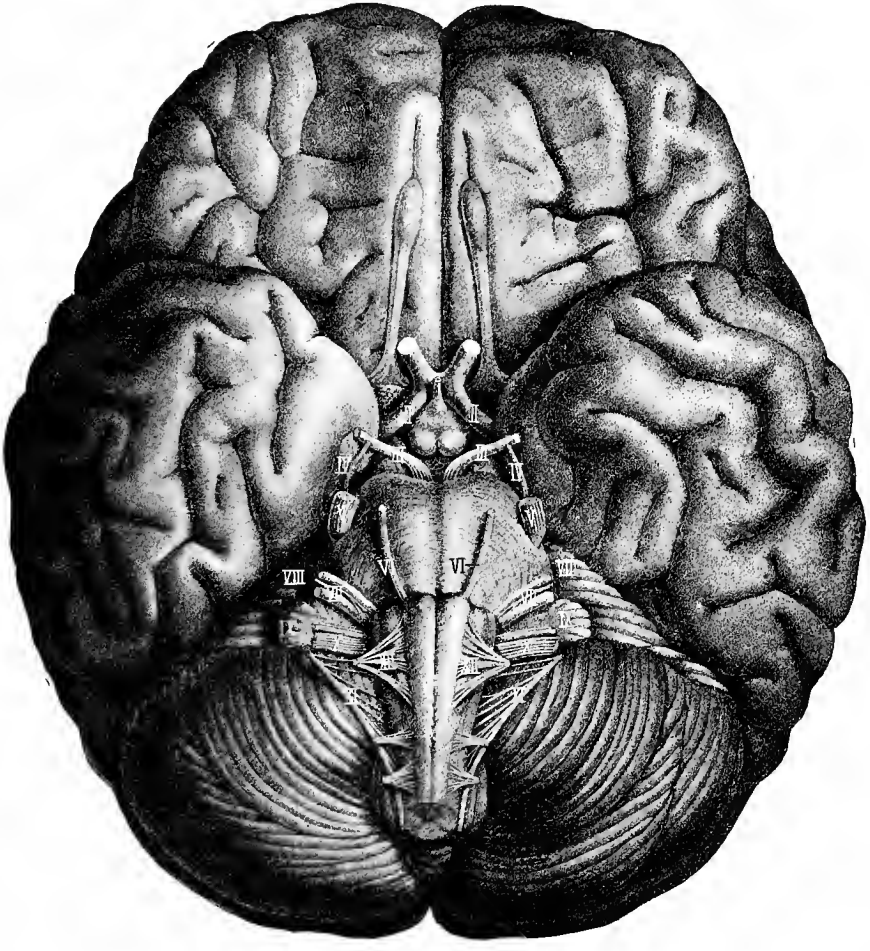


FIG. 39.—Diagram showing the general arrangement of the motor tract and the effect of lesions at various points. (Ormerod.)

into the pons Varolii. Here the fibers which have hitherto travelled together divide into two parts, namely, those from face and tongue centre, which pass to the opposite side and become connected with the nuclei of the facial and hypoglossal nerves, which act as minor centres governing the face and tongue, and the fibers for the arm, leg, and trunk of the body, which continue on down to the medulla

PLATE II.



Base of the Brain, showing the Cranial Nerves.

The crura cerebri are seen on either side of the posterior perforated space and under the third nerves. The various cranial nerves are numbered with Roman numerals. (Modified from Arnold.)

oblongata, where they form the so-called pyramids, and having done so most of the fibers cross to the opposite side of the spinal cord (the crossing of the pyramids), and so form the crossed or lateral pyramidal tracts. (See Fig. 37.) A smaller number of fibers, however, pass directly down to the spinal cord from the medulla oblongata, and form what is called the direct or anterior pyramidal tract. Direct, because it does not cross; anterior, because it lies along the

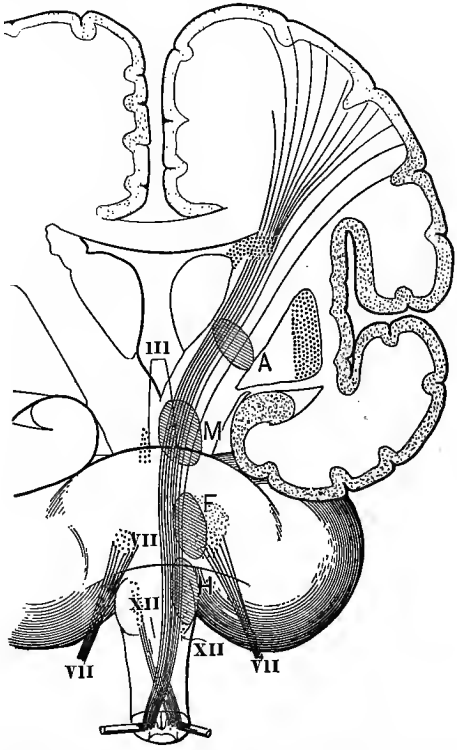


FIG. 40.—Showing the mechanism of different hemiplegias. A lesion at *A* causes complete hemiplegia by destroying the motor tract. One at *M* causes paralysis of third cranial nerve (motor oculi) by destroying its nucleus or root on same side, and paralysis of arm and leg on opposite side. A lesion at *F* causes facial palsy on same side, hemiplegia on opposite side. In a lesion at *H* the hypoglossus would be affected on one side, with hemiplegia on the other. (Modified from Edinger.)

edge of the anterior fissure of the cord; pyramidal, because it comes down from the pyramid. This is sometimes called Türck's column. (See Fig. 37.) It is by means of these two tracts in the spinal cord that motor impulses pass down to the nerve trunks and muscles.

We can understand, therefore, that if a small lesion occurs at the peripheral endings of the corona radiata—that is, on the cerebral

cortex—it will only produce a limited paralysis. Thus, as seen in Fig. 37, a clot at the arm centre would only involve the arm fibers. If, however, the lesion be lower down where the fibers of the corona radiata are getting closer and closer, as, for example, in the internal capsule, then even a small lesion will produce widespread paralysis, since it will involve a large number of fibers running ultimately to widely separated areas in the body, and, if large enough, produce hemiplegia. (See Fig. 37, lesion *H*, and Fig. 40, lesion *A*.) If the lesion be situated in the pons on one side, it will cause facial paralysis on that side and hemiplegia on the opposite side of the body, because, as shown in the diagrams (Figs. 39 and 40, *M*), it will, under these circumstances, destroy the facial fibers after they have crossed, and the remaining motor fibers before they cross. The various tracts, motor and sensory, in the spinal cord are shown in Fig. 41.

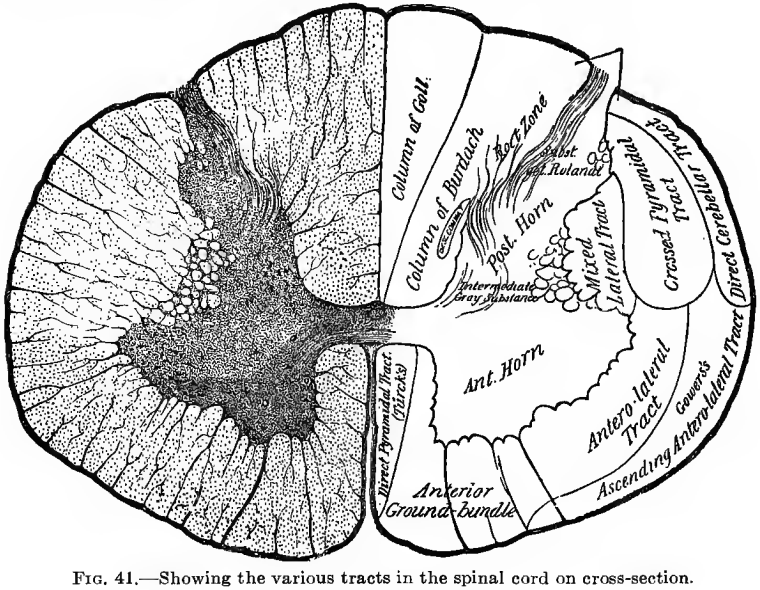


FIG. 41.—Showing the various tracts in the spinal cord on cross-section. (Nervous Diseases, Mills.)

Hemiplegia from Hemorrhage is characterized by sudden onset in most cases, by more or less mental disturbance and disorders of motion, sensation, and of the special senses according to the site of the leaking vessel. The skin reflexes are apt to be markedly decreased and the deep reflexes increased, but the bladder and rectum are not usually paralyzed, although in the first shock of the accident there may be vesical and rectal incontinence. The mental disturbance usually amounts to a rapidly oncoming unconsciousness in hemorrhagic hemiplegia.

The question of the location of the lesion is very important. In the great majority of cases it is situated above the point at which the decussation of the motor fibers takes place in the medulla, and is, therefore, on the opposite side of the body from that on which the hemiplegia exists. If, however, the lesion be below the decussation, the paralysis and lesion are on the same side, as just described. The most common site for the lesion in hemiplegia is in the knee or posterior limb of the internal capsule, owing to the fact that the middle cerebral artery in one of its lenticulo striate branches perforates the internal capsule, and ends in the caudate nucleus, and this artery is so commonly ruptured that Charcot has called it the "artery of cerebral hemorrhage" (Fig. 42). If the hemorrhage does not involve the posterior third of the internal capsule, there are no sensory symptoms associated with the motor loss, but the

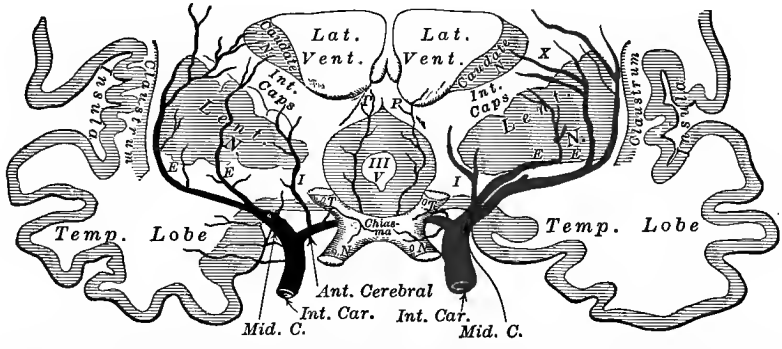


FIG. 42.—Showing distribution of bloodvessels to internal capsule. The artery marked *x* is the so-called artery of cerebral hemorrhage, and it is readily seen how its rupture destroys the fibers in the internal capsule. (Modified from Gordinier.)

paralysis will be practically universal on that side, involving the leg and arm, and the lower part of the face, so that the mouth is drawn toward the healthy side. (Explained by Fig. 38.) The symptoms associated with hemiplegia due to this cause often become very severe, because the hemorrhage is so profuse that the lateral ventricle becomes filled with blood, and from it the blood passes to the third and from there to the fourth ventricle, where, by pressure on the vital centres, it speedily produces death. In such cases deep unconsciousness, stertorous breathing, a slow, full pulse, and a flushed skin, becoming somewhat cyanotic, may be present. Recovery never occurs, for the secondary inflammation, or softening, following the outflow of blood produces fatal results, even if the patient survives for some days

In cases in which the hemorrhage is very limited consciousness may be lost for only a brief period, and at most there may be only

mental confusion. Often in mild cases there is a slight return of power in the affected side within a few days, and the temperature of the affected part, which has been raised, approaches the normal. Finally, after six to eight weeks, the dominant symptoms consist in partial loss of power of the arm and leg, and the facial paralysis has perhaps entirely disappeared, although the tongue when protruded may tend to go over to one side. If the case does not pass to such favorable results, instead of recovery of power at this time there are developed contractures and secondary rigidity from degenerative processes extending to the pyramidal tracts. (See Fig. 37.) Hitzig has shown that these conditions are apt to be least marked in the morning. Wasting of the paralyzed muscles only ensues from the disuse and not from true trophic change.

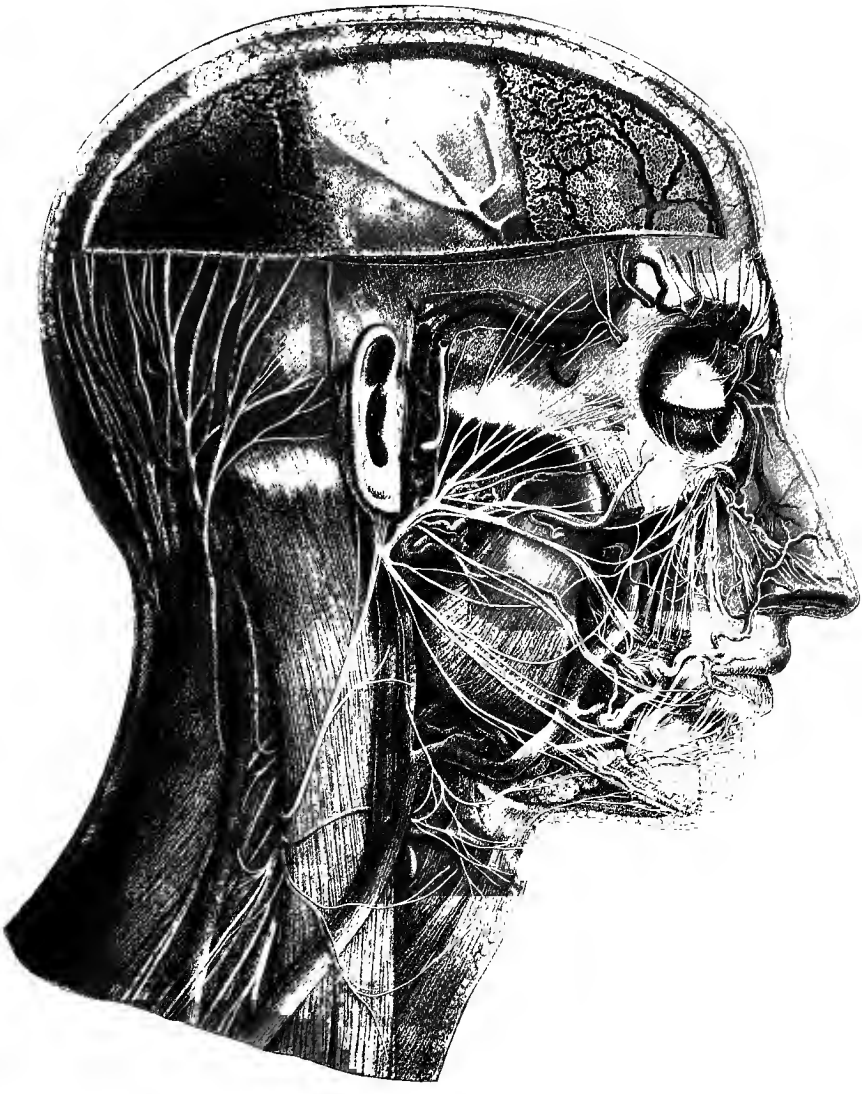
When the case is not of the very severe type which causes death in a few hours, and yet the lesions are such that recovery is not going to take place, the patient at the third or fourth day becomes unconscious a second time, his temperature rises, he mutters, and grows restless, finally becomes comatose, then develops respiratory failure, or a hypostatic congestion of the lungs, and dies.

When a patient is seized with headache, dizziness, vertigo, and vomiting, and rapidly oncoming hemiplegia and hemianesthesia, attended at first with no loss of consciousness, but in a day by unconsciousness and coma, he is suffering from what has been called "*ingravescent apoplexy.*" The hemorrhage, under these circumstances, begins in the knee of the internal capsule, proceeds backward until it involves the sensory fibers in the internal capsule, and, finally, breaks into the lateral ventricle, soon after which death ensues.

When a *hemiplegia is followed by rigidity* very early, with sensory involvement and convulsions, the lesion is probably cortical, or, more correctly is secondarily cortical to a deeper hemorrhage, and spreads over the centres for the face, arm, and leg. Most commonly, cortical hemorrhages are due to injuries, although they may arise from unprovoked vascular rupture. In any case, they are usually ushered in by convulsions. (See next page.)

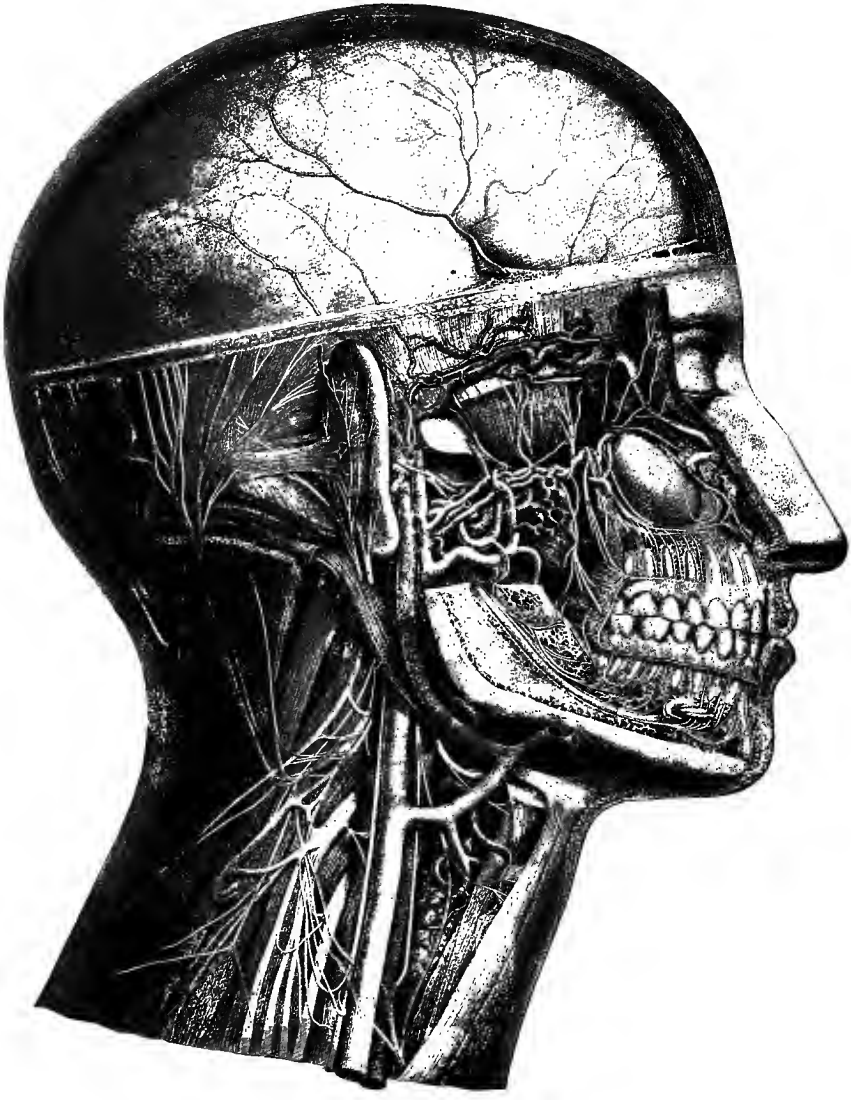
If in the development of symptoms of cerebral hemorrhage there be little hemiplegia and temporary unconsciousness, followed in some hours by a sudden aggravation of the symptoms, it may be that in the beginning of the attack the lesion has been in the frontal lobes, but has gradually extended backward until it has ruptured into the lateral ventricle. So, too, a hemorrhage into the occipital lobe or the posterior part of the parietal lobe is rarely marked by much hemiplegia, and, if present, the leg is more paralyzed than the arm. A characteristic symptom of this lesion is, however, well-marked hemianesthesia (see chapter on the Skin) and hemianopsia. (See

PLATE III.



Showing the Area of the Middle Meningeal Artery, in the Inner Table of the Skull, Injury to which is Sometimes the Cause of Hemiplegia. (Arnold's Atlas.)

PLATE IV.



The same Artery with the Inner Table of the Skull Removed. (Arnold's Atlas.)

chapter on the Eye.) Generally, however, such changes result from a thrombosis.

Hemiplegia developing after an injury to the head is usually due to rupture of the middle meningeal artery or one of its branches. (See Plates III and IV.) Owing to the fracture of the skull, the vessel is ruptured. A pathognomonic sign of this accident is the fact that there is a period of consciousness between the time of injury and the development of unconsciousness. The patient may be momentarily stunned, then recover consciousness, and then relapse into a second period of unconsciousness which rapidly deepens into profound coma. It is important to remember that while the paralysis is on the opposite side of the body from the hemorrhage, the hemorrhage may be on the opposite side from the external injury, since the fracture of the skull may be a fracture by *contre-coup*. The paralysis is usually progressive from part to part, and convulsions may occur as a result of cortical irritation produced by the hemorrhage. A wide dilatation of the pupil on the side of the hemorrhage ("Hutchinson's pupil") may be present. Such a hemorrhage is said to be supradural. Sometimes the hemorrhage arises from the ascending frontal or ascending parietal artery, and is then subdural.

The following table, from Mills' *Nervous Diseases*, gives the differentiation between ventricular and meningeal bleeding:

Ventricular Hemorrhage.	*Meningeal Hemorrhage.*
No antecedents to explain the lesion.	Antecedents: for the newborn a prolonged or hard labor; for the adult a traumatism.
Premonitory symptoms not rare.	Premonitory symptoms generally absent.
Gradually deepening unconsciousness.	When traumatic, unconsciousness followed by partial restoration of consciousness and then again by unconsciousness.
Cephalalgia rare.	Cephalalgia frequent.
Paralysis when present is usually hemiplegic.	Paralysis ordinarily generalized; hemiplegia exceptional.
Varying pupillary conditions.	Unilateral dilatation of pupil common.
Deviation of the mouth and tongue very common.	Deviation of the mouth and tongue rare.
Contractures very common.	Contractures, although frequent, less common.
Convulsions may be present, but not common.	Convulsions the rule.
Vomiting not frequent.	Vomiting frequent.
No symptoms of secondary meningitis with fever.	Symptoms of secondary meningitis with high fever toward the third or fourth day.
Death rapid.	Life, as a rule, prolonged several days.

When there is developed in cases of hemiplegia, *aphasia* or disordered speech, there is probably a lesion in the neighborhood of the third frontal convolution, or the island of Reil. (See chapter on Speech.)

Hemiplegia may be due to *cerebellar hemorrhage*, in which case there are loss of consciousness deepening into profound coma, contracted pupils, vomiting in many of the cases, and finally death when hemorrhage breaks into the lateral ventricle. The diagnosis of cerebellar hemorrhage is very difficult.

Of the *irregular forms of hemiplegia* there are several. Sometimes the leg is from the beginning more affected than the arm, and remains paralyzed long after the face and arm have recovered. The leg may become rigid and distorted by contractures, and there will often be found present marked anesthesia of the skin of the paralyzed leg and arm, with hemianopsia and aphasia. Such symptoms indicate a lesion of comparatively small size involving the leg fibers and some of the sensory fibers in the internal capsule, and result from rupture of the lenticulo-optic artery. When the arm suffers most the symptoms just described as occurring in the leg are found in it, and motor aphasia, if the lesion is on the right side, is often very marked, as is also facial paralysis. This is supposed to be due to the anterior frontal artery, a branch of the inferior anterior cerebral artery, becoming diseased.

When *posthemiplegic chorea* attacks the paralyzed limbs there is often a focal lesion in the posterior extremity of the internal capsule.

Embolism and Thrombosis.—The symptoms which have just been detailed may also arise, as already said, from *embolism* or *thrombosis* of the cerebral vessels. How are we to separate the hemiplegias of hemorrhage and occlusion? In many cases this is impossible, but there are some differential points which may aid us. In the first place, thrombosis is a condition of advanced age, while hemorrhage may occur at any time from thirty years of age on. The presence of hemiplegia in a young man, therefore, is probably not due to a thrombosis. Again, hemorrhage occurs often after exertion or the drinking of stimulants, and occurs rarely in sleep, whereas thrombosis not rarely comes on under these circumstances, and often develops during the night, so that the patient awakes paralyzed, but a patient may have both thrombosis and apoplexy. In hemorrhage, consciousness is generally lost, whereas in thrombosis it is often only dimmed. Vomiting and contracted pupils from pressure on the lower centres indicate hemorrhage, while their absence may point to thrombosis. Finally, the general systemic shock and febrile movement are apt to be greater in hemorrhage than in thrombosis. The history of syphilitic infec-

tion, producing an endarteritis, also points to thrombosis, although hemorrhage may arise from this cause also.

The diagnosis of *embolism* producing hemiplegia from the paralysis due to hemorrhage is always more or less difficult, but the presence of chronic or ulcerative endocarditis or their results, or other cause for the formation of emboli, aids the diagnosis. Where the cause is embolism the onset is sudden, whereas in thrombosis it is sometimes more gradual. The paralysis from embolism is more commonly on the right side of the body, owing to the fact that it is more easy for an embolus to pass into the left middle cerebral artery than into the right.

Additional Causes of Hemiplegia.—*Spastic hemiplegia* may be due to cerebral tumor, and is often associated with convulsions, particularly if the growth be cortical. Very often the paralysis of cerebral tumor will be found, from the history, to have come on gradually. Thus, the history may be that at first the side of the face has been paralyzed, then the arm and then the leg, and that the complete loss of power has not been sudden but gradual in the part affected, or that a convulsion has left that side, which was previously only impaired in strength, totally paralyzed.

Hemiplegia also comes on as a result of *cerebral syphilis*, and, aside from a history of specific infection and response to specific medication, presents few characteristic signs. The presence of intense headache, convulsions of an epileptiform type, and the fact that the paralysis occurs in some cases in early adult life, point to its origin.

Another cause of hemiplegia is *diffuse cerebral sclerosis* of one hemisphere (not multiple sclerosis), in which the most constant symptoms are, in addition to the paralysis, evidences of motor irritation, such as epileptoid convulsions of a bilateral or unilateral character, rhythmical or arhythmical twitchings of the muscles like chorea, and dementia.

Hemiplegia arising from *acute infantile cerebral paralysis* has many of the distinctive features already described when discussing the paraplegia due to this diseased state. The age of the patient, the occurrence of epileptiform convulsions and of athetosis in the affected parts, and the patient's history are the important points to be recalled in making a diagnosis. The lesion is always due to a cerebral hemorrhage or to embolic softening.

When hemiplegia occurs in *locomotor ataxia*, it depends not upon the disease, but upon a complicating hemorrhage, embolism, or thrombosis.

A slowly developed hemiplegia sometimes results from *disseminated sclerosis*, the pathological process involving the side of the pons and spinal cord; but the intention tremor, the peculiar speech,

the nystagmus, and the very excessive reflexes aid us in the diagnosis of their cause of the loss of power. (See chapter on the Hands and Arms, part on Tremor.)

A form of hemiplegia which is often very misleading is that occurring in *general paralysis of the insane* or paretic dementia. In this disease the patient often has attacks of vertigo, unconsciousness, and more or less marked hemiplegia or monoplegia, sometimes with aphasia if the right side is paralyzed. This form is also liable to be wrongly diagnosticated by reason of the epileptiform convulsions, which frequently occur, and which in connection with the paralysis give the impression in the first attack that there is a hemorrhage of the cerebral cortex. The altered disposition of the patient, the loss of memory and intelligence, the peculiar stumbling speech, and the curious changes in the handwriting are some of the symptoms which complete the diagnostic picture.

In this condition we should not forget the possibility of hemiplegia or monoplegia occurring with suddenness in the course of renal disease producing *uremia* The paralysis may be permanent or only transient, but the urine will be found to be albuminous, and the other signs of renal disorder may be manifest.

Hemiplegia sometimes comes on in *purulent meningitis.* The history of a head injury or of a pyemic or infective process, the cerebral symptoms, the stiffness of the back of the neck, the impairment of the normal movements of the eyeball, and the optic neuritis, associated with the convulsions, make the diagnosis possible.

Crossed Paralysis.—When there is paralysis of the arm, trunk, and leg on one side, with facial paralysis and anesthesia on the opposite side of a well-marked type, associated with early rigidity of the paralyzed side, conjugate deviation of the eyeballs, a very marked rise in bodily temperature, a contracted pupil, and convulsions, with difficulty in swallowing and in speech, the lesion is to be found in the pons Varolii on the side opposite the paralysis. This is due to the fact that the injury is below the decussation of the facial nerve. (See Figs. 30 and 31.) If both sides of the face are paralyzed, with hemiplegia elsewhere, the lesion is in the pons where the facial fibers cross. *Pons paralysis* is nearly always associated with giddiness, vomiting, conjugate spasm with nystagmus, albuminuria, glycosuria, and marked disturbances in the respiration and heart. Pontile hemorrhages are, however, very rare, and usually are rapidly fatal.

If in a case of hemiplegia there is not only paralysis of the arm and leg and of the lower part of the face on one side, but in addition paralysis of the upper part of the face on the opposite side with ptosis on that side due to paralysis of the facial and oculomotor nerves, and with these symptoms impaired sensibility and vasomotor

changes in the limbs, the lesion is probably in the crus cerebri on the side of the upper facial paralysis—that is, on the same side as the ptosis. This is only true if the two paralyses have been simultaneous in occurrence, as it is possible for two lesions occurring at different times and at different places to produce paralysis of the face on one occasion and hemiplegia on another. (See Ptosis in chapters on the Face and the Eye.)

A very rare form of paralysis, in which the arm on one side and the leg on the other side are involved, is due to a bulbar lesion just where the decussation of the pyramids takes place. This is one of the forms of crossed paralysis, and is due to cutting off of one set of fibers before they cross, and the others after they have crossed. (See early part of this chapter and chapters on the Hands and Arms, Feet and Legs, and Face and Head for further information as to crossed paralysis.)

CHAPTER V.

THE TONGUE, MOUTH, PHARYNX, AND ESOPHAGUS.

The general appearance of the tongue—Its coating—Its appearance in poisoning—Fissures and ulcers of the tongue—Eruptions on the tongue—Atrophy and hypertrophy of the tongue—Paralysis—Tremor and spasm of the tongue — Tonsillitis — Diphtheria — Pharyngitis—Disease of the esophagus.

THE TONGUE.

THE appearance of the tongue is recognized as indicative of the general condition of the patient, and is a valuable diagnostic aid in many diseases other than those associated with disorder of the gastro-intestinal mucous membrane. In examining this organ the physician should take note of the condition of its surface, its shape as it lies in the mouth or is protruded, and the character of its movements. He should also see that it is well protruded, and examine the back of it more than the tip, as the latter is the part giving the least information.

Before discussing the precise appearance of the tongue in the various disorders in which it becomes altered in appearance it is well to remember that its surface is covered by mucous membrane, which differs in various parts. The epithelium is scaly and rests upon the corium or mucosa. The mucosa also supports many papillæ, which are thickly distributed over the anterior two-thirds of the tongue on its upper surface. These papillæ give the peculiar roughness which is so characteristic of this surface, and occur in three forms, namely, the circumvallate or large papillæ, the fungiform or mediate, and the filiform. The circumvallate are only eight or twelve in number, and are arranged at the back of the tongue in the shape of the letter V, with the point toward the root of the organ. The fungiform papillæ are scattered freely over the tongue, mostly at the sides and tip, and appear as deep-red eminences, the bases of which are smaller than their free extremities. Their epithelial covering is very thin. The filiform papillæ, which cover the anterior surface of the tongue, are very minute, and arranged in lines corresponding in direction with the two rows of the circumvallate papillæ. From their apices project many fine, filiform processes which are of a whitish tint, owing to the density of the epithelium

of which they are composed. There are, in addition, many simple papillæ which cover the surface between the peculiar ones already described. The fungiform papillæ are those seen most commonly in cases of disease, for they become large and prominent, and because of their red color show through the coating as red dots.

The appearance of the surface of the tongue varies greatly even in health according to the condition of its mucous membrane and the epithelium covering it. The most common alterations in its appearance are due to mere superficial coatings or fur, which consist of dead epithelial cells, microörganisms of many kinds, and abnormally shaped living epithelium. Small particles of food may also be present. Butlin believes that the coating is chiefly due to microorganisms. The question as to how characteristic of a particular disease any one coating or fur may be has been warmly discussed. Some have gone so far as to assert that the coating of the tongue is not indicative of any state in particular, while others, of whom the author is one, are convinced that, while an absolute diagnosis of disease in other organs cannot be based upon the appearance of the tongue, great aid can be gained by its study.

There are, however, very few conditions of the coating of the tongue which are pathognomonic of any one disease, since the coating is produced by the local conditions of the mouth rather than by the disease itself.

Taking up for consideration the various forms of coating, we find that the area at the base between the circumvallate papillæ is always somewhat coated even in the best of health, and that in disease the heaviest coating is generally found in this region, while the tips and sides, even in those diseases in which the coating is heaviest, are generally fairly clean. This is in part due to the character of the epithelium in different parts, and to the fact that the tip and sides are generally scraped clean by the movements of the tongue. Further, it should be remembered that the development of coating, aside from digestive derangements, depends chiefly on three factors: First, immobility of the tongue, so that it is not kept clean by rubbing; second, mouth breathing, whereby the surface becomes dry and less easily cleansed; and, third, fever, which not only dries the surface of the tongue, but interferes with salivary secretion. Additional local causes are a decayed or ragged tooth or follicular tonsillitis, which infects the lingual epithelium, lack of cleanliness, and habits, such as smoking. In the last class of patients, the smokers, a heavily coated tongue in the morning is very common.

The tongue of the *typhoid state*, and of typhoid fever in particular, is quite characteristic, because the prolonged illness, the great exhaustion, and the general apathy of the patient all conspire to produce a peculiar coating on this organ. Early in the disease the

surface of the tongue may be more or less foul, resembling the coating associated with biliousness, in that the back part is coated evenly and with a paste; but very soon a characteristic sign appears, namely, that the tip of the tongue and its edges become red, and the coating becomes most marked on each side of the median fissure, which increases in depth from before backward. The tongue also becomes narrow instead of broad and flabby, as it is in biliousness, and is drier. If the attack be mild, this condition may remain until convalescence is established; but if the disease runs a severe course, the coating becomes very heavy, more dry, rough and brown from exposure to air and medicine. The furred appearance becomes almost shaggy at the back portion, and the drying proceeds until the underlying epithelial layer is cracked and fissured, so that tiny exudations of blood add to the lingual discoloration. The reddened edges become dusky in hue, and may be cracked and fissured also. The tongue is very slowly protruded on request, partly from mental apathy, partly from feebleness and because its surface is so stiffened that to move it is difficult. It is equally slowly withdrawn for similar reasons, and while protruded is often markedly tremulous. Toward the close of the attack the tongue cleans off through exfoliation of the dead epithelial accumulation, and this is a favorable or unfavorable sign according to whether the remaining surface is red and moist or dusky and dry. Sometimes these characteristic coatings do not appear, the tongue being brown and rough all through the disease. The heavily coated dry tongue just described is, however, rarely seen in those cases of intense fever which are treated by cold bathing. A small, triangular patch devoid of coating is often seen at the tip of the tongue in *relapsing fever*.

In *uremia* the tongue is often dry, brown, cracked, and furred. The patient, if conscious, complains of a foul taste and the breath may smell like stale urine.

In *biliousness* the tongue is coated almost uniformly by a whitish-yellow, pasty coat, extending from back to tip and side to side. The tongue is broad and flabby, and sometimes indented by the teeth, while the breath is foul and heavy. A similar tongue is seen in severe tonsillitis, except that it seems even more foul and less yellow in tint. Similarly in *jaundice* of the acute catarrhal type we have a coating still more yellow in some cases, because, as Fothergill asserts, the coat has been stained by the taurocholic acid eliminated by the salivary glands. The circumvallate papillæ are often prominent and stand above the coating, which is easily removed on scraping.

A broad, white, heavily-coated, moist tongue is often seen in *acute articular rheumatism*, becoming dry if the fever is high and the attack prolonged.

The white tongue of a person who takes large amounts of milk is generally not smooth and pasty, but rather rough in appearance. If the tongue be suffering from an attack of *thrush* (saccharomyces albicans), the white coating will consist of irregular white masses of the growth, which, if in great number, often coalesce and make a fairly even surface. The soreness of the mouth, the local heat, the salivation, and the age of the person—generally a young child—render the diagnosis easy.

A grayish diphtheritic looking coating of the tongue, occurring in adults, may be due to the growth of various forms of *mycoses*. Thus, a fine network of leptothrix in threads and tufts often spreads over the tongue, particularly in the region of the circumvallate papillæ. The growth may be quite dark in color, but it is separated from the exudate of diphtheria by microscopic study and the absence of systemic disturbance.

Sometimes on examining the tongue of a child we find that it is broad and flabby and covered by a gray coating, which is smooth and fairly moist. Scattered throughout this coating are patches in which the coating and epithelium have been shed, leaving red spots with sharply defined edges, which spots are said to be "worm-eaten" in their appearance—that is, to have the irregular outline of the marks on a worm-eaten leaf. In these areas are to be seen enlarged and reddened fungiform papillæ. Such a tongue is typical of what has been called, by Eustace Smith, "*mucous disease*," a condition in which there exists a more or less marked chronic catarrhal process in all the mucous membranes. If, on the other hand, there is a comparatively light coating, dotted irregularly by bright-red spots, which are not raised above the surface, but are very numerous, and the patient is a child, the diagnosis may be made of acute or *subacute gastric catarrh*.

The so-called "*strawberry tongue*" is one in which the organ is entirely denuded of coating and superficial epithelium, while the fungiform papillæ are swollen or enlarged and stand our prominently. This appearance of the tongue is seen commonly in *scarlet fever*, but is not, as has been thought, pathognomonic of that disease. The fungiform papillæ in the strawberry tongue of scarlet fever are, however, particularly prominent and erect.

When the tongue is excessively furred or rough in appearance, the coating is due to abnormally long and projecting papillæ covered by an excess of living and dead epithelial cells; it may denote grave disease of the viscera, but in rare instances possesses no diagnostic importance, unless coupled with other symptoms. This tongue is sometimes seen in scrofulous children in whom strumous manifestations are marked.

Should the tongue be denuded not only of coating, but, in addi-

tion, of its normal epithelium, so that it appears dry, hard, and harsh to the touch, it denotes, as a rule, grave and advanced disease of an exhausting nature, such as renal, hepatic, or gastric disorder about to cause the death of the patient. Sometimes this condition is seen in *advanced phthisis, diabetes,* or *gastric carcinoma,* and is of evil omen.

When the tongue is bereft of epithelium, beefy and red looking elongated and narrowed, and shows a peculiar roundness when protruded, severe disease of the abdominal organs, such as dysentery, or hepatic abscess, or carcinoma, will often be found or, in some cases, this condition develops to add to the discomfort of cases of advanced pulmonary tuberculosis or acute peritonitis. This tongue is sometimes called the "parrot tongue."

In this connection the point should be noted that *dryness of the tongue* in the presence of grave disease is always an evil omen, and returning moisture of the tongue a favorable one.

Unilateral coating of the tongue may be due to a decayed or ragged tooth, or to hemiplegia, which prevents that side of the tongue from being cleaned through movement. Hillow and Fairlie Clark both assert that morbid conditions of the second division of the trifacial nerve cause unilateral coating, and that abnormalities of the third division do not produce these changes as we would expect.

The coating of the tongue is often so stained by extraneous substances as to be entirely changed in appearance. If the coating be black, the color may be due to the ingestion of iron, of bismuth, charcoal, ink or blackberries, mulberries, cherries, or red wine. In very rare cases it is black, not from the growth of a fungus as has been thought, but from overgrowth of the epithelium with the deposit of a black pigment of unknown origin. Usually this brownish-black discoloration is confined to the middle of the tongue. The affected surface is often rough, due to the enlarged papillæ, and the edges of the spot are less black than the centre. In professional tea tasters the tongue may be orange-tinted.

The coating may be stained brown from the chewing of tobacco, from licorice, nuts, prunes, or chocolate, and yellow from the ingestion of laudanum or rhubarb.

The color of the tongue itself, aside from discoloration of its epithelium, is an important diagnostic aid. It is exceedingly pale in all forms of anemia, particularly those due to lack of hemoglobin, such as chlorosis or acute anemia from hemorrhage, and in pernicious anemia, when well advanced, it has a remarkable pallor. It is livid and cyanotic in cases of pulmonary disease interfering with oxidation of the blood, or in cardiac disease with similar difficulty and when very large doses of coal-tar drugs have been taken.

Purple spots, which may be almost black, may be present on the tongue in *Addison's disease*. Sometimes they are bluish black, and always well defined and even with the surface.

Very rarely the tongue is discolored by infarcts, blood stains, and bruises.

When the tongue has its edges dotted with yellowish patches of a slightly elevated character, the condition is xanthelasma, and the liver will often be found to be disordered.

In cases of poisoning by *corrosive sublimate* the tongue presents a most characteristic appearance, for it is white and shrivelled, and the papillæ at the base are unusually large.

When *sulphuric acid* has been swallowed the tongue has a parchment-like appearance, is at first white and then gray or brownish gray, and finally is covered by a black slough, which as it separates leaves a swollen, excoriated patch. In *nitric-acid* and *chromic-acid* poisoning the tongue is shrivelled and lemon yellow in color, as it is when hydrochloric acid has been swallowed. The tongue of *carbolic-acid* poisoning is very characteristic indeed, for the mucous membrane is shrivelled and puckered into folds. The spots where the acid has touched it are brownish if impure acid has been swallowed, or white if the pure acid has been taken. In the course of a few hours this spot becomes surrounded by a red zone, and finally becomes dark brown or black in the centre. After *oxalic acid* is taken the tongue may be covered by a thick white coat and looks as if it had been scalded. *Caustic potash* and soda soften the mucous membrane, so that it is pulpy and easily detached, and looks pearly, red, or yellow in hue. When *ammonia* is swallowed the color is white, but superficial edema may make it pearly in appearance, and acid nitrate of mercury renders it very red. Cantharidal poisoning produces large lingual blisters and sores.

Aside from the coating and color of the tongue, its surfaces should be examined to discover fissures, cracks, ulcers, sloughs, and swellings. The tongue is often seen to be superficially and irregularly *fissured* in old persons, particularly in those who have used large quantities of strong alcoholic drinks or strong tea, or who have chewed tobacco incessantly for many years. The fissures cross each other in every direction, although the central fissure which runs longitudinally is generally deepest and longest. If the furrows are very deep, they may indicate the early stages of what Wunderlich has called *dissecting glossitis*, which in turn may be due to syphilis,[1] although, as a rule, the fissures of the tongue due to syphilis are deepest at the edges of the organ, and are due to pressure by and from irritation from the teeth or to ulceration, and subsequent

[1] This is denied by Demarquay and doubted by Butlin.

cicatrization of small *syphilitic nodules* or gummata. The cervical glands are rarely involved in such cases. If only one ulcer is present it may be chancre, which will have the peculiar Hunterian hard base, and, in such a case, the cervical glands will probably be enlarged. An *epithelioma* may also have an indurated base with secondary glandular enlargement. Lingual ulcers may also be present as the mucous patches of syphilis, or be due to wounds from the teeth, a broken pipe-stem, or a fork. When these become chronic their separation from those due to syphilis and tuberculosis is practically impossible on superficial examination. Sometimes an ulcer of the tongue is due to epithelioma; but if this is the case, the patient will probably be past thirty years of age. As deep syphilitic ulcers heal sclerosis of the tongue may develop.

Multiple ulceration of the tongue may be due to tuberculous disease, which is very rarely primary, but rather secondary to its presence elsewhere. The sores are often stellate in shape, and there is always swelling of the cervical lymphatics, whereas in multiple syphilitic ulceration of the tongue the glands generally escape. The diagnosis between tuberculous ulcer and that due to epithelioma is more difficult, since in both diseases the cervical glands are involved. Both are more common in men than in women. The age of the patient, the presence of tuberculous disease elsewhere, and the absence of induration point to tubercle. The tuberculous ulcer is not surrounded by much inflammation, is covered by grayish purulent mucus, and may contain bacilli of tubercle, and is often associated with tuberculous nodules which have not broken down.

Ulcers of the tongue may also be due very rarely to *lupus*. A very similar tongue is seen in a tropical disease with intestinal disorder called by Thin "psilosis." An herpetic eruption appears on the tongue, which leaves large areas devoid of epithelium, while sinuous furrows or fissures develop. These fissures then heal, the patches become pallid, and recovery takes place.

The various ulcerated surfaces so far described might be confused with ulcerative stomatitis, but their chronic character and insensitiveness as compared to acute ulcers of the tongue, associated with a specific history or manifestations of tuberculosis or syphilis elsewhere, render the diagnosis clear.

An *ulcer on the frenum* may be due to whooping-cough, in which disease the edge of the lower incisors may injure the tongue in the paroxysm of cough, or, in adults, it may indicate the presence of a ragged tooth, which produces constant irritation, or, if the patient is advanced in years, represent the early stages of epithelioma, or that a broken pipe-stem has produced a wound.

Very rarely the tongue partakes of the ulceration of the tonsils and roof of the mouth which is seen in cases of Schönlein's disease,

accompanied by purpuric eruptions on the skin and evidences of septicemia.

Should the tongue be *marked by bites* from the teeth the patient may be an epileptic. Even if he denies that he is affected by the disease, the attacks may be unknown to him, because they are nocturnal. If the tongue is frequently bitten, the patient may be suffering from the early stages of glossolabiopharyngeal paralysis.[1]

The surface of the tongue may be attacked by various eruptions, such as measles, variola, eczema, herpes, erysipelas, pemphigus, zoster, or hydroa, and from the rupture of the vesicles or bullæ so formed ulcers may arise.

If the sore is herpetic, de Mussy asserts that the eruption will be found in the distribution of the lingual branch of the chorda tympani along the under border at the side.

Sometimes the surface of the tongue is here and there devoid of epithelium, and in some of these patches excoriated. Pain may or may not be present. The condition is called *chronic superficial glossitis* by Hack, and is considered by some to be the same disease described by Kaposi as glossodynia exfoliativa. It is more common in men and lasts many years.

Urticaria of the tongue has been reported by Laveran and xeroderma pigmentosum by Keating.

The presence of a plaque on the anterior portion of the dorsum of the tongue to one side of the median line, which is raised, not ulcerated, but red and irritated looking, may be due to excessive smoking, the smoke irritating the local epithelium. It is always very smooth, later covered by a yellowish-brown coat, and is sometimes called "smokers' patch." It may extend over the whole tongue and last for years.

When the tongue has on its dorsum and edges dull-white or slate-colored dots, patches, or lines, which are elevated, hard, and horny to the touch, but not painful, the condition is known as *leucokeratosis buccalis*, or leucoma or ichthyosis, and this may arise from smoking or glass blowing. It rarely begins in persons under twenty or in those over sixty years. It is often a strong predisposing agent toward cancer of the tongue. These spots are arranged on the tongue in longitudinal lines. Hyde asserts that they are due to excessive keratinization of the epithelium covered by an adherent and dense pellicle. The history is chronic, and ultimately by the stiffness of the spots the tongue may become cracked, and this in turn, perhaps, gives rise to carcinoma.

When the tongue is covered by smooth, dense plaques and disks

[1] It may be pointed out in passing that if there be fits, and biting of the tongue never occurs, and the patient is a female, the attacks are probably hysterical.

or rings, the condition may be *lichen planus*, but the diagnosis of lichen planus from leucokeratosis buccalis is difficult, if not impossible. The plaques are most commonly seen in males between twenty and forty years. Closely allied to this is the rare condition of hardening of the tongue due to *scleroderma*, as described by Kaposi.

A very rare condition of the tongue is one in which its surface is marked by rings or areas on the dorsum, which gradually enlarge until they reach the edge or coalesce. In appearance they are red and smooth, deprived of filiform papillæ, but not of the fungiform variety. Often the border of the circle is more red than the centre, and the very edge is often yellowish. This condition is sometimes called wandering rash, *geographical tongue*, or annulus migrans. Little if anything is known of its cause, save that delicate children are most often affected by it.

Feeble, sickly children sometimes develop upon the tongue, as well as on the lips and cheeks, a condition in which a tenacious exudation is thrown out, the mucous membrane becoming fissured and sore. Gaston and Sebestre have called this *stomatitis impetiginosa*.

Edema of the tongue, with the development upon it of vesicles, and, finally, sloughs, may occur. and is probably identical with the *foot-and-mouth disease* of domestic animals.

Bilateral atrophy of the tongue is due to disease affecting the hypoglossal nerves in some part of their course in or below the nuclei. (See Paralysis of the Tongue.) It occurs as a symptom of glossolabiopharyngeal paralysis, in which case the tongue is shrivelled and atrophied in patches, and in the later stages of the disease the organ has a crenated appearance. In other cases it is present in progressive muscular atrophy, and rarely in locomotor ataxia. It has also been seen in general paralysis of the insane. Unilateral atrophy may also occur from these causes, and Remak asserts that it sometimes arises from chronic lead poisoning. Any disease involving the hypoglossal nerves may so result. (See Paralysis of the Tongue.)

Smooth atrophy of the base of the tongue when developed in a person under fifty years of age is a sign of syphilis. Virchow pointed out this fact, and it has been confirmed by others.

In cases where the tongue is much enlarged the increase in size may be due to malignant growth, to *macroglossia*, which is a form of congenital lymphangioma, inflammatory hypertrophy, and syphilis, or acute inflammation from irritant poisons or foods. It may also be due to dermoid cysts, fibroma, lipoma, papilloma, angioma, myxoma, osteoma, and enchondroma. When it is due to acute glossitis the organ is seen to be several times its normal size, is pro-

truded from the mouth, and marked by the pressure of the teeth. The organ is also clumsy and stiff, and heavily coated on the back portion. There is a profuse flow of saliva, and swallowing and speech are almost impossible. Glossitis may also be due to mercurialism, to septic infection, and may be either unilateral or bilateral. The tongue may be greatly enlarged by actinomycosis. Great enlargement of the tongue may also arise in acromegaly and in myxedema. In the latter disease the organ is broad, flat, and soft.

The Movements of the Tongue.—The movements of the tongue depend upon its innervation and its muscles, and afford valuable information in diagnosis. The rapidity of its protrusion in nervous and excitable persons when they are asked to show the tongue is noteworthy, and its constant rolling is often seen in persons who are feeble minded. In all diseases associated with mental hebetude its protrusion on request is very slow, although the patient will often do this act when all other orders to move parts of the body fail to produce a response. In the various forms of coma due to apoplexy, diabetes, uremia, and cerebral congestion this condition obtains, and it is very characteristic of typhoid fever. Often the tongue which has been partially protruded is left so, even when the patient is told to draw it in. When the patient finds it difficult or impossible to remove food from between the teeth and cheek by means of his tongue, and complains that the power of speech is interfered with, because the tongue is clumsy in its movements, he may be suffering from the disease known as glossolabiopharyngeal paralysis or progressive bulbar paralysis. These lingual disorders are often the earliest signs of the disease. More rarely this disability of the tongue may arise from pseudobulbar paralysis, or what has been called glossolabiopharyngeal cerebral paralysis, a disease in which foci of softening occur in that portion of the cortico-muscular tract in which are the fibers which supply the muscles used in swallowing and speaking. This false type is separated from the true bulbar palsy by its sudden onset, an apoplectiform seizure, and other evidences of cortical disease. The tongue affords the most important points for differential diagnosis when a differential diagnosis is to be made under these circumstances, for in the false disease it does not waste or develop the reactions of degeneration, whereas in true bulbar paralysis these changes always speedily develop.

Paralysis of the Tongue.—In apoplexy the tongue is protruded toward the paralyzed side, as it is also in the condition, already described, of hemiatrophy. The lesions of the hypoglossus which produce paralysis may be of cortical origin (unilateral), in which case the hemorrhage or other injury may be situated where the middle and inferior frontal convolutions form the anterior central

convolution,[1] or in the supranuclear tract between the cortex and the medulla, or in the hypoglossal nucleus, or, again, in the infranuclear tract within the medulla. Insular sclerosis may very rapidly cause lingual paralysis. Paralysis of the tongue may also result from injury to the hypoglossal fibers outside the medulla through meningitis or syphilitic or other growths. In still other cases pressure upon the nerve in its foramen may cause unilateral paralysis, or wounds of the neck, caries of the first cervical vertebræ, or cervical tumors may so result. Often in such a case the spinal accessory nerve is also involved. Very rarely, indeed, the tongue may be paralyzed by a hypoglossal neuritis. (Erb.) In rare instances hemiatrophy of the tongue is associated with hemiatrophy of the face without hypoglossal injury. (Gowers.) Girard asserts that the sensory part of the trifacial contains trophic filaments for the tongue, and that the unilateral wasting may be due to disease of this nerve.

In paralysis of the facial nerve the tongue may be partially paralyzed through the fact that the lingualis muscle is supplied by means of the chorda tympani nerve.

When a tongue which is paralyzed unilaterally is retained in the mouth, it is seen that its root on the paralyzed side is higher than the other, owing to the paralysis of the posterior fibers of the hypoglossus; but when it is protruded the tongue goes toward the paralyzed side because it is pushed out by the fibers of the genioglossus muscle on the well side. Finally, let us remember that if the tongue is paralyzed on one side the lesion is in the cortex or the pons on the opposite side of the body, or in the nucleus in the medulla on the same side of the body, or in the nerve after it has left the medulla. If it is bilateral paralysis the lesion is probably nuclear, because the nuclei are so closely situated that even a small lesion involves both of them, or it may be due to symmetrical disease of both sides of the cortex, the so-called pseudobulbar paralysis already spoken of.

It should not be forgotten that paralysis of the tongue may occur as the result of diphtheria.

Hirt asserts that the reaction of degeneration may be found in the tongue whether the lesion be cortical or in the nucleus. If the lesion is only cerebral, this reaction will probably appear very late.

Tremor of the Tongue.—A tremor seen in the tongue may indicate a variety of nervous ailments or severe acute disease, as in typhoid and other severe infectious diseases, but the freedom from excessive coating and the absence of the ordinary signs of acute illness will

[1] This is probably a fact, but not yet confirmed by autopsy, unless we consider Edinger's case of softening under this area, which affected the tongue only, as a typical one.

separate the case of tongue tremor of acute disease from the tremor representing nervous ailments.

An important point to be regarded in noting lingual tremor is whether the tremor or fibrillary movement is constant, or whether it appears only when the tongue is moved to and fro or protruded. In typhoid fever the tremor occurs on movement, whereas in glossolabiopharyngeal paralysis when the mouth is open fibrillary movements of the organ are often marked, while the organ lies in the floor of the mouth powerless and beyond the control of the patient. Tremor of the tongue is also seen in a marked form in many cases of alcoholism, and associated with this tremor it will be noted that the protrusion of the organ is uncertain or in jerks.

Spasm of the Tongue.—Spasm of the tongue may be unilateral or bilateral, most commonly the latter. It is seen very commonly in cases of chorea, particularly of the posthemiplegic type, and in hysteria. In the first disease the movements are characteristically choreic. In the latter the spasm may be tonic or clonic or alternately tetanic and irregular.

Often the spasm in hysteria is unilateral. Sometimes it is clonic in puerperal melancholia. Spasm of the tongue is a common symptom in association with the twitching of the lips of general paralysis of the insane. Jerky movements of the tongue may also occur in insular sclerosis, but this is not the cause of the peculiar speech of that affection.

Very rarely the condition of lingual spasm is due to irritation of the hypoglossus by some cause as yet unknown. The tongue is darted in or out or thrown from side to side and often injured by the teeth. The spasms, as a rule, are not constant, but come on in attacks which closely resemble epilepsy, in that they are preceded by an aura. (Remak and Berger.) A very rare affection termed aphthongia (Fleury) is characterized by spasm of the tongue on attempting to speak. Romberg has recorded a case of lingual spasm due to irritation of the fifth nerve from lingual neuralgia.

In that very rare condition called "Thomsen's disease," "characterized by tonic spasms in the muscles during voluntary movements," the tongue may be involved, but in this case the other voluntary muscles will share in the affection

THE TONSILS, SOFT PALATE, TEETH, AND PHARYNX.

Having considered the diagnostic significance of changes in the appearance of the tongue in this chapter, and of the appearance of the lips in the chapter on the Face and Head, there is yet to be discussed the condition of the buccal mucous membrane, the tonsils,

the soft palate, the teeth, the upper part of the pharynx, and the postnasal spaces. As almost all the conditions found in the latter regions are of interest to the rhinologist rather than the general practitioner, only one or two affections of these parts will be included in this work.

The Teeth.—We can sometimes gain some information from the teeth as to the state of the patient. Normally the two lower central incisors are cut about the sixth to the eighth month, then the four upper incisors from the eighth to the tenth month, and the lower lateral and all the front molars from the twelfth to the fourteenth month. The canines are cut from the eighteenth to the twentieth month, and the posterior molars at two to two and one-half years. The first permanent teeth usually begin to come in about the sixth year. In children who are sufferers from rickets the teeth decay very early and rapidly, and if they be sufferers from inherited syphilis, the teeth are often cut in the early months of life.

Caries of the teeth to an undue extent is also seen in many pregnant women and in cases of diabetes mellitus.

If the permanent upper incisors are notched or peg-shaped with notches in the free edge, as if cut out with a small gouge, they are a fairly sure indication of syphilis of an hereditary character (Hutchinson teeth), and if in association with this deformity of the teeth we find middle-ear catarrh and keratitis, we have the "syphilitic triad," which is important as a sign of hereditary syphilis. These notches are not found in the so-called "milk teeth."

The staining of teeth by tobacco or other materials held in the mouth may reveal certain habits of the patient, and a blue line on the gums where they join the teeth is an indication of the presence of chronic lead poisoning. Loosening of the teeth, with bleeding, spongy gums should call to the physician's mind the possibility of scurvy or scorbutus, and the spongy gums are particularly indicative of this affection in bottle-fed babies. If loosening of the teeth occurs in adults, it may be due to mercurial salivation.

Grinding of the teeth in sleep in children usually indicates gastrointestinal irritation from indigestion or worms, and it is sometimes seen in the advanced stages of respiratory diseases, as from pneumonia or diphtheria associated with dyspnea. It takes place in adults in hysteria, maniacal attacks, and in epilepsy.

The Buccal Mucous Membrane.—Swelling and redness of the buccal mucous membrane occur in the various mild forms of stomatitis, and in the ulcerative type of this disease the more severe lesions are often found in this area. In the malignant ulcerative stomatitis called noma the slough which separates from the inside of the cheek leaves a large excavation which may become so deep as finally to perforate the cheek.

It is interesting to note that swelling of the cheek with great inflammation of the buccal mucous membrane is sometimes seen as the result of the formation of a salivary calculus in the duct of Steno, and it is also stated that obstruction from inflammation of this duct often occurs as a result of poisoning by sulphuric acid.

This writer has seen a case of that rare malady called Schönlein's disease, in which, in addition to the multiple arthritis, purpuric eruption, and great edema, the formation of a large ulcer or slough threatened to perforate the cheek, and in healing produced a cicatrix which interfered with the patient's ability to open the mouth. This patient was an adult.

The Tonsils.—If a patient presents himself to the physician with the statement that he is suffering from general pains all over the body, particularly in the small of the back, quite high fever it may be, with much sore throat and difficulty in swallowing, the trouble in the majority of cases will be, in the adult, tonsillitis of the follicular form. If the symptoms are exceedingly severe, the inflammation may result in suppuration—suppurative tonsillitis. It is to be remembered in all cases that the systemic or constitutional disturbance is out of all proportion to the severity of the local lesions. If it is tonsillitis, the glands can be felt in the majority of cases a little beneath and forward of the angle of the jaw, and pressure upon them may produce considerable pain. If the mouth is well opened and the tongue depressed, there will be found on each side of the throat a more or less projecting and inflamed mass, in the depressions or follicular openings of which will be found a white or yellowish exudate, which in severe cases may spread over the surface of the gland until it slightly resembles the membrane of diphtheria. Pressure on the tonsil may cause the further projection of these cheesy-looking masses.

In the suppurative form of the disease the surface of the gland may be smooth and reddened, and in a day or two become soft and fluctuating, and if lanced pus will escape.

The severe constitutional disturbance, the soreness of the throat, difficulty in swallowing, and the follicular exudate call to mind in all such cases the possibility of the disease being diphtheria; but in tonsillitis the exudate can be easily removed without leaving a bleeding surface behind it, and it has not the dusky, dirty look of diphtheritic membrane. Again, in tonsillitis the exudate is seen on the tonsils only, whereas in diphtheria it spreads over the half-arches and uvula. The general symptoms may make one suspect the onset of scarlet fever, particularly if the patient be a child; but the examination of the throat in scarlet fever shows the intense redness of pharyngeal mucous membrane with comparatively slight enlargement of the tonsils. The intense redness of the throat in

scarlatina and the development of the rash on the skin aid in making a differential diagnosis. The lymphatic glands of the neck may be enlarged in scarlet fever, but are rarely so in tonsillitis.

The Pharynx.—Difficulty in swallowing may arise from involvement of the pharyngeal muscles in diphtheritic paralysis, or from glossolabiopharyngeal paralysis. (See chapter on the Face, or Retro-esophageal Abscess.) Much more commonly it results from tonsillitis or pharyngitis. Not rarely it is due to a stricture of the esophagus, and sometimes to a morbid growth in the walls of this tube, or to the pressure of such a growth situated in the surrounding tissues. If the difficulty in swallowing is due to diphtheritic paralysis the history will be that there had recently been an attack of diphtheria. If due to a lesion of the bulb there will be the symptoms described in the chapter on the Face, as referred to above. The presence of an inflammation of the pharynx or the tonsils is easily discovered by observation of the back part of the mouth, as is also retro-esophageal abscess, which will generally be found associated with disease of the cervical vertebræ. If these states be excluded the diagnosis now lies between a stricture and a growth, and as the growth may be an aneurysm the patient's chest should be carefully examined and the other signs of aneurysm sought for, for should this be overlooked and an esophageal sound passed, the aneurysm, if present, may be ruptured. This examination may also disclose the existence of a mediastinal growth or enlargement of the retro-esophageal glands. If these causes be eliminated the actual search for stricture may be begun. First the physician should listen over the cardiac orifice of the stomach while the patient takes a swallow of water. If the act of swallowing is properly performed this single swallow of water will be heard to descend to the cardiac orifice, and then pause there for six seconds before it falls into the stomach. If there is a stricture this fall will be delayed; if there be atony of the cardia it will be hastened. An ordinary esophageal bougie may be passed. If a point of resistance is discovered that part of the bougie stem opposite the upper incisor teeth is marked, and then the instrument is withdrawn. In this manner we are enabled to tell the part of the esophagus affected. Usually pain will be felt where the bougie is arrested; but care must be exercised that spasmodic contraction of the esophagus is not mistaken for stricture. In the former state gentle and persistent pressure will overcome the obstruction. Usually the stricture exists at a point about six inches from the teeth; or where the left bronchus crosses the gullet, about eight or nine inches from the teeth; or at the cardiac orifice, which is seventeen inches from the teeth in the adult.

If a stricture exists it may be due to a cicatrix the result of an old burn, from the ingestion of alkalies or acids, or from an

ulcer due to another cause. In other cases the lesion is due to syphilis.

If the obstruction be due to cancer the passage of a bougie may do great damage, and, therefore, if any intimation of the existence of such a growth is present, great gentleness must be used. It should also be remembered that the bougie may be arrested by its passage into a diverticulum, or, in other cases, the instrument, by coiling on itself, may give a wrong impression as to the site of the obstruction. If a diverticulum is present the food which is obtained from it is usually alkaline, as it has never entered the stomach, and milk derived from a diverticulum, in which it has tarried a short time after attempted swallowing, will not be coagulated.

In doubtful cases it is advisable to have the patient swallow a large dose of bismuth in mucilage of acacia and then examine the esophagus with the Röntgen rays.

Finally, the physician should not forget, if his patient be a young woman, that there may be hysterical spasm of the esophagus.

If the patient complains of dysphagia, and, on examination, the pharynx is red and the tonsils are covered with patches which speedily spread, as just described, so that by forty-eight or seventy-two hours the tonsils, pillars, and soft palate are covered by a gray membrane, the case should always be diagnosticated as diphtheria and treated as such, unless a bacteriological examination of the exudate shows the infection to be due to a streptococcus and not to the Klebs-Loeffler bacillus. Even if the patient has not true diphtheria, he may be exceedingly ill. Again, it is to be remembered that while some cases of scarlet fever which in their early stages present a membranous pharyngitis or tonsillitis are due to the streptococcus and not to the Loeffler bacillus, in the later stages of the disease the Loeffler bacillus is the cause of the local lesion. The differentiation is to be made chiefly by bacteriological tests, but it is worthy of note that the early formed streptococcic membrane does not spread as does the diphtheritic membrane, and does not return so rapidly when removed. The two diseases, diphtheria and scarlet fever, often exist simultaneously. Rarely the formation of a false membrane due to streptococcus infection, or still more rarely to the diphtheria bacillus, complicates the course of typhoid fever, and also occurs as a grave complication of measles.

If in any case of diphtheria the false membrane extends to the nasal chambers, the prognosis is very unfavorable.

Ordinary sore throat or acute pharyngitis is generally accompanied with little systemic disturbance, the local pain and soreness being the most characteristic symptoms. Inspection will show the pharyngeal wall red and angry looking, and very likely unduly dry.

Care should always be taken, in the case of children particularly,

that the early sore throat of measles and scarlet fever is not taken for simple pharyngitis. Often the rash of measles can be seen on the pharyngeal wall some hours before the rash appears on the skin. A peculiar eruption also develops on the buccal mucous membrane. This eruption consists of small, irregular red spots with a bluish-white centre, and should be looked for in a good light. Koplik believes that these spots are absolutely characteristic, and clinical experience endorses his view. (See Plate V.) Their absence, however, does not exclude the presence of measles. They are to be distinguished from the reddened mucous membrane of scarlet fever, the large, white spots of thrush, and the sore mouth of stomatitis. They do not appear in rötheln.

Pigmentation of the buccal mucous membrane often occurs in Addison's disease.

Sometimes cases are seen in which there are tonsillar pain and irritation, and in which careful examination proves the discomfort to be due to the presence of a small calculus in a follicle of the tonsil.

When swelling of the tonsils is chronic the enlargement of these bodies may produce mouth breathing, with the peculiar facies of that habit (see illustration in chapter on the Face), deficient thoracic and general systemic development, and a peculiar cough, constant in character and worse at night. Often the swollen or enlarged glands extending across the pharynx actually touch one another.

PLATE V

Fig. 1.

Fig 2.

Fig. 3.

Fig. 4.

The Pathognomonic Sign of Measles (Koplik's Spots).

FIG. 1.—The discrete measles spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

FIG. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

FIG. 3.—The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthem on the skin is at this time generally fully developed.

FIG. 4.—Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in hue. Minute *yellow points* are surrounded by a red area. Always discrete.

CHAPTER VI.

THE SKIN.

The color of the skin—Eruptions on the skin—Gangrene, ulcers, and sloughs—Scars, sweating, dryness, edema, hardness—Anesthesia and hemianesthesia—Paresthesia, hyperesthesia, itching.

MUCH information can be obtained by careful examination of the skin in many cases of disease. The examiner should make a note of the color of the integument, of its general nutrition, of its pliability, and of its sensibility. Naturally the eye at once takes in any eruption or scars which may mar its naturally smooth surface, and, as eruptions and scars are often the manifestations of more or less active systemic disorders, an insight into the presence of internal disease may be obtained from them.

COLOR OF THE SKIN.

The color of the skin in health in the white race depends upon the presence of pigment in the cells of the mucous layer of the epidermis, and in the corium in those parts of the body where pigmentation is marked, or to the condition of the subcutaneous circulation or of the blood in the subcutaneous vessels. Thus we often find the skin of the perineum, scrotum, axillæ, and of the lower abdomen much darker than elsewhere in persons in perfect health. Similarly we see a marked reddish or yellowish-brown hue in those parts of the skin which have been exposed to sun and weather, as a result of a deposition of pigment and an increased capillary circulation. With these normal alterations in color, however, we have little to do, for it is the abnormal colorations which interest us from a diagnostic standpoint. The most common of these changes in color due to pigment is jaundice; the next the chloasma of pregnancy or uterine disease, a condition usually limited to the face. Abdominal growths due to tuberculosis, cancer or lymphoma, and tuberculosis of the peritoneum also cause pigmentation of the skin, and in melanotic cancer there is often very dark discoloration, so marked as to be confused with that of Addison's disease. Again, it is not uncommon for persons who have hepatic torpor with constipation to develop what are called liver spots, in which the skin has rather

a dirty hue. Under the name of vagabond's "pigmentation" we sometimes see discoloration induced by the irritation of the skin produced by lice and exposure to dirt and weather, and this is capable of being mistaken for the pigmentation of Addison's disease. Finally, we see the yellowish-brown hue of the skin due to tinea versicolor, the bronzing of the skin in Addison's disease, and the slate-blue hue of argyria or chronic silver poisoning. (See further on in this chapter.)

The changes in color depending upon disturbance of the subcutaneous circulation or on alterations in the blood are either local or general. In extreme nervousness flushing or blushing, due to a local vasomotor relaxation with increased blood supply, may redden the face and neck, or in hectic fever a hyperemia of the skin over the malar bones may give rise to an increase in color, which may be dusky red, due to imperfect oxidation of the blood. Considerable cyanosis of the face and hands in a case of tuberculosis of the lungs is a very grave symptom. Again, we see in pneumonia a peculiar dusky red flushing of one cheek or of the entire face, and in erysipelas the zone of hyperemic redness is characterized by its sharp line of demarcation and its raised edge. In the alterations in color due to changes in the quality of the blood we have, as causes, anemia due to lack of corpuscles or of hemoglobin, arising from the various etiological factors producing such states.

Jaundice.—Taking up the color changes due to pigment, we find that in jaundice the deposition of the biliary coloring matter varies in degree from a slight tinge or almost imperceptible yellowing to a dark citron or olive-green hue.

In examining the skin for jaundice care should be taken not to do so by gas- or candle-light, for the yellow flame masks the biliary color. If the tinge is very slight, it may be made more marked by stretching the skin on the palm of the hand or by pressing upon the skin a glass slide so that the yellow hue shows through it.

Having discovered that yellow coloring matter has been deposited in the rete mucosum, it remains for the physician to decide what the cause of the jaundice may be. In the first place, it must be remembered that jaundice may be hepatogenous—that is, arise from disorder in the liver, or be hematogenous, from disorders of the blood with the setting free of blood pigment. The hepatogenous jaundice is by far the more common of the two conditions, and the most common cause of this form of jaundice is catarrhal inflammation of the smaller bile ducts and the common bile duct which generally occurs in association with gastroduodenal catarrh.

Hepatogenous Jaundice.—As a result of a catarrhal process the bile duct becomes blocked by the swollen mucous membrane and the mucus which is secreted; the biliary coloring matter is absorbed into

the hepatic circulation and general circulation, and is by this means distributed over the body. Another common cause of hepatogenous jaundice is the obstruction offered to the flow of bile by the presence of a gallstone or gallstones in the ducts; and a third cause of obstructive jaundice, so called, is pressure on the ducts by growths or inflammatory products in the immediately adjacent organs, or of adherent inflammation in the ducts themselves, or by the presence of a roundworm in the duct. Very rarely the jaundice may arise from the pressure on the common duct produced by floating kidney.

Jaundice very rarely arises from pressure on the ducts by an aneurysm of the abdominal aorta, or from aneurysm involving the hepatic artery. Three such cases are recorded by Frerichs. Jaundice has also been seen in aneurysms of the superior mesenteric artery as the result of pressure and in cases in which there has been, or is, perihepatitis, with displacement of the liver in such a way that the adhesions cause twisting or dragging on the ducts.

Catarrhal jaundice of the acute type is generally produced by indiscretions in diet associated with exposure. The patient, after more or less marked symptoms of gastric and intestinal disturbance and indigestion, feels wretchedly. There is a premonitory mental heaviness, with languor and malaise, and within forty-eight hours or less the yellowing of the conjunctiva and skin appears. The temperature is generally subnormal to a slight degree. The tongue is heavily coated and often somewhat dry. There are marked loss of appetite, great distress, headache, and depression of spirits. Examination of the hypochondrium may reveal some local tenderness and slight hepatic enlargement, while the abdomen will be in some instances markedly tympanitic as a result of intestinal fermentative processes in the absence of antiseptic bile. The bowels are constipated, often refusing to move except with powerful purgatives. There is little pain, except headache. This condition lasts for a few days or a week, when the color of the skin and conjunctiva usually begins to fade and the normal hue is reached in the course of a week or more.

The presence of persistent jaundice should raise the suspicion that it is due to more serious disorder than simple catarrhal inflammation.

The jaundice from *obstruction by stone* may be due to blocking of the biliary duct, whereby there is a stagnation of the flow with reabsorption of the bile, or to stoppage of the flow by the presence of a stone in the common duct just as it enters the bowel. A differential diagnosis as to whether the stone is in one or the other of these places is usually impossible.

The jaundice of gallstone obstruction may be sudden or gradual in onset. If sudden, it is often, *but not always*, preceded by a violent attack of pain in the hypochondrium, or, in other words,

hepatic colic, in which the agony is excruciating and is accompanied by nausea and vomiting. The area of the pain is, however, distinctly hepatic, and it does not radiate down the inside of the thigh and into the testicle or penis as does that due to renal calculus. In place of the subnormal temperature so often seen in catarrhal jaundice, we find in obstructive jaundice that the temperature is often considerably raised, and this is particularly apt to be the case in those instances in which the onset is gradual and the jaundice persistent, being due to reflex irritation or septic absorption produced by the impacted stone, which may be scratching or ulcerating the lining membrane of the duct. The history of repeated attacks of gallstone colic, the presence of gallstones now and then in the stools, the discovery of gallstone crepitus on deep palpation, the age of the patient, who is generally in or past middle life, and the fact that the patient is a female, all point to gallstone as a cause of the jaundice. As a rule, there is great loss of flesh in all forms of jaundice. Should the jaundice be due to gallstones impacted in the ducts, and producing irritation or ulceration of their lining so that septic absorption or "Charcot's fever" develops, the pulse may become more rapid and running, from the general feebleness which rapidly asserts itself. Rigors of extreme severity, followed by sweatings and marked febrile movement, develop in such cases, the chills occurring daily or periodically in a manner closely resembling those of intermittent fever.

The jaundice of *malignant disease* pressing upon the gall ducts is usually not intense, and is characterized by the physical signs of a tumor, by the marked wasting of the patient, and, as a rule, by the very gradual onset of the pigmentation of the skin. Generally the lesion in such cases is carcinoma of the head of the pancreas. In jaundice resulting from cancer of the liver the growth must be so situated as to compress the ducts, consequently jaundice occurs in only about one-third of the cases.

In connection with this possibility the law of Courvoisier is to be borne in mind. This law has been well expressed by Mayo Robson in the following words: "Jaundice with distended gall-bladder is presumptive evidence in favor of malignant disease, but jaundice without distended gall-bladder favors the diagnosis of cholelithiasis." While this law or rule is a good guide, it must not be forgotten, as pointed out by Kehr, that gallstones produce malignant disease. Thus Courvoisier found gallstones in 87.5 per cent. of malignant cases, Delano Ames in 95.4 per cent. of gall-bladder cancer, and Schroeder has reached similar conclusions.

Jaundice is also seen in hepatic *hypertrophic cirrhosis* to a slight extent in a small proportion of cases, and it is to be remembered that in those cases of this disease in which delirium and muscular

twitching occur the symptoms may resemble acute yellow atrophy of the liver, and that all forms of jaundice produce headache and may cause delirium. In acute yellow atrophy of the liver (see below) the liver is greatly reduced in size, whereas in hypertrophy it is greatly increased in size; and in atrophy the temperature is subnormal, whereas in the jaundice due to hypertrophic cirrhosis it is apt to be above normal. Jaundice also may be a manifestation of acute *poisoning by phosphorus*, which condition is generally accompanied by hepatic swelling and tenderness and with coffee-ground vomiting.

Jaundice is present in all fatal cases of *yellow fever* and often in cases which ultimately recover. It also is a constant symptom in Weil's disease, which is probably in reality a septic icterus, but it is very rarely seen in suppurative hepatitis.

A fleeting and light hue of jaundice is sometimes seen in cases of chronic valvular cardiac disease in which compensation is gradually failing. Rarely this hue becomes deeper as the heart failure increases. This jaundice is due to engorgement of the liver (nutmeg liver), which in time results in catarrh of the bile ducts, with consequent obstruction to the flow of bile.

In *amyloid disease* of the liver Bartholow states that jaundice occurs in about one-tenth of the cases as a result of enlargement of the lymphatics in the hilus with pressure on the hepatic duct. Similarly jaundice may result from the presence of *echinococci*, but this is not a common symptom of the growth of these parasites, and the disease is very rare in the United States.

The association of gastric disorder with evidences of gastric dilatation and jaundice should recall the fact that *gastroptosis* sometimes causes jaundice.

Jaundice sometimes complicates diabetes. Under these circumstances it may be regarded as a coincidence or a valuable diagnostic aid, for, as we have already stated, tumors of the pancreas by pressing on the common duct may cause jaundice, and, as is now well known, widespread disease of the pancreas may cause diabetes. Jaundice in a case of diabetes should, therefore, direct attention to the pancreas.

In this connection it is well to remember that Hanot, under the name of *diabète bronzé*, has described a pigmentation of the skin which contains iron (that of Addison's disease and melanemia does not), and which is associated with diabetes, hypertrophic cirrhosis of the liver, and enlargement of the spleen. The coloration occurs most markedly upon the face, limbs, and genital organs; the glycosuria is abundant and slight ascites may be present, the lower limbs may be edematous, the loss of weight and strength is rapid, and death soon ensues from pneumonia or coma. Hanot and Marie both regard it as a disease distinct from ordinary diabetes mellitus. True diabète bronzé is exceedingly rare. Up to 1897 only 30

cases had been reported. In the 4 cases reported in America it is interesting to note that diabetes was not present. It is probably better to call the disease "hepatic cirrhosis with pigmentation."

Other noteworthy symptoms of hepatogenous jaundice are intense itching of the skin; a very slow pulse when the patient is at rest, due to stimulation of the vagus by the bile in the blood; and staining of the sweat due to the bile pigment may also be present.

When the gallstone produces active suppuration the fever becomes more like remittent fever and the patient rapidly emaciates and presents all the signs of active suppuration.

The urine in all cases of hepatogenous jaundice is heavily bile-stained (see Urine), and the stools are generally clay-colored owing to absence of bile in the feces.

A very rare cause of jaundice is *acute yellow atrophy of the liver*, a disease which is seen somewhat more frequently in women than in men, and particularly in association with pregnancy. The age of occurrence is usually between the twentieth and thirtieth years. The symptoms begin with gastro-intestinal disorder, followed by headache, delirium, muscular twitching, and perhaps convulsions. Simultaneously with the onset of the headache the jaundice appears, the patient becomes typhoidal and dies from exhaustion, although recovery has been known to occur. The stools during the attack are clay-colored, and the urine contains leucin in disks and tyrosin in needle-like crystals.

Hematogenous Jaundice is due, as its name implies, to breaking down of the blood to so great an extent that the liver cannot deal with the waste material with sufficient rapidity, and as a result altered hemoglobin is deposited in the tissues. Any poison which produces excessive *hemolysis*, such as picric acid and the coal-tar products, chlorate of potassium, glycerin, and poisonous mushrooms, may cause this condition to develop, and in extreme malarial disease (remittent and pernicious malarial fever), dengue, relapsing fever, pernicious anemia, pneumonia, and in other infectious maladies jaundice may be produced in this manner. It is particularly apt to occur in cases of marked sepsis.

Jaundice sometimes occurs after severe hemorrhage of a prolonged character and in *prolonged exhausting fevers*, and is then due not to any local hepatic trouble, but to blood changes, with the production of urobilin in excessive amounts. The urine fails to carry off all the urobilin which is produced from hematoidin or bilirubin. This condition is called "urobilin icterus."

In nearly all cases of hematogenous jaundice the discoloration of the skin is very slight, and the important fact is to be remembered that the stools are not light or clay-colored as in hepatic jaundice, but contain a normal or excessive amount of pigment. Again, the

systemic symptoms of catarrhal or obstructive hepatic jaundice are practically absent in the hematogenous variety, and the jaundice is simply a minor symptom associated with more grave manifestations which characterize the individual infectious process. If the poisoning is very marked, convulsions, coma, or active delirium may come on, but it is probable that these symptoms are due more to the poison of the disease than to the broken-down blood.

Vierordt states that a very small amount of biliary coloring matter is often found in the urine of patients suffering from pyemic jaundice, and regards this as an important sign that the discoloration of the skin is due in a given case to blood changes and not to biliary obstruction, whereas an excessive amount of biliary matter in the urine indicates hepatic trouble.

Jaundice sometimes complicates *croupous pneumonia*, and if hematogenous in origin is a most serious condition, for it usually indicates a fatal ending. When such an attack arises from catarrh of the gall ducts during an attack of pneumonia the condition is by no means so grave.

There remains to be considered the jaundice seen in *the newborn*, usually within the first or second day of life (icterus neonatorum), which some believe to be due to a decrease in the blood pressure in the portal vessels subsequent to the arrest of the placental circulation, with consequent absorption of bile into the blood, owing to the comparatively high tension of this fluid in the bile capillaries. Others think this jaundice is due to breaking down of the blood corpuscles shortly after birth as the result of some mild infection. Probably both causes act in some cases. If the cause be altered blood pressure the prognosis is favorable, and recovery takes place in about ten days or two weeks; and if the cause be an infection, the condition often proves rapidly fatal. Should this jaundice of the newborn be very marked the patient may be suffering from congenital stenosis, or absence of the common or hepatic duct (which cause is rare); from septicemia, through infection by way of the umbilicus; from phlebitis of the umbilical vein, or from a hepatitis due to hereditary syphilis. In any of these latter causes death will probably occur, whereas in the mild form of icterus neonatorum the prognosis is very favorable, even though the discoloration lasts for weeks. The mild form of icterus neonatorum, if due to blood changes, is rarely accompanied by great discoloration of the urine, and the feces are usually no lighter than normal in color; but if hepatic disease be present, the urine is bile-stained and the feces are light in hue.

Jaundice sometimes comes on in the course of *acute ulcerative endocarditis*, and has been mistaken for that of acute yellow atrophy of the liver, and it often appears as a symptom of pernicious malarial fever, with vomiting, diarrhea, and grave nervous symptoms.

Rarely jaundice follows *severe fright* or extreme anger, and Da Costa states that it sometimes ensues after concussion of the brain.

Other Changes in the Color of the Skin.—A condition of the skin characterized by yellow, more or less elevated patches, is *xanthoma*, which Murchison states often complicates hepatic disease, but in the writer's experience it is more commonly met with in gouty women or patients suffering from oxaluria. Its favorite distribution is about the eyelids, but it may appear elsewhere. Lesions similar to xanthoma sometimes appear in the course of diabetes (Hutchinson, Besnier), and under these circumstances generally develop suddenly, and spontaneously disappear after some weeks or months.

When the skin of the entire body, the face being particularly affected, is of a livid or bluish-slate color, resembling somewhat the appearance of a person exposed to rays of light passing through blue glass, the condition is that of argyria or *chronic silver poisoning*. This discoloration is so characteristic as to admit of no difficulty in diagnosis, since the absence of any circulatory or respiratory embarrassment excludes the possibility of its being due to cyanosis. Owing to the small amount of silver now given internally by physicians, chronic argyria is becoming rare. The discoloration is due to a deposit of oxide of silver in the rete Malpighii.

Discoloration of the skin of the entire body of a sallow, lemon-yellow tint, sometimes called a "muddy yellow" hue, is seen in persons who are sufferers from *prolonged malarial poisoning*, and in some cases the subjects of prolonged suppurative processes not tuberculous in character. A greasy, yellowish skin does, however, occur as an accompaniment of some cases of pulmonary phthisis, and these cases have, as a rule, a gloomy prognosis. Often chronic hepatic disease, such as cirrhosis, produces this sallow appearance.

A very rare but interesting condition of black discoloration of the skin, making it as dark as if gunpowder were rubbed into it, is met with in cases of *ochronosis*. Associated with this discoloration there is a similar darkening of the conjunctiva, which is particularly well marked at angles of the palpebral fissure and alkaptonuria, that curious state in which the urine reduces copper in Fehling's test. Autopsy reveals an intense darkening of the articular cartilages.

Other changes in the color of the skin, which cannot be said to be due to deposition of pigment, although they seem to be caused by this, are seen most markedly in the peculiar yellowish, cheesy pallor of carcinoma, the greenish-yellow tinge of true chlorosis, the curious cadaveric hue of advanced pyemia, and the yellow skin with a greasy feeling in some cases of paretic dementia.

Local pigmentation of the skin results from many causes, both local and systemic, direct and indirect. When brownish-yellow

spots or streaks appear on the face, so that chloasma is developed, we should look for uterine or hepatic disturbance or pregnancy; they are practically large freckles of a more or less distinct brown hue. Sometimes on the skin of the trunk small yellowish-brown or chamois-skin colored spots appear accompanied by no other symptoms except perhaps slight itching. This is due to *phthiriasis versicolor* or chromophytosis due to the growth of the parasite microsporon furfur. The diagnosis can be settled by painting the infected area with Lugol's iodine solution, when the spot becomes a dark-brown or mahogany color. This is called "Allen's test." In other instances chloasmic spots or localized discoloration of the skin results from injury to the skin, as pressure by clothes, chafing, or after constant severe scratching in the course of eczema or pediculosis or scabies. If the pigment is found in the nuchal and sacral regions, it is probably from the scratching caused by *pediculi;* if on the body in irregular distribution, it may have been caused by *prurigo.* Again, the presence of a brown pigmentation of the skin in clearly outlined patches may indicate the earlier use of a fly blister, a mustard plaster, or other counterirritants, and a brown discoloration of the skin, which might possibly be confused with that of Addison's disease, is produced by the free use externally of oil of cade. Sometimes these spots are produced by the prolonged use of arsenic, and the writer has reported a case in which the coalescence of the spots produced a curious grayish-brown hue of the entire body, so that the man looked somewhat like a mulatto.

Sometimes brown pigmentation of the skin of the neck and face appears as a symptom in *exophthalmic goitre,* and this disease may also produce similar lesions on the chest and wrists.

Very closely resembling these spots is the bronzing of the skin in patches which is seen in persons suffering from *Addison's disease;* but although bronzing of the skin is a somewhat constant symptom of Addison's disease, its presence is neither a positive nor negative sign in diagnosis, for bronzing is sometimes seen in cases in which the suprarenal capsules are normal. In some instances the bronze color deepens into a dark gray or even a black hue, and although the discoloration is generally in patches, it may extend over the entire surface of the skin, even to the edges of the finger nails. The nails, however, escape, as does also the mucous membrane of the lips, although the lining of the mouth itself may be dotted with pigmentation. The color is due to pigmentation of the rete Malpighii, and pressure has no effect on it. The symptoms of Addison's disease to be found associated with these skin changes are "anemia, general languor or debility, remarkable feebleness of the heart's action, and irritability of the stomach." (Addison.)

The slate-colored skin of argyria or chronic silver poisoning can

be readily distinguished from the bronze color of Addison's disease; but if a further test is needed, it will be found that washing the skin of argyria with a solution of iodine changes its color, while that of Addison's disease remains unaltered.

White patches, or *leucoderma*, are also sometimes seen in cases of true goitre, and brown ones in tuberculosis.

In carcinoma of one of the internal organs, or of the breast of an advanced stage, the appearance of the skin is drawn and unusually smooth, often shiny or greasy looking, somewhat gummy and leathery to the touch, particularly where the integument is naturally dense. Although it is difficult to describe, this skin is almost pathognomonic of carcinoma, although it may also be present to some extent in far-advanced cases of pernicious anemia or sarcoma.

Pallor of the skin is due to absence of the normal pigment, to deficient blood, to central or local vasomotor disturbance as is typified by fainting, and far more rarely by Raynaud's disease. As a type of the pallor due to lack of pigment in the skin we see *vitiligo*, while the pallor due to pernicious anemia or pseudoleukemia and malaria is owing to lack of red corpuscles. Similarly, a pallor due to lack of hemoglobin is typified by chlorosis. (See chapter on the Blood.) In all of these diseases the skin may be of ghastly whiteness or tinged with yellow. The skin is apt also to be very white, and even chalky in appearance, in chronic contracted kidney and chronic parenchymatous nephritis.

In *chlorosis* the entire surface of the body is exceedingly pale, and the skin of the face, particularly about the mouth and nose and eyes, is somewhat greenish in hue. The skin of the cheeks may, however, be flushed and the lips abnormally red in hue.

A very important diagnostic point to be remembered is that red cheeks often cause the physician to overlook well-advanced anemia in young women. (See chapter on the Blood.)

In those cases in which the skin is pale from alteration of the subcutaneous circulation there is usually incompetence of the heart or vasomotor disturbance, but the most marked form of general pallor is that due to *myxedema*.

Cyanosis, or blueness of the skin, depends upon the circulation in the subcutaneous vessels of imperfectly oxidized blood. The small veins are often seen to be swollen, particularly those of the face and the hands and feet. The most marked form of cyanosis with which we meet is the cyanosis of the newborn child, suffering from a *patulous foramen ovale*, and in this condition the color may vary from a slate hue to an almost black hue. The lobes of the ears, the tongue, the scrotum, and the toes show the color most deeply. It is important to remember that this form of cyanosis is greatly decreased, as a rule, by placing the child on its right side. Anything which pro-

duces excitement increases the cyanosis greatly, whereas cyanosis due to other causes is not subject to great variations. In the cyanosis of the newly born, males are far more frequently affected than females, in the proportion of about 2 to 1 or 3 to 1, and it is a noteworthy fact that even when the cyanosis is due to a malformation of the heart it may not be present from the time of birth, but may develop several days afterward. J. Lewis Smith records 41 cases in which the cyanosis due to congenital heart lesion came on at periods ranging from two weeks to forty years after birth.

About 35 per cent. of the cases of cyanosis due to congenital defects die in the first year.

In cyanosis of the newborn the chances are about 10 to 1 that the lesion is absence of a properly developed interauricular or interventricular wall.

In the adult or child cyanosis may be produced by serious *cardiac disease,* by pulmonary disease, such as pneumonia, pulmonary congestion, and bronchiectasis with emphysema and associated cardiac dilatation. It also occurs in *laryngeal obstruction* arising from external pressure or intralaryngeal difficulty, and in cases of asthma of a severe form. (See chapter on the Thorax and its Viscera.)

In some cases of *paretic dementia* the skin of the forehead is dull and dusky-looking. In other instances a grayish-blue or cyanotic appearance may arise from the ingestion of drugs which reduce the hemoglobin of the blood, such as antipyrine or acetanilid, and in such instances the discoloration is first seen about the base of the thumb nail or in the skin of the face, particularly if the patient be examined from a little distance.

The condition of the skin, so far as its nutrition is concerned, is of great importance in diagnosis. In profound failure of the vital forces continuing over a great length of time it becomes abnormally dry and scaly, the hair becomes straggling and lustreless, and frequently falls. In young persons suffering from grave disease of the lungs or heart of a chronic type there is often not only an undue dryness of the cuticle, but an abnormal growth of downy hair all over the body and limbs, and more particularly down the spine and over the breast bone.

ERUPTIONS ON THE SKIN.

The influence of age upon the development of skin lesions is very great, and Stephen Mackenzie has summed up the relationship of skin diseases to age in the following amusing manner: "The seven stages of man could be well illustrated by disease of the skin, though we lack a Shakespeare to do justice to the theme. In

the 'mewling and puking' infant we meet with sclerema and edema neonatorum, the 'red gum' or strophulus of the older writers, intertrigo, eczema, urticaria papillosa (lichen urticatus), urticaria pigmentosa, xeroderma pigmentosum, and impetigo; the 'school boy,' with his chilblains and ring-worms, alopecia areata, pityriasis rosea, ecthyma, and 'foot-ball disease;' and then the 'lover,' with his acne and sycosis, and, as a result of irregular sexual excursions, his syphilides; 'and then the justice, in fair round belly,' with acne rosacea, diabetic boils, and pruritus ani; the sixth stage shifts into the 'lean and slippered pantaloon,' with rodent ulcer and 'gouty' eczema; 'last scene of all, sans teeth, sans eyes, sans taste, sans everything'—except an incessant and intolerable itching of the skin which we call senile prurigo."

There are two conditions of the skin in which valuable evidence is given that the patient is suffering from *rheumatism.* One is the presence of *erythema* in one of its many forms, the other is the appearance of *purpura,* or, as it has been called, peliosis rheumatica. That the presence of erythema is often associated with lithemic or true rheumatic infection is proved beyond all doubt, either erythema papulatum, annulaire, marginatum, or nodosum being indicative of the systemic state, but it is worthy of note that the erythema marginatum is most diagnostic and erythema nodosum the least diagnostic of rheumatic poisoning. Sometimes this eruption may be the only manifestation of the disease other than cardiac involvement, and when it is marginate severe cardiac lesions are commonly present. The papulate eruption is most commonly found on the back of the wrists, the hands, and the feet when it occurs as a rheumatic sign, while the nodose variety is generally confined to the front aspect of the legs or the extensor surfaces of the arms. It is to be remembered that these forms of erythema may be distributed anywhere over the body in rheumatism, but that they become especially diagnostic if limited to the areas named.

Purpuric Discolorations of the skin, somewhat resembling minute multiple bruises in appearance, are due to a number of causes and possess a varied significance. In the first place, they are due to the condition known as purpura hæmorrhagica, which may be divided into the acute and subacute forms and that which is secondary as the result of severe infections and certain poisonings. The *acute form* of purpura is probably in all cases a manifestation of an infection by a pathogenic organism runs a rapid course and reaches a fatal result in most cases in a short time. It is a comparatively rare disease and usually attacks young adults, chiefly males, up to twenty-eight years of age. It is sometimes seen in young girls and more rarely in young pregnant women. The chief symptoms consist in hemorrhages from the mucous membrane, purpuric spots

high fever, and a general class of symptoms resembling those of sepsis, as chills, pyrexia, and exhaustion. In other instances active hemorrhages take place into the viscera, and if into the meninges of the brain cause cerebral symptoms at once. The liver and spleen are nearly always enlarged.

The *subacute type* of purpura, while severe, runs a far more favorable course as to its manifestations and results. It usually attacks children or young adult males from twenty to thirty years of age. The patient, after a feeling of wretchedness, and perhaps a chill, followed by the purpuric eruption, is attacked by swelling of the joints and perhaps hemorrhages from the kidneys, bowels, and mucous membranes. If the hemorrhage is from the gums, the teeth are not loosened, as in scurvy. Prostration may be great and the patient may appear as if suffering from typhoid fever. The prognosis is good for ultimate recovery. It is sometimes called peliosis rheumatica or *Schönlein's disease*. This subacute form, however, sometimes occurs in a more severe manner, as "Henoch's disease," in children between nine and twelve years, and is much more common in males than females (5 to 1). In this form we have as additional symptoms marked pain and tenderness in the belly, and bloody stools, with tenesmus and active vomiting. The illness may last a long time, but recovery often occurs, about 25 per cent. dying.

The joint symptoms of the mild forms of purpura may be slight or absent. Often, too, the purpura is accompanied or replaced by erythema.

The question as to whether purpuric eruptions are ever truly indicative of rheumatism has been much discussed. When purpura develops in the course of true articular rheumatism it is often an indication of an associated infection. In many cases, however, the arthritic inflammation is not a true rheumatism but in reality a septic arthritis which is due to the same cause as the purpura. In either case the eruption appears as a rule in the neighborhood of the involved joints, nearly always on the lower limbs, often breaking out before any evidence of articular trouble exists. In other instances the development of the purpura is simultaneous with the disappearance of joint trouble. The eruption usually fades in a few days, but frequent relapses or new crops of it often occur.

Purpuric eruptions may be produced by quinine in persons who have an idiosyncrasy to this drug, and by iodide of potassium, chloral, and salicylic acid. They may also accompany any severe infectious disease and follow the entrance into the body of any poison which destroys the blood, such as the poison of snake bite. They also result from severe jaundice, from profound anemia, from congenital syphilis with vascular changes, in ulcerative endocarditis (a

form of sepsis), and in cases of multiple sarcomata. Rarely purpura has followed fright and severe grief.

Hemorrhages into the Skin occur spontaneously in some cases of hysteria and paretic dementia, and after epileptic attacks, particularly about the eyes, and often from injuries received in other parts of the body during the convulsion. Minute hemorrhages may also occur in the course of severe whooping-cough, and, in the form of petechiæ, result from snake poisoning, septicemia, cerebrospinal meningitis, iodism, ergotism, and after inhaling the vapor of benzine. They are also seen in scurvy and in some cases of profound wasting, as in the course of phthisis and carcinoma.

Petechial rashes closely resembling those of malignant smallpox, typhoid fever, or cerebrospinal fever may be due to the presence of acute ulcerative endocarditis.

Hemorrhages sometimes appear in the skin covering a part which has been affected by a severe pain in a crisis of locomotor ataxia.

A very extraordinary manifestation of spontaneous subcutaneous hemorrhage is seen in what is known as *hematoma auris*, a condition in which a free extravasation of blood takes place into and beneath the skin of the ear. The color of the swollen ear is quite red, if the hemorrhage has been recent, or dark blue if it is an old occurrence. The left ear is more commonly affected than the right, and it is seen more commonly in males than in females.

The development of polymorphic skin lesions, consisting of hyperemia, edema, and hemorrhage, with arthritis occasionally and visceral disturbances, consisting in attacks of vomiting or diarrhea, endocarditis, pericarditis, acute nephritis, and hemorrhages from the mucous membranes, indicates the presence of a condition called *erythema exudativum multiforme*. The attacks are apt to be recurrent. Sometimes the skin manifestations are absent.

Subcutaneous Fibroid Nodules sometimes occur in cases of rheumatism and vary in size from a hemp-seed to a walnut. They are usually situated in the subcutaneous connective tissue, but may be attached to the deep fascia or muscular sheaths.

Intense redness of the skin is seen in acute inflammations of the skin or the subcutaneous tissues, and as the result of hot applications, the redness being marked in proportion to the degree of heat and the time it is applied. Often the prolonged use of high heat will produce a peculiar mottling of the skin like that of an old bruise.

Aside from the redness of the cheeks and forehead from blushing, we should remember the general flushing seen so commonly in persons suffering from phthisis, particularly when they are excited, which differs from the more dusky redness seen over the malar bones in hectic fever.

Urticaria may occur as a manifestation of rheumatism, but it has no diagnostic value. Sometimes it ensues upon the use of salicylic acid or turpentine, and quite commonly follows the ingestion of iodide of potassium. The wheals produced by the latter drug differ from those of urticaria in being unduly red.

Very closely allied in its causes and appearance with urticaria of the severe type is *angioneurotic edema.* In this condition there appear upon the skin numerous patches or plaques of circumscribed puffy swellings, which have a red appearance and vary from the size of a nickel to a silver dollar or larger. There is an absence of itching, an important difference from true urticaria, but the part affected may be tense or hot to the patient. These attacks are generally recurrent, and take place in neurotic persons. They may cause loss of sight through swelling of the eyelids, and, where the mucous membranes of the pharynx and larynx are involved, serious interference with breathing. The swelling of angioneurotic edema does not pit, and it is to be separated from the blue edema and white edema of hysteria. True angioneurotic edema is rare in hysteria, and if localized swellings do result from this condition the physician will generally find marked hysterical signs manifested, such as disorders of sensation or tenderness over the ovaries.

Myxedema.—When the skin is pale and affected by an edematoid swelling, with loss of elasticity, particularly about the face, and also in the trunk and extremities, and if this swelling, which resembles edema, fails to pit on pressure, the physician should remember that *myxedema* or the cretinoid edema of Gull may be present. If in addition to these signs there is a half-idiotic or heavy expression of the face, a slow and labored manner of speech, with thickened, clumsy fingers, the diagnosis is made practically certain. The brain in this disease perceives or grasps ideas very slowly, and all the functions of the body seem torpid.

There are several other diseases in which great thickening of the skin takes place, which cannot, however, be confounded with myxedema. In *elephantiasis* there is an hypertrophy of skin and subcutaneous tissues which is confined to some particular region of the body and arises from local circulatory disturbance in the blood and lymph vessels. The skin is very hard, so that the leg, if affected, feels like a solid mass of wood. The disease most commonly affects one of the legs, rarely both, and the scrotum. In both myxedema and elephantiasis the process develops very slowly.

Scleroderma.—When the skin is dotted with irregular patches or streaks, which may be elevated or tightly stretched, or if the entire skin is thickened, covered with thin scales, or possesses a plaster-like appearance, the physician should recognize these symptoms as indicative of *scleroderma.* If in addition to these signs there is

a fleeting pitting of the skin on pressure, and it cannot be pinched into a fold, the diagnosis is confirmed. Sometimes the skin is sclerodermatous, seems bound down by tense cords or bands of retracted connective tissue to the tissues beneath, and in rare instances even the tendons, muscles, faciæ, and joints may be involved. Generally the impaired movements of the joint depend upon the stiffening of the skin, but in some cases the disease results in atrophy of these deeper parts.

If during the first months of life the skin of an infant becomes edematous, hard, tense, and glossy, varying in color from a white to a reddish or dirty yellowish brown, and if this rapidly involves the entire surface so that the integument becomes cool, immovable, and resistant, the child appearing as if frozen into stiffness, it is probably suffering from *sclerema neonatorum*, a disease entirely different from the scleroderma of the adult. As a rule, death speedily ensues, but before this takes place the parts first affected become thin and lose their swelling and may develop cyanosis and gangrene.

The affection just described is to be separated from *edema neonatorum*, a condition arising in prematurely born children. Within a few days after birth there is discovered a pallid, cold condition of the buttocks, thighs, legs, and arms. The parts speedily become edematous and livid blue. Finally, the edema may become very marked and the skin tense in consequence. Intense drowsiness is a characteristic of the disease. Death commonly ensues, but recovery may occur. While the color of the skin may be identical in edema neonatorum and sclerema neonatorum, the former affection lacks the stiffness of the jaw and other joints, and the pitting on pressure is marked. As scleroderma does not occur before the first year, it can be excluded from the diagnosis.

Another interesting diagnostic sign in the skin is what is known as the "*tache cerebrale*," a condition of vasomotor disorder in which when the finger is gently drawn over the skin of the forehead a red patch speedily develops. It is seen in meningeal irritation, brain abscess, epilepsy, in some cases of exophthalmic goitre, and in paretic dementia. Sometimes it is called "*tache meningeale*."

Erythema or Rose Rash, sometimes called roseola, is a redness of the skin, and occurs in many pathological conditions. It may be localized or diffused. In a number of diseases it aids very greatly in reaching a diagnosis, but the physician should always be cautious in depending much upon it, since it may mislead, owing to the fact that it often appears when devoid of diagnostic importance in so far as the eruptive fevers are concerned. (See page 147.)

The Rash of the Acute Infectious Diseases.—The development of a diffuse, punctated rose rash on the skin of a person who is suffering from malaise, fever, nervous disturbance, and sore throat should

direct the physician's attention to the possible presence of two infectious diseases, namely, scarlet fever, which is more common in childhood, and syphilis, which is more frequent in adults.

Scarlet Fever.—The rash of *scarlet fever* is usually of a bright-red color, and shows itself at the end of the first or on the second day of the disease, first appears on the chest and neck, and then speedily involves nearly the whole surface of the body, although the forehead often escapes and the skin about the corners of the mouth remains nearly always white and free from eruption. On the other hand, the soles of the feet and palms of the hands are very markedly affected. So intensely reddened is the patient's surface that it may have the color of a boiled lobster. This redness depends upon an acute hyperemia of the skin, which though removed by pressure instantly returns when the finger is withdrawn. A noteworthy point is its punctate and mottled appearance, for, while the entire skin may be red, there are points which are more red than the rest of the skin, and also certain areas which are particularly so. The skin is often slightly swollen and feels tense, and itching is commonly present. The rash usually lasts three or four days, and then fades, desquamation of the cuticle speedily setting in, which is complete in about two weeks. Sometimes, however, it remains for ten days to three weeks. Often when the rash can scarcely be seen on the skin its full development will be found on the pharyngeal wall. In the malignant types of scarlet fever petechiæ and subdermal hemorrhages occur. Sometimes in scarlet fever fine miliary vesicles develop chiefly in the neighborhood of the axillæ or on the abdominal wall or thorax anteriorly.

Sometimes, too, in those cases of scarlet fever which have severe symptoms of ulcerating sore throat with ear or nose complications there develops, about the third week of the disease, a dark-red papular or macular erythema on the extensor aspects of the large joints. It is a grave symptom.

Rotheln.—The rash of *rubella or rubeola or rötheln* (German measles) more closely resembles that of scarlet fever in some cases than it does that of measles, but it is never as scarlet, is distinctly maculated, and only at a distance looks homogeneous. Like measles, it is first seen on the face, chiefly about the nose and on the upper lip. Close examination always reveals the rash in oval patches or crescents, and it lacks the diffused character of the rash, the punctation of the skin, the grave systemic disturbance, and the throat symptoms of scarlet fever. Further, the febrile movement is comparatively slight, and the rash lasts only twelve hours or at the most for two or three days. Slight desquamation may, however, occur.

Rötheln, or German measles, is separated from true measles in

many cases by the marked glandular enlargements, chiefly the posterior cervical, axillary, and inguinal; but this is also constantly true of scarlet fever and sometimes of measles. It never presents "Koplik's spots" on the buccal mucous membrane. (See chapter on the Tongue.)

Measles.—The eruption of *measles* is very characteristic, and can be in most cases easily separated from the other exanthemata by close examination. It is a roseola in character, but more dusky than that of scarlet fever. It appears about the fourth day of the illness in association with catarrh of the mucous membrane of the eyes and respiratory tract. Unlike scarlet fever it appears in macules first upon the forehead or face, then on the neck, trunk, and limbs. The macules, which often coalesce, are arranged in crescents which are red, but become somewhat yellowish on pressure. They are slightly raised. There is nearly always to be seen some uninvolved skin, the entire surface not being covered as in scarlet fever. In some instances in which the eruption is aberrant a diagnosis of measles from scarlet fever is admittedly impossible until the case has been watched for some days; but the slow onset of measles, in which the eruption appears on the fourth day as against the first day in scarlet fever, the swollen eyes and nose, the puffiness of the face, the catarrhal condition of the mucous membranes, the curious fall of temperature after the preliminary rise on the first day, the short duration of the rash, all aid in the diagnosis of measles. The dusky eruption of measles can nearly always be found on the pharyngeal mucous membrane. (For the mouth and throat symptoms of scarlet fever and measles, see chapter on the Mouth and Tongue.)

An erythema or roseola sometimes appears on the skin of children *after vaccination,* generally about eight to ten days after the operation. It rarely lasts more than two days, and on its disappearance there is slight desquamation.

It also appears sometimes in cases of *smallpox* previous to the outbreak of the true eruption. Under the latter circumstance it is found most commonly about the groin and inner surface of the thighs and on the hypogastrium, loins, clavicles, and the extensor surfaces. So closely may the early rash of smallpox simulate the aberrant type of measles as to lead to grave mistakes in diagnosis. Sometimes an immediate diagnosis is impossible, even by the most experienced, but the rash of measles commonly appears on the face, therefore this difference, coupled with a history of exposure, the gradual development of the peculiar "shot under the skin" sensation of variola, and the ultimate distinct papulation, vesiculation, and pustulation of smallpox soon remove the doubt from the physician's mind.

DAY OF ERUPTION OF THE VARIOUS EXANTHEMATA.

Day.	Disease.	Area.
First to second day . .	Rötheln or German measles.	Face first.
	Varicella or chickenpox.	Face or trunk.
Second day	Scarlet fever.	Neck and chest
Third to fourth day	Measles or morbilli or rubcola.	Face.
	Variola or smallpox.	Forehead, face, and wrists.
Fourth to fifth day.	Typhus or ship fever.	Trunk.
Seventh to ninth day. . . .	Typhoid or enteric fever.	Abdomen.

The remembrance that the incubation period of variola is twelve days, that of varicella seventeen days, of measles ten days, of rubella twenty-one days and of scarlet fever two to four days, will aid the diagnosis if a history of exposure can be obtained.

A diagnosis between the eruption of measles and variola often can be made by stretching the skin between the fingers, when, if it be measles, the papule cannot be felt, whereas, if it be variola, it persists. This is called the "grisolle sign."

An erythema resembling scarlet fever, not only in its appearance, but also by its association with swelling of the lymphatic glands and reddening of the mucous membranes of the mouth, sometimes develops about the second or third day in cases of *dengue* or break-bone fever.

In children there are several other conditions than scarlet fever which are associated with rose rash, and these are prone to lead to grave errors of diagnosis not only because they may be mistaken for scarlet fever but chiefly because a mild attack of scarlet fever may be considered as of their nature. The most frequent of these is *erythema roseola*, or roseola of acute indigestion, or that following the use of a food to which the patient has an idiosyncrasy. It is generally, but not always, widely diffused and is often associated with acute and severe febrile movement and vomiting, but it can be separated from scarlet fever by the facts that there is an absence of severe constitutional and nervous symptoms (except in neurotic children), there is no sore throat or enlarged cervical glands, and the rash does not come out on the clavicles and gradually travel down the body. Roseolous eruptions also appear in persons with delicate skins after coming in contact with irritant plants, and Dukes asserts that they may develop from handling caterpillars.

The severe cases of scarlatina are easy of diagnosis. It is those in which the rash and sore throat are mild that are difficult of determination. In these cases the physician must delay his diagnosis until the subsequent course of the malady enables him to marshal

before his mind's eye most of the characteristic manifestation of true scarlet fever.

Another condition closely resembling scarlet fever is rarely seen, namely, acute exfoliating dermatitis, called, in its mild form, *erythema scarlatiniform*, which has a sudden onset with febrile movement and a rash which rapidly spreads over the entire body and lasts four or five days, finally ending in desquamation So closely may this disease resemble scarlet fever that a diagnosis during the first attack may be impossible for the first few days, but the condition of the throat and tongue does not resemble the condition seen in scarlatina. Desquamation is often even more complete than in scarlatina, and the hair and nails are frequently shed. Relapses are very common and give rise to the reported cases of repeated attacks of scarlet fever.

Among other diseases in which rose rash appears we find diphtheria, septicemia, cholera, typhoid fever, malarial poisoning, and Bright's disease. In *diphtheria* it may lead the physician to a diagnosis of scarlet fever with severe faucial manifestations, and only a careful examination of the throat, the rapid subsidence of the rash, and the bacteriological examination of the false membrane will settle the diagnosis. Sometimes, however, a roseola appears late in the course of diphtheria, probably as a result of septic absorption. In other instances the injection of antidiphtheritic serum or other antitoxins produces this result. The presence of a very high temperature, of nervous irritability, and the predominance of the throat lesions of scarlet fever ought to decide the diagnosis in favor of scarlet fever.

The physician should also recall the fact that the injection of *antidiphtheritic serum* sometimes causes a roseolous eruption, associated, it may be, with pains in the joints. The general illness caused by diphtheria, plus these symptoms, may point to a complicating scarlet fever or measles. The antitoxin rash is not, as a rule, so persistent as that of scarlet fever, lasts a short time, and is rarely followed by desquamation, except in fine scales. While it may resemble measles in its characteristics, the patient does not present the eruption on the pharyngeal mucous membrane nor the peculiar coryza of that disease, nor the bronchitis or other evidences of respiratory catarrh.

The roseola of early *syphilis* resembles that of scarlet fever in that it first appears on the trunk; but it is not bright scarlet, but rather dusky red. It appears in patches and is not diffuse, and it ensues about six weeks or three months after the appearance of the initial lesion, occurs in an adult, as a rule, is not associated with high fever, and soon involves the face and forehead. These symptoms aid us in separating it from scarlet fever, although the rash

often appears in full blast in the palms of the hands and soles of the feet; but a roseolous rash in these areas in an adult is always suspicious of specific trouble. These patches speedily change from rose rash to other more marked lesions in cases of syphilis, and one of the first changes that they undergo is to become circinate. They fade and reappear, last an indefinite time, fade in the centre, and so change into marginate or circinate erythema.

When roseola develops after a surgical operation or after delivery in a puerperal female, it is not a manifestation of scarlet fever, but is due to sepsis ("surgical scarlet fever"), although it is, of course, possible for scarlet fever to attack such cases at any time. The rash is usually found over the abdomen and inner sides of the thighs. The absence of sore throat, the presence of a septic process, and the absence of a strawberry tongue all help to exclude scarlatina. Sometimes late in an attack of cholera a rash like surgical roseola appears in the same areas, or in the period of reaction comes out on the forearms, backs of the hands, and rarely on the back.

The *roseolous rash of typhoid* is sometimes widely distributed and almost like measles in appearance; but, as a rule, it is limited to a few or many rose spots on the abdomen, chest, or back. These rose spots disappear on light pressure, but immediately return when the pressure is removed, and are most marked in typhoid fever about the seventh to the tenth day of the disease. They may become slightly papular. In this connection it should not be forgotten that the rose rash of typhoid fever may be so profuse, particularly in persons with a delicate skin, as to resemble scarlet fever; and, further, it is to be borne in mind that very rarely scarlet fever and typhoid fever may complicate one another. The abdominal symptoms of typhoid fever and the throat symptoms of scarlet fever aid in the differential diagnosis. It should be remembered, however, that the exhaustion following an attack of scarlet fever may render the general appearance extremely like typhoid. In the relapse of typhoid fever the rose spots often appear as early as the third or fourth day. In typhus fever they are much more plentiful and often form petechiæ.

In *Bright's disease* a roseola often appears over the feet and ankles, wrists and hands, and sometimes spreads to the skin of the chest and abdomen. Desquamation may take place, but absence of febrile movements and the presence of renal trouble render the diagnosis easy. This manifestation has not a dangerous import.

A dusky-red rash rapidly spreading over the neighboring skin, above the level of which the affected area is raised, and which is separated from the sound skin by a sharp line of demarcation which can be both seen and felt, is characteristic of *erysipelas*. The skin soon becomes brawny to the sight and touch, and the lines of demar-

cation feel markedly indurated. Most commonly the disease appears on the face, starting from the inner canthus of the eye, the nostril, or the corner of the mouth. Rarely erysipelas affects the skin of the trunk. The fever may be quite marked, even in mild cases, and usually falls by crisis on the sixth day. In severe cases with fatal tendencies there may develop in place of crisis a typhoid state with low fever and delirium. If the disease be severe, blebs and bullæ form, edema of the skin becomes very profound, and finally suppuration may occur, forming what is known as phlegmonous erysipelas. (See also Glanders.) Erysipelatous inflammation of the skin without systemic disturbance may follow the application of arnica. A condition also closely resembling erysipelas in its raised surface is urticaria, which, however, differs so materially in other respects that a diagnosis is readily made. Aside from the absence of systemic disturbance in urticaria the swelling of the skin is not red, but pale and pearly in hue, although it may be surrounded by an erythematous blush; the onset is extraordinarily sudden, so that a skin seemingly normal at one moment, after a slight bruising by the finger or rubbing by the clothes, develops the complete eruption in a moment.

A marked roseola or dermatitis involving the insides of the thighs or the scrotum or vulva should give rise to the belief that the patient is suffering from a failure to properly pass or retain the urine, which, on escaping, irritates the skin. This is particularly apt to result if the urine is that of a diabetic.

It is an interesting fact that in some cases of *tuberculous peritonitis* an erythematous rash appears on the abdominal wall around the navel.

The presence of a roseola or erythematous rash often indicates the *untoward influence of some drug*, following its external or internal use. We find that it very commonly follows the ingestion of copaiba, and, as many persons suffering from venereal disease take this drug, the physician must use care not to be led into a diagnosis of syphilitic roseola. It also follows the use of quinine, opium, antipyrine, and many other drugs, such as digitalis and chloral.

The roseola caused by the use of copaiba appears by preference on the upper and lower extremities, and particularly on the backs of the hands, about the knees, the ankles, and on the chest, and it is often accompanied by fever. Indeed, the eruption caused by copaiba may closely resemble a papular syphilide; but its sudden onset, itching, and disappearance when the drug is stopped separate it diagnostically from the specific disease.

The roseola following the use of bromide of potassium is, according to Veiel, very rare, and is distributed over the lower limbs. In children it may closely resemble measles.

The roseola or erythema caused by quinine is to be separated from that of scarlet fever by the absence of fever, of the scarlet tongue and sore throat, and by the fact that there are no prodromes or circulatory disturbance except the characteristic evidence of cinchonism. In doubtful cases this is still further confirmed by analysis of the urine or by the use of the following simple test: Observe the disappearance of the fluorescence of the urine caused by quinine, after the sodium chloride has been removed by precipitation by nitrate of mercury, or after separating the quinine as an iodide by the addition to the urine of a solution of two parts of iodine, one part of iodide of potassium, and forty parts of water. The iodide of quinine can be again dissolved by the application of heat.

A distinct diffuse roseola sometimes follows the use of arsenic. Roseola may be caused by the use of salicylic acid and strychnine, and a scarlatiniform rash sometimes appears in blotches over the face and body in persons who are taking turpentine.

Roseola also ensues in some persons after the application of surgical dressings containing iodoform, corrosive sublimate, and carbolic acid, being due either to a local effect of these drugs or to their absorption from the dressings. Arnica tincture applied for sprains or bruises may produce marked roseola, or even erythematous and erysipelatous swelling of the skin, as already stated.

An important drug exanthem is that caused by atropine, the rash produced by it being very like that of scarlet fever, except that it lacks the red punctations of that disease. This rash may be associated with a slight rise in temperature and be followed, rarely, by desquamation. The face of a child suffering from an overdose of atropine is very characteristic. The eyes are bright, the pupils widely dilated, and the skin over the malar bones is red, but striking lines of pallor reach from the corners of the mouth to the nose. There may be active, talkative delirium and very mild convulsions from overdoses of atropine, thus making the resemblance to the onset of scarlet fever very striking. The brief duration of the rash, its lack of punctation, the absence of high fever, and the history of the patient having taken atropine or belladonna, all help to make the differential diagnosis.

Roseola, followed by desquamation, has been known to follow the hypodermic injection of mercury. Sometimes the use of blue ointment produces a widespread rash resembling measles, and this resemblance may be increased by the development of a febrile movement. A similar eruption may ensue from the ingestion of opium.

Erythematous rashes, too, frequently follow slight irritation of the skin in persons who use chloral.

Acne of the skin, particularly on the face, is often produced by the use of bromide or iodide of potassium, or of any preparation

containing bromine or iodine. That produced by iodine is generally sudden in its onset and profuse in its distribution. The base of the pimple is bright red, the top speedily becomes pustular, and Fournier states that it may be hemorrhagic. Stopping the ingestion of the drug speedily relieves, or at least decreases, the eruption. The acne due to bromine is often very profuse, and the pimples in severe cases may coalesce, making sloughs of considerable size with an indurated base.

In some persons, generally females, there is developed an acne on the face, breast, and back, as the result of taking iron as a tonic.

In addition to the acne caused by drugs or their compounds, mention should be made of the acne and furuncles appearing in

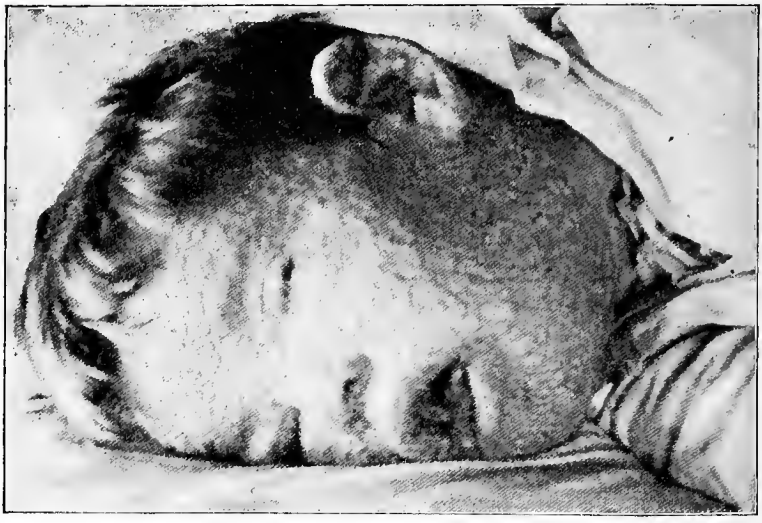


FIG. 43.—Smallpox eruption on the seventh day.

persons working in paraffin, which is due to blocking of the sebaceous glands.

Smallpox.—Closely associated with the eruption called acne is that which is characteristic of smallpox and chickenpox. The eruption of smallpox appears on the second or third day in the form of tiny specks, resembling flea bites. These rapidly become papules, which have an indurated base, so that they feel as if shot were under the skin (Fig. 43). After about thirty-six hours these papules become vesicles, containing a turbid fluid, which speedily becomes purulent, forming a pustule. Generally this process of maturation takes three days, and, with the development of the pus, the so-called secondary fever, which may be even higher than the primary fever of invasion,

sets in. After a period of eighteen to twenty-one days the pustules drop off, having become dried up, leaving, if the attack has been severe or the skin delicate, deeply pitted scars. The vesicles of variola soon become umbilicated, are multilocular, and are difficult to rupture with the finger nail. Although the eruption of smallpox appears on the forehead, which is the favorite seat of acne in many cases, a differential diagnosis is not difficult, since the grave systemic disturbance, febrile movement, and rapid involvement of the skin of the limbs speedily indicate the true nature of the disease. The early appearance of the rash on the hands in variola is a valuable diagnostic sign, as acne in this part of the skin is practically unknown. Then the sudden development of the eruption in smallpox is entirely different from the gradual onset even of the most intense acne.

It is not to be forgotten that cases of smallpox develop in which the symptoms of systemic disturbance are so mild that it seems impossible for true smallpox to be present, and also that acuminate syphilide accompanied by febrile movement may develop about six or seven months after infection, which may be accompanied by such marked systemic disturbance as to resemble smallpox.

Pustular syphiloderm sometimes resembles smallpox so closely as to almost defy an immediate differentiation. The history of syphilitic infection or of certain syphilitic eruptions, and the absence of severe systemic symptoms, may aid us, but fever may be present. As the pock develops in syphilis it does not become so well umbilicated, nor does it leave deep pits in the skin.

In some cases a purulent acne of the forehead develops in syphilis.

The separation of variola from measles has already been discussed, and it is only in the papular stage that the former disease can be confused with the latter, while the reddened mucous membranes and swollen face of the case of measles soon determine the diagnosis. The rapid formation of vesicles and the shot-like sensation of the eruption show that the rash is not measles.

Vaccinia.—The appearance of the eruption of vaccinia following vaccination must be next described. Three or four days after the vaccination a single or several papules arise on the scarified surface, which by the sixth day are changed into umbilicated vesicles, which soon unite and form one vesicle the size of a five-cent piece. This vesicle finally forms a scab, which falls off after the expiration of about three weeks from the inoculation. A "good primary take" is always surrounded by an areola of rosy red of several inches in width. Rarely severe inflammation and sloughing ensue (Fig. 44). When the vaccination is a secondary one, the "take," if it occurs, often produces no symptoms until the ninth and tenth day, and the local lesions are then very mild.

Chickenpox.—In chickenpox the eruption appears on the first or second day, and keeps coming out for several days. It is rose-colored and occurs as papules, which immediately become vesicles. They last but four or five days, which is the time that it takes the eruption of smallpox to develop, and are usually associated with very mild febrile disturbance, the child remaining but little indisposed if well cared for and nursed. Unlike smallpox, varicella does not become umbilicated unless it grows about a hair follicle which

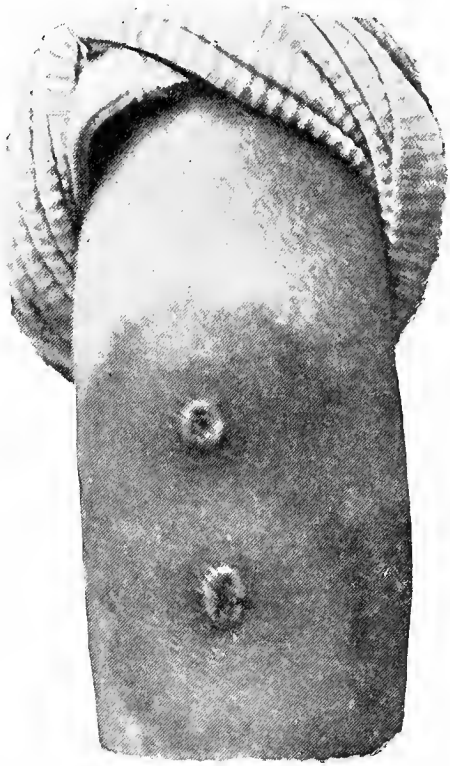


FIG. 44.—Typical vaccine vesicles of a "primary take." Tenth day.

holds the centre of the pock, and rarely leaves pits in the skin unless the vesicles are picked at by the finger nails. Neither do the vesicles become pustules unless infected by picking or the child is in a condition of debility or suffers from struma. Varicella is separated from variola by the absence of severe systemic disturbance, by the rash first appearing on the chest and neck instead of the forehead and hands, by the presence of other cases of the disease in an epidemic, by the repeated crops of the eruption in vaccinia, so that several sets of vesicles may be present at one time, and, finally, by

the fact that it attacks children who have been well vaccinated, whereas smallpox does not. The history of exposure is, of course, an important point to be investigated.

The profusion of the eruption is not of diagnostic aid, as it may be scant in variola and profuse in varicella.

Impetigo.—An eruption somewhat resembling chickenpox or smallpox is that called impetigo contagiosa, in which there are found multiple, flattened or slightly umbilicated, roundish or oval vesicles, pustules or blebs, which form after some days dry yellowish crusts. It occurs in childhood or early adult life, and is often associated with some degree of fever. The areas involved are the face, neck, buttocks, hands, and feet. The lesions of the skin are larger than in chickenpox, but often follow this disease. As its name indicates, the disease is contagious, and the occurrence of a series of cases in close proximity to one another should not mislead the physician into a diagnosis of variola or varicella. The eruption lasts about two weeks, and Kaposi asserts that swelling of the submaxillary glands is always present. We can further separate impetigo contagiosa from varicella by the localization of its eruption to one area, as a rule, by the fact that the eruption becomes bullous or purulent, and by the larger size of the vesicle. From smallpox we can separate it by the absence of severe pain in the back, the grave systemic disturbance, and the secondary fever of that disease, accompanied as they are by the smallness of the pox, the peculiar odor of the patient, and the history of exposure to variola.

In the presence of a papular, pustular, or vesicular eruption of the skin it must be remembered that *quinine* sometimes develops these lesions in susceptible persons. In some instances where it involves the hands it may indicate that a local effect has been produced by working with the drug.

Eczema in its various forms may appear as the result of the use of quinine internally or locally, or of the employment of mercury internally or externally. When it arises from the use of iodide of potassium, which is very rare, it chiefly affects the scalp and scrotum. The development of an eczematous irritation of the skin sometimes follows the use of chloral.

Herpes Labialis is a very constant lesion associated with croupous pneumonia, and its development is said to be a favorable sign. It is also an important sign for the separation of epidemic cerebrospinal meningitis from meningitis due to other causes, as it is not commonly present in the non-epidemic form. It sometimes arises as a result of using salicylic acid.

The presence of herpes rather excludes tuberculosis, typhoid fever, and simple pleurisy from the case in instances in which the diagnosis is doubtful, since it is rarely met with in these maladies.

In the cases of herpes zoster the skin lesion often has its origin in compression of the spinal cord, or in such diseases as tabes, spinal meningeal irritation, and peripheral neuritis.

Furunculosis.—The development of recurring crops of boils in persons not exposed to paraffin or tar should cause the physician to suspect the presence of diabetes mellitus, or at least that there is general debility, and particularly an absence of lime salts from the system in the proper quantity. When the ordinary boil passes into a condition of marked induration about its base, with sloughing of the subcutaneous tissue and necrosis of the skin, which becomes perforated by the openings of several sinuses, we have to deal with a carbuncle or anthrax simplex. The disease usually appears on the back of the neck, on the back, or the lip. The systemic disturbance is very great and the exhaustion profound. The skin covering the area involved becomes grayish or bluish black, and then separates as a large mass, while the subcutaneous tissue comes away in shreds. It is a dangerous disease in all persons, but particularly so in those who are already weakened by other diseases or excess.

Anthrax..—The development of a painless macule on the skin of the hand or foot, followed by an acutely inflamed papule which itches and is soon changed into a relaxed vesicle containing bloody serum, in which there is a hard nucleus which rests upon an indurated base, is the initial manifestation of anthrax maligna or malignant pustule. The lymphatics soon become swollen, and metastatic abscesses speedily form elsewhere, as in the axillary glands. The systemic symptoms are severe, sometimes being manifested in high fever, in other cases by a typhoid state. Death is very commonly the sequel (65 per cent.), even if prompt surgical interference takes place. There is generally a history of exposure to infected animals or their hides. Malignant pustule is to be separated from carbuncle by its fulminating character and peculiar appearance.

Glanders.—When an erysipelatoid rash with swelling of the skin and the development of papules, vesicles, pustules, and bullæ appears in association with induration of the skin, with sloughing eventually taking place, the disease may possibly not be erysipelas of a phlegmonous form, but glanders or equina. Numerous inflammatory foci appear in the skin in glanders which end in local abscesses and hemorrhagic nodules, and profound systemic infection is always present. The presence of a sanious discharge from the nose aids in confirming the diagnosis. Death usually comes in a few days in this acute form. Should the course of glanders be chronic, pustules somewhat like those of smallpox, except that they are not umbilicated, lie on an indurated base, and in them is formed a viscid or sanious pus of offensive odor. This disease is rare. Both forms arise from infection from a horse suffering from the malady. Glan-

ders may be confused with variola or the pustular and ulcerative gummatous stages of syphilis.

Pemphigus.—The development of pea-sized or larger bullæ upon the skin may indicate the presence of pemphigus, or if there is central nervous disease involving the spinal cord and resulting in trophic lesions similar bullous eruptions may take place. The bullæ, if they contain dark bloody fluid and are situated upon a limb in which there is an abnormally high temperature, are peculiarly indicative of central nervous lesions, particularly if there is a tendency to dilatation of the capillaries of the skin on slight irritation; but if the temperature of the entire body be raised, the physician should remember that pemphigus is a disease in which there is often marked febrile movement. Sometimes these bullous manifestations are followed by gangrene in cases of neuritis or other disease causing trophic lesions, such as myelitis and paretic dementia

Bullæ on the face may follow the ingestion of antipyrine or iodine compounds.

The development of a pemphigus-like eruption in the skin may follow the use of salicylic acid or copaiba.

Glossiness of the skin, in which its minute creases become smoothed out and it appears unduly shiny, often results from chronic disease involving some portion of the nervous system connected with the government of nutrition. Very commonly it results from peripheral neuritis. In addition to glossiness there are often redness and marked thinning or thickening of the cuticle and subcutaneous tissues.

Gangrene of the skin may follow nerve injuries or central nervous lesions. Thus, it may follow upon division of a nerve trunk, or be due to cerebral abscess, in which case the gangrene will be with the other localizing symptoms on the opposite side of the body. The cerebral form develops suddenly and without the prodromal redness of bed-sores as seen in prolonged illness. Similar rapidly developing sloughs and ulcerations of the skin are seen in cases of acute myelitis and in the second and third stages of paretic dementia.

A very interesting condition is the so-called spontaneous gangrene of hysteria. On the skin, generally of the breast of a young girl, a spot develops which feels to her to be hot and burning. The skin soon becomes very white, then in a few hours very red and forms a wheal. This rapidly becomes dark and bluish black, looking like a burn of sulphuric acid, and a slough finally comes away, leaving a permanent cicatrix.

The development of gangrene of the fingers and toes sometimes follows the prolonged use of bread made from rye which is infected by ergot.

Sometimes gangrene of the skin follows severe attacks of the

exanthemata in children who are strumous or very feeble, or who are syphilitic.

Ulcers about the base of the finger nails should arouse the suspicion of the excessive use of chloral.

Bed-sores may develop whenever by long-continued pressure upon any part of the body the local circulation is disturbed, particularly if in addition there is general systemic debility from some exhausting disease, such as typhoid fever. They also develop very speedily, and apparently almost spontaneously, in the course of acute transverse myelitis. Under these circumstances the sacrum is the area most severely affected. Sometimes these sloughs have been known to develop as early as six hours after the beginning of the attack. Associated with the involvement of the soft tissues the bones may break down, and cellulitis about the rectum and bladder place the patient's life in immediate jeopardy. In hemiplegia, particularly in that which is due to cerebellar hemorrhage, bed-sores often form on the buttocks, and in paraplegia from other causes than transverse myelitis, upon the sacrum. They also appear on the heels, inside of the knees, and about the hips in some cases of paraplegia.

Sudden sloughing of the skin of the nates sometimes occurs in cases of intracranial hemorrhage, and is said by Joffroy to be connected with lesion of the occipital lobes.

Gangrene of the skin follows upon diabetes mellitus, and may involve the scrotum or vulva if the irritation of these parts by the urine is constant. More commonly the toes are affected, and there is this important differential point, that in the gangrene of old age with bad vessels the lesion is usually at the tip of the toe, whereas in diabetic gangrene it is frequently about the ball of the big toe or on the sole or dorsum of the foot. Previous to the development of gangrene there are developed bullæ and other inflammatory changes in the skin which is about to be affected. Kaposi describes a serpiginous form of gangrene affecting the leg in diabetics and a variety of tissue break-down in which a dermatitis, followed by ulcers and a lupus-like formation, also occurs in diabetes. Perforating ulcer of the foot occurs in locomotor ataxia and in paretic dementia.

Raynaud's Disease.—Closely related, yet quite distinct from angioneurotic edema, is that condition called Raynaud's disease, symmetrical gangrene, or local asphyxia, according to its severity. The fingers and toes or the nose, with or without exposure to cold, are found to be pale and livid, looking like a hand from which all the blood has been removed by the use of an Esmarch bandage. The part often feels as if "asleep," and is more or less numb and without sensation. To the touch the part is cold and waxy, and it does not bleed when pricked. With the onset of these signs there are often general chilliness and malaise. Often this manifestation speedily disappears, leaving the

skin apparently normal; but if it persists, the skin becomes glossy, shrivelled, and looks as if it had been soaked in hot water for hours. When the disease is more severe the pale waxiness is supplanted by cyanosis until the finger tips, for example, look as if dipped in blue ink; there is often local swelling; the skin is frequently found to be sweating freely and is distended with blood. The skin may rapidly separate from the deeper tissues and become necrotic in patches or *en masse*, and the entire tip of the finger, after becoming black, shrivels up into a condition resembling dry gangrene, which is separated from the sound skin by a sharp line of demarcation. Sometimes small necrotic patches slough out, which leave cicatrices telling of the attack. The prognosis is not bad. The most interesting complication of the disease is paroxysmal hemoglobinuria.

Rupia.—The value of roseola and rupial eruptions in the diagnosis of syphilis has already been dwelt upon. When the roseola becomes transformed into slightly elevated or bean-shaped spots, irregularly scattered, but sometimes forming groups which are apt to be circular, and these circles become marginated and then scaly on the edges, resembling lepra or psoriasis, or even go further than this and develop bullæ and blebs, and when the sores which form are filled with a clear liquid which may become sanious or turbid and on drying leave crusts, the removal of which reveals deeply excavated sloughs, the area of the slough often being as large as a silver dollar, but often irregular in outline, syphilitic rupia is probably the lesion. There is, however, this important differential point, namely, that in specific rupia there is an essential feature, a peripheral ring of induration, whereas in the non-specific form this induration is absent.

If, in addition to these variations, the eruptions are dusky red and leave behind them on healing copperish-looking discoloration of the skin, and appear on areas, such as the flexor surfaces, where ordinary skin eruptions are rarely seen, the diagnosis of syphilis is highly probable. If the eruption is chiefly tuberculated and the tubercles are large and more marked than usual, and if they ulcerate and become deep sores, and finally form on healing well-marked cicatrices, tertiary syphilis is to be considered the probable cause.

If, again, we find small nodules under dusky-red skin, which finally breaks down and discharges bloody serum, or pus which in burrowing forms discharging sinuses, syphilis of the third stage may be regarded as a likely cause.

The appearance of hard, dark-brown, infiltrated areas in the skin may be due to the excessive use of bromine, and as they gradually become depressed in the centre closely resemble in some cases the nodules of syphilis.

SCARS OF THE SKIN.

Scars of the skin often give us much useful information. Early tendencies to struma or tuberculosis may be found in the scars resulting from suppurating cervical glands. In the groin such scars may be an evidence of venereal infection, although it should be remembered that suppuration of these glands usually takes place as a result of chancroids and not from true chancre. It has already been shown that syphilitic skin lesions often leave scars to mark their site. Scars upon the head tell us of possible injuries to the brain in suspected traumatic epilepsy, or of falls in epileptics. Similarly, other traumatisms in the history of the patient may be discovered by scars elsewhere.

The presence of numerous regularly arranged fine scars on the chest or elsewhere may develop the fact that the patient has at some time been wet-cupped for some pulmonary or other disease; or if the peculiar three-pointed scar of the leech is seen, another good evidence of a bleeding is presented.

When the skin of the abdominal wall exhibits striæ or scars arranged in parallel series, it indicates that it has been stretched very considerably by pregnancy, ascites, or, more rarely, by excessive corpulence. Sometimes these striæ appear on the lower limbs in pregnant women or in persons with dropsy. Very rarely they may develop on the arms or legs or elsewhere during convalescence from some grave disease, such as typhoid fever.

SWEATING OF THE SKIN.

Sweating of the skin, aside from the normal and almost imperceptible exhalation of moisture, takes place in health as a result of severe muscular exertion, whereby the peripheral circulation is increased and the bodily temperature raised, or when the body is very heavily clad or exposed to external heat in excess. In all these cases the sweating is to be regarded as a physiological effort on the part of the body to reduce its temperature by increased evaporation from the surface. In disease sweating provides us with very important information in many conditions.

During the course of fevers which naturally end by crisis the occurrence of a profuse sweat (generally associated with a fall of temperature) gives us the first sign of beginning convalescence, and in irritative fevers, or those due to cold and congestion, the artificial production of sweat is decidedly a good omen. The sweat of crisis is perhaps most marked in croupous pneumonia. Profuse sweating

is also a characteristic symptom of relapsing fever, pyemia, acute ulcerative endocarditis, phthisis, malarial fever of the distinctly periodic type, and of typhoid fever and collapse. Constant profuse sweating is marked in some cases of acute articular rheumatism, and it is worthy of note that, while sweating generally occurs in febrile diseases at a time when the temperature is falling, in rheumatism the febrile movement may even increase during the sweat rather decrease.

Profuse so-called colliquative sweats often occur in sleep in debilitated persons without the presence of any febrile movement, and are an evidence of profound nervous and vasomotor relaxation. Moderate sweating sometimes is seen from similar causes in feeble persons after taking anything in the food or drink which produces circulatory or nervous excitement. Localized sweatings occur almost solely in subjects of nervous disorder, which is often organic, as in paretic dementia, and sometimes functional, as in hysteria or Raynaud's disease. They depend upon perverted vasomotor influences sent to the glands and their supplying vessels in particular areas. Localized sweating of one side of the face or neck or chest is often a most important sign of a thoracic aneurysm pressing on the cervical sympathetic. Bromidrosis may occur in hysteria, or the head may be the only part affected in Graves' disease and in migraine. Profuse sweating of the head of an infant when sleeping may be indicative of rickets. In cases of the toxemia of cholera or of renal disease there may be profuse sweating, which takes the place of the dry and hot skin seen more commonly in this condition. The surface of the entire body is usually involved in the sweat.

The quality of the sweat varies greatly in many persons. In cases of deficient renal activity it often contains urinary elements, smells uriniferous, and may even deposit particles on the skin in small white scales, particularly on the forehead and nose. This is called uridrosis. In jaundice the sweat may be bile-stained.

EXCESSIVE DRYNESS OF THE SKIN.

Excessive dryness of the skin is seen in grave forms of renal disease, in nearly all acute fevers with a high temperature, and in cholera and diabetes, in which diseases the dryness is largely the result of drainage of liquids from the body. Sometimes after a prolonged dryness of the skin during high fever, as soon as sweating begins hundreds of little blisters develop, due to retained sweat under the epiderm. These are called miliaria or sudamina.

When the skin is dry and harsh, and the naturally thickened portions have in their folds a peculiar white appearance as if filled with

meal, diabetes should be sought for. Rarely the physician may be deceived by profuse sweating in diabetes, in which disease the skin is usually very dry.

DROPSY AND SWELLING OF THE SKIN.

Swelling of the skin and subcutaneous tissues occurs most frequently as a result of dropsy, in which condition the lymph spaces become filled by liquid. The skin in the area involved is not only swollen but doughy, or if the effusion is very great the skin may be of almost board-like hardness, so tensely is it distended. Pressure with the tip of the finger upon such an area will result in pitting, and this is one of the more important signs separating dropsy or true edema from the swelling of acute inflammation, which, while it may be very tense, does not pit. Further, the swelling of inflammation is usually localized, reddened, and feels hot to the touch, whereas the dropsical swelling is more diffuse, is pale, and the temperature of the part is lower than normal.

When the effusion of liquid is limited to one portion of the body it is usually called edema or localized dropsy, whereas if the entire body is boggy it is designated general anasarca. Dropsy is to be differentiated from myxedema by the facts that in the latter disease the onset is very slow, the swelling does not pit on pressure and is universal and fairly equally distributed over the body, the thyroid gland will often be found diseased, the subcutaneous tissues are not boggy but resistant, and there is anesthesia of the skin. When the subcutaneous tissues are distended by air, instead of liquid, they are even less resistant than in dropsy, the swelling is usually very localized and does not pit, and the part crackles or crepitates on gentle pressure. The presence of dropsy is indicative of many widely separated diseases. In the first place, it may indicate a deficient circulation of blood, either by reason of a feeble or diseased heart or because of obstruction by the pressure of growths, thrombi, or emboli.

It may be due to disease of the walls of bloodvessels and lymphatics, as is generally the case in renal disease, or it may arise from disease of the blood itself. Again, in some cases it is due to disordered nervous control of the vessels, by reason of centric or peripheral changes which may be organic or functional.

General Anasarca.—The significance of a widely diffused general dropsy or anasarca is generally that there is well-marked *renal disease*, and this probability is greatly strengthened if the edema of the face be well marked, particularly in the morning on arising, disappearing as the day goes on. The skin in such cases will usually be quite pale, and an examination of the urine will reveal the presence of the

signs of nephritis. The next most common cause of general anasarca after renal disease is *heart disease*. When due to this cause it will be found that the ghastly pallor of renal anasarca is replaced by cyanosis, and often by engorgement of some of the superficial veins, while the physical signs of cardiac disease will confirm the diagnosis. General anasarca may rarely arise as a result of a *multiple peripheral neuritis*, and it also occurs as a symptom of beriberi and from the excessive use of large amounts of arsenic. This arsenical anasarca may be due to the neuritis produced by the drug, although Wood thinks it is due to a cellulitis. Rarely we find general anasarca in cases of advanced *cancerous cachexia*, and care must be exercised that the hemic murmur due to anemia does not mislead the physician into a diagnosis of heart disease.

Local Dropsy.—The most common seat of localized dropsy or edema is the feet and legs, particularly about the instep, the ankles, and the tibiæ. When it is bilateral it is generally indicative of *cardiac failure* or more rarely of renal disease. Nearly always, if it be renal, a careful examination will discover edema in other parts of the body, although it may be most marked in the feet and legs. In many cases the various serous sacs, such as the pericardium, peritoneum and pleuræ, will be found to contain more liquid than normal, and the tissues generally will be found infiltrated.

Other causes of edema of the feet and legs are *anemia*, and obstruction to the return of blood from the lower limbs by reason of *growths* in the abdomen pressing upon the iliac veins or inferior vena cava. Thus, cancer of the pancreas sometimes causes edema of the feet and legs in this manner. Very rarely edema of the lower extremities follows hepatitis or *hypertrophic cirrhosis* of the liver as a primary symptom. Usually such lesions produce ascites alone, or if the legs are involved they become so by reason of the pressure of fluid in the pelvis during the time that the patient is sitting up or standing. This latter cause of bilateral edema of the lower limbs is, however, rare. Sometimes edema of both legs and feet comes on in persons who, though feeble and relaxed, remain standing with little muscular movement during many hours in the pursuit of their occupation, as in typesetters and salesmen, or in young persons who have subjected themselves to excessively severe muscular exercise. In other instances, very much more frequently, edema of the feet and legs comes on in the course of profound anemia resulting from slow hemorrhages or other causes. It is also seen in the cachectic stage of cancer, owing to the anemia which is present. General swelling of a leg in a puerperal woman is probably due to *phlegmasia alba dolens*, and this affection may also be bilateral. Both Herman and Cameron Kidd have each reported a case of bilateral phlegmasia alba dolens occurring in a virgin with

anemia. When it occurs in males it is most commonly unilateral and a complication of convalescence in typhoid fever. It is due to thrombosis of the left femoral vein, as a rule.

Dropsy diffused or localized in the feet and legs occurs in *scurvy*.

When the face is edematous the swelling is most marked under the eyes, the lower lids of which are particularly puffy in the morning and nearly normal in appearance at night. This form of edema is most marked in, and is almost pathognomonic of, renal disease. Its only other causes are the excessive taking of arsenic and angioneurotic edema. More alarm should be felt at a slight swelling of the face of this character than if the feet are markedly puffed. Sometimes edematous swelling of the side of the face and scalp which has been involved in a severe attack of neuralgia takes place.

When edema of one or both eyelids occurs, with protrusion of the eyeball, the swelling extending to the rest of the face as time goes on, it forms an important symptom in obscure cases of suspected cerebral thrombosis, and is caused by the intimate association between the intracranial vessels and those of the face.

Sometimes edema of the eyelids comes on in neurotic subjects and may extend to the forehead. This may be seen in children, most commonly about puberty, and is probably the result of a neurosis.

Edema of the upper extremities alone only results from causes interfering with the flow of blood, such as are produced by morbid growths in the chest, as mediastinal growths, and in cases of aneurysm. When the swelling is limited to one arm or leg it is a sign that there is interference with local circulation, as, for example, the obstruction of the femoral vein by thrombus, as in phlegmasia alba dolens following labor or enteric fever, or, when the edema is in the left leg, by cancer of the sigmoid flexure. If the swelling of the arms and head is manifested suddenly, it may be due to that rare condition in which an aortic aneurysm ruptures into the vena cava; whereas if it develops slowly, it is due to pressure by a growth.

There remain three forms of local edema of some diagnostic significance, namely, that occurring in a limited area over some deep-seated suppurative process, as in the skin back of the ear in cases of mastoid abscess or thrombosis of the lateral sinuses, that over the ribs in cases of purulent exudation into the pleura, and that on the thigh in the abscesses which sometimes follow typhoid fever.

ADIPOSIS DOLOROSA.

No better place can be found in which to mention that condition in which irregular and numerous masses of fat are to be found in the subcutaneous tissues of middle-aged persons, usually women;

these masses are more or less painful, and occur in the body and extremities. The skin itself is not altered. Dercum first described this state and gave it the name of *adiposis dolorosa.*

The ocular appearance and touch of the skin having been studied in so far as its surface affords evidence of more deeply seated disease or functional disturbance, we next pass to a study of its sensibility, having the same diagnostic objects in view

SENSATION IN THE SKIN.

Before considering the various perversions of its sense it is important to remember that the sensibility of the skin may be divided into four parts, namely, its tactile sense, its pain sense, its thermic sense, and its sense of pressure. Any one of these senses may be perverted or in abeyance without the others being affected, and it is noteworthy that, while corresponding areas of the skin in all individuals have practically identical sensibilities, each part of the skin has a sensitiveness of its own, so that while in some parts the slightest touch is felt, in others severe irritation must be produced to cause much of a result. These differences have been carefully studied by many observers, the most thorough being Weber, who has found that the average ability to separate points brought in contact with the skin is about as follows: at the finger tips points can be separated at from 2 to 3 mm., on the lips 4 to 5 mm., on the tip of the nose 6 mm., on the cheeks and backs of fingers 12 mm., and on the forehead 22 mm.

The skin on the neck separates points at 34 mm.; that on the forearm, on the lower leg and back of foot, at 40 mm.; on the chest at 45 mm.; on the back at 60 mm., and on the arm and thigh at 75 mm. If tests be frequently repeated in a single individual, the ability to separate the points increases with training. Care should always be taken that the pressure on both points is equal, applied simultaneously, and that the points are equally sharp.

In testing tactile sensibility, not only should points be used, but also objects. Often single points may be applied without any abnormal manifestation, and, in some cases of disease, the skin, which seems devoid of sense on ordinary touch, is found to be excessively hyperesthetic if the hand is drawn lightly over it.

The best apparatus for testing tactile sensibility is the esthesiometer of Carroll, which is a pair of double-pointed compasses connected by a graduated scale. (See Fig. 45.)

The ability to distinguish pain-giving and thermal applications is

most acute in the normal skin of the hands, in which tactile sense is also most acute.

The methods by which we test the pain sense are several, but chiefly by pricking the skin, more or less deeply, with some sharp-pointed instrument, such as a pin, or by pinching the integument.

The thermal sense is studied by applying bodies which are hot or cold against the skin, such as a cold knife, a small piece of ice, or a test tube which contains very cold or hot water. In all such tests the physician should use both hands simultaneously. With one hand he should apply his instrument to the suspected area, and with the other a similar instrument to an area known to be healthy, in order that an actual comparison as to the sensations may be noted by the patient. Thus the face may be used as the normal area in a spinal lesion, and the skin of the arms as a control surface in a lesion involving the legs. The eyes of the patient should be blindfolded, and if tactile sense is being tested the instrument must be of the same temperature as the body.

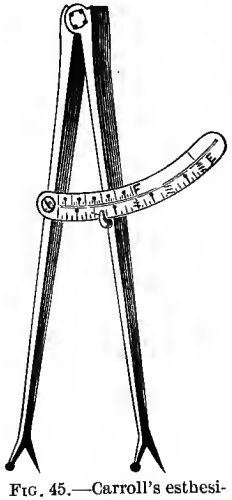


FIG. 45.—Carroll's esthesiometer.

Closely connected with the subject of tactile sense is what is known as stereognosis, or the ability to recognize objects by contact and grasp. By this means healthy persons are able without looking at an object to judge of its character, and in the blind this sense is very highly developed. Manifestly the entire sensory apparatus must be intact for stereognosis to be performed, since any interference with the sensory system will produce that condition which is known as "astereognosis." Not only does stereognosis involve the tactile sense but also muscle sense, since it is by the grasping of the object that information concerning it is gained, as well as by its coming directly in contact with the hand. At the present time the discovery of astereognosis in a patient has not very definite clinical significance, so far as localization of the lesion producing this condition is concerned, but when astereognosis does not depend upon a disease of the sensory nerves, it depends upon involvement of the cerebral cortex, in the middle third of the posterior central convolution and in the adjacent part of the inferior parietal lobule. Of course, it is possible for a lesion in the sensory fibers of the internal capsule to cause astereognosis; but a lesion of any size in this region will usually produce signs of motor paralysis, owing to the juxtaposition of the motor and sensory fibers.

Disturbances in the sensation of the skin may arise from functional

or organic disease involving the peripheral nerves, the sensory tracts in the spinal cord, similar tracts in the lower part of the brain, and, finally, the subcortical or cortical parts of the cerebrum itself.

The sensory pathway or the afferent fibers pass upward, starting with the peripheral sense organ in the skin, or elsewhere, and after forming part of the nerve trunk and entering the ganglion on the posterior root, enter the spinal cord by what is called the posterior root, which is shown in Fig. 46.

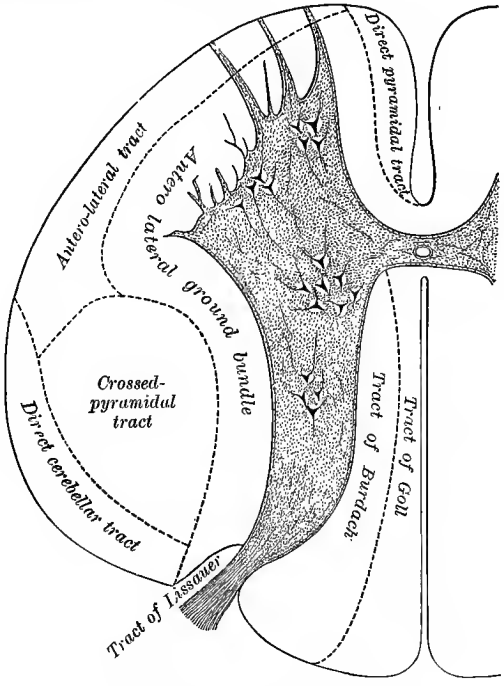


FIG. 46.—Columns of the cord. (Gray.)

The posterior root enters the cord in three sets of fibers; one of these, the one lying nearest the posterior median fissure, is composed of coarse fibers and is called the median bundle, and passes obliquely into the lateral part of the column of Burdach. As soon as they have entered this column they turn at right angles and run upward for some distance, thereby helping to form the column of Burdach. Some of them also run downward a short distance. Some of these fibers also enter the column of Goll.

The second set, near the side of the cord, goes directly into the gray matter of the posterior horn through the substance of Rolando,

and the third set, nearest the side of the cord, enters the cord very superficially, and, turning at once at a right angle, goes upward to form Lissauer's zone. Here they pass upward chiefly in the column of Goll (posterior median) to the medulla oblongata. Before reaching the medulla, however, the column of Goll ends in the gracile nucleus and the column of Burdach in the cuneate nucleus.

These nuclei which have received the fibers of the two sensory columns give origin to fibers which pass to the brain. They sweep forward to the front of the central canal of the medulla and decussate at a higher level than the motor tracts. A great majority of these fibers pass upward to the brain, but some pass forward, and finally join the restiform body on the posterior aspect of the medulla. Those which pass upward from the so-called fillet pass into the crus cerebri, in that part of it called the tegmentum, and thence into the posterior part of the posterior limb of the internal capsule, whence they spread out in the corona radiata to the occipital lobe and temporosphenoidal lobes.

The duty of the physician in all cases is to determine first whether the disorder of sensation is functional or organic, and then where the lesion producing the symptoms is situated.

The two chief manifestations of perverted sensibility in the skin are anesthesia and hyperesthesia, and the minor ones are paresthesia or numbness, tingling and formication, and analgesia, or the failure to feel pain. Whatever the cause of these symptoms may be, the history of the patient and his general symptoms should be carefully studied when examining these signs, as frequently a diagnosis is impossible with them alone as guides.

Anesthesia.—Anesthesia of the skin is indicative of a very large number of conditions arising anywhere in the sensory apparatus. In other words, anything which interferes with the transmission of an impulse to the perceptive centres in the brain may be its cause. Of the functional causes, the most frequent is hysteria, and the presence of cutaneous anesthesia in a female should always arouse a suspicion of its being due to this cause. Rarely it is seen in hysterical males. The organic causes of anesthesia of the skin are cerebral hemorrhage, cerebral tumor, hemorrhage in the pons or tumor of the pons, hemorrhage in the cord, tumor of the cord, myelitis (transverse), locomotor ataxia, cerebrospinal meningitis, spinal meningitis; compression of the cord by vertebral caries, by fractures, by dislocations; and hemorrhage into its membranes. Additional causes are pressure on the posterior nerve roots by reason of caries and growths, inflammation of the nerves (neuritis), injuries to the nerves by blows, pressure, or cutting, and, finally, by paralysis of the nerve endings from cold or the action of drugs.

Anesthesia, according to its area of distribution, may be divided

into hemianesthesia, crossed anesthesia, bilateral anesthesia, irregular but complete anesthesia, and partial anesthesia.

Hemianesthesia occurs most frequently as a result of hysteria, next commonly from lesion of the posterior part of the internal capsule, and more rarely from spinal injuries or growths in the cord of a unilateral character.

The hemianesthesia of hysteria involves, as its name implies, one side of the body, and is usually universal on that side, except that here and there may be patches of hyperesthesia or tenderness, dotted-like oases in the midst of the absence of sensation. This anesthesia is often unaccompanied by motor paralysis, and its area is separated from the opposite side of the body by a sharp line of demarcation, which runs along the middle of the trunk and face. The presence of such a well-defined line of separation in a young woman is of great significance. The anesthesia is generally absolute, and severe injury may be done to the skin in some cases without the patient feeling it; but, notwithstanding its degree, it is a noteworthy fact that the anesthesia may transfer itself to the opposite side of the body with great suddenness, and equally suddenly return to its former site. In a great majority of cases, for some unexplained reason, the left side is the one affected by anesthesia, and hyperesthesia on the opposite side increases the contrast which exists between it and that in which sensation is lost. (See Hyperesthesia.) In some cases of hysterical hemianesthesia the paralysis of sensation involves the nerves of special sense; and loss of smell, taste, and hearing, and impairment of sight may ensue on the same side. The visual changes are so characteristic that they practically decide the character of the case when they are discovered in any instance of doubtful diagnosis; they consist in a loss of the color vision (first, violet is lost, then blue, and then red), and there is a great limitation of the visual field, whereas in the hemianesthesia due to an organic lesion in the internal capsule, so situated as to involve the nerve fibers connected with vision, there is hemiopia. Hemianopsia due to hysteria is so rare as to be denied an existence by most authorities, but Lloyd and de Schweinitz have seen a case. Generally the loss of vision on the anesthetic side is a total one for both sides of the eye in hysterical blindness. (See chapter on the Eye.) Nearly always in hysterical hemianesthesia a spot can be found over the shoulder which is not anesthetic. The age of the patient, the sex, the general expression of the face, and the history of the illness, associated, as is frequently the case, with some or all of the hysterical symptoms detailed farther on in this chapter, will generally decide the diagnosis in favor of hysteria.

A form of hysterical hemianesthesia very apt to lead to an error in diagnosis is that seen in persons who have suffered from infantile

cerebral paralysis with the resulting deformity (a disease not characterized by sensory disturbances), but who have in later life, superimposed upon the old picture of disease, that of hysteria with this sensory manifestation.

Anesthesia irregular in its distribution, or absolute hemianesthesia, may occur in the course of chorea. The presence of the motor manifestations of chorea clears up the diagnosis as to the cause of the loss of sensation.

Hemianesthesia when not hysterical is nearly always due to an organic lesion in the posterior part of the hinder limb of the internal capsule on the opposite side of the brain from the anesthesia, and the additional symptoms which sometimes accompany it depend for their existence upon whether the lesion is large enough to involve not only the fibers from the cutaneous areas, but also those of special sense, such as sight, hearing, or taste. Nearly always the area destroyed is sufficiently large to result not only in hemianesthesia, but also in loss of motion on the same side. The loss of sensation in such a case is rarely as complete as in hysteria, and the sole of the foot and palm of the hand are often not affected. In rare instances, however, the hemianesthesia of capsular disease may be absolute and universal, or, more rarely still, occur in patches, thereby closely resembling the anesthetic areas seen in hysteria.

Hemianesthesia may also be produced by a large lesion of the cortex in the occipital, temporal, and parietal lobes, in which case it will involve the side of the head as well as the trunk, and will be associated with such definite evidences of apoplexy or injury that the diagnosis will be readily made. If it is widespread, all the special senses will be involved.

Sensory disturbances of the skin are more frequent in softening of the brain than in hemorrhage into the brain, and most commonly are associated with subcortical, rather than cortical lesions.

In this connection it should be remembered that the irregularity of distribution of the lesions in disseminated sclerosis may cause a hemianesthesia, partial or complete.

Anesthesia resulting from tumor of the brain occurs in about 20 per cent. of the cases, and may be unilateral and confined to the paralyzed side, or appear as an isolated symptom without motor paralysis. When of the latter form it is often associated with lesions in the neighborhood of the fissure of Rolando, and in tumors involving the posterior parietal region and the posterior part of the internal capsule.

Autopsies and experiments show that hemianesthesia may arise from a lesion in the optic thalamus, but such an occurrence is very rare.

A very important and essential factor in making the diagnosis

that the anesthesia is cerebral in origin is the history of the beginning of the attack, which has been sudden if due to hemorrhage, embolus, or thrombus (see Hemiplegia), and characteristic of the condition which we call apoplexy.

An important point to be noted in the diagnosis of cerebral anesthesia is the fact that the reflexes are preserved, though the patient may not feel the touch or painful impression; that is to say, irritation of the skin causes movement in the arm or leg, not by any intention of the patient, but owing to the fact that the sensory centres in the cord receiving an impulse cause the corresponding motor centres to send out impulses which contract the muscles.

Unilateral anesthesia associated with motor paralysis, both being somewhat irregular in their distribution, may be due to a lesion, such as a tumor in the pons or medulla oblongata, but death so commonly ensues soon after the apoplexy that the symptom is often overlooked or cannot be developed when this accident is the cause. Further, the discovery of such anesthesia does not positively localize the lesion in the pons, for we do not know much about the course of the sensory fibers in this part. If, however, the area supplied by the trifacial nerve, namely, the face, is anesthetic, and these symptoms are associated with it, then it is fair to assume that the trouble lies in the pons and has involved the nucleus of the fifth nerve. (See Anesthesia of the Face.)

Anesthesia of irregular distribution or confined to one limb may result from cerebral or spinal lesions, or be due to a neuritis, of which we shall speak farther on. If it is a mono-anesthesia from cerebral disease, which is very rare, the anesthesia is most marked at the distal part, and gradually fades off as the trunk is approached. It is evenly distributed, so far as circumference is concerned, and has no sharp line of demarcation.

When such an anesthesia is due to spinal disease the cause may be tumor of the spinal cord, the symptoms depending in their character on the area involved; but in any event the upper border of the area involved is sharply outlined and a constriction-band sensation is often present.

The irregularly distributed form of anesthesia due to hysteria has the same general peculiarities of migration as are seen in hemianesthesia from this cause, and in its symmetrical form it closely resembles the anesthesia due to multiple neuritis. Thus, in the hand the area of anesthesia may be that covered by a gauntlet glove, in the foot that covered ordinarily by a sock, the line of normal sensation being present just above the place to which these protections usually extend.

Crossed Anesthesia.—When sensory paralysis of one side, associated with partial paralysis of motion or paresis on the same side,

comes on, and with it there is hyperemia of the skin on that side from vasomotor paralysis, there is a strong probability that there is a lesion in the cerebral peduncle of the opposite side. If there is at the same time paralysis of the muscles supplied by the oculomotor nerve on the opposite side from the anesthesia—that is, on the same side as the lesion, this diagnosis is still further confirmed; and if the tongue and half of the face on the anesthetic side of the body are paralyzed, still further confirmatory evidence of a peduncular lesion is obtained. Thus, there might be hemianesthesia and paralysis of the right side of the body, including the face and right half of the tongue, and ptosis, from oculomotor palsy, on the left side of the face. The paralysis of the body, face, and tongue would be on the side opposite to the lesion, but the oculomotor paralysis would be on the same side as the lesion.

Crossed anesthesia of the limbs and face—that is, anesthesia of one side of the body with anesthesia of the opposite side of the face—can only occur in lesions involving the upper part of the pons in such a way that the fibers of the trifacial are diseased on one side, and the path for sensory impulses of the other side of the body is also destroyed. (See chapters on the Face and Head, and on Hemiplegia.)

Partial hemianesthesia, with partial hemiplegia on the opposite side in crossed paralysis, may occur from lesions on one side of the spinal cord, and if high up, involve a large part of the trunk and lower limbs. (See chapter on the Feet and Legs, part on Myelitis.) These cases have been explained by a theory of Brown-Séquard, which has recently been doubted owing to the studies of Mott and others. Thus, until recently it was considered as proved that sensory impulses entering the cord crossed to the opposite side almost at once, at least in greater part, passing to the lateral columns in front of the pyramidal tract, and that a very small number entered the posterior columns, while a few ascended in the gray matter. The studies of Mott seem to prove that the reverse is the case, and that the greater part of the sensory impulses do not cross the cord, only a few fibers passing to the opposite side on entrance. He believes that the main pathway for heat and cold sensations is in the gray matter, while the tactile pathways are in the posterior columns, although it is possible that some few isolated fibers may exist in the lateral columns and that these cross in the cord about the level of entrance.

Bilateral Anesthesia.—Anesthesia of hysterical origin involving both legs, and sometimes the lower part of the trunk on both sides, may occur, and, aside from the typical signs of hysteria in general which distinguish it, may be discovered by the fact that in hysteria the failure of sensation does not involve the skin of the genitals, as

it does in organic lesions producing somewhat similar symptoms. In addition it will be found that in hysteria a V-shaped piece of skin over the sacrum is not anesthetic. Anesthesia of this variety, corresponding in the sensory organs to what we call paraplegia in the motor apparatus, is practically never produced by a cerebral lesion, and, if not hysterical in cause, must be spinal; but it is much more rare than is motor paralysis in these parts from lesions in the spine. When it does ensue from spinal causes motor paralysis will in the great majority of cases be found associated with it, at least to some extent. To express it concisely, the characteristic of a typical spinal anesthesia is that it is bilateral and usually involves both sides quite symmetrically; that motor paralysis is generally associated with it; that the reflexes are greatly perverted; and that trophic changes may be present as a result of an involvement of the trophic cells in the anterior cornua coincidently with the disease of the sensory parts of the cord.

The diseased conditions of the cord which result in symmetrical anesthesia of the skin of the legs and trunk are, first and most prominent, locomotor ataxia; second, myelitis; hemorrhages, tumor of the cord or its membranes, meningitis, or injuries which cause pressure on the sensory tracts by producing fracture of the vertebræ or dislocation. Very rarely, however, a lesion of the pons may so result.

Anesthesia of the lower portions of the body and legs occurs in the later stages of locomotor ataxia, and is usually preceded by forms of paresthesia. (See Paresthesia.) The anesthetic areas are most marked in the soles of the feet and about the malleoli, according to Belmont. In other words, blunting of sensibility is seen in nearly all cases of tabes dorsalis late in the disease. In some cases the sense of touch is preserved and the sense of pain lost (analgesia), while in others the opposite condition is present. Again, we find loss of tactile sense and of pain sense without loss of heat and cold sense, and *vice versa*. A very characteristic sensory symptom of tabes is the delay in the recognition of an irritation of the sensory nerves, so that if the patient be blindfolded and then pricked with a pin he will not make an exclamation or draw his foot away for several seconds. In other instances the patient complains of repeated pricks when only one has been given, or, when asked the number of points pricking him, states that there are four or five instead of the one really present. If, in addition to these sensory disturbances, we find Romberg's symptom (see Legs), Argyll-Robertson pupils (see Eye), and loss of patellar reflex (see Reflexes), and a number of other diagnostic peculiarities of tabes, the decision as to the cause of the anesthesia is easily made.

An important early diagnostic sign of locomotor ataxia is the

development of areas, unilateral or bilateral, of diminished sensibility. This is particularly apt to be found in the areas supplied by the mid-dorsal nerves (level of fourth intercostal space). They are very constant symptoms. When the disease is advanced the anesthesia extends down the inside of the arms and forearms. The sense should be tested by a warm finger tip applied to the skin in a very gentle manner.

Slight anesthesia, retardation of the transmission of sensory impulses from the skin, and perversion of temperature sense may be rarely developed late in the course of Friedreich's ataxia.

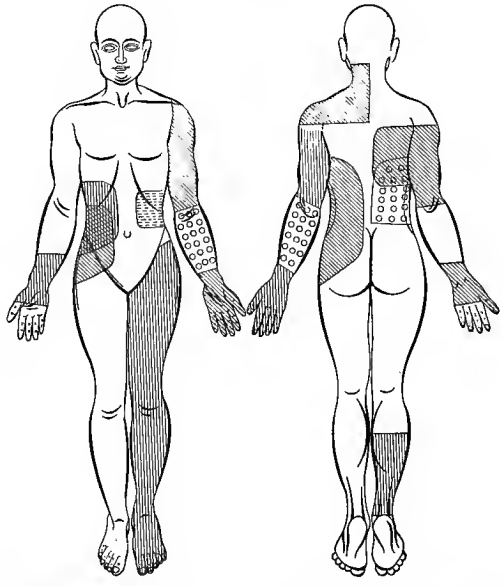
Bilateral anesthesia of the character just discussed, as caused by locomotor ataxia, may also occur as a result of acute or chronic myelitis. The first change under these circumstances is a mere obtunding of sensitiveness, which gradually deepens until loss of pain sense, pressure sense, and, lastly, complete anesthesia is developed. The development of these symptoms indicates involvement of the posterior columns. Loss of reflex activity in the legs is developed in direct proportion to the destruction of the motor and sensory nerve tracts in the cord. The predominance of motor paralysis, the fact that the lower limbs are both involved, and the absence of the characteristic symptoms of locomotor ataxia all tend to make the diagnosis certain, while the absence of the pains of tabes and of the other signs of that disease still further excludes its presence from the case. Further than this, the myelitis creeps up the cord, involving new areas, and new parts of the skin become anesthetic. An important point, too, in regard to the anesthesia of acute myelitis is this, namely, that while in the upper extremities the loss of sensation and motion is associated, so that both functions are lost in the same area, in the lower extremities these two functions are not lost in the same areas. Thus, myelitis of the lumbar enlargement in its lower part is accompanied by anesthesia of the gluteal area and motor paralysis of the anal muscles; and, again, anesthesia of the gluteal region, the back of the thigh, and the back of the calf is associated with loss of power in the muscles that move the foot, while in lesions of the upper part of the lumbar segment the anesthesia involves the thigh, the inner side of the leg, and the foot, in association with paralysis of the quadriceps extensor and deeper muscles of the thigh. (See chapter on the Feet and Legs, part on Myelitis.)

The development of sudden bilateral anesthesia, which is accompanied by severe pains of a tearing or burning character, creeping rapidly up the body, is indicative of acute hemorrhage into the spinal membranes, or it may be due to that very rare lesion, hemorrhage in the cord. In either case motor paralysis is present. Anesthesia, or the milder perversions of normal sensibility of the skin,

may be present in cases of compression of the cord by caries, and by spinal curvature, tumors, or aneurysms producing erosion. Sometimes, while tactile anesthesia is complete in these cases, severe pain is constantly suffered (anesthesia dolorosa), and this is often the case, according to Wood, in cancer of the spine.

Partial anesthesia of the skin of the trunk and arms of a bilateral character, associated with progressive muscular atrophy, scoliosis, and trophic lesions in the skin, points strongly to syringomyelia. The loss of pain and temperature sense is usually the first symptom. The areas of anesthesia are best shown in Fig. 47.

FIG. 47



Sensory chart, showing areas of . *Thermo-Anaesthesia* *Analgesia* *Thermo-Anaesthesia and Analgesia* *Tactile Anaesthesia*, and areas in which the patient's answer to tests of temperature showed reversal *Cold-Hot; Hot-Cold.* in a case of syringomyelia. (Dercum.)

Localization of the Spinal Lesion.—Having considered the general spinal causes of anesthesia of the skin, it yet remains to determine what part of the cord is involved by the pathological process; and this is, fortunately, possible, chiefly through the very accurate and noteworthy studies of M. Allen Starr, Thorburn, and Head, not to mention collateral ones of great value by Horsley and many others; but the field is only partly covered, and some of our uncertainties depend upon lack of knowledge as to the course of the sensory fibers in the cord.

Roughly, we may state that disease of the cervical cord generally produces disturbances of sensation in the arms, hands, and fingers; disease of the dorsal cord, disturbances in the sensation of the back and trunk, which may radiate into the thighs; and disease of the lumbar cord gives rise to these symptoms in the legs and feet.

Again, it is to be remembered that, as a rule, in a transverse lesion of the spinal cord the anesthesia begins at a level which is three or four inches below the lesion in the cord (Horsley and Gowers); this being due, as proved by Sherrington, to the fact that

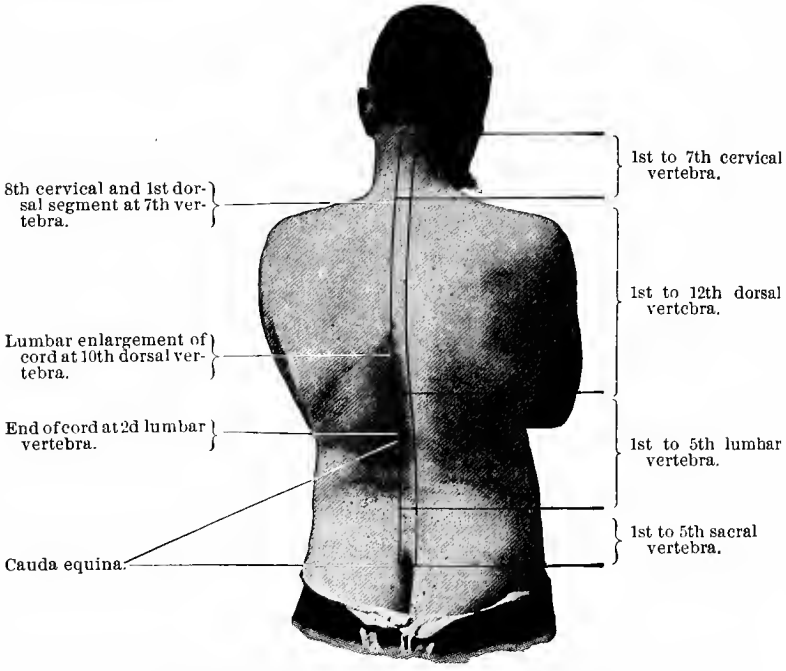


FIG. 48.—Showing the surface areas of the back corresponding approximately to the areas of the spinal cord supplying the trunk and limbs.

each area of skin is supplied by three nerve roots whose peripheral filaments overlap one another.

For the ready study of the subject the cord has been separated into segments corresponding with the vertebræ covering it. The areas of anesthesia produced by spinal injury or disease are best described by Starr's well-known article and diagrams, from which we quote. In this connection the reader should refer to the tables on pages 82 and 83, showing the localization of the functions of the segments of the spinal cord. (See chapter on the Legs and Feet.)

The anesthetic areas included in zones I and II in Fig 49 are due to a lesion involving the conus medullaris and the fourth and fifth sacral segments of the cord. These zones include the peritoneum, the posterior part of the scrotum in males, the vagina in females, and the mucous membrane of the rectum. Anesthesia in zone III is due to lesion of the third, fourth, and fifth sacral segments, and includes a large part of the buttock and the upper part of the thigh, posteriorly, in a triangular space. Zone IV is practically an enlargement of zone III in every direction, particularly toward

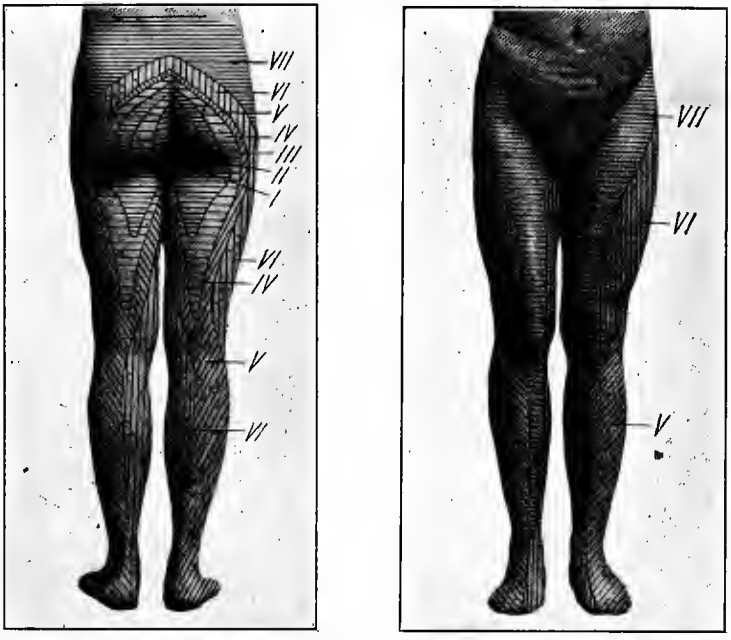


FIG. 49.—Areas of anestbesia in lesions at various levels of the spinal cord from sacral V to lumbar II. (After Starr.)

I. Sacral V.
II. Sacral IV.
III. Sacral III.
IV. Sacral I.
V. Lumbar V.
VI. Lumbar III.
VII. Lumbar II.

the popliteal spaces, and is probably due to a lesion in the first and second sacral segments; but this needs confirmation by autopsy, as Starr points out. Zone V includes all the first four zones just named, and extends down through the popliteal space in a band-like shape; after it passes this space it descends the outer side of the leg and foot, sometimes ending at the ankle, sometimes at the sole or the three outer toes and half the next toe. Such an area indicates a lesion involving all the segments of the sacral cord, and extending into the lumbar cord to the fifth lumbar segment. Zone

VI is caused by a lesion extending to the third lumbar segment, and when it is present the anesthesia covers the back of the thighs and legs and also the front of the thighs, except in an area which extends from above downward along the shin, sometimes to the foot, as in Fig. 49. If the foot is involved, the lesion in the lumbar cord is probably above the third lumbar segment. Zone VII, which is larger than all, follows a lesion in one of the four lumbar segments—

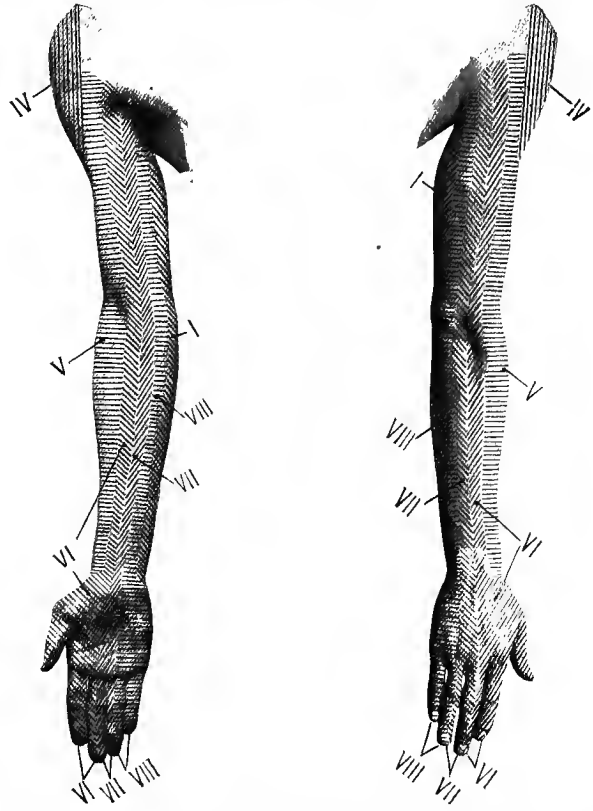


FIG. 50.—Areas of anesthesia from lesions at various levels of the spinal cord from the second dorsal to the fifth cervical. (After Starr.)

that is, all but the first. The line of anesthesia, Starr tells us, is lower in front than behind. When the abdominal wall is involved in the anesthesia the first lumbar segment is probably diseased.

The area of the anesthesia from the level of the abdomen corresponds very closely to the levels in the cord if we allow for the space already mentioned, of two to three inches for the interlacing anastomosis of the nerve fibers of the posterior roots.

They are about as follows, according to Thorburn: Wehn the

anesthesia is as high as the anterior inferior spine of the ilium, the lesion is at the twelfth dorsal vertebra; if at the umbilicus, at the eleventh and twelfth dorsal vertebræ; if up to the lowest floating rib, the whole eleventh dorsal vertebra; if from one to four inches above the umbilicus, the ninth and tenth dorsal, and perhaps part of the eighth dorsal vertebra; if as high as the nipples, the fourth dorsal vertebra; and if to the third rib, the lesion is as high as the second dorsal vertebra.

Starr has also given us, in another paper than that already quoted, equally good ideas of the areas of anesthesia occurring above those

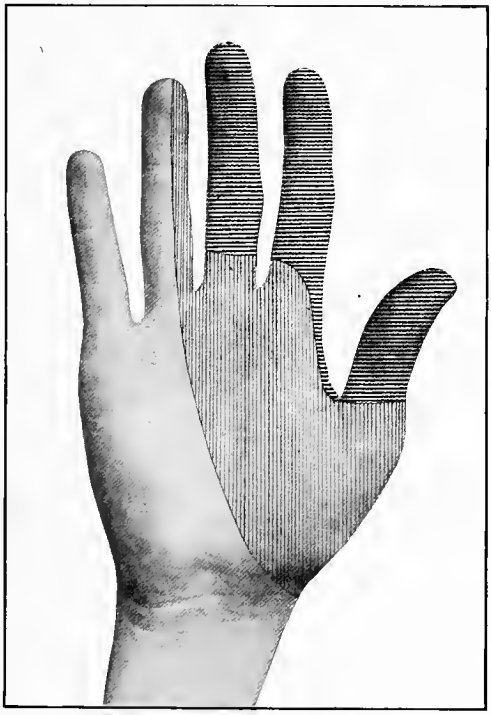


FIG. 51.—Area of anesthesia from injury of the median nerve. Palmar surface.

just described (Fig. 50). When the anesthesia extends to the arms and is found upon the inner side of the arm and forearm, reaching to the wrist, but not to the hand, and also involves a small zone on the extensor and flexor surfaces of the arm and forearm, the second dorsal region is the site of the lesion. If the anesthetic area includes the ulnar side of the hand, the palmar and dorsal surfaces of the same, and the little finger, and extends in a narrow strip up to the axilla on both the anterior and posterior surfaces of

the arm and forearm, the lesion is probably at the level of the eighth cervical vertebra. When the zone involved extends to the middle of the central figure on the palmar and dorsal aspects, and runs up the centre of the forearm and arm, the seventh cervical area is diseased. Again, when the remaining skin of the hand up to the wrist and a narrow strip of skin up the forearm and arm on both surfaces to the axilla is affected, the lesion is at the sixth cervical vertebra, while anesthesia of the forearm and arm on the outer surface as high as the deltoid insertion indicate the fifth cervical vertebral area

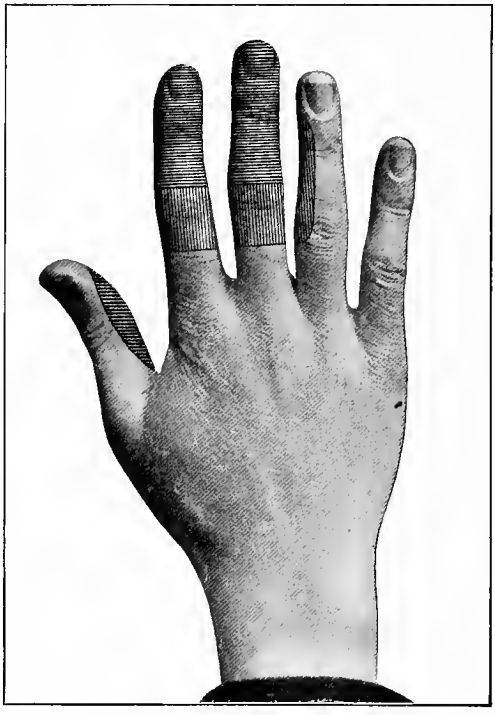
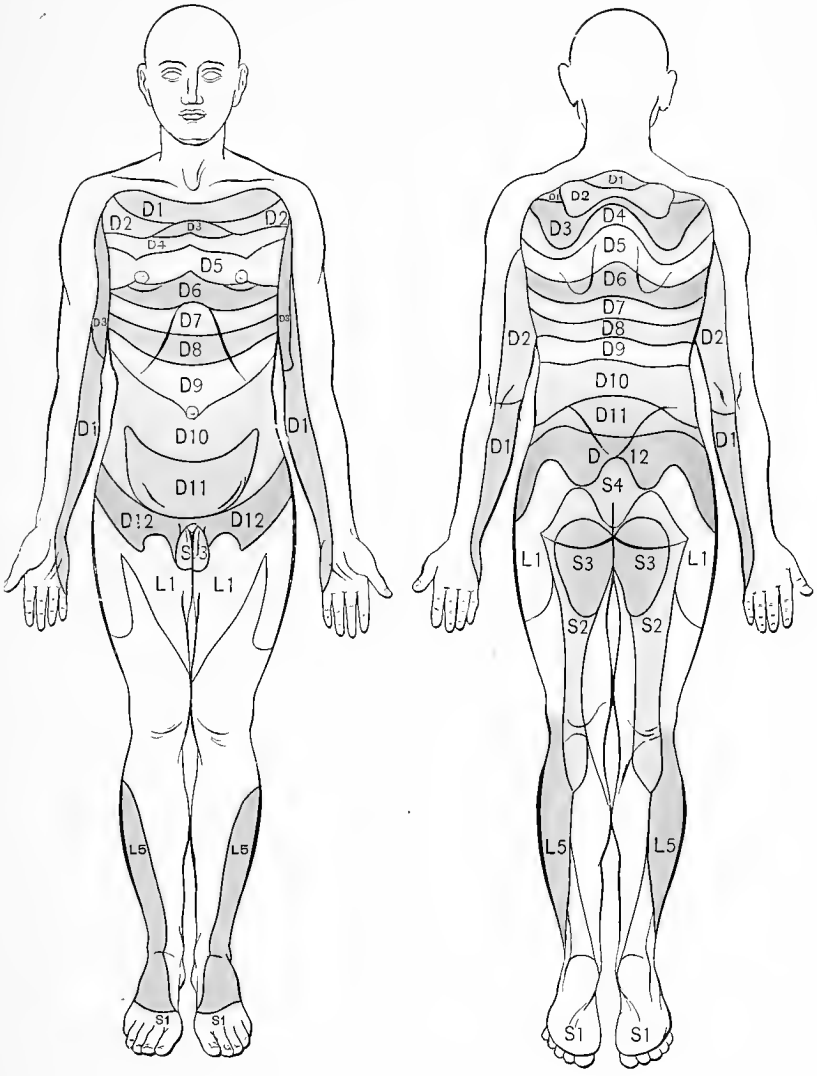


FIG. 52.—Area of anesthesia from injury of the median nerve. Dorsal surface.

in trouble. Lesions higher than this usually produce death before it is possible to test sensibility.

Neuritis as a Cause of Anesthesia.—Anesthesia of the skin in any part of the body may be due not only to cerebral or spinal lesions, but also to neuritis or inflammation of the nerve trunk, or to some injury which impairs its functional activity by pressure, bruising, or cutting. As a rule, loss of sensation from neuritis occurs late in the disease, hyperesthesia of paresthesia being the earlier manifestations; but in some cases these are absent, and anesthesia begins at once.

PLATE VI.



Cervical Roots are represented by the letter C, Dorsal Roots by the letter D, and Lumbar Roots by the letter L. (Chart after Head.)

The characteristic of such an anesthesia is that it is confined to the area supplied by the affected nerve, although the presence of a multiple neuritis may produce such a universal anesthesia by involving all the nerves that this sign is masked. While a mono-anesthesia may be due to other causes, it is in the great majority of cases due to neuritis. The signs of an anesthesia due to neuritis are loss of motion and sensation, tenderness on pressure over the nerve trunks supplying the affected area, trophic changes in the tissues of the part, with the development of reactions of degeneration and pain in the involved

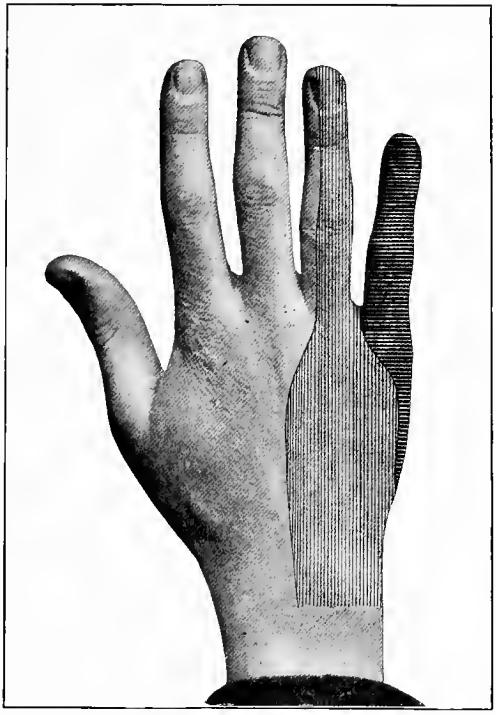


FIG. 53.—Area of anesthesia in injury of the ulnar nerve. Dorsal surface.

nerves or parts supplied by them. Somewhat similar symptoms occur in anterior poliomyelitis, but pain is not commonly present in this disease, and there is no anesthesia, either in children or adults. (See chapters on the Hands and Arms, and on the Feet and Legs.)

Toxic peripheral neuritis producing anesthesia may arise from poisoning by arsenic, lead, alcohol, or mercury, from septic states of the body, and from the infectious diseases, particularly diphtheria, influenza, and typhoid fever.

That due to the mineral poisons has in each case certain differen-

tial points of importance. The anesthesia of arsenical poisoning is more marked than in lead poisoning, in which condition it is often almost absent, and the lower extremities are very apt to be involved, whereas in lead poisoning, as is well known, the nerves of the arm are particularly susceptible. (See chapter on the Arms and Hands.) Arsenical neuritis may also produce pigmentation of the skin. In alcoholic neuritis the temperature of the anesthetic areas is often subnormal and there are nearly always mental disturbances represented by delusions. In mercurial poisoning, shaking like paralysis

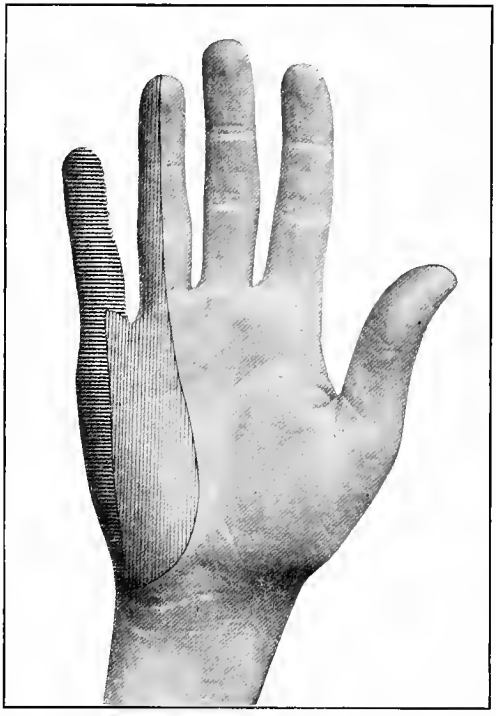


FIG. 54.—Area of anesthesia from injury of the ulnar nerve. Palmar surface.

agitans may be present. An analysis of the motor symptoms in all these cases is important, and the discovery of any one of these poisons in the urine, with the history of the patient, generally makes the diagnosis possible.

Diphtheritic neuritis is quite common, and in 50 per cent. of the cases in which it occurs sensibility is lost or disturbed in the areas supplied by the involved nerves.

Great care is needed in all cases of neuritis lest the mistake be made of diagnosticating the condition as one of locomotor ataxia, when in reality it is pseudotabes.

It has already been stated that in neuritis the area of anesthesia is that supplied by the affected nerve. For this reason we can determine what nerve trunk is affected by studying the area of anesthesia, always remembering, however, that the sensory fibers of the nerves, particularly in the hands and feet, anastomose so freely with those of adjacent nerves that the area of the anesthesia may not be exactly

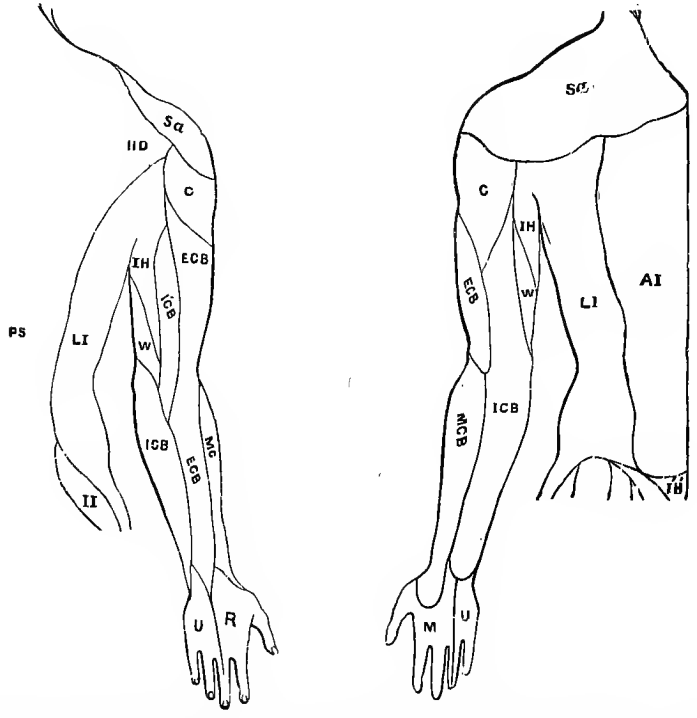


FIG. 55.—Cutaneous nerve supply of the trunk and upper extremity. (Fowler.) SA. Supraclavicular nerve. IID. Second dorsal. PS. Posterior branches of the spinal nerves. LI. Lateral branches of the intercostal nerves. AI. Anterior branches of the intercostal nerves. C. Circumflex nerve. IH. Intercostal humeral. W. Nerve of Wrisberg. ICB. Internal cutaneous branch of musculospiral nerve. ECB. External cutaneous branch of musculospiral nerve. ICB. Internal cutaneous nerve. MC. Musculocutaneous nerve. R. Radial nerve. U. Ulnar nerve. M. Median nerve.

that supplied by the nerve involved; or, in other words, the presence of loss of power in a region supplied in health by a nerve which has been divided is constant, but very often sensation is not disturbed, even though the divided nerve be the sensory as well as the motor supply to the part.

It is well to remember also that sensory disturbances of the skin following injuries of nerves are often not nearly so great as the

motor disturbance, even where there is no sensory transmission by anastomosis, and where they are present they usually disappear, more rapidly than the motor loss, as recovery takes place.

The following facts are, therefore, of diagnostic interest. If the anesthesia is found to be due to a neuritis and to involve the palmar surface of the thumb, fore and middle fingers, the median nerve

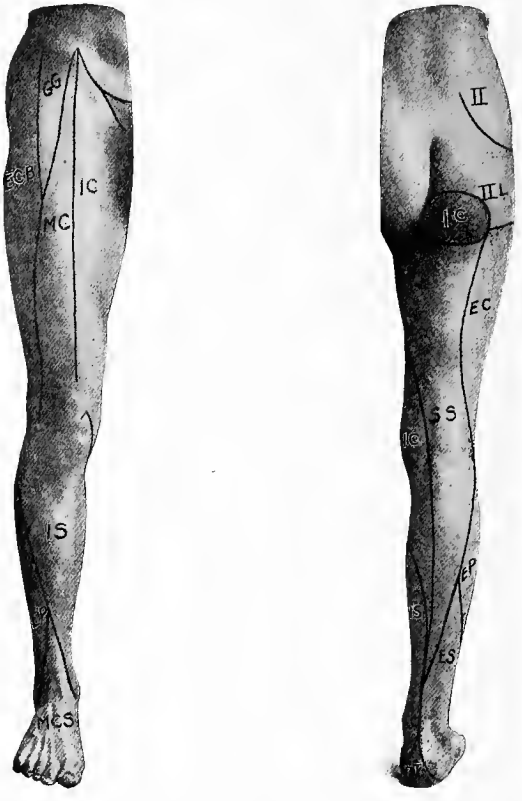
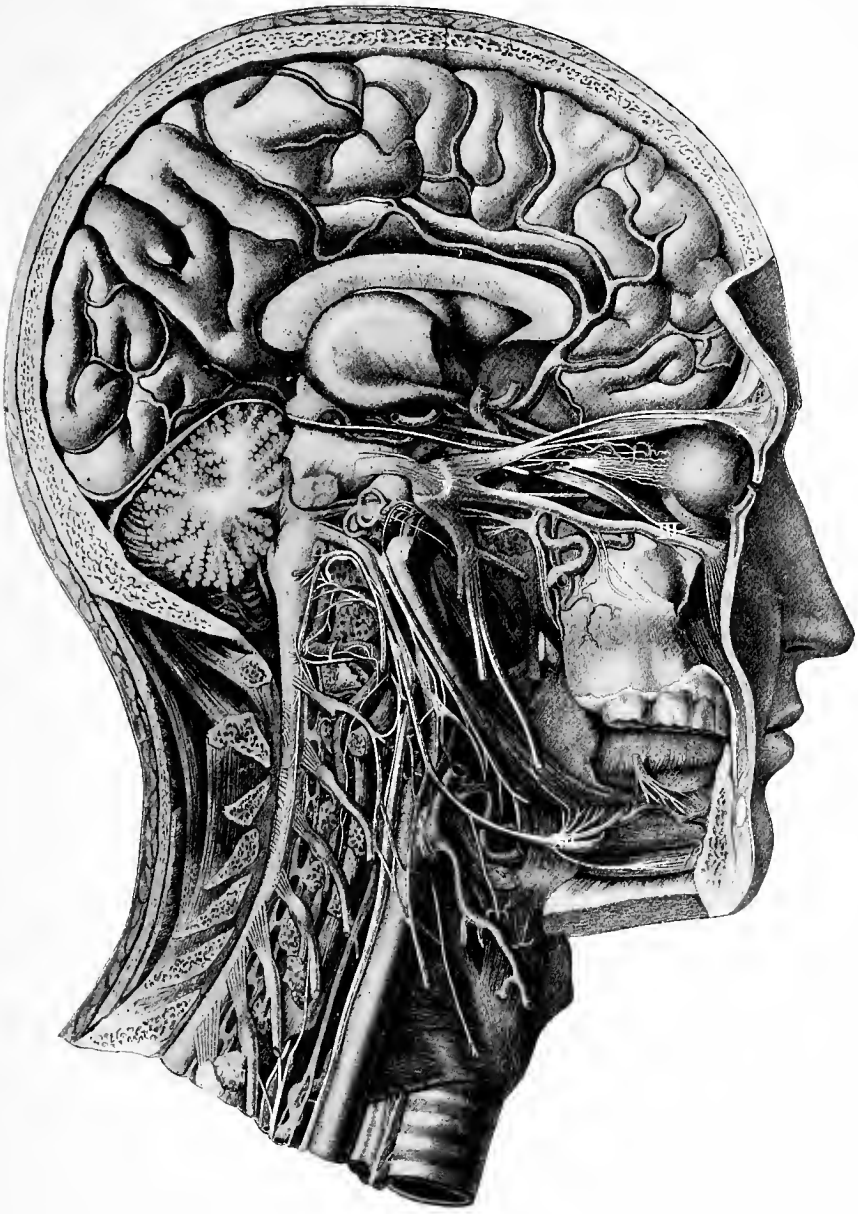


FIG. 56.—Cutaneous nerve supply of the lower extremity. II. Ilio-inguinal. IIL. Second lumbar nerve. GG. Genitocrural. EC. External cutaneous. MC. Middle cutaneous. IC. Internal cutaneous. IS. Internal saphenous. SS. Small sciatic. EP. Branches from external popliteal. ES. External saphenous. MCS. Musculocutaneous. PT. Branches of posterior tibial. (Modified from Gerrish.)

is probably the one at fault (Figs. 51 and 52), and the area may even include in rare instances the backs of these fingers at their bases and the half of the third finger nearest the thumb. When there is disturbance of sensation in the ulnar side of the ring finger

PLATE VII.



Showing the Distribution of the Cranial Nerves, particularly the Fifth.
(Modified from Arnold.)

III. Branch of oculomotor to inferior oblique. V. The Gasserian ganglion, composed of the fibers of the sensory root of the nerve. (The plate would seem to indicate that the three branches of the nerve arise from this ganglion, but they do not, for the motor fibers do not enter the ganglion, but join the sensory fibers in the third branch, after they leave the ganglion.) XII. The hypoglossal nerve.

and in the skin of the little finger, there may be ulnar neuritis (Figs. 53 and 54). (See also chapter on the Hands.) The nerve supply of the skin of the entire upper extremity is well seen in Fig. 55.

The development of sensory disturbances in the feet, resulting from neuritis, is as follows: When there is perverted sensation of the inner side of the foot from the tip of the big toe to the heel, and thence up the inside of the calf to the knee, the nerve involved is the long or internal saphenous. When the dorsal surface of the foot has its cutaneous sense disturbed the nerve involved is the musculocutaneous, a branch of the external popliteal. Disturbance of sensation on the outer side of the foot and calf indicates failure of function in the external saphenous, which is composed of the cutaneous branches of the external and internal popliteal nerves. Disturbed sensation on the posterior surface of the calf also indicates trouble in the external saphenous nerve and communicans peronei, while when the sensation of the skin of the heel is disturbed the plantar cutaneous nerve, a branch of the posterior tibial, is involved (Fig. 56).

In the skin of the thigh the anterior surface is supplied by the middle cutaneous nerve, which is a branch of the anterior crural; on the inner side by the internal cutaneous, also a branch of the anterior crural; and on the outer side by the external cutaneous, which arises from the second and third lumbar nerves. Laterally the external cutaneous gives the supply. Posteriorly the small sciatic gives the nerve supply to the skin.

Anesthesia of the greater portion of the skin of the thigh, except in a narrow strip on the back part and in the area supplied by the internal saphenous nerve, often occurs as the result of paralysis of the anterior crural nerve, arising from pelvic tumors, psoas abscess, and vertebral disease.

Facial Anesthesia and its diagnostic meaning are still to be considered. When it occurs it indicates that the fifth nerve, or its nucleus, is involved.

If the area be that of the forehead, the upper eyelid, the conjunctiva, and the nostril, the ophthalmic branch of the fifth nerve is at fault, and the lesion is probably at the sphenoidal fissure or within the orbit, and reflex winking of the eye no longer takes place because the conjunctiva is anesthetic.

If the skin of the upper part of the face is anesthetic, the superior maxillary branch is involved; and if the skin of the temporal region and that of the jaw and the under lip are anesthetic, the inferior maxillary branch is diseased. When both of these branches are paralyzed there is probably a tumor of the superior maxillary bone; and if the entire area of the three branches is anesthetic, the Gasserian ganglion may be the part affected, and this will be accom-

panied by trophic changes in the anesthetic parts. The most common cause of anesthesia of the trifacial is, however, neuritis.

Romberg makes the following differential statement:

(*a*) The more the anesthesia is confined to single filaments of the trigeminus, the more peripheral the seat of the cause will be found to be.

(*b*) If the loss of sensation affects a portion of the facial surface, together with the corresponding faucial membrane, the disease may be assumed to involve the sensory fibers of the fifth pair before they separate to be distributed to their respective destinations; in other words, a main division must be affected before or after its passage through the cranium.

(*c*) When the entire sensory tract of the fifth nerve has lost its power, and there are at the same time derangements of the nutritive functions in the affected parts, the Gasserian ganglion, or the nerve in its immediate vicinity, is the seat of the disease.

(*d*) If the anesthesia of the fifth nerve is complicated with disturbed functions of adjacent cerebral nerves, it may be assumed that the cause is seated at the base of the brain.

Other Disturbances of Sensation than Anesthesia.—The other disturbances of sensation of the skin than anesthesia, which are usually subjective rather than objective, are paresthesia, hyperesthesia, and analgesia.

Paresthesia—numbness, tingling, or burning—is seen in nearly all cases in which anesthesia ultimately develops as a result of organic lesions. When a patient complains that he cannot feel the contact of clothing about his feet and legs, or that the feet when he walks feel as if wrapped in some thick material, or as if he were walking on moss, or that the soles of his feet feel as if they were numb and at the same time tickled by ants walking over them, the characteristic sensory disturbance of the skin seen in locomotor ataxia is present.

Often there is tingling or numbness of the fingers, particularly of the ring and little fingers, and a sensation as if a girdle were about the patient is common. These are the subjective disturbances of sensation in tabes dorsalis, and, as they are often the earliest manifestations of the disease, possess great diagnostic importance. The objective sensory perversions consist in the discovery by the physician, when studying the sensibility of the skin, of areas of anesthesia, analgesia, and hyperesthesia which are usually bilateral. Belmont has stated that we also find these areas in spinal syphilis, either on one or both sides. Numbness, tingling, and formications affecting the skin are also often early symptoms of brain tumor in the area supplying the affected part, and this possibility is increased if there is associated spasm. The actual objective sensibility of the

skin may be preserved for some time after these symptoms appear, or it may be impaired almost at the outset, owing to the involvement of all or part of the sensory tracts in the cord. Similar symptoms are often seen in the early stages of myelitis. They are very frequently seen after injuries to nerves, and severe tingling in its acute variety occurs when the "funny bone" of the elbow is knocked against an object, owing to bruising the nerve. It is also seen in cases of aconite poisoning, and when the hands have been exposed to carbolic acid. Paresthesias are also frequently seen in cases of neurasthenia.

Perversions of sensation in the skin sometimes take a curious form, as, for example, that known as *allochiria*, in which a sensory impulse in one hand is referred by the patient to the opposite hand. This is seen in tabes dorsalis, myelitis, multiple sclerosis, and hysteria. In other cases, as in paralysis agitans, this perversion takes place in the form of failure to distinguish heat and cold, and subjective sensations of extreme heat are felt. The part affected may actually have its temperature raised several degrees.

Magnan asserts that a sensation as if a worm or bug were crawling under the skin is indicative of cocaine intoxication.

Very closely associated with the numbness of hysteria or neurasthenia, and lying between functional and organic disease of the nerves, is that condition called *acroparesthesia* or waking numbness. This state is usually seen in women past middle life, but may occur in men. On waking in the morning marked formication and numbness of the fingers are present, which usually pass off as the day progresses, but as the condition becomes more marked they may last all day. While there is no anesthesia, strictly speaking, the disturbed sense of touch renders sewing or performing any small act with the fingers almost impossible. These sensations may be confined to the area of one nerve, as the ulnar, or involve all the skin of the hands, or more rarely of the feet. General nervous excitability is usually associated with the local manifestations. Sometimes the scalp may be the area involved.

Acroparesthesia is to be separated from the sensory disturbances of hysteria by its irregular outline, for generally in the latter disease the areas are distinctly outlined, by the fact that the hysterical condition is usually unilateral, and by the absence of the characteristic general hysterical symptoms. From organic disease it is separated by the absence of the signs of neuritis about to be described, and by the absence of tenderness, pain, and loss of power. From cerebral or spinal disease it is separated by the absence of symptoms produced by lesions in these parts, and by the facts that in both these lesions there is paralysis of motion in association with the sensory disturbance, and in the case of spinal lesions the symptoms

are usually in the legs, while acroparesthesia generally manifests itself in the hands.

Closely associated with paresthesia, if not an actual form of it, is the "girdle sensation;" that is, the patient feels as if a tight belt were strapped around a limb or the trunk. This is seen as a prominent symptom in locomotor ataxia, myelitis, and tumors of the cord or its envelopes. When the lesion is in the lower cervical or dorsal region the sensation is in the chest or abdomen; but this relationship between the growth and the sensation of constriction is not always constant. (See chapter on the Feet and Legs.)

Hyperesthesia of the skin is an important symptom of both hysteria and neurasthenia, and its discovery in association with the peculiar symptoms which occur in the former morbid state confirm a diagnosis most positively. The most important and curious of these hyperesthesias are the so-called hysterogenous zones, or, in other words, areas involving the skin and subcutaneous parts, which possess great sensitiveness, and which, when pressed upon, cause in many cases convulsive seizures of the hysterical type. Not only is this true, but in addition it is a noteworthy fact that after the nervous disturbance produced by this means is set in motion, a second pressure on the hysterogenous zone may arrest the seizure. These zones commonly exist over the ovaries, in the groin, about the periphery of the mammary glands, or upon the spine in the lumbar or dorsal region. (See chapter on Pain.)

The hyperesthesia due to neurasthenia is to a great extent spinal in character, but the skin of the rest of the back, particularly over the great muscles on each side of the spine, may also be involved. Often the neurasthenic patient or one who has phosphaturia will complain that in brushing or combing the hair pain or extreme sensitiveness is developed upon the scalp, and there may be tender areas on the chest. These areas in neurasthenics can hardly be confused, even by the careless, with the hyperesthetic zones of hysteria, and the personal history and characteristics of the individual aid still further in separating the two conditions.

Hyperesthesia of the skin, aside from that seen in hysteria and neurasthenia, occurs in peripheral neuritis and locomotor ataxia, the skin of the back being particularly tender in the latter disease, and the excessive sensitiveness is frequently seen in a zone extending a little above the anesthetic areas of transverse myelitis, this hyperesthetic area being soon rendered anesthetic by the progress of the disease. Hyperesthesia in the skin of the limbs is also rarely seen in myelitis, and when there is motor paralysis of one side and sensory paralysis of the other it is commonly found on the side on which motion is lost. A condition of excessive dermal hyperesthesia is also present in cerebrospinal meningitis, in which disease it is

often a very early symptom. It usually appears first in the legs, then in the hands and arms, and, finally, the skin of the face and head become involved.

Hyperesthesia of the skin occurs, often associated with skin eruptions, in that very rare condition called chronic leptomeningitis.

Motor symptoms are nearly always present if the cord becomes involved.

Hyperesthesia of the skin is considered by some authors to be, when found in association with other characteristic symptoms, almost pathognomonic of brain tumor. It may be found on the scalp, over a large part of the body, or in the part which is paralyzed. It is also found during the convalescence of typhoid fever and in relapsing fever. It also appears in the paralyzed side of persons suffering from hemiplegia, in the area supplied by a nerve suffering from neuralgia, particularly that of a migraine type, in the scalp of persons suffering from gout, and in the same area in women about the time of the menopause.

General tenderness of the skin or deeper tissues is quite frequently seen in cases of rickets and scurvy, the child crying whenever it is moved, as if sore and tender, and tender spots often appear over the ribs in cases of pleurisy.

Sometimes in a neurotic girl about the time of puberty, or in a woman, one breast becomes exceedingly painful and tender, and the skin of the breast becomes so hyperesthetic that the slightest touch causes pain. The whole breast is, moreover, tender, and movement of the arm may be impossible, owing to pain thereby caused in the gland. This hysterical breast can be separated from the painful breast due to a tumor by the general diffuse character of the swelling, the failure to outline any distinct mass, the neurotic character of the patient and her age.

The hyperesthesia of chronic alcoholism may be both dermal and deep, and is well marked along the course of the peripheral nerves, particularly where they emerge from deeper structures. It is also seen in the neuritis of lead and arsenical poisoning.

Increased sensibility of the skin may follow the use of opium or ergot, and is met with in the course of, or as a sequel of, influenza, and in some cases of profound anemia.

In some cases hyperesthesia is an early sign of the onset of non-tuberculous leprosy, and will generally be found in the course of the ulnar or sciatic nerves in such cases.

A very interesting fact from a physiological and diagnostic point of view is that disease of the internal organs or viscera often produces areas of hyperesthesia or tenderness upon the skin, which may in future be used to aid in the localization of the lesions. This subject has been well studied by Head (*Brain*, 1893 and 1894),

from whose researches much information may be derived, but the results of which will have to be confirmed in many cases before they can be used as diagnostic guides. (See article on Pain.)

Pain in the skin is very various in its manifestations, and nearly always is due to functional nervous troubles. Duhring has noted a boring sensation in some cases. It should direct the physician's attention to the possibility of hysteria or tabes dorsalis.

Pruritus, or intense itching of the skin, may be due to contact with some irritant, but its presence, if persistent, particularly if widespread or near the genitals, should always raise a suspicion of diabetes mellitus, or chronic lead poisoning, or gout, or chronic contracted kidney. Very rarely opium may produce a pruritus, and jaundice is nearly always accompanied by some itching. Pruritus about the anus is often due to piles, gout, and diabetes.

Finally, one important point is to be remembered, viz., we cannot attempt to make a general diagnosis merely from a study of the areas of anesthesia or other perverted sensibility of the skin in any case. The results obtained from studies of the sensation of the skin are only to be used as additions to the motor and other symptoms which will be found discussed under the chapters on the limbs

CHAPTER VII.

THE THORAX AND ITS VISCERA.

The inspection of the normal and abnormal chest—Their topography—Alterations in the shape of the thorax—The rhythm of the respirations—The results of using inspection, palpation, percussion, and auscultation in health and disease—The characteristic signs and symptoms of the various diseases of the thoracic organs.

THE chief contents of the thoracic cavity consist of vital organs, which are, unfortunately, only too often subject to disease. A careful study of the signs associated with the normal functions of these parts is, therefore, of importance, as is also that of the symptoms indicating pathological changes. While it is true that in many instances patients present themselves to the physician with well-marked objective and subjective symptoms pointing to abnormalities in the organs of the chest, it is also a fact that in many others none of these signs exist, or they exist in such an indefinite manner that the physician's attention is not attracted to them, and as a result important thoracic changes from the normal are overlooked or made light of. We base our diagnosis of the character of a case on the changes which we find in the thorax as to its contour and as to its movements, on the respiratory and cardiac sounds, and on the other physical signs about to be described.

The measures used in the physical diagnosis of the diseases of the thoracic organs are Inspection, Palpation, Mensuration, Percussion, and Auscultation.

INSPECTION.

Before we proceed to the study of the alterations produced by disease in this portion of the body, we must have a clear conception of the appearance of the chest in health.

Inspection of the normal chest when free from clothing will reveal the fact that it is conical in form, the broader part of the cone being in the upper portion. Above the clavicles there is usually a slight depression (the supraclavicular fossa), and below the clavicles, which may be somewhat prominent, there is a slight convexity which extends as far down as the fourth rib. This convexity varies considerably according to the muscular development of the individual, the formation of the bony portion of the chest wall, and the deposit

of fat in the subcutaneous tissues of the chest. The nipple is by no means as definite a landmark as is sometimes thought, as its position, in respect to the ribs under it, varies greatly in different individuals; and it is still further altered in its position by the presence of much fat under it, or, again, in multiparous women by the relaxation of the breast. In the average adult male or virgin female the

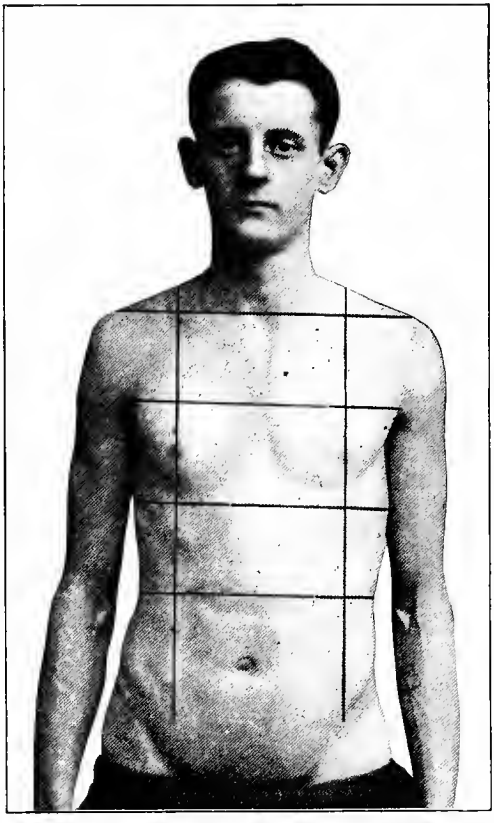


FIG. 57.—The regions of the anterior aspect of the chest. The vertical lines are called the mammillary lines. The upper squares form the superior thoracic area; lower squares form the inferior thoracic area, and the lowest square the epigastric.

nipple is on a level with the fourth rib or fourth interspace. The ribs in a well-developed person are not prominent in the upper two-thirds of the chest, but in the lower third are more readily seen, particularly at the sides, because of their thin covering by muscles and the subcutaneous tissues and the skin. The sternum in front and the spine behind are normally in the middle line. Over the top of the sternum is a depression called the episternal notch.

The result of lateral examination of the normal chest when compared with the front view will show that the anteroposterior diameter is less than the lateral diameter.

The surface of the chest anteriorly, posteriorly, and laterally has been arbitrarily divided by imaginary lines into spaces, as shown in the accompanying figure (Fig. 57). The lines running from the middle of the clavicles downward through the nipple are called the mammillary lines. The parasternal line, not shown in the figure, is a vertical line half-way between the middle of the sternum and the mammillary line; and a line running down the side from

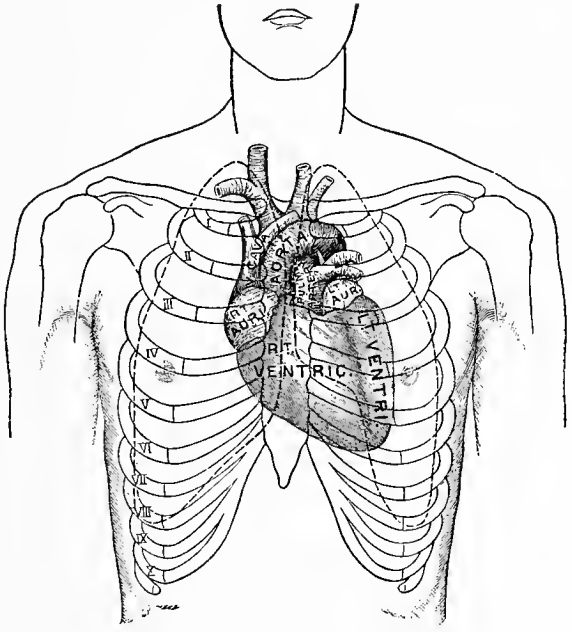


FIG. 58.—Position of heart in relation to ribs and sternum. (Tyson.)

the axilla is called the midaxillary line. These artificial divisions enable us to describe the locality of signs and symptoms.

If we could see through the chest wall, we would find that the lungs extend above the clavicles. Immediately back of the inner end of the left clavicle is the beginning of the innominate vein, and back of this, again, the common carotid artery. On the right side the innominate artery bifurcates just behind the junction of the sternum and clavicle. The figure above shows the relation of the cavities of the heart and its great vessels to the chest wall (Fig. 58).

Anteriorly the lung extends downward as far as the sixth rib on the

right, but the dome of the liver reaches to the level of the fourth interspace. On the left side the lung extends a little lower than on the right side. Laterally the lung on both sides extends to the ninth rib in the midaxillary line. Posteriorly on the right side the lung extends as low as the tenth rib, and on the left side as low as the ninth.

Marked variations in the shape of the chest occur in healthy individuals without possessing any direct pathological significance. Thus, it is very common to see one shoulder slightly higher than the other, and, in the case of clerks or persons who work much at

FIG. 59.—The alar chest of phthisis.

FIG. 60.—Side view of same patient.

a desk, the left shoulder is very apt to be somewhat elevated. Occupations which cause the individual to assume certain positions, or to use certain muscles continually, also cause variations in the contour of the thorax.

Inspection of the Abnormal Chest.—The configurations of the chest which show a tendency to disease or the results of attacks of disease are numerous.

The most familiar of these is the so-called *phthisical chest,* which has been called the "alar chest," because the scapulæ stand out from the back like wings (Fig 59). The anteroposterior diameter, particularly in the upper two-thirds, is very slight, and instead

of convexity of the anterior surface there may be flattening or hollowness (Fig 60). This area scarcely moves on inspiration, but the lower third, which is bulging, moves markedly with the respiratory efforts, as does also the epigastrium. The shoulders are very sloping; the neck, anteriorly, recedes at the episternal notch, but springs forward toward the Adam's apple and the chin. The ribs in the phthisical chest fall downward toward the belly from their points of origin, instead of coming forward in a normal curve (Fig. 61).

If, on the other hand, the chest bulges anteriorly and posteriorly to such an extent that the anteroposterior diameter is greater than,

Fig. 61.—Phthisical chest.

or equal to, the lateral diameter, and if this bulging is fairly uniform, the shoulders being elevated, the back rounded, and the neck short in appearance from the raised shoulders, the patient is probably a sufferer from *emphysema* of the lungs. This chest is often called the "barrel-shaped chest." (See Fig. 63.) The chest wall moves very little or not at all with the respiratory movements, which are chiefly diaphragmatic.

Bulging of the chest wall results, in its most diffused type, from the presence of *chronic pleural effusion* and *pneumothorax;* bulging of a limited area also arises from *cardiac hypertrophy,* particularly

that occurring in childhood; from *aortic aneurysm,* causing bulging by pressure (Figs. 62 and 63); from *pericardial effusion;* and, finally,

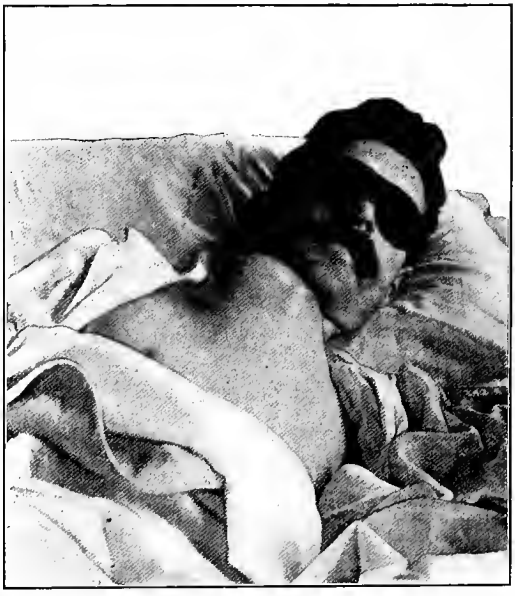


FIG. 62.—Aneurysm of ascending arch of aorta.

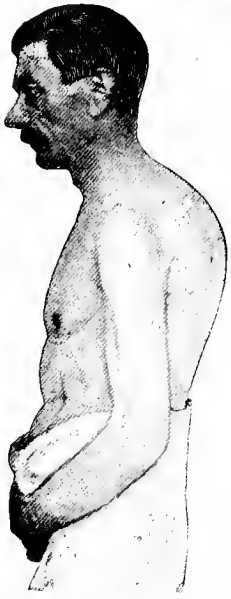


FIG. 63.—Emphysema of the lungs. Shows barrel-shaped chest.

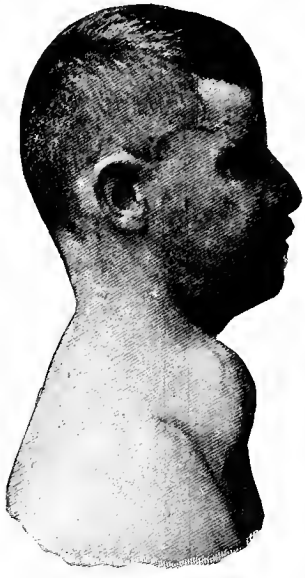
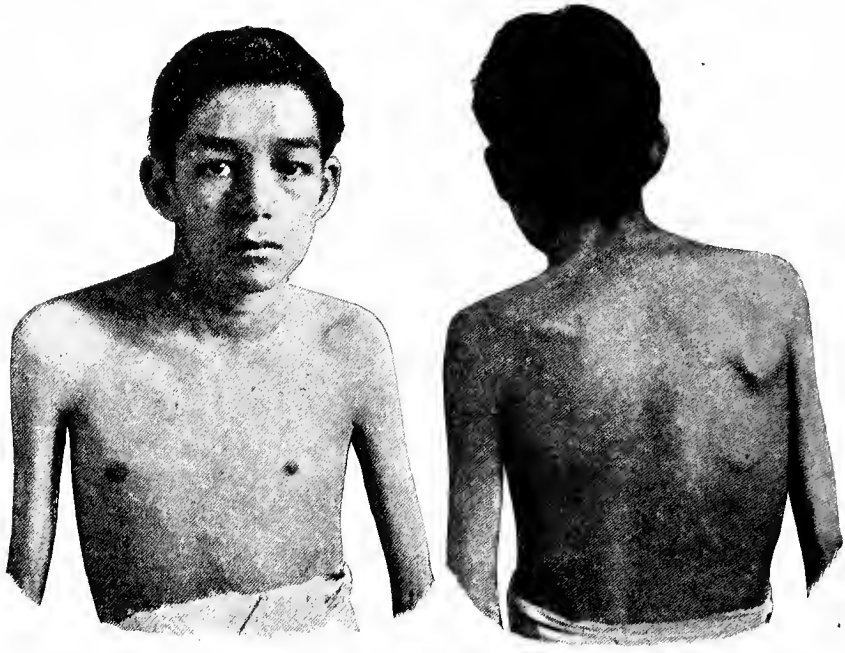


FIG. 64.—Bulging of the chest wall, with erosion of ribs, from aortic and innominate aneurysm.

FIG. 65.—Rachitic rosary. (Barbour.)

from *mediastinal growths*. Marked bulging over the lower part of the chest on the right side should cause us to look for some *hepatic affection* as well as to examine for pleural effusion, and, if the bulging is low down on the left side, to examine for pleural effusion or enlargement of the spleen.

Bulging or protrusion of the sternum and the cartilaginous portions of the ribs attached to it is called "pigeon breast," and is due either to *rickets* or to the presence of some obstruction to respiration of a more or less chronic character during the time the chest



FIGS. 66 and 67.—Showing shrinkage and partial collapse of left side of chest and distortion of spinal column due to chronic tuberculous pleurisy in a boy of fifteen years. (From the author's wards in the Jefferson Medical College Hospital.)

wall was soft and capable of being moulded. Sometimes on each side of the sternum, over the costal cartilages, there is seen a groove or depression as the result of rickets. In other cases a depression or groove extends from the ensiform cartilage backward on either side toward the spine. This is called "Harrison's groove," and is developed in children with poor bony systems, as the result of repeated attacks of asthma or other obstructive respiratory difficulty.

When examining the chests of children the physician will often notice swellings of the tissues at the costocartilaginous junctions, which look and feel to the touch like large beads under the skin.

These beaded ribs are indicative of *rickets*, and are a manifestation of the general tendency to epiphyseal enlargement. This beading is usually most marked on the lower ribs (Fig. 65).

Finally, unilateral bulging of the chest may be due to curvature of the spine, which part of the body should always be examined before a diagnosis as to deformity of the chest is attempted.

Shrinkage of the chest in one part may be due to the contraction of old pleural adhesions (Figs. 66 and 67). It is sometimes seen

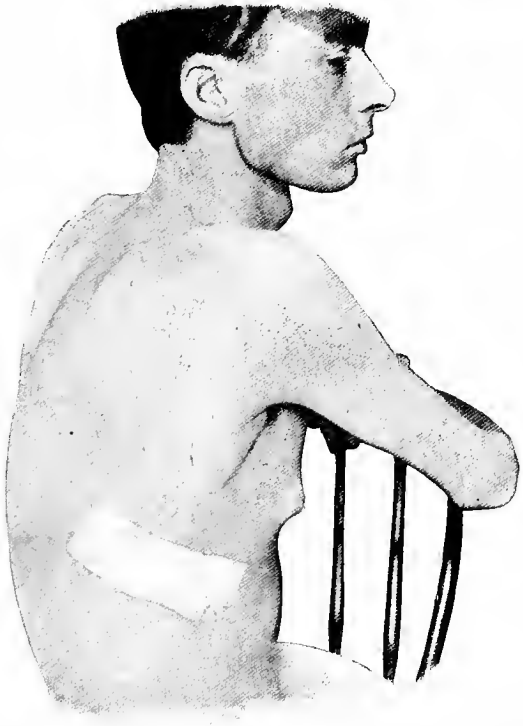


FIG. 68.—A case of empyema of the right side, showing obliteration of the intercostal spaces and hypertrophy of the mammary gland. (From the author's wards.)

over the diseased area in pulmonary tuberculosis, and may be apparently present, but in reality due to wasting of the tissues covering the part.

Marked enlargement of the mammary gland on the affected side is sometimes seen in pulmonary tuberculosis, particularly in males (Fig. 68).

The shape and surface of the chest having been studied, we can go farther and learn much from its movements in respiration: first, from the rapidity of respiration; second, from the respiratory rhythm;

third, from the character of the breathing; and, fourth, from the movements of the ribs.

When counting the respirations the physician should always endeavor to do so without letting the patient know what he is doing, since it is difficult for many persons not to control their breathing when their attention is called to it. Generally the eye can detect the frequency of the breathing by simply watching the movement of the chest, or the information can be gained by resting the hand on the abdomen or thorax, while the wrist is also held and the doctor is apparently taking the pulse. In the newly born child in perfect health the respirations are often as high as 44, but in the adult male at rest they are usually about 14 to 16 per minute. During sleep the number may fall to 8 or 10. The ratio of pulse to respiration is usually 4 to 1, but in rare instances in disease it may be 1 to 1.

Rapid respirations not due to any recent sudden exertion are nearly always indicative of respiratory trouble, primary or secondary. If the primary trouble be in the lung, it will probably be due to croupous pneumonia, catarrhal pneumonia, severe bronchitis, asthma, tuberculosis, pulmonary abscess, or tumors of the lungs. If it be due to secondary lesions in the lung, it may rise from pulmonary edema due to nephritis, from congestion or hypostatic exudation as the result of a weak heart, from pulmonary embolism, from a pleural effusion which seriously interferes with the action of the lung or lungs, from growths in the mediastinum pressing upon bloodvessels and so causing exudation into the lungs or pleura, and from ascites or abdominal growths pressing upon the diaphragm. Usually in these states the respirations will be not only more rapid than normal, but difficult or labored. Sometimes in hysterical rapid breathing the respirations reach 150 per minute. This is not voluntary, and the diaphragm moves very little, the chief breathing being costal. If the lungs be clear of trouble, then the difficulty may be present in the trachea or larynx, either as the result of spasmodic contraction of these passages or because they are occluded by growths, such as papilloma or malignant growth, inside or outside, or aneurysm which may act by pressure, thereby narrowing the tube. Any agency which interferes with the proper oxygenation of the blood causes rapid breathing unless at the same time the respiratory centre is depressed.

There are, moreover, several other causes which affect the character of the respiration without affecting the larynx or lung tissues directly or indirectly. These are fever, which acts as a respiratory stimulant, and excitement, nervous or mental, particularly that of hysterical patients. Again, apoplectic seizures, uremia, and diabetic coma may be accompanied by rapid breathing.

The *respirations are slowed* or decreased in number by great obstruction to the entrance of air into the lungs from any cause, so that it is difficult to inhale the air, by the action of poisons made in the body, as the poisons of uremia and diabetes; by the effect of poisons swallowed or absorbed in other ways, notably opium, chloral, aconite, chloroform, or antimony.

The *rhythm* or relative time of inspiration, expiration, and the pause is in health in the mouth and trachea as follows: If 10 represents a complete respiratory cycle, inspiration is represented by 5, expiration by 4, and the pause by 1. If it is difficult for air to enter the chest, as in spasmodic croup, the inspiration is much prolonged. This prolongation is also sometimes very marked in cases of paralysis of the posterior crico-arytenoid muscles. If there is difficulty in expelling the air, the expiration is prolonged, as in asthma and in emphysema.

The most remarkable change in rhythm is the so-called Cheyne-Stokes breathing, in which the patient after a pause of several seconds begins to breathe with gradually increasing rapidity and depth, and then, after reaching an acme of hurried respirations, gradually decreases their rapidity and depth until they fade to nothing, when, after a pause, the same process is repeated. This breathing is seen commonly in apoplexy, in uremia, in brain tumor, in cerebrospinal fever, in meningeal tuberculosis, in some rare cases of cardiac valvular disease, probably as the result of embolism, and in hematuric malarial fever. Rarely it occurs in cases of acute febrile disease, as typhoid fever, scarlet fever, pneumonia, whooping-cough, and puerperal septicemia. It may also be met with in the course of diabetes. Its presence is an exceedingly bad prognostic sign, but cases of recovery after its onset have been observed, and Murri has reported a case in which Cheyne-Stokes breathing lasted forty days, and Sansom one in which it lasted 108 days. If the cause be an acute disease, recovery is more common after this symptom than if it be due to some chronic process with an acute exacerbation.

Labored breathing (dyspnea) is seen in all cases in which the blood cannot be provided with sufficient oxygen owing to obstruction to the entrance of air into the chest, to spasm of the bronchioles, or to the occluding of the air vesicles by any form of exudate, croupous, catarrhal, or serous. These conditions may be primary or secondary to disease elsewhere, as in uremia or cardiac disease. Inspection of the chest in such a case shows great activity of the accessory respiratory muscles, such as the sternomastoid, the scaleni, the pectorals, and the abdominal recti. The nostrils are dilated and the face is anxious. The posture of the patient is that of sitting up in bed.

Sometimes when the chest is flexible, as is that of a child, the

inspiration is jerking when there is obstruction to breathing. This is due to the fact that the chest is forced into expansion by muscular effort, and at the same time is subjected to the external atmospheric pressure, while the air enters the lung slowly and irregularly owing to the obstruction.

The function of breathing and the movements of the chest are closely associated. In men the respiratory movements chiefly affect the lower ribs and the abdominal walls, owing to the fact that as the diaphragm descends it pushes the abdominal contents downward, so causing abdominal bulging. In women, however, this is not so marked, and the breathing is chiefly costal, the upper part of the chest moving more than the lower (costal breathing). If abdominal breathing is absent in a man and is replaced by breathing of the costal type, we can be assured that the movements of the diaphragm are impaired by the pressure of fluid in the abdomen (ascites); by peritonitis, causing fixation of the diaphragm, owing to pain; by the presence of large growths in the abdomen, or by great enlargement of the liver and spleen. Other possible causes would be a subphrenic abscess or a greatly enlarged cystic kidney, or hydronephrosis.

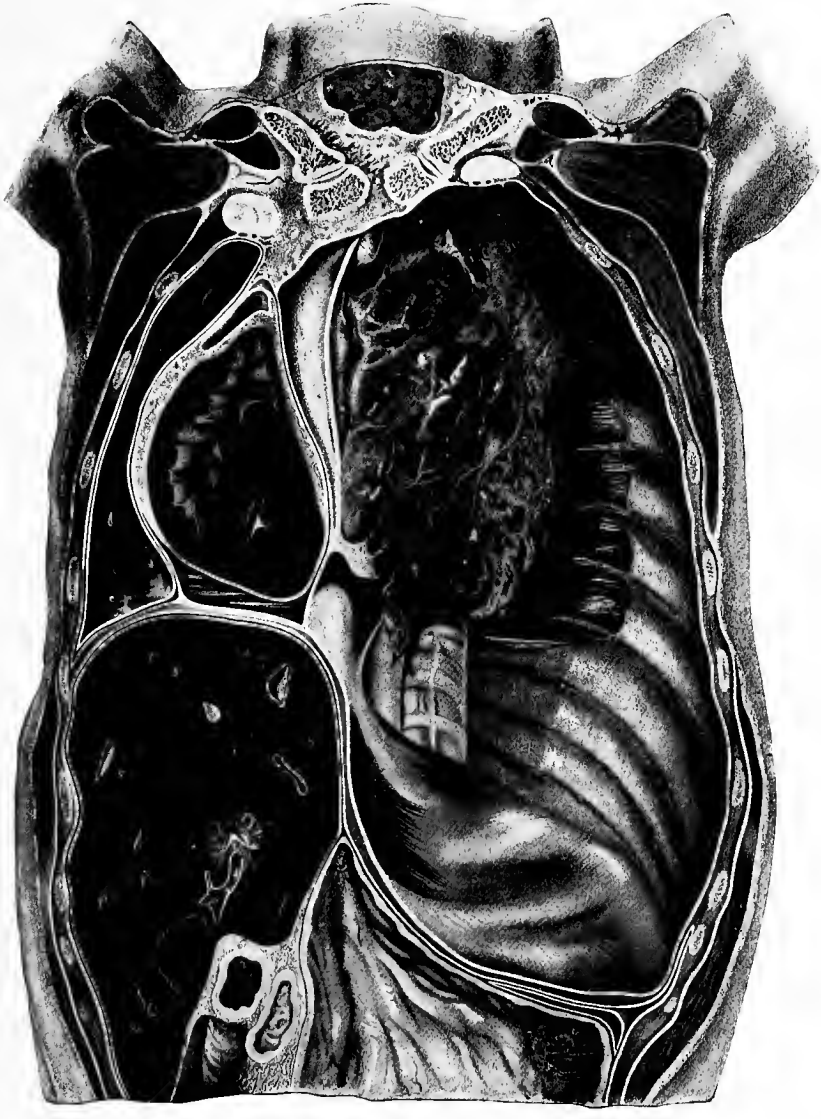
If the costal breathing of a woman is absent, there is nearly always some pulmonary cause for it, such as faulty development, or, if due to disease, its absence arises most commonly from tuberculosis or pleurisy, or old pleural adhesions which bind down the chest wall.

In this connection should be mentioned the "*wavy breathing*" met with most commonly in pneumonia, a condition in which inspiration and expiration do not seem to occur regularly or evenly all over the chest, one part filling or emptying a moment before the other. This usually indicates a grave pulmonary condition.

It is also necessary to notice the extent of the chest movements. These are very limited in the characteristic chest of a person having a tendency to tuberculosis, and in the barrel-shaped and rigid chest of emphysema of the lungs. Deficient respiratory movement is not only a predisposing cause of lung disease, but an important diagnostic sign. When one side of the chest moves more than the other to a considerable extent, we suspect, in the side which moves slightly, a pneumonia, a pleuritis, a pleuritic effusion or adhesion, tuberculous consolidation or fibroid lung, provided that the patient has not naturally a greater development on one side than the other, or has not pursued a trade or occupation causing unilateral hypertrophy.

While inspecting the surface of the chest the physician should also note the presence or absence of enlarged or *pulsating blood-vessels* on its surface or about the base of the neck. The cervical

PLATE VIII.



A frozen section made in a plane lying 5 cm. in a posterolateral direction from the nipples and too near the anterior surface to show the large vessels of the chest and neck. The patient died of acute left-sided pneumothorax, resulting from tuberculosis. The section reveals the collapsed left lung, the air space about it, the heart pushed far to the right in its sac, and the anterior border of the right lung compressed by the heart. The right lung is also greatly displaced, and the air has pushed the diaphragm downward on the left side. (Ponfick.)

vessels are commonly seen to be distended in cases of advanced emphysema of the lungs and in chronic bronchitis. Systolic pulsation of the jugular veins indicates tricuspid regurgitation. Again, in cases of thoracic aneurysm pressing upon the superior vena cava and innominate veins we find spongy venous masses above the clavicles, and the veins of the trunk and arms may be engaged. Intrathoracic growths produce similar symptoms.[1] Pulsation in the cervical vessels is also sometimes seen in cases of severe anemia and in cases of aortic dilatation with regurgitation.

Sometimes when a patient is placed flat on his back with his feet pointing straight toward a window (cross-lights being excluded) and the chest exposed, the following phenomenon can be observed during forced respiration: along both axillæ a sort of shadow is seen to descend during deep inspiration from about the seventh to about the ninth ribs, passing up again during expiration. It is best seen in spare, muscular young persons of either sex. The observer should stand with his back to the light. It is called *Litten's sign.*

This phenomenon is nearly or entirely absent in the following conditions: (1) Fluid or air in the pleural cavity. (2) Obliteration of the pleural cavity by adhesions. (3) Advanced emphysema of the lungs. (4) Pneumonias of the lower lobe. (5) Intrathoracic tumors low down in the chest.

Slight and limited pulsations on the chest wall elsewhere than over the apex beat may be due to many causes.[2] (See Palpation.) When they are seen in the second or third interspace on the right side they are due as a rule to displacement of the heart, which has been drawn to the right by mediastinopericarditis or contraction of the right lung and pleura as a result of chronic disease. If the pulsation is lower than this, it is usually due to a dilated right auricle, or displacement of the entire heart, as in a left-sided hydro-, pneumo-, or pyothorax. (See Plate VIII.) If the pulsation be on the left side of the sternum, then it may arise from a displaced apex beat due to effusion, or to retraction of the pleura which has become adherent to the pericardium, or fibrosis of the lung may be the cause. Finally, there may be marked epigastric pulsation. This is due to displacement of the heart by left-sided pleural effusion, which pushes the heart to the right and downward, to hypertrophy of the right ventricle, to pulmonary emphysema, often a cause of enlargement of the right side of the heart, and, finally, it may be due to transmitted pulsation of the abdominal aorta. If the latter is the cause, it can usually be determined by deep palpation that the pulsation arises from this vessel. Rarely it is due to the transmission of the

[1] See "The Pathology, Clinical History, and Diagnosis of Diseases of the Mediastinum," by the author. Fothergillian Prize Essay of Medical Society of London for 1888.

[2] These are spoken of here because they can often be seen yet cannot be felt with the finger tips.

aortic impulse by a tumor which overlies the artery. If this is the case, it will be found that when the patient takes the knee-chest posture the pulsation disappears because the growth falls away from the bloodvessel.

If the epigastric pulsation is in the nature of a systolic retraction, then it is probably due to indurative mediastinopericarditis.

Rarely, on inspection of the chest anteriorly, a curious retraction of the interspaces near the level of the apex beat is noticed to occur with each systole of the heart. This is usually indicative of an adherent pericardium, and when on inspection of the posterior surface of the chest such retraction is seen at the level of the eleventh interspace it is called "Broadbent's sign" of adherent pericardium. This movement is supposed to be due to the heart pulling on the central tendon and muscular portion of the diaphragm.

(For the further discussion of the significance and position of cardiac pulsations and thrills, see Palpation below.)

PALPATION.

Palpation of the chest is usually performed by placing the finger tips or the whole hand, palm downward, on the chest. This method reveals alterations in its contour and in its elasticity. It will also reveal the ability of the thoracic viscera and the chest wall to transmit vibrations produced by the voice (vocal fremitus). This so-called vocal fremitus depends upon the fact that below the vocal bands lies a column of air which reaches to the vesicular portions of the lung, and when an individual speaks this column of air is put into vibration, and these vibrations are in turn transmitted to the chest wall. Of course, a chest wall greatly thickened by fat or by highly developed muscles will not transmit these vibrations as readily as a thin chest wall; but aside from these causes of variations in fremitus in health we have a number of causes in disease which greatly modify vocal fremitus. It must be remembered, too, that this vibration is more marked in men than in women and children, because the voice of a man is so much louder and has greater volume. Vocal fremitus is also greater on the right side than on the left, because the principal bronchus supplying this lung is larger than that of the left side, is joined to the trachea at a less acute angle, and is nearer the vertebral column; and, again, as recently emphasized by Cary, the bronchus going to the right upper lobe is given off at a point very near the origin of the right bronchus, and in many cases "fully two and a half inches above the corresponding left bronchial tube." Sometimes this upper tube comes off the trachea directly.

Palpation of the Abnormal Chest.—The conditions of the *lung* which cause a decrease in vocal fremitus are pleural effusions of any kind, which not only cut off the transmission of sound, but by their contact prevent vibration of the chest wall; pneumothorax, which causes collapse of the transmitting medium, the lung; any condition which causes occlusion of a large bronchus, such as a tumor or a large mass of mucus, and great pleural thickening. When the vocal fremitus is increased it is an indication of pneumonia, of tuberculous thickening or consolidation of the lung, of the presence of a cavity or of tumor in the thorax touching the chest wall. Fremitus is increased in these conditions because the consolidated lung transmits the vibrations of the air in the bronchial tubes to the chest

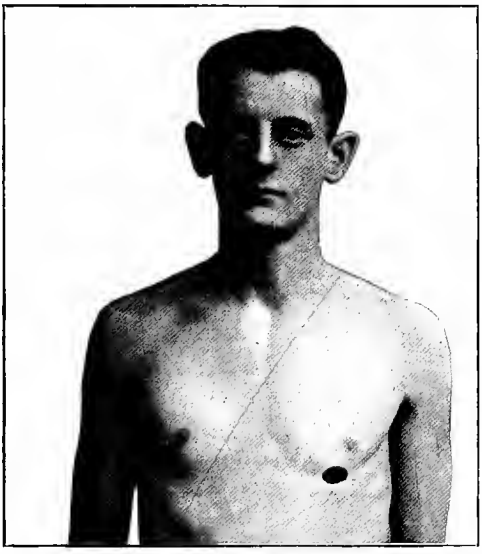


FIG. 69.—Area of normal apex beat.

wall, or, in the case of a cavity, the sound is transmitted directly to it, and it there causes so great a vibration of the air in the hollow space that the vibration of the chest wall is marked. (In this connection, see part of this chapter on Auscultation.)

Palpation of the chest wall will also give information as to the *position* and *character of the cardiac pulsations.* Thus, the apex beat of the heart in persons standing erect will usually be felt, in those who are not inordinately fat and who are healthy, between the fifth and sixth ribs, about two inches to the left of the sternum (Fig. 69). If the apex beat is below this level, its depression may be due to enlargement of the heart (hypertrophy or dilatation), to effusion in the pleural cavity on the left side, to pulmonary emphysema

causing abnormal descent of the lung and diaphragm, and with it cardiac hypertrophy. Sometimes tumors in the chest produce a similar depression of the apex beat. On the other hand, if the apex beat of the heart is felt above the fifth interspace, the heart may be raised by pericardial effusions or adhesions following inflammation, by pleural adhesions or effusions, by abdominal effusion (ascites), by tumors, by distention of the colon with gas, and by great enlargement of the spleen. Displacement of the apex beat to the left is generally associated with downward displacement, and is commonly due to hypertrophy of the left ventricle, to pleural adhesions, and particularly to pleural effusion on the right side. Displacement to the right is due to adhesions and to hypertrophy and

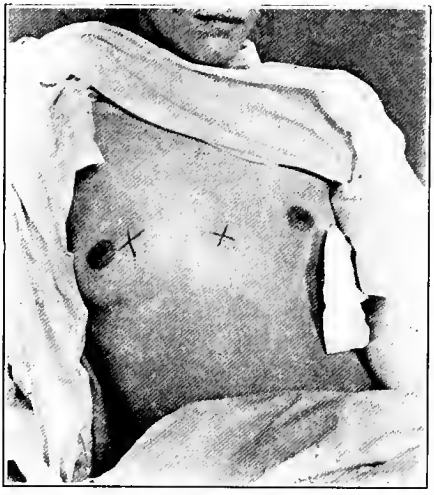


FIG. 70.—Case of large empyema of left side. The *x* mark near the right nipple indicates the displacement of the apex of the heart to the right. The *x* mark in the middle line indicates the apex of the heart after the pus had been evacuated. (From a patient in the author's wards.)

dilatation of the right ventricle, so that the apex beat is felt in the epigastrium or against the edge of the sternum. Pleural effusion or pneumothorax on the left side may also cause this displacement even as far as the right nipple (Fig. 70). (See Plate VIII.)

The area of the normal apex beat is about one square inch. In disease this area often extends over several square inches, generally as the result of hypertrophy and dilatation of the ventricles.

The force of the apex beat in health depends largely upon the depth of the chest and the thickness of its wall. It is increased in hypertrophy of the heart, when the heart is fully under the influence of stimulants, as, for example, digitalis in full doses, in the early stages of acute fevers, and in great nervous excitement. If hyper-

trophy is the cause, the apex beat is usually lower, and more to the left than normal, and the impulse is more or less diffuse and powerful. The apex beat is decreased in force in cases of dilatation of the heart without compensatory hypertrophy, in degeneration of the myocardium, in cases of effusion into the pericardium and in the presence of pulmonary emphysema, which causes the projection of a part of the enlarged lung between the heart and the chest wall.

Thrills felt in the chest wall over the heart may be due to abnormalities in the blood current when valvular disease or aneurysm is present. Thus we find thrills in the precordium, or in the neighborhood of the apex, in disease of the mitral valve, both regurgitant and obstructive, but they are usually much more marked in stenosis than in regurgitation, which latter condition does not cause much palpable thrill, as a rule, except in children. A well-marked thrill at the apex is usually to be considered a sign of mitral stenosis if it is presystolic in time. It is then the so-called "diastolic thrill of mitral stenosis." Thrills in the neighborhood of the second right costal cartilage indicate an aortic lesion, generally that of aortic stenosis, of aortitis, or of aortic aneurysm. When thrills are felt in the tricuspid area, namely, in the midsternal region, or a little to the right of it, the lesion is probably tricuspid regurgitation, as tricuspid obstruction is very rare. Sometimes a thrill in this area is due to aneurysm of the descending part of the aorta.

Pulsation is felt in the chest wall in some cases of empyema. In nearly every instance this pulsation, when it occurs, is found on the left side. It is produced by the impulse of the heart against the effusion, and occurs in two forms: the internal, in which the effusion transmits a heaving impulse to the chest; and the external, in which there is a pulsating tumor external in the chest wall. Sometimes this is called "pulsating empyema."

PERCUSSION.

Percussion of the chest is commonly performed by placing one finger, generally the middle one of the left hand, on the chest wall and tapping it on the back with the tip of the bent finger of the right hand, the movement of the striking hand being entirely a wrist movement. Sometimes percussion is made by directly striking the chest with the fingers or palm of the hand (direct percussion). Many physicians also employ a percussion hammer with a rubber head and a plexiimeter, or chest piece, of ivory, celluloid, or glass. Glass is by far the best material for the chest piece, as it does not produce a note of its own when struck by the hammer, as do the other materials. The disadvantage of this means of percussion is

that the physician cannot determine the degree of resistance offered by the surface percussed, which is of the greatest service in many cases of doubtful character, as, for example, in a case in which pneumonia is suspected and the results of the percussion will decide the diagnosis. Care should be taken in performing percussion: First, that similar points on the chest wall on each side are carefully compared; second, that the finger which is applied to the chest is

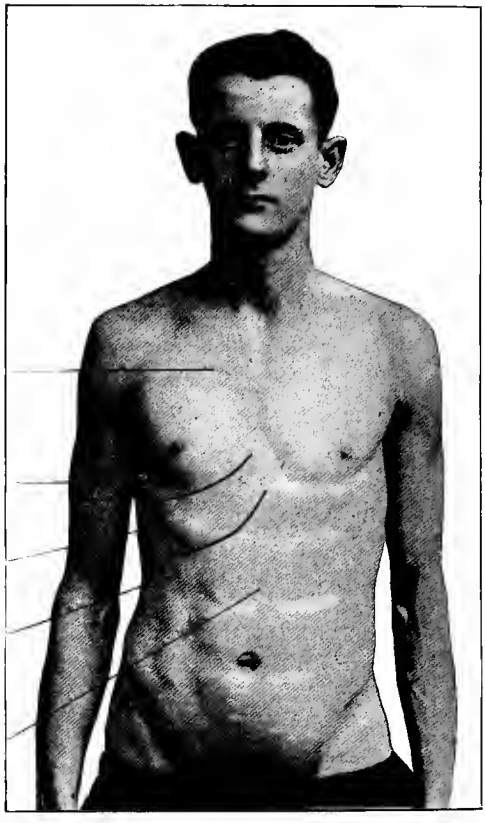


FIG. 71.—The effects of heavy and light percussion in outlining organs.

placed in the same relation to the ribs, or interspaces, on each side when it is struck; and, finally, in studying the effects of percussion the physician should always employ it both during forced inspiration and forced expiration, in order to determine the resonance of the chest with its full quota of air and when it has only residual air.

The resonance produced on percussion is due to three things: First, to the vibrations of the air in the lungs; second, to the vibrations of the chest wall when it is struck; third, to the vibrations in

the pleximeter placed on the chest. The last need be considered as a factor only when a piece of celluloid or ivory takes the place of the finger, for the finger itself does not vibrate enough to alter the note developed. The note produced by vibration of the chest wall can also be excluded as of little importance unless the chest is very pliable and resilient, as in a thin child, and the blow be delivered very hard. The most important factor in the production of the percussion note is that first named, viz., the vibration of the air in the chest caused by the blow delivered on the chest wall. A large part of the percussion note depends, therefore, upon the amount

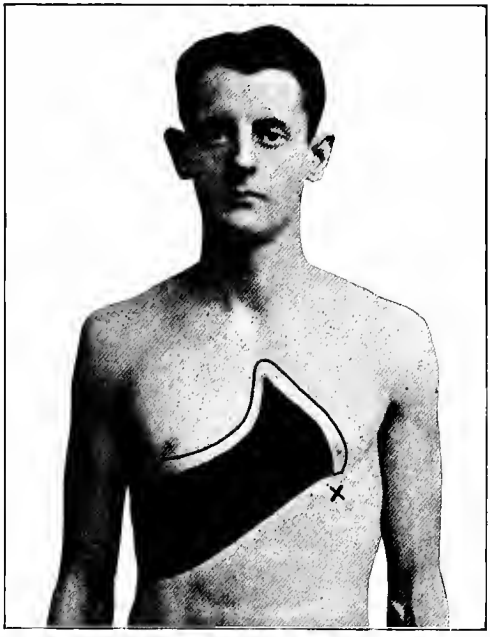


FIG. 72.—Showing area of partial hepatic dulness (outline) and absolute hepatic dulness (solid), merging into cardiac dulness in enlargement of liver.

of air in the chest, the tension of the chest wall, and the condition of the pulmonary tissues. The sound produced when the healthy chest is percussed is called the normal pulmonary resonance.

In percussion, very different results are obtained by the use of light and heavy blows, and when percussing the chest it is wise, as a rule, to use light percussion, since a heavy blow may produce some resonance in a distant healthy part, and so cause the physician to overlook a small localized area of consolidation, which light percussion might discover. Further than this, it is of great importance that the sense of resistance offered to the finger placed on the chest

in percussion be carefully observed, and many experienced men gain more information from this sensation than from the character of the note elicited. In consolidation of the lung the elasticity and resiliency of the chest are impaired, and in pleural effusion they are still more destroyed. The resiliency and elasticity over cavities are marked. The various results produced by heavy and light blows are well shown in Fig. 71.

Percussion of the Respiratory Organs.—On percussing the right side of the chest anteriorly in the mammillary line we find in health normal pulmonary resonance as low as the fourth interspace or fifth rib, at which point the resonance begins to be impaired, so that at the sixth interspace or seventh rib we find dulness due to the upper

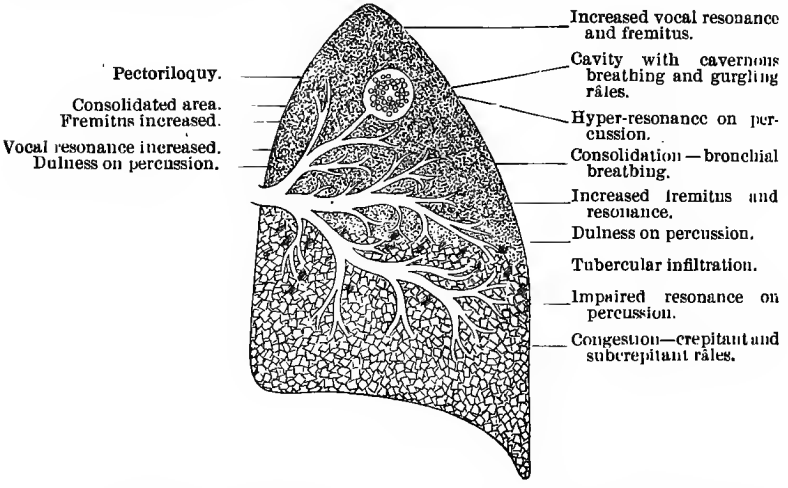


FIG. 73.—Tuberculosis at various stages in one lung, the physical signs depending on the stage. The upper part is consolidated, and contains a cavity communicating with a bronchus.

border of the liver. (See Fig. 71.) The area of partial and absolute hepatic dulness is shown in Fig. 72.

Posteriorly we find on percussion of the right chest that the normal pulmonary resonance begins as high as the suprascapular area, and ends as low as the tenth or eleventh ribs. It is much less resonant as compared with the percussion notes obtained from the anterior aspect of the chest, by reason of the thickness of the chest wall and the presence of the scapulæ. For this reason pulmonary resonance is best developed posteriorly at the bases of the lungs below the scapulæ. Before percussing the back the patient should be made to lean forward and fold the arms, in order to stretch the tissues and make them tense and as thin as possible.

We can divide the normal sounds produced by percussion into

the tympanitic, the dull, and the flat. We can also develop by percussion of the chest in disease what is known as a "cracked-pot sound."

A tympanitic sound is best produced in its most typical form by percussing the epigastrium when the stomach and colon contain some gas. When this sound is produced by percussing the chest it is due to one of several causes, such as a large cavity, pneumothorax, collapse of the lung, or adhesions of the pleural surfaces. A high-pitched note on percussion is also a valuable sign in localizing a deep-seated consolidation, for while consolidation produces dulness on percussion when it is near the chest wall, it is a common thing for

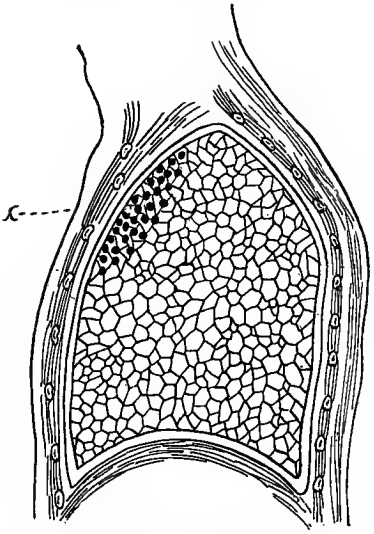


FIG. 74.—Moderate dulness on percussion at x over tuberculous infiltration.

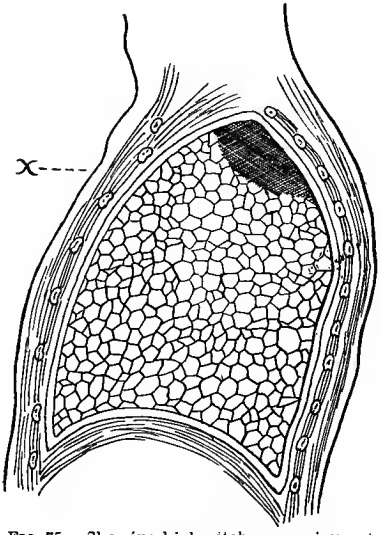


FIG. 75.—Showing high-pitch percussion note anteriorly from consolidation posteriorly. The shaded area is the consolidated part; x indicates the position anteriorly where the percussion sound is raised in pitch.

hyperresonance to be found in the chest over the area which is consolidated if healthy lung supervenes.

If the cavity be in the lung itself, it must be of some size and be near the surface to produce a tympanitic note, and, if it communicates with a bronchus, the character of the note will change when the mouth is closed or opened (Fig. 73). If the case be one of pneumothorax, with fluid in the chest, changes in the posture of the patient will greatly alter the character of the note in a given locality.

Consolidation of the lung, as in pneumonia and tuberculosis, as just stated, generally gives a dull rather than a tympanitic note; but if the consolidated area surrounds a very superficially placed bronchus, the percussion stroke may produce vibration in the air in this tube, and this will cause a note, high pitched in charac-

ter, which varies as the mouth is closed or opened. (See Fig. 75.) Collapse of the lung causes a tympanitic or high-pitched note, because the comparatively little air in the lung vibrates as a whole, its vibrations not being stopped as in health by the tense septa and vesicular walls. This note is best elicited in cases of pleural effusion over the apex of the chest, into which the collapsed lung has been pushed by the effusion. This is sometimes called "Skodaic resonance." If the compression is sufficient to consolidate the lung, the tympanitic note is lost. This note is not altered by opening and closing the mouth.

The "cracked-pot sound" is produced in an adult by the sudden expulsion of the air from a cavity through a small opening by the

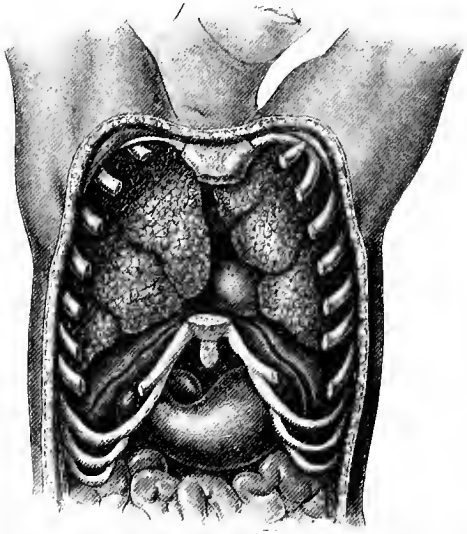


FIG. 76.—Position of heart partly covered by lungs. (Modified from Aitken.)

force of the percussion stroke. It occurs on percussing a healthy child when its mouth is open, the air being forced by the blow from the lung through the glottis. In adults who have a diseased lung the cracked-pot sound most commonly results from the presence of a cavity, t may also be heard in cases of pneumothorax with a fistulous tract opening externally or into a bronchus, in a few cases of pleural effusion in thin-chested persons, and, in rare instances, before consolidation has occurred in pneumonia.

In cases of pleural effusion a flat note on percussion is produced over the effusion, and it is of very much the same character as the sound elicited by percussion of the solid tissues of the thigh.

Percussion of the Heart and Great Vessels.—On percussing the chest anteriorly on the left side at the fourth interspace, it will be found that the resonance is decreased by the presence of the heart. At the apex of the chest on this side percussion develops normal resonance, but as we descend in the line situated half-way between the mammary line and the midsternal line we find an impairment of resonance at the third rib which becomes in the next inch of descent a very marked dulness, which is produced by the presence of a solid organ, the heart. The impairment of resonance is not complete at the upper border of the heart, because of the fact that the edge of the lung intervenes between the heart and the chest wall, and so the

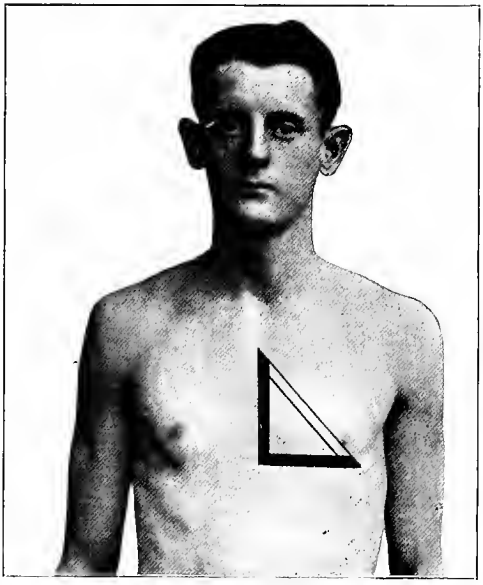


FIG. 77.—The cardiac triangles.

note which results on percussion is neither the normal resonance of the lung nor the dulness produced by the presence of the heart (Fig. 76). The outlines of the normal cardiac dulness on percussion are shown by the above diagram, and they form what have been called the "cardiac triangles" (Fig. 77).

The large triangle begins at the level of the second left costal cartilage, and extends down the midsternal line to the level of the sixth costal cartilage. The base then extends to the apex beat, normally situated in the fifth interspace just inside of the clavicular line. The hypothenuse of the triangle joins these points. In this area we have included the partial and total cardiac dulness.

The small cardiac triangle, of absolute cardiac dulness, begins at the third costal cartilage and extends to the sixth. The base line extends to within one and one-half inches of the nipple, and the hypothenuse joins this point with the third costal cartilage at the midsternal line. (See Fig. 77.) The borders of the heart really extend farther than this, but are not near the chest wall and are partly covered by lung tissue as already stated. (See Fig. 76.)

The greater part of the cardiac dulness on percussion is due in health to the presence of the right ventricle, which is nearest the chest wall. The right auricle also is well forward, while the left

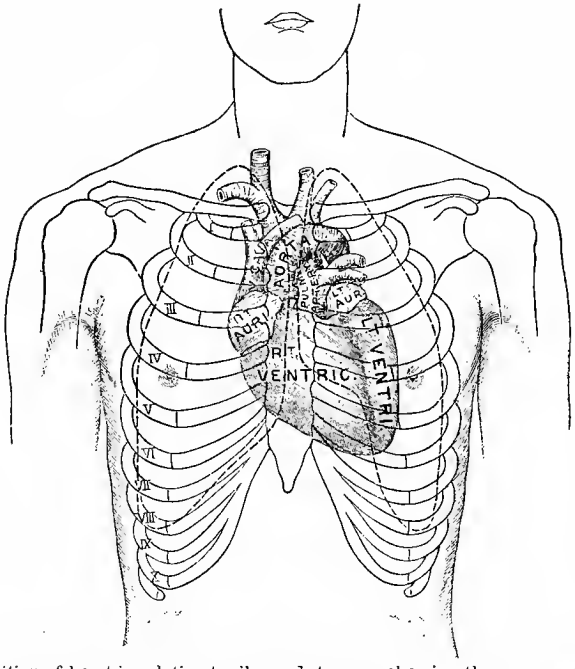


FIG. 78.—Position of heart in relation to ribs and sternum, showing the comparatively small area of cardiac dulness due to the left ventricle.

ventricle only fringes the edge of dulness to the left. This is well shown in the accompanying diagram (Fig. 78).

When hypertrophy or dilatation of the heart occurs, it may be found that the area of cardiac dulness extends to the right of the sternal line and to the left of the long side of the triangle, and the apex beat is apt to be displaced downward and to the left. On the other hand inability of the physician to discover any such increase in the area of cardiac dulness to the left by no means proves that it does not exist for the enlarged left ventricle often seems to bury itself in the left lung even when this organ is normal in size. If the lung

be emphysematous the difficulty of finding the true left outline is greatly increased, and in emphysema of the lungs the cardiac triangles may be obliterated by the extension of the lung between the chest wall and heart. They may also be distorted by reason of pleural effusions pressing the heart upward and to the right, or in the case of right-sided pleural effusion the heart may be pushed unduly to the left. Pneumothorax may cause similar results, or, again, old pleural adhesions and conditions may so displace the lungs or heart that the triangles cannot be found. (See Plate VIII.)

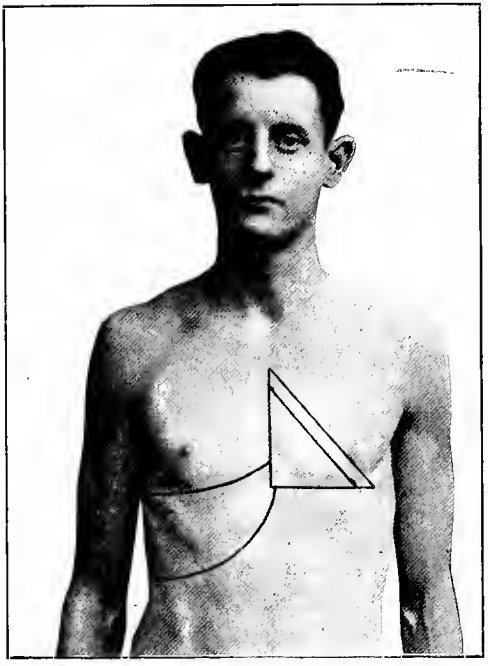


FIG. 79.—Areas of cardiac and hepatic dulness.

Great distortion of the triangles occurs as the result of pericardial effusion (see Fig. 80), but in this case the heart sounds will be distant on auscultation and the apex beat very feeble or lost, whereas in hypertrophy they are exaggerated and the apex beat forcible. The diagnosis of pericarditis, after the stage of dryness and friction sound has passed by, is by no means as easily made as some of the text-books would make it appear. One of the most reliable signs of pericardial effusion is that of Rotch, namely, that any considerable dulness in the fifth right intercostal space near the sternum means pericardial effusion, provided pulmonary consolidation and pleural effusions or adhesions are excluded. The writer has, however, often

seen this sign present in marked cardiac dilatation; but if, as Ewart has pointed out, there is obliteration of the normal acute angle between the right border of the cardiac dulness and the line of liver dulness, this sign is of value. (See Fig. 79.) In dilatation of the heart the area of the apex beat is usually diffuse, and the heart sounds, while feeble, are clearly heard.

In this connection the following summary, prepared by Sansom, of the differential diagnosis between dulness due to pericarditis and that due to dilatation of the heart, is of interest:

	Pericarditis with Effusion.	*Dilatation of the Heart.*
Outline of dulness	Dulness pear-shaped, and enlargement chiefly upward.	Dulness not pear-shaped, and enlargement chiefly downward.
Rate of development of dulness	Often rapid, and then characteristic.	Usually very slow, though a rapid dilatation of the heart sometimes occurs.
Impulse and apex-beat	The impulse when present is in the third or fourth interspace; apex-beat tilted upward and outward, or effaced.	Impulse can usually be felt to the left of the lower end of the sternum or in the epigastrium.
Relation of dulness to left apex-beat	Dulness may extend to the left of the apex-beat.	Dulness does not extend to the left of the left apex-beat.
Pain over præcordia and tenderness in the epigastrium	Often present.	Usually absent.
Pulsation in the veins of the neck.	May be present if endocarditis complicates.	Often present when right heart dilated.
Etiology	Usually acute, in course of acute rheumatism, cirrhotic Bright's disease, etc.	Usually chronic; often associated with chronic valvular lesions, fatty and fibroid degeneration.
Fever	Often present.	Absent unless from some complication.

The same author also tabulates the facts in the differential diagnosis between increased dulness due to pericarditis and hypertrophy of the heart as follows:

	Pericarditis with Effusion.	*Hypertrophy.*
Rate of development	Usually rapid.	Usually slow.
Impulse; apex-beat	Impulse, when present, is in the third or fourth left interspace, and is feeble; apex tilted upward and outward, or beat effaced.	Impulse powerful; if left ventricle hypertrophied, apex displaced downward and outward; if right ventricle hypertrophied, apex displaced downward and inward beat may be in the epigastrium.
Pulse	Weak and quick; may be irregular.	Character of the pulse depends on the side of the heart which is hypertrophied and the cause of the hypertrophy. When left ventricle hypertrophied and no aortic obstruction or mitral regurgitation, the pulse is large and powerful.

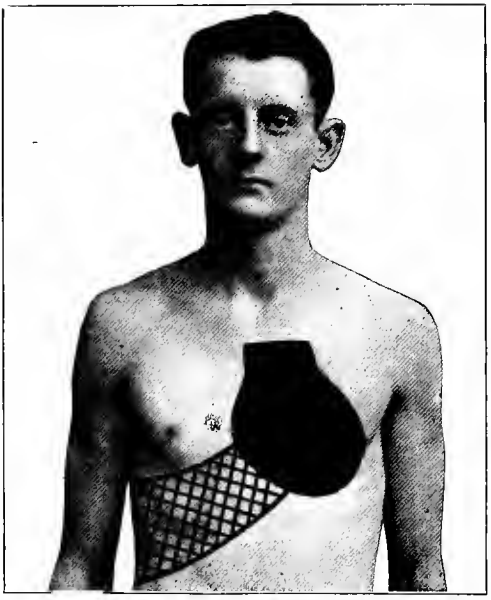


FIG. 80.—Outline of percussion dulness in extensive pericardial effusion. The light shading is the liver dulness.

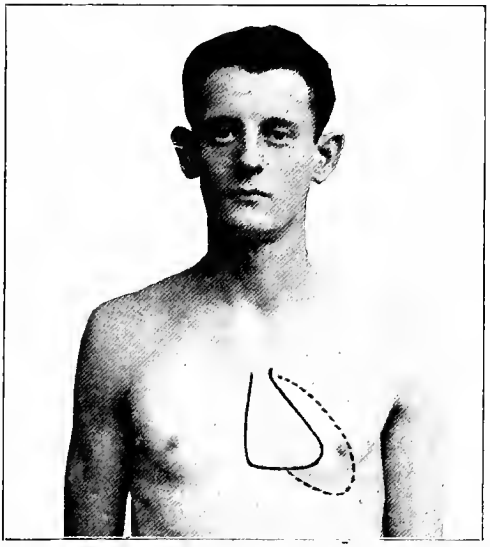


FIG. 81.—Diagram of the area of the heart in aortic obstruction and regurgitation. The dotted lines show enlargement of the left cavities, especially the ventricle, while the solid lines show the normal area.

The various valvular and other lesions of the heart result in alterations in the size of the various cavities without the entire viscus being equally affected. Thus aortic regurgitation causes enormous enlargement of the left ventricle (dilatation and hypertrophy), and aortic stenosis also causes the same enlargement, as a rule, in less degree. Mitral regurgitation causes hypertrophy and dilatation of the left ventricle and some enlargement of the left

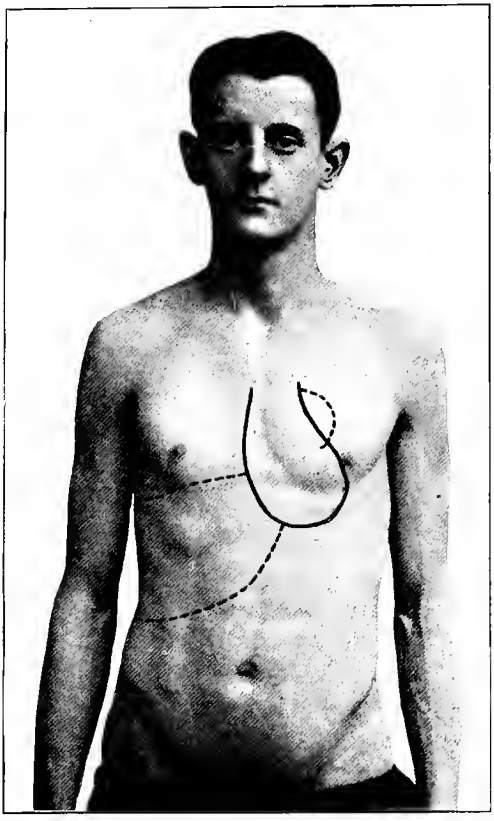


FIG. 82.—Area of the heart in mitral stenosis. The dotted lines indicate the enlargement of the left auricle and enlarged liver. The solid line shows enlargement of the right cavities.

auricle, and the left auricle is also enlarged in mitral stenosis. Tricuspid regurgitation causes hypertrophy and dilatation of the right auricle and hypertrophy of the right ventricle, and mitral stenosis often has a similar influence over the right side of the heart by damming back the blood into the lungs and right side of the heart. The following diagrams illustrate the deformity of the area of cardiac dulness under these various conditions (Figs. 81 and 82).

AUSCULTATION.

Auscultation of the normal chest reveals in health two sets of sounds: the respiratory and the cardiac.

Auscultation of the Respiratory Apparatus.—The respiratory sounds occur in two varieties, namely, vesicular breathing and bronchial breathing. The vesicular sound is heard in its most typical form over the apices of the lungs anteriorly, the latter at the inferior angles of the scapulæ posteriorly. We may listen to these sounds by placing the ear directly against the chest, or by the use of a single or a binaural stethoscope, but neither of these instruments is satisfactory for this purpose. The patient must be in an unconstrained position, as should be that of the physician, and if the ear is placed against the chest, or a single stethoscope is used, the face of the physician should always be turned away from that of the patient, because the breath of a sick person is often very disagreeable and the breath of the doctor may be equally annoying to the patient. Care should be taken in the use of the stethoscope to see that the edge of the bell in its entire circumference is in close contact with the chest wall.

The respiratory sounds consist, as already stated, in the vesicular murmur and the bronchial or blowing sounds, which are sometimes designated by the term tubular breathing. In the vesicles the air is subdivided into many minute parts, whereas in the bronchial tubes it moves along in a column. Whatever may be the actual cause of the production of normal "*vesicular breathing*," we know that when it is present it signifies a healthy pulmonary parenchyma, and when absent one more or less diseased.

Bronchial breathing, normal in the bronchial tubes, becomes an abnormal sign when it is heard in an area in which vesicular breathing should be present, as will be shown shortly.

After determining the fact that the sounds of normal vesicular breathing are present in the anterior parts of the chest, or that those of bronchial breathing can be heard between the shoulders, we next take note as to the relative duration of the inspiratory and expiratory sounds. Normally in the perfectly healthy chest the ratio of the expiratory sound to the inspiratory sound is as 1 to 3, although if the volume of air itself be measured the duration of expiration is 6 to 5. In other words, so far as auscultation of the vesicular portion of the lung is concerned, inspiration is far longer than expiration. Just at this point we learn one of the most important points in the physical examination of the chest, namely, that while the expiratory sound may be entirely absent in health, any marked increase in its length and loudness, so that it equals or exceeds the inspiratory sound, is a sign indicative of some diseased

state which impairs the elasticity of the lung, such as early tuberculosis, pneumonia, and emphysema.

The other variations in the vesicular respiratory sounds differing from those of health are harsh, or, as it is sometimes called, puerile breathing, and irregular breathing. In children, as the term "*puerile breathing*" indicates, the normal vesicular breathing is loud, clear, and harsh, because of the great elasticity of the lung and the thinness of the chest wall. If it is exaggerated in a child or present in the area of normal vesicular breathing in adults, it usually indicates some irritation of the bronchial mucous membrane. If it is found in the apices of the lungs in a marked degree, and expiration is prolonged, it is an important and fairly sure sign of early pulmonary tuberculosis, or in acute cases of catarrhal pneumonia or influenzal congestion.

Sometimes physicians speak of "bronchovesicular breathing," meaning a breath sound consisting of both bronchial and vesicular sounds. It is sometimes heard in a healthy person when he breathes superficially, and in disease usually indicates the early stages of pneumonia or early tuberculosis of the lungs. It is of value as a diagnostic sign only when localized in one part of the lung. This harsh breathing of exudation and thickening differs from normal puerile breathing in this important particular, namely, that in the latter expiration holds its normal ratio to inspiration, whereas in disease it is greatly prolonged.

Irregular, "cog-wheel" breathing occurs in the chest of a healthy, sobbing child and in that of an hysterical woman, but it possesses pathological significance if it occurs when a full breath is taken, and it is often present as an early sign of incipient pulmonary phthisis.

Bronchial breathing in health is best heard in the posterior part of the chest, as already stated, between the scapulæ and the seventh cervical to the fourth dorsal vertebra. When this bronchial or tubular breathing is heard in other parts of the chest it is a sign of disease, for while the bronchial tubes are distributed to all parts of the lung, the breath sound which is in them is masked by the sounds of vesicular breathing and muffled by the lung tissue surrounding them. If this vesicular tissue becomes consolidated by disease, the vesicular murmur is lost and the solid lung transmits the bronchial sounds directly to the ear of the examiner. Bronchial or tubular breathing, or, as it is sometimes called, "blowing breathing," heard in the part of the lung in which vesicular breathing is normally heard, is, therefore, a sign of tuberculous or pneumonic consolidation (Fig. 83), or of compression or collapse of the lung above a pleural effusion. Bronchial breathing is also heard in that area of the chest in which vesicular sounds normally predominate, in cases of cavity of the lung, because in such a lung the bronchial sound

is transmitted directly to the cavity, and thence to the ear without being impaired by the intervention of healthy lung tissue. In other words, consolidated tissue and cavities transmit sound better than the normal vesicular portion of the lung, which is a combination of air and vesicular wall. If the cavity be large, we have a loud sound developed by the transmission of the bronchial sound into its open space and by the passage of air through it. This is called "*cavernous breathing.*" If the cavity is not very large, or is peculiarly situated in relation to the supplying bronchus, we have what is called "*amphoric breathing*"—that is, a sound like that produced by blowing over the mouth of an empty bottle. This

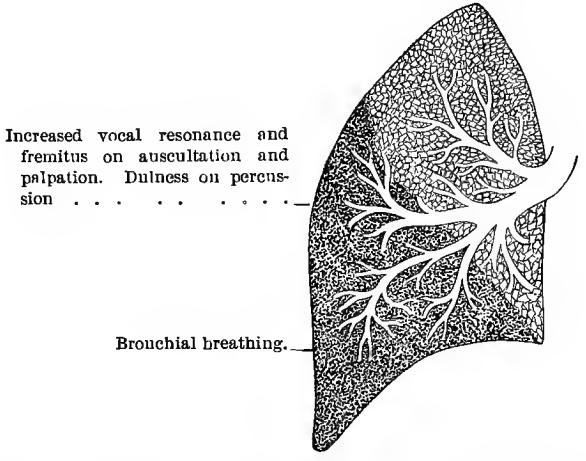


FIG. 83.—Pneumonia of the lower lobe with the physical signs of consolidation.

sound is also rarely heard in cases of pneumothorax in which the bronchial tubes, running near to the pleural cavity, transmit their sound to the air in the pleural space.

It is never to be forgotten that in auscultating the chest the two sides must be compared, since the well side often gives a standard for that affected by disease, and in doing so it must be remembered that disease not only modifies the signs in the lung in which the morbid process is situated, but also changes the normal signs. Thus pneumonia or pleurisy or pleural effusion causes a louder vesicular and bronchial breathing on the healthy side than is normal, because this lung has to take in more air to make up for the loss of activity on the diseased side. Great care should, therefore, be exercised that the loud, harsh breathing of the healthy part in such a condition is not mistaken for the harsh breathing of disease.

There are a number of other sounds heard in the chest in cases of disease of the air passages. These consist in rales of various kinds,

voice sounds (vocal resonance), friction sounds, and succussion notes or sounds.

Rales are divided into two chief classes, moist and dry. The moist are subdivided into the crepitant, or crackling, the fine bubbling, and the coarse bubbling. The dry are called sonorous, or sibilant and hissing. Sometimes the sonorous rales are called rhonchi. The fine crepitant rale is best imitated by moistening the thumb and finger tip, pressing them tightly together and then separating them while they are held near the ear. Another method of imitating the fine crepitant rale is to roll the hair where it grows above the ear between the thumb and fingers; and still another imitation is the crackling of salt when thrown on a fire. This rale is due to the separation of the vesicular walls, which have become adherent because of exudate. It occurs, of course, during the latter part of inspiration, and is an important sign of croupous pneumonia in its early stages before consolidation has occurred. It also is heard in cases of pulmonary collapse and edema, but not always in any of these diseased conditions. Care should be taken that the fine rales sometimes heard in the chest at the bases, posteriorly, in a person who has been long in one position in bed, are not thought to be indicative of pneumonia, as they are due only to congestion or accumulation of secretion due to the dorsal decubitus.

Fine bubbling rales occur chiefly in the smaller bronchioles and the coarse bubbling rales in the larger bronchioles, and they are caused by the passage of air through liquid or mucus. These are commonly heard in bronchitis and in pulmonary edema in the lower parts of the chest, chiefly posteriorly. If such rales are heard anteriorly or in the area for vesicular breathing, they indicate the stage of resolution of a pneumonia, or if this disease has not been present, or is long gone by, they possess the serious import of breaking down of tissue from tuberculosis in the lung. Sometimes these rales are limited to inspiration or expiration. In convalescence from an attack of asthma they occur with a to-and-fro character, and are often musical or tinkling.

There is another condition in which metallic tinkling is heard very clearly, and that is hydropneumothorax. In this condition there is a continual dropping of liquid from the apex of the chest, or, more correctly, from the compressed lung in the apex of the chest, and as the drops fall through the air in the chest they strike the surface of the watery effusion with a tinkling sound (Fig. 85).

Rales are often removed or altered in character, if not crepitant, by coughing.

It has already been pointed out that dry rales may be divided into the coarse and sonorous and the small or fine sibilant rales. They are produced by the passage of the air, in the large or smaller

bronchial tubes, through partly inspissated and sticky mucus. If they are sonorous, the larger tubes are the part involved; if sibilant, the small bronchioles are affected.

If a cavity has formed and liquid is in it, we may hear in the chest a peculiar hollow tinkling, called by Laennec "*metallic tinkling.*" These sounds are sometimes heard over the stomach when this viscus is in motion and contains a little liquid and air.

It should not be forgotten that harsh breath sounds made in the mouth or in the nose may cause the transmission of rough sounds or rales into the lungs, which will mislead the physician in his diagnosis if he thinks they arise in the pulmonary tissues.

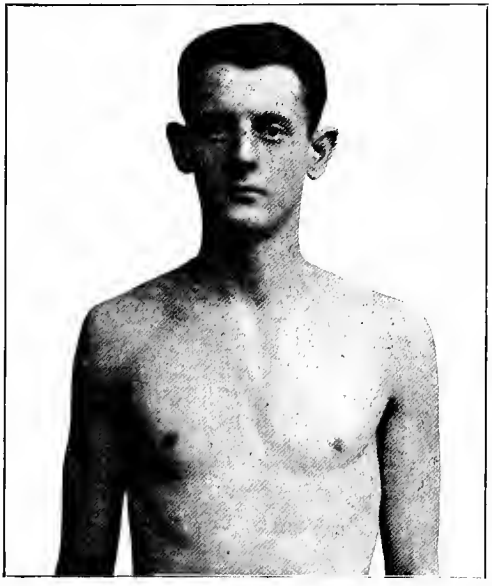


FIG. 84.—Area in which a pleural friction sound is usually heard clearly.

Friction sounds in the chest depend upon disease of the pleura or of the pericardium, generally the former. Normally the visceral and parietal layers of the serous membranes glide over one another noiselessly, but when they become roughened by disease a sound of friction is developed. The friction sound is sometimes so slight as to be almost inaudible, and again so harsh as to sound like a loud creaking, which not only can be heard, but will convey a sensation to the hand when it is placed on the chest. As a rule, friction sounds due to pleuritis are best heard toward the close of inspiration, and occur only in the early stages of the disease, ceasing with the development of the effusion and perhaps reappearing as the effusion

is absorbed. The place where the sound is usually most audible is near the axilla. (See Fig. 84.) An indistinct pleural friction sound may be emphasized, as pointed out by Abrams, if the patient lies on the affected side a few minutes, and then quickly sits up and stops breathing. The physician places his ear to the chest wall, and directs the patient to take a long breath, when the sound is developed.

If a friction sound is heard at the apex of the chest, tuberculosis will often be the cause of its existence.

Care should be taken that fine rales are not mistaken for friction sounds. They can be separated one from the other by the recollection of the facts that rales are modified by coughing, are not affected by deep pressure on the chest wall, and are usually well diffused, while the friction sound is not modified by coughing, is intensified by pressure on the chest wall, and is usually limited to a narrow area.

An excellent imitation of a friction sound is produced by laying one hand over the ear, and then firmly stroking the back of this hand by the fingers of the other hand.

Vocal resonance is closely allied to the sensation called vocal fremitus which is felt on palpation, as already described in this chapter. It is due to the transmission of the voice sounds down the trachea into the bronchial tubes and bronchioles, and thence through the various portions of the lungs. If a stethoscope is placed in the episternal notch while the patient speaks, and the ear of the examiner which is not closed by the instrument is closed by the pressure of his finger, the voice of the patient will be very clearly heard. If the stethoscope be placed between the vertebral column and the scapula posteriorly—in other words, over the bronchial tubes—the voice also will be clearly heard, but not as clearly as over the trachea, for two reasons: first, because the sound has already been divided into the different bronchial tubes, and, second, because the thickness of the chest wall muffles it. If the stethoscope be placed over the anterior part of the chest toward the sides in the area of typical vesicular breathing, the sound of the voice will be still more modified, because the sound, like the air that conveys it, is now minutely subdivided, and the vibrations are decreased by the multitude of vesicular walls. Of course, the degree of transmission of vocal resonance is governed largely by the character of the voice, and for this reason it is more distinct in men than in women.

If the patient being examined is a man and has a well-developed voice, it is usually best to have him speak in a whisper, because the full volume of his voice is so great that it will be heard all over the chest, and the nice differences between the transmission of the sound in the healthy lung and in the diseased area cannot be distinguished. Usually we get the patient to speak by asking him to repeat his name or count "one, two, three." The unemployed ear of the

physician should always be closed by the tip of the finger, and the counting or speaking should be continued only when the physician is actually listening to the chest.

In diseased states of the lung we find the resonance is increased by those changes which aid in the transmission of the sound and decreased by those changes which obstruct its transmission. As pointed out when speaking of vocal fremitus, a solidified lung and the opposite state—namely, a cavity—transmit sound better than healthy tissue, which is partly air and partly lung tissue. We find, therefore, that the vocal resonance, or the sound of the voice of the

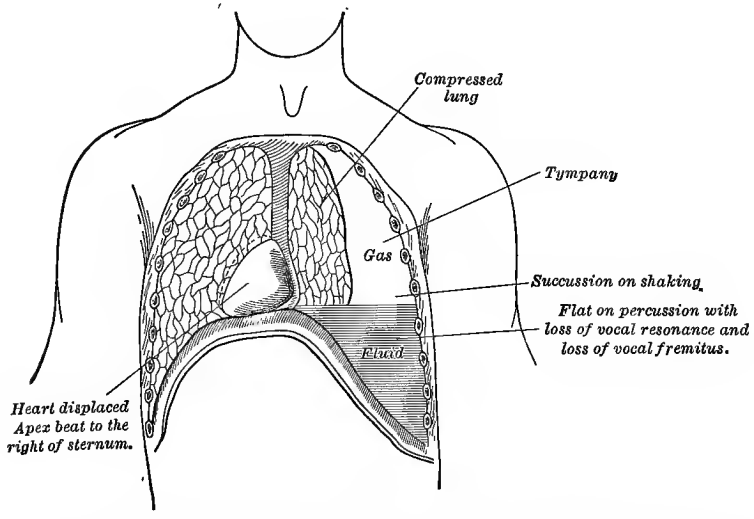


FIG. 85.—Left pyothorax. Metallic tinkling may be produced by the fluid dropping from the apex of the chest into fluid below. (After Maydl.)

patient when he speaks, is increased in pneumonia, in tuberculous consolidation, and in cavity; and is decreased in cases of emphysema, or in cases in which a pleural effusion separates the lung from the chest and deadens sound. Vocal resonance, however, may be increased over pleural effusions, particularly the resonance of the whispered voice. This is called "Baccelli's sign," and Baccelli claims that it serves to separate serous effusions from purulent effusions, because in his experience it is absent in the latter class of cases and present in the former.[1]

When a cavity is situated near the surface of the lung so that the sound of the voice is transmitted to it and from it through the chest

[1] This sign is mentioned here for what it is worth. The writer has never been able to use it with success.

wall with unusual clearness, the sound so clearly heard is called "*pectoriloquy.*" It is usually very marked over a cavity connected with a bronchial tube.

Sometimes when the voice sounds through the chest wall as if it were of a bleating character it is called "*egophony.*" Egophony is usually heard at the angle of the scapula, near the margin of a pleural effusion, and is supposed to be caused by compression and partial occlusion of a bronchus.

Finally, in pyo- or hydropneumothorax, if the ear be placed against the chest and the patient is shaken, we have developed a splashing or slopping sound, called "*Hippocratic succussion.*" (See Fig. 85.) It is not always heard in these cases, and may be developed when a large cavity in the lung is partly filled with liquid.

THE DIAGNOSIS OF RESPIRATORY AFFECTIONS.

The healthy physical signs, and the variations from the normal signs met with in diseased conditions of the lungs, have now been discussed. The next step is to group these various signs with other characteristic symptoms in order that we may obtain a complete picture in the diagnosis of a given disease.

Croupous Pneumonia.—Let us suppose that a patient, previously in health or without any serious pulmonary complaint, is found, after a physical examination of his chest, to have rapid breathing, a somewhat anxious expression, a bright eye, and a dusky flush on one or both cheeks. Palpation discovers a hot, fevered skin, which is dry or more rarely moist, and increased vocal fremitus over both sides of the chest, more marked on one side than the other. Percussion reveals impairment of resonance over the area where fremitus was found most increased, and auscultation in this area shows bronchial breathing, fine crepitant rales, and increased vocal resonance. Under these circumstances we have before us the physical signs of *acute croupous pneumonia.* The pulse is apt to be rapid, but not so fast as the respiration would lead us to suspect, for it is a characteristic of this disease that the respirations are out of proportion to the pulse. The diagnosis is confirmed by the presence of pain in the side affected, by the cough, the rusty, sticky sputum, and the history that the illness was sudden in onset and was initiated by a chill which may or may not have followed exposure. After a few days the rales disappear as consolidation becomes complete in the affected part, and the area which gave impaired resonance on percussion now gives a dull note, while the bronchial breathing in the affected part becomes more marked. The lips are apt to be attacked by herpes. With the fall of temperature, or crisis, which may be reached

by the third to the ninth day, the rales return (*rales redux*) and become more and more loose, coarse, and moist as resolution progresses, until the lung becomes entirely clear, and only a slight roughening of the breath sounds is to be heard. Bad symptoms in such a case are delirium, a feeble pulse, a feeble heart with distant heart sounds, or one in which the action is labored and irregular. Prune juice sputa, or, as the disease progresses, purulent sputa, are bad signs also. If the temperature falls to normal about the fifth day and then rises again, forming a pseudocrisis, the attack will probably be prolonged. When a child is affected by croupous pneumonia it is very common for us to find all the ordinary objective symptoms without any of the physical signs just named. The dulness on percussion is difficult of development, because the chest is so resilient that the percussion blow makes the whole chest resound, and it is noteworthy that percussion of the chest on the diseased side quite commonly develops a high-pitched tympanitic note such as we often find above a pleural effusion.

Care should be taken in all suspected cases of croupous pneumonia that another common cause of the same symptoms does not mislead the physician. This cause or condition is *acute pneumonic phthisis.* In many cases only the finding of tubercle bacilli in the suptum, and the fact that resolution is delayed, and that finally the lung breaks down and cavities are formed, will permit a diagnosis of acute tuberculosis to be made, although the profuse sweats and rapid loss of weight may before this occurs reveal the true state.

There are two areas in the lung often affected very early in pneumonia, particularly of the croupous type, and in pulmonary tuberculosis, which are apt to be overlooked, namely, the axillæ and the septum between the upper and middle lobe on the right side, an area exposed to percussion and auscultation only when the right hand of the patient is placed on top of his head in such a way that the angle of the scapula is drawn away from the vertebral line (Fig. 86). If this is done, the inner border of the scapula will approximate the line of the septum, and along this line there will often be found in tuberculosis of this portion of the lung marked dulness on percussion or, on auscultation, rales, and the other physical signs of consolidation, even though the physician is unable to find elsewhere any evidence of local disease to account for the general systemic symptoms. Very often careful auscultation of the axillary area will also reveal signs not to be found elsewhere, which account for the illness, such as those of pneumonia or pleurisy, for here, as a rule, the friction sounds of the latter affection are best heard.

There is another state that gives dulness on percussion, crepitant rales, and the other physical signs of pneumonia, namely, *pulmonary congestion* dependent upon the action of a feeble heart in the course

of prolonged exhausting fevers or mitral disease; but the history of the illness, the feeble heart, and the development of these signs in the dependent parts of the chest effectually preclude the idea of any acute inflammatory process in the lung.

Finally, we frequently have after a pulmonary infarction an area of consolidation in the lung; but if this be the case, we also have,

FIG. 86.—Area of dulness found in many cases of obscure pulmonary tuberculosis, when the arm is raised so that the scapula no longer covers the septum.

as a rule, a history of recent hemoptysis. This condition is, however, comparatively rare.

The condition of croupous pneumonia cannot readily be confused with any other disease except acute pneumonic phthisis, because of its characteristic symptoms, but catarrhal pneumonia and tuberculosis of the lung often are confused.

Catarrhal Pneumonia.—In catarrhal pneumonia the patient usually presents a history of recent illness of which the pulmonary state is a sequence. The disease rarely begins with the marked and startling symptoms of the croupous form, but is insidious and accom-

panied by a milder but more prolonged fever. Percussion often will not give the positively dull note which can be elicited in croupous pneumonia, and only impairment of resonance may be developed. There are increased vocal fremitus on palpation and increased vocal resonance on auscultation; there are also increased bronchial breathing and more bronchial rales than in the croupous form, for the disease is a bronchopneumonia involving the bronchial tubes and adjacent tissues. The signs are generally diffuse, very often heard best at the bases posteriorly, and clear tubular breathing, such as is heard in the croupous form, is rarely to be found. The sputum is not sticky or rusty; the fever does not end by crisis, but rather by lysis; and the lung returns to its normal state very slowly, its condition often remaining almost stationary for weeks at a time.

The separation of these symptoms of catarrhal pneumonia from those of early pulmonary tuberculosis is practically impossible by the physical signs until the case has progressed to a well-advanced stage, and the sputum should be well watched for tubercle bacilli.

Pulmonary Tuberculosis.—Often catarrhal pneumonia merges into tuberculosis, and very often the diagnosis of catarrhal pneumonia proves to have been made in a case in which the disease is really primarily tuberculosis.

We have to rest the diagnosis of tuberculosis chiefly on the family history, the personal history, the fact that recovery does not speedily take place, and that the patient loses weight more or less rapidly, and, more important than all, the presence as the case becomes well advanced of tubercle bacilli in the sputum, and later on yellow elastic fibers which indicate a breaking down of the lung tissues.

In other instances a patient after complaining of a persistent "cold" with more or less cough fails to get better, and suffers from loss of appetite, loss of weight, and develops chills and fever, also becoming more or less anemic. In such cases it is particularly necessary that the physician be on the *qui vive* for the physical signs of *pulmonary tuberculosis*, as this is the stage in which the disease is still curable in most instances. In such patients a slight prolongation of expiration, with harsh inspiration, and impairment of resonance on percussion, may be the only, yet very important, physical signs.

If the malady be tuberculosis and progressive, we soon find in the chest and sputum signs which make the diagnosis clear. On inspection the costal breathing is less than normal; the hand placed upon it feels, when the patient speaks, that there is not only increased fremitus, but a bubbling feeling from coarse rales, and auscultation also reveals loose rales, the signs of the breaking down of lung tissue. Finally, when a cavity is developed the percussion sound over it becomes high-pitched, and, if the cavity be large,

almost tympanitic, although all around it dulness may be present. If the cavity is large, it will often be found that it is possible to develop a still more tympanitic percussion note if at the same time that the chest is percussed the patient holds his mouth open. This is called "Wintrich's change of note." The breath sounds now become more tubular or amphoric, and vocal resonance may be increased to such an extent that bronchophony or pectoriloquy becomes marked even in that part of the lung in which in health the vesicular sounds are heard most typically (Fig. 88). Prolongation of expiration is also present, and sweats, irregular hectic fever, and great loss of flesh ensue.

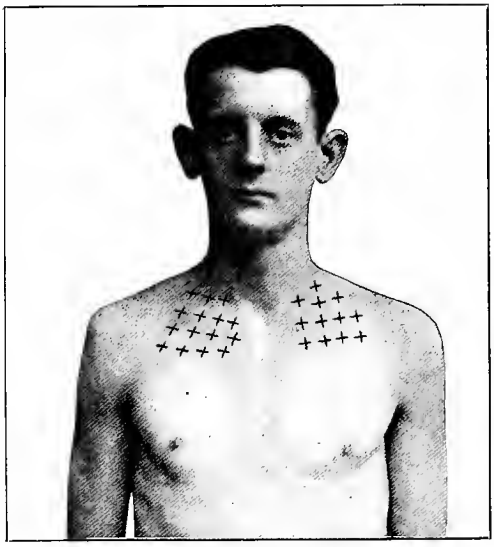


FIG. 87.—Areas of fine moist rales in early stages of tuberculosis of apices of lungs with impaired resonance on percussion and prolonged harsh expiration.

When there is *fibroid phthisis* of the lungs, inspection of the thorax often shows marked retraction of one side of the chest, more often the left side in front near the apex. The supraclavicular fossa and the costal interspaces are apt to be retracted, the scapula drawn nearer the spine, and that side of the thorax is poorly expanded on inspiration. The respiratory murmur is feeble and distant, and resonance on percussion is below normal. If secondary pleural contractions occur, the heart may be drawn from its normal position and the other lung is apt to be emphysematous. This form of pulmonary tuberculosis is usually very slow and lasts for years.

In striking contrast to the form just described is *acute pneumonic phthisis*, which often begins with the suddenness of an acute croupous

pneumonia, with a chill, blood-tinged sputum, and pain in the chest. When the consolidated lung fails to undergo resolution the sputum is examined and tubercle bacilli reveal its true nature. In other cases an attack of true croupous pneumonia sets free tubercle bacilli which have been walled off from the rest of the lung, and so precipitates acute pulmonary tuberculosis.

In some cases of suspected pulmonary tuberculosis the Röntgen rays can be used with great success in aiding difficult diagnosis. It is best to employ the fluoroscope rather than the radiograph, since with the former different parts of the chest can be rapidly compared, and the focus readily changed for deep and superficial tissues. Consolidated areas are dark and shadowy; cavities are light surrounded

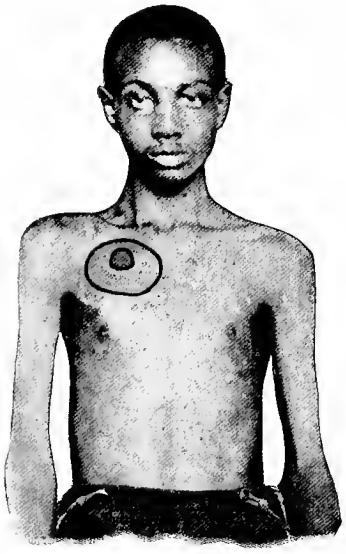


FIG. 88.—Case of pulmonary cavity due to tuberculosis. The central ring is the area giving the physical signs of cavity, with cavernous breathing and whispering pectoriloquy, and the outer ring that of consolidation (dulness), with rapid breaking down of the lung tissue (moist rales).

by shadow. So, too, abscess of the lung produces a distinct shadow in many cases.

Finally, it is to be recalled that mitral stenosis or even regurgitation, may, by producing engorgement of the lung, produce physical signs closely resembling pulmonary tuberculosis, for there are continued cough, dyspnea, blood-spitting, and loose rales on auscultation. The absence of tubercle bacilli, and the presence of large cells filled with brown pigment in the sputum show the cause to be cardiac.

Pulmonary Abscess.—The history of the case and its symptoms are our chief means of separating pulmonary abscess from pulmonary

tuberculosis with the development of cavity, for the physical signs are about the same. In cases of abscess we find that the patient has suffered from pneumonia or from pyemia with embolic infarction. In other cases discharges from the nose and throat entering the lungs produce such lesions. The symptoms of abscess, which separate it from cavity due to tuberculosis, are as follows: in abscess the lesion exists in the lower lobe, as a rule, while the tuberculous cavity is usually found at the apex or in the upper lobe. The constitutional disturbance in abscess is often very slight, whereas in tuberculosis it is usually severe. In abscess the sputum is copious and purulent, and often coughed up in gushes, whereas in tuberculosis it is often scanty, and not markedly purulent, as a rule. Again, in the last-named disease tubercle bacilli may be found, but they are absent in abscess unless tuberculous infection is simultaneously present.

The use of the fluoroscope is of great value in localizing the pus in some cases.

Pulmonary Gangrene.—If the patient has the signs of cavity of the lung, and in addition an exceedingly fetid breath, with great wasting, the case is one of pulmonary gangrene. Gangrene is usually found at the base of one lung, as is abscess. The sputum is usually brownish.

Bronchiectasis with fetid breath is occasionally met with, but the fetor after coughing is never so exceedingly offensive as it is in cases of gangrene.

Pulmonary Edema.—The physical signs of pulmonary edema may develop suddenly as a result of an injury to the vagus, or in acute disease of the kidneys, lungs, or heart. Their onset is insidious, but the rapid breathing, the crepitant rales, the limitation of these signs chiefly to the lower part of the chest, the bilateral character of the signs combined with dulness on percussion, the absence of fever, the frothy sputum, and, it may be, the associated presence of renal or cardiac disease, all point to the true state of affairs. Such a state not rarely develops in the course of pulmonary congestion in old persons, particularly if influenza is the cause.

Pleuritis.—Let us suppose that a healthy man is seized with pain in the thorax and a chill followed by fever. An examination of his thorax will reveal on inspection deficient breathing on the affected side, which is fixed because of pain produced by the inflamed pleural surfaces moving over one another on inspiration. Exaggerated breathing will be found on the opposite side to compensate for this fixation, and auscultation on the painful side will reveal a friction sound, probably best heard in the axilla. (See Fig. 84.)

Pleural Effusion.—The signs of pleural effusion are impaired mobility, or fixation of the affected side and obliteration of the

interspaces where the fluid exists. The chest is flat on percussion at the most dependent part of the pleural sac, namely, at the base of the lung posteriorly. This area of flatness on percussion gradually rises higher and higher until the effusion is completed. It extends anteriorly, and may be demonstrated as well here as it can be posteriorly and laterally, although, if the patient lies on his back or is partly recumbent, the entire anterior surface of the chest may be hyperresonant, owing to the fluid leaving the front of the chest and going

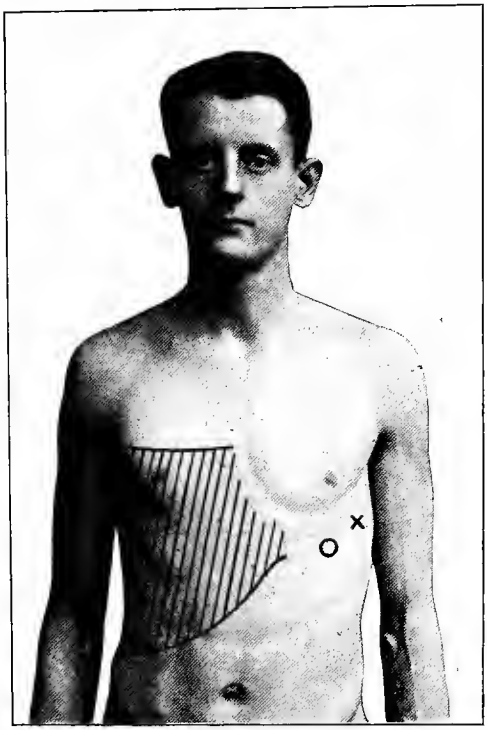


FIG. 89.—Right-sided pleural effusion. The area of flatness on percussion in the thorax merges with that of liver dulness, and the lower border of the liver is below its normal level. The apex beat is displaced to the left at *x*. The circle represents Traube's semilunar space, which is obliterated in left-sided effusion, but not in right-sided effusion.

to the more dependent parts. In other words, in cases of nonsacculated serous pleural effusion changes in the position of the patient cause alterations in the area of flatness on percussion, unless the effusion is large enough to fill the chest entirely, when, of course, it is immovable. Inspection will show an increase in the size of the chest on the diseased side, with bulging of the intercostal spaces.

A curious yet important point in this connection is the fact that

the line where flatness on percussion ceases at the top of the effusion posteriorly is wavy or sigmoid (S-shaped). Above the level of the effusion percussion over the compressed lung gives a somewhat hollow note or hyper-resonance, called "Skodaic resonance," and the sense of resistance to the percussed finger is less at this point than over the effusion where the resistance is great. Percussion and palpation will reveal the lower margin of the liver depressed if the effusion is on the right side. (See Figs. 89 and 90.) Inspection and palpation will show the apex beat of the heart displaced to the right and downward in cases of effusion into the left pleura, and to the left in cases of right-sided effusion. Again, if the effusion be on the left side, it will be found on percussing "Traube's semilunar space," a space directly in the mammillary line and a little below the nipple that the usual tympanitic resonance normally found in this area is extinguished through the downward pressure of the fluid.

In many cases of pleural effusion careful percussion posteriorly at the base of the opposite side of the chest from that of the effusion close to the spine will reveal marked dulness. This area of dulness is triangular in shape with the base of the triangle near the diaphragm and its apex several inches up the side of the vertebral column. This is called Grocco's sign or the "paravertebral triangle of dulness of pleural effusion."

In auscultating the chest in the area in which flatness has been developed by percussion the breath sounds are very indistinct, except in the back near the vertebral column, where there may be marked blowing breathing. If the patient speaks, there will be found loss of vocal resonance and of fremitus over the effusion, but along the margin of the spine on the diseased side there may be heard in some cases bronchophony, or even the bleating voice sound called egophony.

It is a noteworthy fact, however, that the physical signs on auscultation vary with the method of examination which is employed, for if the stethoscope is used the breath sounds are often inaudible, whereas, if the unaided ear is used bronchial breathing may be distinctly heard. The presence of bronchial breathing over an area supposed to contain fluid does not negative this diagnosis if the unaided ear is employed. While it seems paradoxical that sounds can be heard with the naked ear which are inaudible with an aided ear the paradox is explained by the fact that when the stethoscope is used only a very limited portion of the thoracic contents is auscultated.

If the effusion be accompanied by *pneumothorax* (see next page), we will find three sets of physical signs, namely, those of effusion, which will be at the lowest part of the chest, next above this an area in which percussion gives a clear tympanitic note due to the air in the pleural cavity, and above this the physical signs of the compressed

lung in the apex of the chest cavity. In this condition we may also hear succussion or splashing sounds, if the patient is shaken while the physician's ear is against the chest wall, and the metallic tinkling, or dropping sounds, as the fluid falls from the top of the chest cavity into the effusion. (See Fig. 85.) Again, we may use what has been called "coin percussion." This consists in having an assistant place a large silver coin against the chest wall on the diseased side anteriorly, and then the physician listens at the posterior aspect of the chest, his unused ear being closed by his finger. The assistant now strikes the silver coin with the edge of another silver coin. It the

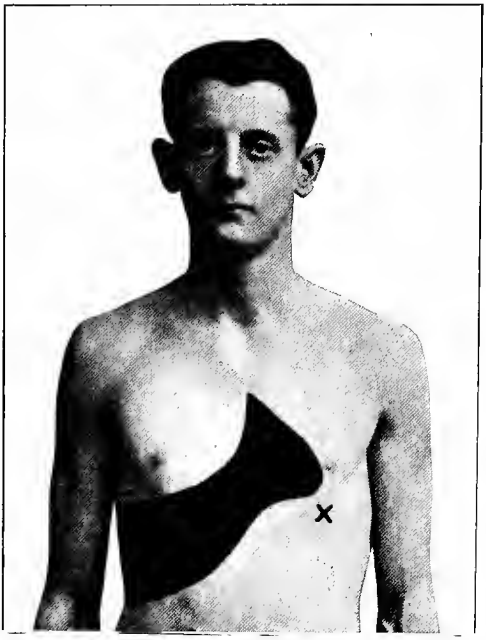


FIG. 90.—Showing at *x* mark the so-called area called Traube's semilunar space, where in health percussion gives a tympanitic note, which becomes flat in left-sided pleural effusion. The solid block represents hepatic and cardiac dulness.

coins be struck together below the surface of the effusion, very little of the metallic sound will be transmitted through the chest. If the coins are struck together at the level of the layer of air, the sounds come through the chest cavity with startling clearness; but if at the level of the lung, they are less clearly heard than at the level of the air, but more so than at the level of the effusion.

The reasons for this are obvious, for the liquid prevents transmission of the metallic sounds, as does also to some extent the compressed lung at the apex of the chest, whereas the space filled with air conveys the sounds directly to the ear,

Finally, if the effusion is absorbed by unaided nature, the area of flatness on percussion becomes less and less from above downward, the expansion of the chest on inspiration increases, the interspaces cease to bulge, and the friction sounds may return for a brief period.

Particular attention should be called to the possibility of pleural effusions coming on insidiously. There is probably no other massive pathological change anywhere in the body so often unsuspected or overlooked, and it is noteworthy that, when pleural effusion is insidious in its onset and devoid of prodromes, it is often due to an undiscovered tuberculosis, whether the exudate be found to be serous or purulent. Again, the fact that tubercle bacilli cannot be found in the effusion when it is aspirated in no way proves that the effusion is not tuberculous in origin, since they are rarely found in the fluid even when tuberculous pleurisy is most active.

Serous pleural effusion, single or double, may occur as part of the dropsical condition in renal or cardiac disease, from disease of the blood itself, or it may result from thrombosis of the vena azygos. The latter cause is particularly apt to come on in patients suffering from typhoid fever or other exhausting diseases. A right-sided effusion may arise from heart disease which primarily enlarges the right side of this viscus which in turn presses upon the root of the right lung and so compresses the vena azygos major.

Transudations can be separated from the effusions due to inflammation by the method of Pohl and Rosenbach. This consists in withdrawing some of the fluid by an aspirating needle after the patient has received doses of iodide of potassium. A few drops of fuming nitric acid are added to the fluid, and it is then agitated with chloroform, when, if the effusion be a transudation, the iodine will be seen of a red color sinking to the bottom of the test tube with the chloroform. If it be an inflammatory exudate, the iodine will not be passed into the effusion.

Additional information may be obtained by the following methods, none of which, however, are positive but only indicative in their results. The first consists in an estimation of the proportion of leukocytes contained in the aspirated fluid (cytodiagnosis). It may be said that if the fluid contains a relatively large proportion of polymorphonuclear cells (60 to 90 per cent.), its exudation has resulted in all probability from infection by the pneumococcus, the streptococcus or the staphylococcus, and that there has been an acute inflammation of the pleura. In such cases small masses of fibrin may be found in the fluid. The number of polymorphonuclear cells and the quantity of flakes of fibrin are, as a rule, in direct proportion to the severity of the inflammation. If the fluid contains a relatively large proportion of lymphocytes, it is probably due to tuberculous

infection. Such findings are only of value as confirming an opinion based upon other points in the history of the case, or those which may have been discovered by a careful study of the physical signs, and they are liable to mislead if too much reliance is placed upon them. Thus, in some cases of tuberculous infection a large proportion of polymorphonuclear cells may be present in the first fortnight during which the effusion has taken place, although later they diminish as the lymphocytes increase. Again, if the infection by the tubercle bacillus is mixed, that is to say if, as so often happens, other microörganisms are active, such as the pneumococcus, then the presence of these organisms may in a case of tuberculosis cause a polymorphonuclear leukocytosis.

The technique advised for this method of examination is as follows:

The fluid having been drawn into a sterile vessel several sterile glass balls are placed in it and shaken to break up any clot which may, by forming, entangle the leukocytes in its meshes. The contents of the vessel is centrifugalized for five minutes, after which the supernatant fluid is entirely decanted, only a few drops remaining in the vessel or test tube. From this remaining fluid and sediment after they are thoroughly mixed by stirring them with a platinum loop, a drop is removed by means of the loop and a smear is made between two cover-glasses as in ordinary blood examinations. After the smear is dry the cover-glass is dipped in alcohol to fix the cells, which are then stained by immersing the glass for thirty seconds in a fluid composed of Wright's blood stain three parts, and pure methyl alcohol one part. The stain is then diluted by adding to the cover-slip a few drops of water, and is allowed to remain in contact for two minutes, when the glass is washed with pure water several times. After this, it is dried by waving it over a Bunsen flame, at such a distance that the fingers can stand the heat. The cover-glass is now mounted in xylol balsam and placed under an oil-immersion lens.

A third method of gaining some idea of the cause of an effusion consists in studying its specific gravity. If the effused fluid has a specific gravity of from 1.010 to 1.020 it is probably the result of an acute infection by the pneumococcus or the streptococcus or staphylococcus; whereas if its specific gravity is as low as 1.008 it is probably a transudate.

A fourth method nearly related to that just named is the estimation of the albumin content of the fluid. There are several methods by which this estimation can be made. Only one of them lends itself to employment by the practitioner, namely, that of Brandberg. That of Kjeldahl and that of Reuss are too complicated.

Brandberg's method consists in a determination of the rapidity

with which cloudiness appears at the point of contact of nitric acid and the fluid tested as in urinary analysis. When the cloud develops in from two and a half to three minutes the fluid contains about 0.033 per cent. of albumin. If more albumin than this is present the cloudiness develops in a shorter time, and if less than this is present a longer period of time elapses before the cloud appears. Effusions due to inflammatory processes contain 4 per cent. or more of albumin, whereas those due to transudation contain from 1 to 3 per cent. A fallacy underlying this method depends upon the fact that an inflammatory process may be responsible for a part of the fluid when another part is the result of transudation resulting from venous stasis produced by pressure arising from the inflammatory process. In this manner a low albumin content is developed.

A fifth method is by determining the freezing point of the fluid. This requires so much apparatus and special skill that it is not practical for the average physician. This is called "cryoscopy."

A sixth method is called "inoscopy," or the determination of the presence of tubercle bacilli by bacteriological methods that do not lend themselves to the hand of the general practitioner since the fluid and its contents requires careful treatment before the staining for tubercle bacilli is attempted.

If on aspirating the fluid in the chest it is found to be hemorrhagic in character, the cause may be one of the diseases which produce marked asthenia, notably carcinoma, nephritis, one of the acute infectious diseases in a malignant form, or tuberculosis. The cancer may or may not be in the chest. Rarely such an effusion occurs in otherwise healthy men without these causes. The possibility of the hemorrhagic effusion being due to a leaking aneurysm, or to leakage from an ulcerated bloodvessel in tuberculous disease of the lung is to be remembered.

Sometimes *interlobar pleurisy* develops and effusion of fluid or of pus takes place in such a position that it lies between the two lobes without escaping into the general pleural cavity. Under these conditions the symptoms of an ordinary pleuritis may be present, and the physical signs will consist of an area of flatness on percussion, which is sharply outlined, and is bordered *above* and *below* by an area of high-pitched resonance on percussion, and tubular breathing due to compression of the lung.

Empyema.—If the effusion is purulent, the patient is apt to lose flesh and strength, to have chills, fevers, and sweats, and to present all the evidences of an accumulation of pus in some part of the body. Particularly is this result apt to follow a pleurisy complicating one of the acute infectious diseases, such as scarlet fever, typhoid fever, pneumonia, and in many cases in which tuberculosis is responsible for the illness. In children empyema is not so serious as it is in adults.

In the former it is usually due to the pneumococcus, in the latter to the streptococcus. The following statistics, compiled by Netter, show the frequency with which these organisms produce empyema in adults and children:

	Children.	Adults.
Pneumococcus .	53.6	17.3
Pneumococcus and streptococcus	3.6	2.5
Saprophytic organisms . .	10.7	. .
Staphylococci	.	1.2
Tuberculosis	14.3	25.0
Streptococci	17.7	53.0

Empyema may also be due to the bacillus typhosus, the colon bacillus, the microörganism of influenza, the gonococcus, and actinomycosis.

Pneumothorax.—The development of sudden dyspnea and thoracic pain, with pallor and cyanosis, a subnormal temperature and a rapid pulse, during the course of a case of tuberculosis of the lung in particular, should lead us to suspect pneumothorax. Inspection shows distention of the affected side, bulging of the intercostal spaces, and a sensation of distention of the chest to the physician's hand. The act of respiration moves the involved side but slightly, while the opposite side is moved greatly. The apex of the heart is displaced and percussion gives a loud hollow note. Auscultation of the affected side reveals absence of breath sounds, and the hyper-resonance on percussion with absence of respiratory murmur makes a very pathognomonic combination of symptoms. Metallic sounds are often elicited in this condition arising from unknown causes. It will be found that if the plexímeter is struck with the handle of a percussion hammer, while the physician auscults the chest elsewhere, a clear distinct metallic sound is transmitted to the ear. When a pneumothorax communicates freely with a bronchus we often have gurgling or bubbling sounds due to bronchial secretion, or if pus is in the thorax, we find moist rales in the bronchial tubes and purulent expectoration. It is remarkable how differently patients suffer when affected by pneumothorax. Some are in the most urgent dyspnea, but others after a very short time seem to be able to take some exercise without grave embarrassment.

Bronchitis.—If after exposure to cold there is a sense of soreness in the chest, with more or less oppression and a hard cough, which seems to tear the bronchial tubes, the cough not being associated with expectoration, and the febrile movement but moderate, we suspect the presence of an acute bronchitis; a diagnosis which will be confirmed if we find the following physical signs:

There is marked roughening of the breath sounds all over the chest, particularly over the bronchial tubes at the back, between

the scapulæ, without any increase in vocal resonance and fremitus or any impairment of resonance on percussion. As the disease progresses these sounds of harsh breathing give way to rales, which are at first fine and moist, then coarse and sonorous, as the second stage, or stage of secretion, develops; and, finally, they decrease little by little, as health is approached and the mucus is expelled by coughing. Care should always be taken to determine in examining a case of suspected bronchitis that the symptoms are not due to a bronchopneumonia.

Should the case become chronic, the sounds of coarse and more or less sonorous rales will persist and become constant. Such cases usually become worse in winter, and the sputum is sometimes very profuse (bronchorrhea).

The physician should always be careful in these cases to see to it that renal disease or a feeble heart is not the cause of the bronchial disorder. The health suffers but little in simple chronic bronchitis; but if bronchiectasis develops it may be much impaired.

Too often the careless diagnosis of chronic bronchitis is made when the trouble is due to pulmonary tuberculosis or a feeble heart.

Under the name "putrid bronchitis" we have a state in which the sputum is foul and expelled in a liquid form, in which float little yellow plugs (Dittrich's plugs). This condition may end in pulmonary gangrene or cause metastatic abscess.

Emphysema.—The presence of a barrel-shaped chest, with almost immovable walls and marked abdominal breathing, points to the presence of emphysema of the lungs, and this opinion is confirmed if on auscultation of the chest we find *marked prolongation of expiration*, diminished vocal resonance and fremitus, and increased resonance on percussion. The face is often quite cyanotic, the superficial veins of the neck turgescent, the abdominal respiratory movements abnormally great, and the superficial veins in the epigastrium enlarged. If bronchitis or bronchiectasis is associated with the emphysema, as is frequently the case, we find more or less marked rales all over the chest, particularly posteriorly. Sometimes a systolic murmur can be heard over the tricuspid area, due to regurgitation on the right side of the heart. Cardiac dulness is generally obliterated by the enlarged lung, and the apex beat cannot be felt except in the neighborhood of the ensiform cartilage or in the epigastrium. Both the hepatic and splenic dulness are found to begin and extend lower than normal, owing to the expansion of the lung. We may also find accentuation of the second sound in the pulmonary artery. The tricuspid regurgitation not rarely develops as a result of a damming up of the blood in the right ventricle.

Asthma.—If it is asthma, there will be labored breathing in which all the accessory muscles of respiration in the neck and trunk aid

the ordinary respiratory muscles. The posture of the patient will usually be that of sitting up in bed and somewhat leaning forward. The face will be flushed, the vessels of the face and neck turgid, and the lips may be cyanotic. Often the patient, while sitting up, supports himself by resting on his hands, which are placed at his side in order to raise his shoulders and fix the chest walls for contraction of the muscles which are endeavoring to drive out the air, for it is to be remembered that the respiratory difficulty in asthma depends more upon the fact that the patient cannot empty the lungs than upon the fact that he cannot fill them. As a matter of fact, they are too full of air which has been used.

Inspection not only shows these signs in asthma, but also reveals, in cases in which emphysema has not developed to such an extent as to cover the heart with the lung, that the apex beat is diffused and the heart laboring. Palpation reveals little except when coarse rales are present in large numbers, when some bubbling may be felt.

Percussion usually gives an increased resonance, because the chest is inordinately full of air, and auscultation reveals very loud blowing breathing, musical notes, or squeaking or creaking noises, both on inspiration and expiration. Finally, as secretion begins to be established, musical and cooing rales may be heard, in well-marked cases, all over the chest before the ear is placed against the patient. At first these rales are heard chiefly on expiration, but very shortly they occur equally loudly on both inspiration and expiration.

If on auscultating the chest we find it filled with musical and cooing rales heard in every part, although most marked in the bronchial tubes, we may be fairly sure that an attack of asthma is about passing away; but if, on the other hand, the attack is beginning, the manifest difficulty in breathing, the prolonged expiration, with comparatively few rales, the harsh bronchial sounds, and the general objective symptoms of the case will explain the cause of the pulmonary condition. Toward the end of the attack coughing brings up a limited amount of sputum, which contains Curschmann's spirals and Charcot-Leyden crystals. (See chapter on Cough and Expectoration.)

As asthma is a symptom, not a disease in itself, the physician should always examine the nose, with the object of discovering some source of reflex irritation in the nasal mucous membrane, or test the urine to discover whether renal disease is present, or the heart to determine if a cardiac lesion accounts for the symptoms. Sometimes gastric disorder is responsible for the attack.

Care should be taken that a catarrhal pneumonia developing after an attack of asthma is not overlooked until the patient is dangerously ill.

When a patient is seized with a violent attack of dyspnea its cause

may be asthma, a foreign body in the air passages, or laryngeal spasm.

Foreign Bodies in the Air Passages.—The dyspnea due to a foreign body in the air passages, whether it be a piece of meat or a false membrane, is quite different from that of true asthma, for in this case the difficulty is commonly in the entrance of air. The onset of the attack is usually sudden, and inspection will show that on inspiration the costal interspaces are greatly drawn in, as is also the epigastrium. There will be practically no signs in the chest which are not evidently due to the efforts at forced breathing, and a history of having had a foreign body in the mouth or of some laryngeal disease will usually be obtainable. Obstruction may, however, be present, and the history of a foreign body be absent in cases in which an abscess has burst into the air passages from the mediastinum or through the posterior pharyngeal wall. In such a case, however, there would be, in all probability, purulent expectoration.

Laryngeal Spasm.—Laryngeal spasm producing difficult breathing causes symptoms precisely like those of a foreign body in the larynx, except that in spasm the cough is often constant and is very brassy or ringing. The patient will show by a gesture with his hand that the obstruction is in the larynx, if unable to speak. Such obstruction when seen in children is, as a rule, due to spasmodic croup, and, if so, probably depends upon one of three causes, namely, laryngeal catarrh, rickets, or digestive disturbance. If in an older person, it is probably due to aneurysm pressing on the recurrent laryngeal nerve, to a laryngeal crisis in locomotor ataxia, or to growths in the mediastinum producing pressure on the nerve trunks going to the laryngeal muscles. Sometimes great enlargement of the retrobronchial glands will cause reflex laryngeal spasm.

Tumors of the Chest.—Tumors occur in the chest generally as mediastinal growths, and are most commonly sarcomata or lymphadenomata. There will be found, if the growth be large, evidences of its pressure upon the chest wall, such as bulging and dulness on percussion over the swelling. This level of dulness is unaltered by changing the posture, as it may be in pleural effusion. Generally there will be evidence of pressure on the bronchial tubes, which causes dyspnea, and of pressure on the thoracic vessels, which produces signs of impaired circulation as shown by cyanosis, venous engorgement, and flushing of the skin of the face and neck. Often such growths cause pleural effusions by pressure on the bloodvessels, or produce pulmonary consolidation by causing an exudation in the lung tissue.

The diseased conditions from which it is necessary we should distinguish mediastinal growths during life are as follows: (1) From aneurysm; (2) from abscess; (3) from pleural effusion; (4)

from chronic pneumonia. There are several subdivisions of these diseases that might be made, but to all intents and purposes these are sufficient. Pericarditis may, perhaps, be named as the fifth lesion to be thought of. On general principles it may be said that primary mediastinal growths are so rare that they can be excluded on this ground in many cases.

Deeply seated aneurysm in the thorax is sometimes extremely difficult of absolute diagnosis from tumor that but few rules can be laid down for its differential diagnosis from growths in the mediastinum, for deeply seated aneurysm in this region cannot be said to possess any pathognomonic symptoms. The various portions of the aorta in which aneurysm occurs make its symptoms different in almost every case, and we are forced to rely more upon general conditions than absolute signs. Thus, if a patient has no direct symptoms of aneurysm, and none of those conditions present which we know predispose to such a lesion, such as atheroma of the blood-vessels, or syphilis, or a history of violent exertion or severe toil, we may with a certain degree of assurance look farther for symptoms of mediastinal trouble of another sort than aneurysm. (See Aneurysm, in this chapter.)

Unfortunately, the most common age for aneurysm is much the same as that for mediastinal disease, although mediastinal disease seems to occur more frequently in youths than does aneurysm, or, in other words, is scattered over a wider range of years. The pain in some cases of aneurysm is often more violent than that of any other thoracic lesion except angina pectoris. If the aneurysmal sac be large enough to give a wide area of dulness on percussion, there ought to be an expansile movement. Hemoptysis is not in any way a differential sign, since in the one case it may be due to aneurysmal leakage, and in another to ulceration of small blood-vessels by pressure exercised by a tumor, be it aneurysmal or malignant, or even benign. Aneurysm is relatively the more frequent lesion. (See latter part of this chapter.)

Mediastinal abscess is to be separated from mediastinal tumors with care. In the first place, in abscess we generally have a history of infection, or, if the case be one of cold abscess, it is commonly associated with a history of struma or spinal disease. If the abscess be acute, there is generally the history of pain, followed by a chill more or less severe, and fever; or, if cold, then we frequently have irregular febrile movements, with long-continued anorexia and loss of flesh. Cold abscess, too, is generally in the posterior mediastinum, while acute abscess generally occurs in the anterior space.

Pulsation may frequently occur, owing to the transmission of the aortic or cardiac impulses, and affords no better diagnostic point here than elsewhere. In some cases where the presence of aneurysm

is extremely doubtful and the likelihood of abscess extremely probable, an exploratory needle may be used, either through a hole drilled in the sternum or passed between the ribs; but a careful review of the history of the case should certainly always be made and used as a basis from which to draw conclusions.

AUSCULTATION OF THE HEART AND VESSELS.

On attempting to study the heart sounds, we usually auscult the neighborhood of the apex beat and find, if the heart be healthy, two

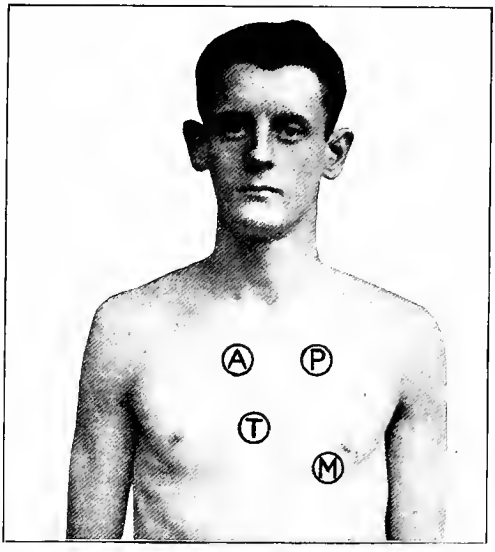


FIG. 91.—Showing the areas in which the various heart sounds are best heard in health. *A* is the area for the aortic valve; *P*, that for the pulmonary valve; *T*, for the tricuspid valve; and *M*, for the mitral valve. The pulmonary circle is a little high.

sounds, occurring one immediately after the other, which resemble "lub dup," the "lub" being the so-called first sound of the heart, produced by the contraction of the heart muscle and the tense valves, and the "dup" being chiefly caused by the slapping to of the aortic valves. After listening in this region we next place the ear over the second right costal cartilage, in order to come as near as possible to the point of greatest intensity of the sound, produced by the aortic valves. If the heart is normal, we find only these sounds, "lub dup," and nothing else. If it is feeble from exhausting disease, from fainting, or by reason of fatty degeneration, we find that the sound "lub" is feeble, and the "dub" sound is also feeble, because the valves do

not slap back into place with as much force as is normal. If, on the other hand, the heart is hypertrophied or stimulated, we find these sounds accentuated, and it is of importance to remember that marked accentuation of the aortic second sound, showing forcible closure of the aortic valves, indicates a condition of high arterial pressure, often the result of vascular spasm arising from chronic contracted kidney or general arterial sclerosis. On the other hand, if the pulmonary second sound at the third left intercostal space is accentuated, it indicates an increase in pulmonary pressure due to impediment to the flow of blood in the lungs. This pulmonary second sound is

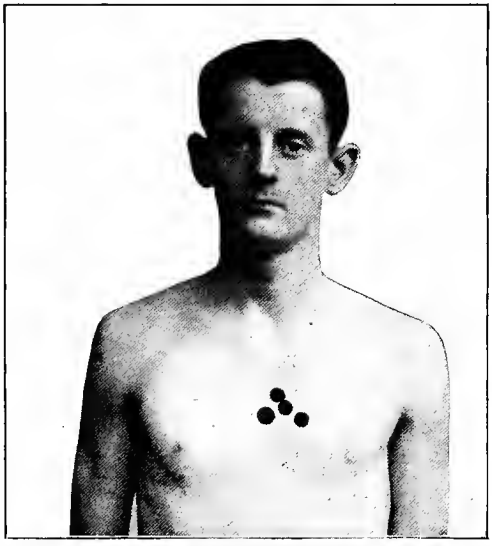


FIG. 92.—Approximate positions of the valves of the heart. To the left the mitral, in the centre the pulmonary, to the right the tricuspid, uppermost the aortic. Compare with this the figure showing where the sounds of these valves are actually best heard (Fig. 91).

markedly accentuated in both mitral obstruction and regurgitation and in some cases of pneumonia and emphysema.[1]

The sound produced at the various orifices of the heart are heard best at or near the following points (Fig. 91), although the approximate positions of the valves are shown in Fig. 92. The mitral valve is heard best at the apex beat; the aortic valve at the second right costal cartilage, the tricuspid valve over the sternum on a line drawn from the third left intercostal space to the fifth right costal cartilage, and the pulmonary valve at the third left intercostal space. All the heart sounds may be reduplicated in health and in disease. When

[1] By accentuation, I do not mean necessarily any increase in the loudness or volume of the sound, but an increase in its sharpness.

the first sound is reduplicated, it probably is due to asynchronism of the ventricles, and when the second sound is doubled there is asynchronous tension of the two sets of sigmoid valves. If disease of the valves be present, we are apt to find reduplication of the second sound in cases of mitral stenosis and in pulmonary disease producing an abnormally high tension in the pulmonary circulation. Such reduplication is also seen in some individuals suffering from aortic stenosis.

The Significance of Heart Murmurs.—Supposing that on listening to the heart in the mitral area—that is, in the neighborhood of the apex beat—there is heard in place of the normal sounds ("lub dup"), or with them, a murmur. What does it mean? It means that, friction sounds being excluded, either valvular disease, a relaxed mitral orifice, aneurysm of the aorta, or marked anemia is present. Still more rarely the sound may be due to what is called a *cardio-pulmonary* murmur, which may occur at any time in the cardiac cycle. This sound is produced not by the movement of the lung in the respiratory act, but by the movement of the lung by the action of the heart, a movement which occurs approximately seventy times a minute, instead of fourteen times, as it would if respiratory. The sound is produced, not in the heart, but by the displacement of air in the lung, and the murmur can often be arrested or altered in character by changing the posture of the patient. The absence of any signs of cardiac difficulty, such as are met in true cardiac disease, and the fact that holding the breath on expiration or inspiration may stop the murmur, aid us in suspecting that the murmur is one of the cardio-pulmonary type, particularly as it is apt to be musical in character.

The *anemic murmur* is particularly apt to be heard in the case of a feeble child suffering from chorea, or, if in an adult, in association with other signs of disorder of the blood, which should make the physician suspect this condition to be the cause. Further than this, an anemic murmur is apt to be soft and purring, and associated with rather feeble heart sounds, probably due to the fact that the heart muscle is not well nourished; such a murmur will generally be found most marked at the left margin of the sternum near the third interspace.[1] (See chapter on Blood for illustration.)

Having found that there is a murmur, and having excluded the causes just named, it is now necessary to determine at what orifice of the heart it is produced, and the rule is to be remembered that a murmur is nearly always heard loudest at about its point of origin. We therefore place the ear over the aortic cartilage (second right). If the murmur be mitral in origin, it will not be heard at this place, unless it be so loud as to be transmitted from the apex. If it is aortic

[1] It must not be forgotten that murmurs due to endocarditis also are frequently found in choreic children.

in origin, it will be louder here than at the apex. If it is tricuspid, it will be loudest in the tricuspid area; if pulmonary, loudest at the pulmonary area (Fig. 91). As murmurs at the tricuspid and pulmonary valves are rare, we nearly always have to deal with aortic or mitral murmurs, or both. In this way, therefore, we can determine the origin of the murmur, and that it is a mitral or an aortic murmur.

Mitral Murmurs.—Let us suppose that it is mitral. We must determine whether it is that of mitral regurgitation or obstruction, or, as they are also called, incompetence and stenosis.

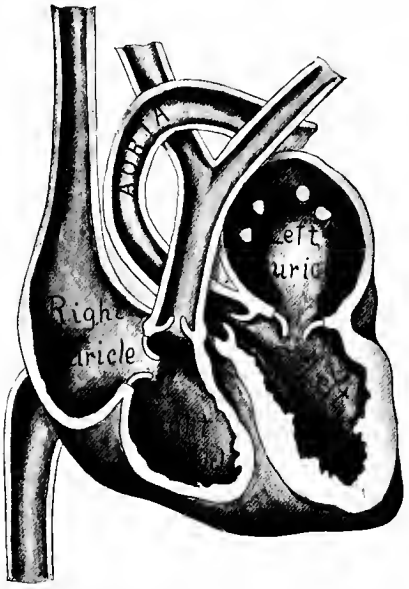


FIG. 93.—Diagram modified from Page to show the relation of the various valves. A study of this diagram will render clear the time of the various cardiac murmurs. Thus in mitral regurgitation the blood passes back from the left ventricle to the left auricle during systole, and is dammed up in the pulmonary veins, the openiugs of which are seen in the auricular wall, producing pressure on the pulmonary valves, the sound of which is thereby accentuated.

The probabilities are that it is the *mitral regurgitation*, because this is by far the most common murmur heard in the heart; and if to this probability we add the fact that it is transmitted well into the axilla, and even heard at the angle of the scapula, our diagnosis is greatly aided, for this is the area of transmission of the murmur of mitral regurgitation (Fig. 94). The most important diagnostic point, however, is the discovery that the murmur occurs *simultaneously with the first sound of the heart*, or with systole—that is, with the apex beat or the carotid pulse. If it does, and the other signs of mitral disease are present, it is almost certainly one of mitral regurgi-

tation. This murmur occurs with the first sound, or systole, because the ventricle in contracting drives most of the blood in the normal direction into the aorta, and also forces some of it back through the left auriculoventricular orifice into the auricle, causing a regurgitant murmur. (See Fig. 93.) Sometimes there will be found in such cases a very marked accentuation of the second sound at the pulmonary orifice, due to the increased pressure in the pulmonary veins by reason of the blood, which has been dammed back into them by the distention of the auricle due to the blood which regurgitates into it. The area of greatest intensity of the mitral regurgitant murmur is shown in Fig. 94.

FIG. 94.—Showing at x the apex beat where the murmurs of mitral regurgitation and obstruction can be best heard. The arrow pointing to the axilla indicates the direction in which the regurgitant murmur is transmitted, and the arrow pointing to the sternum the direction of transmission of the obstructive murmur.

In adults inspection and palpation will rarely reveal much of a thrill over the precordium in mitral regurgitation, but in children this thrill is rarely absent and is usually well marked. Percussion will show that the area of cardiac dulness (see earlier part of this chapter) is broadened, extending beyond the right edge of the sternum and to the left of the mammillary line.

If the pulse is irregular and asynchronous with the heart beats, the heart enlarged by dilatation, and the urine scanty, we recognize that compensation is lacking and treatment needed.

In the diagnosis of the mitral regurgitant murmur the physician must not be misled by a loud aortic systolic murmur transmitted down the sternum to the area of the apex beat.

If the murmur is due to *mitral stenosis*, it will be found that it does not occur with systole, but just before it, and is not transmitted into the axilla, but to the right, over to the midsternal line, and it is presystolic in point of time (Figs. 94 and 95). This murmur can often be exaggerated by placing the patient in a prone position, and occurs before systole, or the first sound, because it is produced by the blood passing through an obstructed left auriculoventricular orifice, and, as the ventricle does not contract (systole) until it is filled, the murmur must be made while it is filling,

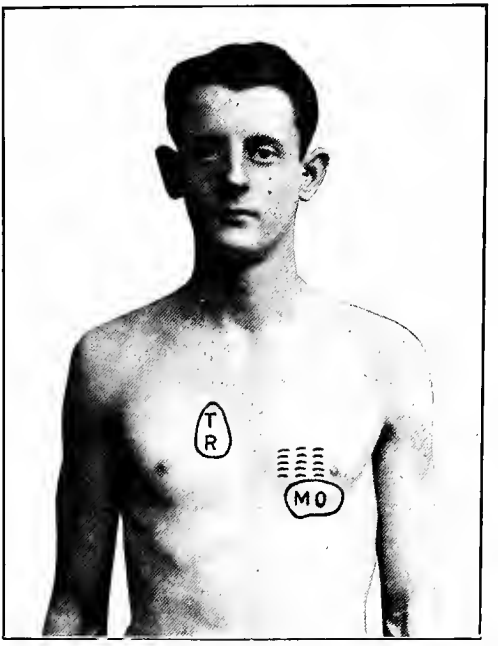


FIG. 95.—MO shows area of greatest intensity of a mitral obstructive murmur. TR shows area of greatest intensity of a tricuspid regurgitant murmur; both lesions were present in this patient. The fine lines indicate the area in which is felt the characteristic thrill of mitral stenosis. (From the author's wards.)

and so is presystolic in time. Palpation of the precordium in such a case will usually reveal a marked diastolic thrill in the fourth or fifth interspace. If the compensation of the heart in a case of mitral stenosis is broken, these signs are accompanied by a very irregular action of the heart, the first sound becoming accentuated and the murmur disappearing or being inconstant, because the auricle is too feeble to drive the blood forcibly through the orifice. In some cases what is called a "gallop rhythm" develops, the heart sounds being reduplicated in such a manner as to make a sound somewhat resem-

bling that made by a horse's feet when it is galloping. If in addition to these physical signs the physician finds cyanosis, jugular distention, congestion of the lungs and liver, and dyspnea, indicating a failing heart, for the venous system is engorged and the arterial system starved, the diagnosis of mitral stenosis with failure of compensation is established.

In the diagnosis of mitral obstruction the physician must not be misled by the possible presence of what is known as Flint's murmur, a presystolic murmur heard in the mitral area, and due to relaxation of the mitral valves, which are thrown into vibration during diastole by blood regurgitating from the aorta in aortic regurgitation.

Aortic Murmurs.—If, however, we have found the murmur to be aortic in origin, we must determine whether it is that of aortic regurgitation, aortic roughening, aortic obstruction, or aneurysm. *Aortic obstruction* or *roughening* is so common that it must be excluded from the diagnostic possibilities before any further steps are taken.

This murmur occurs with the systole of the ventricles, and the carotid pulse and apex beat; it is harsh, as a rule, and is transmitted up into the carotid, and it may be into other arteries of less importance (Fig. 96). It is produced by the contraction of the ventricle driving the blood through a narrowed or roughened aorta or aortic orifice. A similar murmur may arise from vegetations on the aortic valves. Considerable hypertrophy of the left ventricle is usually produced, and the apex beat is strong and forcible if compensatory hypertrophy is present.

As a matter of clinical fact, true aortic obstruction, due to vegetations on or contractions of the aortic valves themselves, is always associated with a certain degree of aortic regurgitation, even though the regurgitant murmur may not be discoverable. Aortic systolic murmurs entirely free from an aortic regurgitation are really due to aortic roughening produced by atheromatous plaques on the aortic wall.

If the murmur occurs after the systole or the apex beat and is aortic, the murmur is that of *aortic regurgitation,* and is called the diastolic aortic murmur. It is heard loudly at the aortic cartilage (second right), often equally marked to the left of the sternum, and it is transmitted down along the sternum very clearly and into the left ventricle, so that it is plainly heard at the apex (Fig. 97). In this condition we have usually marked dilatation of the heart with hypertrophy (the so-called "ox-heart"), and a peculiar trip-hammer pulse (see chapter on Pulse), sometimes called the "water-hammer" or Corrigan pulse. This murmur is due to incompetence of the aortic valves, which allow the blood to regurgitate into the heart after it is driven out into the aorta. If in association with this murmur we find irregularity of the action of the heart, a lack of sharpness

in its sounds, displacement of the apex beat downward and to the left, extension of cardiac dulness to the right, and a feeble pulse, then we know that the heart is failing. Finally, a soft mitral systolic murmur and the development of rales in the chest at the base of the lungs show still greater failure, the result of dilatation of the mitral orifice, which is the result of the strain produced at this area by the constant regurgitation and consequent dilatation of the left ventricle. The objective symptoms of valvular disease are described later on.

FIG. 96.—Showing the area of greatest intensity and the direction of transmission into subclavian and carotid arteries of the aortic obstructive murmur.

FIG. 97.—Showing the area in which the murmur of aortic regurgitation can be most clearly heard.

Aortic Aneurysm.—The characteristic symptoms of *aortic aneurysm* vary greatly with the site of the lesion, as will be pointed out below. The most typical signs are a "bruit" or angry murmur, systolic in point of time, thrill over the growth, dulness on percussion over the area of this thrill, dyspnea, cardiac hypertrophy, and functional disturbance of the heart. Finally, a history of alcoholism, syphilis, and severe strain or injury will be a valuable indication of a causative factor. It is to be remembered that small aneurysms deeply situated, which do not press upon other organs, may produce no symptoms for years, and finally be discovered only at autopsy. When the ordinary signs of aneurysm are not clear, an examination of the radial pulses may reveal that one of them is delayed, and this is a confirmatory sign of aneurysm.

Although the general symptoms of aortic aneurysm have just been

described, there are others which depend upon the seat of the aneurysm, and which materially modify the importance of the points so far named in diagnosis. Let us suppose that a patient presents himself with great engorgement of the vessels of the head and neck and arm of the right side, with perhaps edema of that arm. The heart may be pushed downward and to the left and the voice may be lost or partially impaired by pressure on the recurrent laryngeal nerve of the right side. The pupil of the eye may be widely dilated through irritation of the sympathetic, and there may be unilateral pallor of the face from this cause. This influence of the cervical sympathetic has

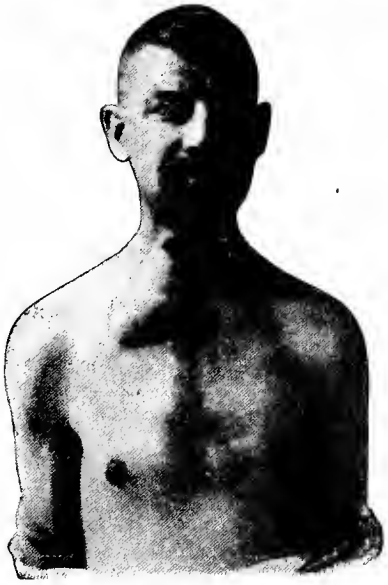


FIG. 98.—Case of aortic and innominate aneurysm, with erosion of the clavicle and ribs from the author's wards in the Jefferson Medical College Hospital. This case is of extraordinary interest because this picture was taken thirty-five months after an arrest of the growth of the aneurysm by electrolysis.

recently been denied and the condition is ascribed to syphilis, which often causes mydriasis, but this theory does not explain the fact that the mydriasis and aortic lesion are on the same side. If the pupil is contracted, then the ciliospinal fibers are paralyzed by pressure. In such a case pain is apt to be a prominent symptom and so severe as to be almost like that of true angina. Percussion over the second right interspace will give impaired resonance, and auscultation of the area of the pulmonary valve may show a pulmonic systolic murmur, due to pressure on the pulmonary artery, which in turn causes hypertrophy and dilatation of the right ventricle. There may be

bulging of the first, second, or third interspace on the right side. Generally such symptoms will be due to an aneurysm of the greater curvature of the ascending aorta, although they may be due to a tumor in the anterior or middle mediastinum; but the expansile pulsation, the bruit, and the history of the case will usually make the differentiation possible.

Again, let us suppose that the patient has a ringing, brassy cough, difficulty in swallowing, and expansile pulsation in the episternal notch and epiclavicular space of the left side, and dulness on percussion over the first and second left intercostal spaces. The left side

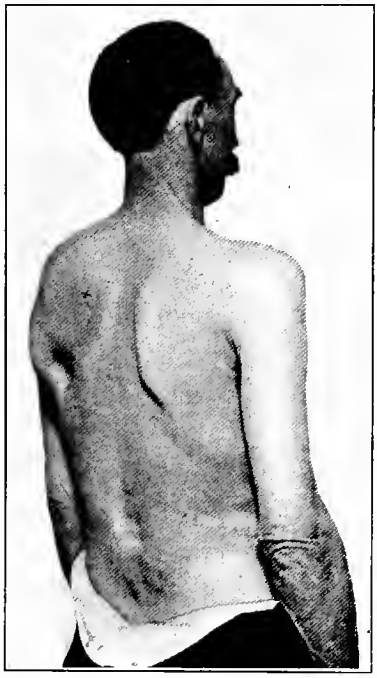


FIG. 99.—Pointing of an aneurysm of the descending part of the aorta between the vertebræ and shoulder blade.

of the face and neck may be engorged from pressure on the left innominate vein. Pupillary symptoms similar to those already named may be present. There is difficulty in breathing, particularly on inspiration, owing to the pressure of the growth on the trachea, the paralysis of the left vocal cord, and the pressure on the left bronchus, and there is dysphagia due to pressure on the esophagus. The voice is altered from paralysis of the vocal cord due to pressure on the left recurrent laryngeal nerve. (See Fig. 100.) These symptoms indicate a lesion of the transverse arch.

If, however, none of these important signs are present anteriorly, we must search for some of them posteriorly, particularly the bruit and the expansile pulsation, and, if these are found to the left of the vertebral column opposite the angle of the scapula, we can rest assured that the aneurysm involves the descending aorta. Severe intercostal pain is often felt in these cases.

In this connection it is well to recall the fact that aneurysm of the ascending portion of the aortic arch is by far the most common condition. Out of 953 cases of aortic aneurysm, analyzed by myself and one of my former assistants, Dr. Holder, no less than 570 were situ-

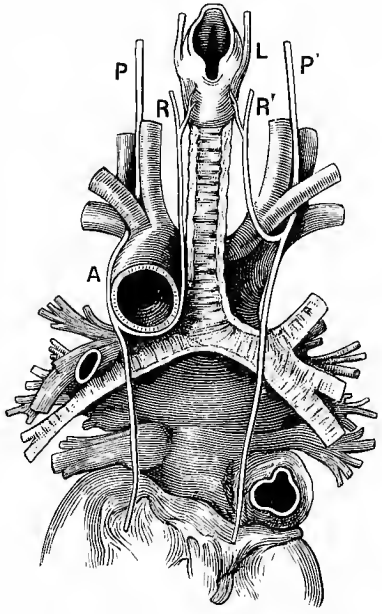


FIG. 100.—A posterior view, showing how an aneurysm of the transverse aorta presses on the recurrent laryngeal nerve. The cut shows the relation of the aorta, where it is surrounded by the loop of the recurrent nerve, to the left bronchus and the lower part of the trachea. *A*, aorta; *P*, pneumogastric nerve; *R*, recurrent nerve; *L*, larynx. (Dieulafoy.)

ated in the ascending part of the arch; of these, 544 were sacculated and 26 fusiform. Aneurysm of the transverse arch occurred 104 times, and of the descending part 110 times. Of the 544 cases, 400 were in males.

There are other symptoms connected with aneurysm which should not be overlooked. The first of these is "tracheal tugging," a sign which is found in some cases but not in all. The patient being in the erect position, the fingers of the physician grasp the cricoid cartilage and gentle upward traction is produced. If aneurysm is present, a distinct tug will be felt with each beat of the heart. It

is important for us to remember, however, that this sign of aneurysm is not pathognomonic, for, as Sewell has pointed out, it may occur in patients who have adhesions of the left pleura or diminished extensibility of the lung, or both combined. Further, in healthy persons the descent of the heart with the diaphragm on deep inspiration may press the aortic arch on the left bronchus, and so transmit to the trachea a tug not due to aneurysm. Another sign emphasized by Osler is the loss of pulsation in the peripheral vessels, the result of the loss of the heart's impulse in the aneurysmal sac.

Doubtful cases of aortic aneurysm should be subjected to the use of the radiograph and fluoroscope, since this will often reveal the growth, particularly if the sac contains a firm clot.

Tricuspid Murmurs.—If the examination has shown that the murmur is loudest in the tricuspid area, it is to be remembered that in the vast majority of cases the condition is one of *tricuspid regurgitation*, for tricuspid stenosis is an exceedingly rare lesion. The time of the murmur of the tricuspid regurgitation is identical with that of the mitral regurgitation (systolic), because the tricuspid valves are the counterpart in the right side of the heart of the mitral valves in the left. (See Fig. 93.)

This murmur is best heard in Fenwick's triangle, the base of which extends two inches to the right of the sternum on the line of the sixth chondrosternal articulation, the apex of the triangle being at the level of the fourth chondrosternal articulation. (See Fig. 95.)

Pulmonary Valvular Murmurs.—Actual disease of the pulmonary valve is exceedingly rare, and usually congenital; pulmonary regurgitation is almost never met with. The signs of *pulmonary stenosis* due to congenital defect are cyanosis, hypertrophy of the right ventricle, a systolic murmur at the left side of the sternum, which is not transmitted upward, and a weak pulmonary second sound. The murmurs sometimes heard, and the thrills sometimes felt, in the pulmonary area are generally due to anemia, the puerperal state, or some neurosis, or to congenital narrowing of the pulmonary artery, or to compression of the vessel by the heart. If the last two causes are present, the ventricular septum is usually deficient and cyanosis is noticeable.

The following rules, laid down by Hochsinger, may be used for making the diagnosis of congenital cardiac disease.

1. In childhood loud, rough, musical heart murmurs, with normal or slight increase in the heart dulness, occur only in congenital heart disease. The acquired defects with loud heart murmurs in young children are almost always associated with great increase in the heart dulness.

2. In young children heart murmurs, with great increase in the

cardiac dulness to the right and feeble apex beat, suggest congenital changes. The dulness to the left is only slightly altered. On the other hand, in the acquired endocarditis in children, the left heart is chiefly affected and the apex beat is visible; the dilatation of the right heart comes late and does not materially change the increased strength of the apex beat.

3. The entire absence of murmurs at the apex, with their evident presence in the region of the auricles and over the pulmonary orifice, is always an important element in differential diagnosis, and points rather to septum defect or pulmonary stenosis than to endocarditis.

4. An abnormally weak second pulmonic sound associated with a distinct systolic murmur is a symptom which, in early childhood, is to be explained only by the assumption of a congenital pulmonary stenosis, and possesses, therefore, an importance from a point of differential diagnosis which is not to be underestimated.

5. Absence of a palpable thrill, despite loud murmurs which are heard over the whole precordial region, is rare, except with congenital defects in the septum, and it speaks therefore against an acquired cardiac affection.

6. Loud, especially vibratory, systolic murmurs, with the point of maximum intensity over the upper third of the sternum, associated with a lack of marked symptoms of hypertrophy of the left ventricle, are very important for the diagnosis of a persistence of the ductus Botalli (ductus arteriosus), and cannot be explained by the assumption of an endocarditis of the aortic valve.

Associated Murmurs.—In the diagnosis of all murmurs in the heart we must remember that several orifices may be diseased, producing associated murmurs. Some discussion as to the relative frequency of these associations has arisen, but the results of H. J. Smith derived from the London hospitals are usually accepted as correct. His results are as follows, in the order of frequency and association:

1. Aortic regurgitation and stenosis and mitral regurgitation.
2. Mitral stenosis and regurgitation.
3. Aortic stenosis and mitral regurgitation.
4. Aortic regurgitation and mitral stenosis.
5. Aortic regurgitation and stenosis.
6. Aortic regurgitation and stenosis; mitral stenosis and regurgitation.
7. Mitral regurgitation and tricuspid regurgitation.
8. Aortic regurgitation and stenosis; mitral regurgitation; tricuspid regurgitation.
9. Mitral stenosis and regurgitation; tricuspid regurgitation.
10. Aortic stenosis; mitral stenosis and regurgitation.
11. Aortic regurgitation; mitral stenosis and regurgitation.
12. Aortic stenosis; mitral regurgitation; tricuspid regurgitation.

13. Aortic regurgitation and stenosis; mitral regurgitation; pulmonary regurgitation.
14. Aortic stenosis and regurgitation; mitral stenosis.
15. Aortic regurgitation; mitral stenosis.
16. Aortic regurgitation; mitral regurgitation; tricuspid regurgitation.
17. Mitral stenosis; tricuspid regurgitation.
18. Aortic stenosis; mitral stenosis and regurgitation; tricuspid regurgitation.
19. Aortic stenosis; mitral stenosis.
20. Aortic regurgitation and stenosis; mitral stenosis and tricuspid regurgitation.
21. Aortic regurgitation; mitral stenosis and regurgitation; tricuspid regurgitation.
22. Aortic regurgitation and stenosis; mitral stenosis and regurgitation; tricuspid regurgitation.
23. Aortic regurgitation and stenosis; mitral stenosis and regurgitation; tricuspid stenosis and regurgitation.
24. Aortic stenosis; pulmonary stenosis.
25. Aortic stenosis; mitral stenosis and regurgitation; tricuspid stenosis and regurgitation.
26. Mitral stenosis and tricuspid stenosis.

The relative gravity of heart lesions is, according to Walsh, as follows, the least dangerous being placed last and the most dangerous first:

Tricuspid regurgitation.
Mitral obstruction and regurgitation.
Aortic regurgitation.
Pulmonary obstruction.
Aortic obstruction.

Pericardial Friction Sound is, of course, heard best in the precordium at the base of the heart—that is, at about the third rib. It is separated from pleural friction by its frequency and by the fact that it continues when the patient holds his breath. (See Fig. 101.)

Laennec likened this friction sound to the noise made by the leather of a new saddle when used for the first time. Sometimes it sounds like the crunching of dry snow under the shoe. It is usually a to-and-fro sound.

General Symptoms of Valvular Disease with Ruptured Compensation.—The general symptoms, subjective or objective, which a patient suffering from the various forms of valvular lesion presents, in some instances, have not been spoken of up to this point, because it is to be distinctly understood that murmurs produced by any form of valvular lesion may exist with great intensity without there being any systemic disturbance or the patient being conscious of their presence.

On the other hand, the murmur may be so faint as to be almost indistinguishable, and yet the general symptoms of heart disease be very marked. This is because the development of general symptoms depends entirely upon the question of compensation by hypertrophy. If there is a leak in a valve or a constriction of an orifice, this leak or obstruction must be overcome by compensatory hypertrophy of the heart muscle. If the heart muscle can make up for the regurgitation or obstruction by increased effort, the circulation is unimpaired; but if it cannot do so, we have developed more or less rapidly, according to the lesion present and the condition of the heart muscle, characteristic symptoms.

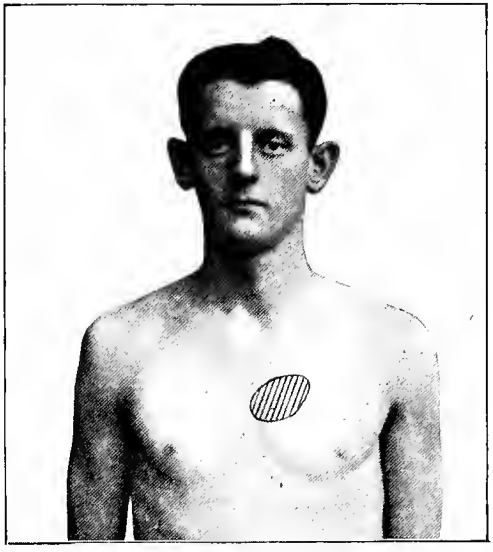


FIG. 101.—Area in which pericardial friction sound is best heard.

Let us suppose that the valvular lesion is that of *mitral regurgitation* with failure of compensation. The first and one of the most prominent symptoms is shortness of breath on exertion; the lips and ears do not possess their normal red hue, but are a little bluish; and if the congestion of the auricle and pulmonary veins is great, bronchitis may be constant or attacks of hemoptysis may develop. Palpation of the heart will also be complained of; and if the patient has developed the lesions in early life, the finger tips are apt to be clubbed. If the rupture or failure of compensation is more complete, all these symptoms become more marked, and the shortness of breath even when lying down, becomes most distressing; indeed, the patient may be comfortable only when sitting up. Dropsy of the lower extremities now comes on; the liver becomes enlarged from portal

congestion, and this results in disordered gastric digestion by the catarrh of the stomach which ensues, while the urine become albuminous, not necessarily from any true renal lesion, but as the result of engorgement of the kidneys with blood.

The general symptoms of *mitral obstruction* are identical with those just described. For the differential physical signs see page 249.

The general symptoms of *aortic obstruction* are also much like those described as resulting from mitral regurgitation, but in addition there are apt to be present, early in the process of failing compensation, some lightness of the head, dizziness or vertigo, or faintness, owing to a deficient blood supply to the brain. Very commonly, too, it will be found that in association with the aortic stenosis there also exists mitral regurgitation, which speedily produces in its turn well-marked pulmonary symptoms. Dropsy is very rarely seen in patients with aortic stenosis. On the contrary, they present, as a rule, the lean and poorly-nourished appearance so often found in the adult, well advanced in years, who has atheromatous tendencies in the bloodvessels.

The association of ruptured compensation with *aortic regurgitation* presents more typical general systemic symptoms than any of the ordinary valvular lesions of the heart. In addition to headache, vertigo, and a tendency to syncope associated with palpitation and a sense of cardiac oppression, we often have a great deal of cardiac pain, of a dull, aching character in rare instances, but more often intensely sharp and lancinating, often darting down the left arm, particularly at night. The dyspnea is often extreme, the patient suffering from terrible attacks of shortness of breath and often sitting day and night in a chair with his head resting on the back of a chair placed in front of him. As time goes on the constant struggling for breath exhausts him, and he falls asleep, only to wake in a few moments gasping for air. Long before any of these grave symptoms arise we may, however, find a number of interesting signs of this heart lesion, chief among which is the "water-hammer" or "trip-hammer" or "Corrigan pulse," the throbbing arteries, and capillary pulsation in the skin and mucous membranes is to be seen. The last is best developed by drawing the thumb nail sharply across the forehead, thereby causing a red mark, which can be seen paling and flushing with each beat of the heart (Quincke's sign), or by pressing a glass slide on the inner part of the lower lip, when the same capillary pulsation will be found. Ophthalmoscopical examination will often reveal similar pulsation of the retinal arteries.

Dilatation of the Heart.—Beyond valvular lesions we have a number of other causes which seriously disturb the action of the heart and the general circulatory condition. The first of these is dilatation of the heart, independent of associated valvular disease.

Let us suppose that a man presents himself with a history of shortness of breath on exertion, so great that his activities are greatly reduced and his usefulness impaired. He gives a history that he was well until he made some extraordinary exertion. Since that time his symptoms of heart failure have been marked. He may perhaps have attacks of syncope. Examination of his heart reveals on inspection a diffuse thrill in the region of the apex; but this thrill is too feeble to be felt, though well marked to the eye if his chest is thin. Percussion shows that the area of cardiac dulness is increased vertically and laterally, and auscultation will discover feeble heart sounds. If the dilatation of the muscular portion of the heart is associated with dilatation of the orifices, a murmur may be present, most commonly that of mitral regurgitation, without there being in association any actual disease of the mitral valves. Sometimes tricuspid regurgitation is also found. The first sound, before it becomes very feeble, may be short and flapping like the ordinary second sound. Marked arhythmia of the heart is often present.

The influence of severe strain in producing cardiac disease deserves careful study on the part of the physician. The study can be divided into three parts: The condition of the heart immediately after acute overstrain, the condition after chronic overstrain, and the final condition often met with months or years after the occurrence of the strain.

It is now well known that immediately after severe muscular effort an examination of the heart will often reveal in entirely healthy persons a distinct increase in the area of cardiac dulness, and, not infrequently, a murmur which disappears with rest. The increase in the area of dulness is, of course, due to more or less cardiac dilatation and the murmur to the same causes, since the dilatation results in stretching of the circular muscular fibers governing the mitral or tricuspid orifices, so that even if the valves be healthy they cannot close the orifice (Fig. 102). In some cases this action seems to be in the nature of a safety valve, in others a sign that the heart has been unduly strained. In the first class the murmur disappears at once or very shortly after the exertion ceases; in the other case it persists until after a long period of rest, when the heart has had a chance to recuperate and regain its normal tone. Persons having the latter condition ought to be advised against such forms of exercise, particularly if they are old in actual years or prematurely aged.

But the physician who regards physical strain as being the only kind of strain which is apt to be productive of grave cardiac damage is much astray. It is quite true that sudden cardiac failure due to great nervous or mental shock is not uncommon, but such attacks being chiefly due to vasomotor relaxation are quickly recovered from. On the other hand we meet with hearts which are distinctly

damaged by prolonged nervous worry. I have met with this most frequently in men who have been very successful in business or in their professional life, men whose success has been largely due to great persistency of effort, who have had great mental strains in carrying through some business deal, and have simultaneously used a good deal of physical force in long journeys or busy days on foot at the same time. Not only do such cases develop the various cardiac neuroses, but not rarely such lives seem to bring on the development of actual organic and degenerative changes in the heart muscle and bloodvessels. That these changes are the result of nerve strain more than to infection or exposure is shown by the frequency of these states in the officers of great corporations and their infrequency in the

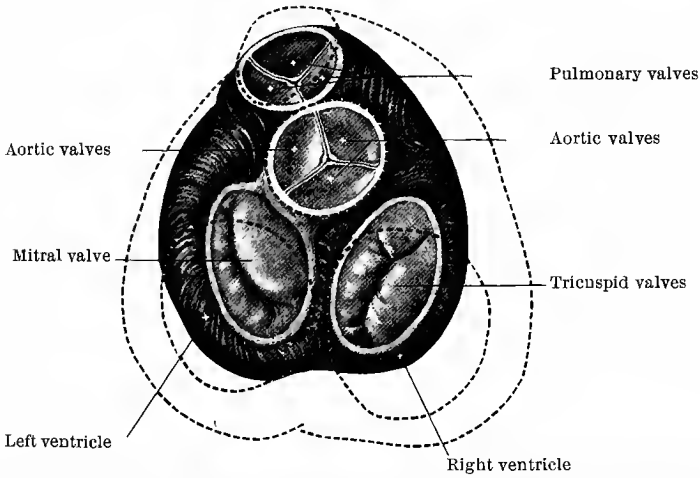


FIG. 102.—The dotted lines show the dimensions to which the heart and its orifices increase in diastole, and in pathological dilatation this may be permanent. (Modified from His and Spalteholz.)

employes who perform the manual labor. There can be no doubt that one cause of this lies in the great increase in the heart action required by mental effort, accompanied as it is by nervous stress without the compensation of long periods of rest, of outdoor exercise, and fresh air. Recent studies with instruments of precision have shown that increased mental activity causes circulatory activity to a greater degree than is generally thought. Such persons run at a high tension for years, develop a state of chronic vascular spasm and high arterial tension, and break down when the heart finds itself tired out with the stress laid upon it. These patients, too, when failure of energy first begins to creep upon them, use their will power to drive them to greater effort, and often very unwisely use stimulants to act as a spur to greater effort.

Closely associated with these patients is that class who suffer from prolonged feebleness of the heart after an attack of true influenzal infection, a feebleness which may last for many years. In many of these patients it is most difficult to determine how much the feebleness of the heart depends upon real changes in its muscular fibers and how much upon lack of proper nerve supply to this organ. Where there is a history of previous cardiac difficulty there can be little doubt that the poison of this disease magnifies it and produces far too often a permanent increase in the disease. In other instances where the heart is primarily healthy the condition is usually a fleeting one, although it is not to be forgotten that undue muscular activity on the part of the patient soon after, or during, an attack of influenza may be provocative of permanent cardiac incompetency.

What the exact changes in the heart are under these circumstances has not, so far as I know, been determined, but during life we certainly meet with them clinically. Such patients often suffer from faintness and precordial distress on exertion, and physical examination usually reveals a feeble and distant first sound, and a second sound lacking in tone because of lack of force in the systole of the heart. These patients also have edema of the lower extremities, sometimes a more or less decided trace of albumin in the urine, but no casts unless there be associated renal lesions, the albuminuria being due to stasis in the kidney. They are often pallid and relaxed, and really need rest and hydrotherapeutic measures more than the internal use of drugs. Such patients, too, particularly if they are advanced in years, not rarely progress rapidly into a gradually increasing circulatory feebleness and so to death.

There is another heart condition closely allied to that just described, at least in one sense, namely, the feeble heart of tuberculosis. Too little attention is paid to this organ in this disease, probably because the mind is centred upon the lungs.

Hypertrophy of the Heart.—Again, we have hypertrophy of the heart occurring in persons without valvular lesions, sometimes as the result of excessive and severe toil. It is seen most commonly by the author in medical students, who, during their holidays, devote their time to severe athletic sports, or to much manual labor, and who, on leading sedentary lives in the winter, develop irregular cardiac action, palpitation, and some shortness of breath. Examination of the precordium in such cases shows a forcible impulse of the apex of the heart against the chest wall, some bulging of the chest wall if the hypertrophy be very great, and no murmurs, but in their place heart sounds very much louder than normal. Palpation shows the apex beat to be lower than normal, and on percussion an increase in the area of cardiac dulness is also found.

Cardiac Neurosis.—Again, let us suppose that a patient presents himself with the statement that he has attacks in which he suffers from a very rapidly beating heart. His skin is alternately red and pale, and sweats without cause, but a careful examination of the heart fails to reveal any murmurs or organic abnormality. There are considerable shortness of breath on exertion and marked palpitation and arhythmia. Such a case may be suffering from a condition in which there is some deficient action of the pneumogastric nerve, whereby the heart is not properly controlled, or the irregular cardiac action may be due to sudden vasomotor relaxations, which by dilating the blood paths reduce the normal arterial resistance. (See chapter on Blood Pressure and Pulse.) This is a condition seen in association with some neuroses and very commonly met with in persons who use tobacco to excess. The symptoms of the so-called "tobacco heart" are indeed chiefly those of arhythmia due to pneumogastric disorder.

Bradycardia.—Rarely because of irritation of the vagus nerves or centres a state of bradycardia develops, in which the heart beats very slowly, perhaps only thirty or even as slowly as twelve times a minute. Bradycardia, or great slowness of the heart, may be due to a neurosis of the vagi, and also occurs in some infectious diseases. It is also seen in jaundice.

Stokes-Adams Disease.—When a patient suffers from attacks of extremely slow pulse with vertigo, syncope, apoplectiform or epileptiform seizures, associated with pulsation of the veins of the neck which are often far more frequent per minute than the beat of the ventricle, the condition is called the "Stokes-Adams syndrome." This incoördination of the auricles and ventricles is due to disease of the auriculoventricular muscle bundle of His. Graphic tracings of the apex beat, the auricular beat, and the jugular beat may be made by the use of the multiple sphygmocardiograph of Jacquet. (See Fig. 108.) A lever of this instrument works as does the ordinary sphygmograph of Dudgeon, the other two by columns of air in tubes connected with tambours which are placed on the chest wall. (See chapter on Pulse).

Tachycardia.—One of the most common causes of tachycardia, or rapid heart, is exophthalmic goitre, in which condition we have not only exophthalmos and enlargement of the thyroid gland, but, in addition to the tachycardia, a marked thrill over the carotid arteries, in which vessels a purring murmur of considerable intensity can often be heard. The patient often suffers from considerable nervous excitement or mental depression.

An exceedingly irregular arhythmical action of the heart coming on in the course of an acute infectious disease, or in any state productive of sepsis, points to the possibility of the patient having an

embolism or thombosis of one of the coronary arteries. If the vessel is suddenly plugged, death occurs; but if the process is gradual, an anemic and feeble necrosis or white infarct is produced.

Fatty and Feeble Heart.—Before discussing the signs of so-called fatty heart we must decide what is meant by this term. True fatty heart—that is, that condition of the heart in which this organ has undergone true fatty degeneration—has no pathognomonic signs, so far as the heart itself is concerned. In these instances we base our diagnosis upon the presence of fatty degeneration of the more superficial organs, such as the arcus senilis in the eye,[1] the presence of atheromatous bloodvessels, the feeble heart sounds at all times, and the evident feebleness of the heart on exertion. The history of poisoning by any one of the poisons causing fatty degeneration is also to be sought after in some cases. Marked fatty degeneration is often present in cases of pernicious anemia. It is not possible to make a differential diagnosis from the physical signs between fatty and fibroid heart.

Another state quite distinct from true fatty heart, but with somewhat similar symptoms, is seen in cases in which an excessive amount of fat has been deposited around the heart and between its fibres as well as in or around the other organs of the body. Here there is little or nothing the matter with the heart muscle, except that it is overloaded with a weight of fat.

When a man shows signs of general degenerative changes, has a feeble heart, some dyspnea, and perhaps some edema of the lower extremities, we may conclude that he has, unless valvular disease is discovered, degenerative myocarditis. Valvular disease may, of course, be found associated with the myocardial lesion. Such cases make up the greater number of sudden deaths, called popularly "death by sudden cardiac failure."

Great feebleness of the heart and of the general system, loss of flesh (or sometimes maintenance of weight), and pigmentation of the skin and buccal mucous membranes point strongly to Addison's disease. (See chapter on the Skin.)

Sudden attacks of cardiac feebleness sometimes come on as cardiac crises in locomotor ataxia and in glossolabiopharyngeal paralysis.

[1] Ophthalmologists and many clinicians deny that arcus senilis has any significance of this character. (See chapter on the Eye.)

CHAPTER VIII.

THE PULSE AND BLOODVESSELS.

Feeling and counting the pulse—The condition of the bloodvessels on palpation—The quality, force, tension and volume of the pulse in health and disease.

ONE of the first things that the physician does when he is studying the condition of a patient is to feel the pulse, even if the symptoms which are present do not indicate circulatory disturbance, because the pulse is an index of the condition of the heart as to its power, its valvular action, and its nervous state. The pulse very often gives information of the presence of renal disease, and it will frequently give a general idea of the tone or degree of debility of the patient. By feeling the pulse we also gather valuable information as to the condition of the arteries, and this is a very important part of the diagnosis, for, to use an old saying, "A man is only as old as his arteries;" and if he is sixty years of age and has good vessels, he is, as a rule, younger in health than another man of thirty with bad vessels, because it is by the bloodvessels that the tissues of the body are nourished, and, as life depends upon this process of nutrition, the better the vessels the better the vitality.

When examining the pulse of a patient who is well enough to be up and about, the physician should wait until sufficient time has elapsed after exercise for the pulse to become steady, and the patient should be in a sitting or reclining posture in order to prevent overaction of the heart. This is particularly important in the case of nervous individuals. An entirely erroneous conception of the circulatory state may be reached if this precaution is ignored, and it is well to insist on perfect rest in bed for several hours prior to the examination in grave cases.

Often when called to see a sick child or a nervous woman, who may be sleeping at the moment of the physician's arrival, a true estimate of the pulse can be made without disturbing the patient by gently putting the tip of the finger on the temporal artery where it passes over the zygomatic process. This fact is of considerable importance, because in some patients the excitement of the doctor's visit may produce marked alterations in pulse rate. This artery may also be used for this purpose in cases of tremor, chorea, delirium, or mania, in which the hand is constantly moved about so that the radial artery cannot be felt.

In counting the pulse it is best to count it for the entire minute, or to count it for fifteen seconds and then multiply the result by four. If the pulse is irregular, it is always best to count it for a minute. If the pulse is very irregular and running, and so difficult of counting, the estimate should be made by listening at the precordium for the apex beat.

Before considering the significance of arterial changes and the qualities of the pulse in health or disease, it is well to understand what these are due to and the manner in which the circulation is carried on. The bloodvessels consist of the arteries, arterioles, capillaries, venules, and veins. These vessels all contain blood during life, and the function of the heart is to propel the blood through them. The flow of blood is maintained, first, by the force expended by the heart, and, second, by the elasticity of the bloodvessels. If the bloodvessels of the body become relaxed, as in death, all the blood is readily held by the ones most relaxed, namely, the abdominal, thoracic, and other veins. We find, therefore, that the vessels are only filled with blood when their walls are to a certain extent constricted by the contraction of their muscular and elastic fibers; and that this contraction is maintained by the action of the vasomotor centre in the medulla oblongata, which also controls many minor centres governing small areas of vessels.

The arteries are very elastic in health, and when filled with blood are slightly distended. Behind the column of blood, which being a fluid confined laterally is practically a solid, for fluids are incompressible, is the heart, and in the arterioles are circular muscular fibers, which by their contraction regulate the flow of blood into the capillaries, from which the nutritional processes are carried on. The blood in the arteries is, therefore, subject to three chief pressures, namely, that of the heart behind the column, that of the elastic and muscular arterial walls on the sides of the column, and the resistance of the contracted arterioles in front of the column. By these means blood pressure or tension is maintained. If the heart beats more strongly or the arterioles contract more tightly than normal, the blood stream is under a greater pressure than before. If the heart is feeble or the arterioles lax, the pressure falls, because the blood is not pressed upon behind or obstructed in its flow in front. If the tension is above or below normal, the interchange of food and oxygen and carbonic acid between the tissues and the blood in the capillaries is perverted, for the rate of flow in the capillaries depends largely upon the blood pressure in the arteries. As the capacity of the capillary system of vessels is many times greater than that of the arteries, if the arterioles relax, the capillaries and veins will retain all the blood and it will stagnate and become useless.

The individual pulse beat is not the wave of blood sent out by the

heart, but it is the transmission of the force of the heart beat sent along the blood column, and the character of the beat gives us, therefore, an idea of how forcibly the heart is driving another quantity of blood into the aorta, and also how much blood is being sent out at each beat.

Supposing, therefore, that on feeling the radial pulse we find that the artery is tense and hard, and that the individual beat is strong and its volume great; this signifies that there is an excited vasomotor centre, causing contraction of the vessels, and that an excited, overacting heart is forcing the blood into the already tense vessels.

Arterial Tension in Health and Disease.—One of the most important acts of the physician is to make a skilful determination of the arterial pressure of his patient, since this is of even greater importance than the study of the pulse rate. Years of experience enable the physician to determine the tension of the arteries by his finger tips, and often the degree of tension gives him far more information than any other physical sign presented by the patient.

When we come to the study of abnormal arterial tension we find that we have to consider cases in which the tension is too high and others in which it is too low.

Temporary High Tension.—Taking up cases of high tension first we find that we can consider it as normal and pathological. The normal cases are those in which the tension is raised to a point above normal by exercise or excitement, such tension being a natural or physiological response. This form of high tension possesses no interest for the clinician unless it is associated with the presence of a weak heart or is so great and prolonged that it produces cardiac distress or injury. It is the form of high tension that frequently damages the heart of the sedentary man who suddenly decides that he needs exercise and takes it in strenuous and excessive degree. Another type of normal high tension, in one sense of the word, is met with in the stage of onset of acute febrile diseases when it may be an effort on the part of the body to supply more blood to ce tain areas for protective purposes, for all large capillary networks are poison destroyers. Still another normal or beneficial type of high tension is the high tension of intracranial injury or disease, in which states Cushing has shown us that the rise is essential to the preservation of life. In early CO_2 poisoning the rise of tension is designed to send more blood to the respiratory centre, and the rise due to severe pain, as in renal colic, lead colic, and labor, may all be advantageous. In the latter case (labor) I have elsewhere pointed out that it is this rise of tension produced by pain that permits women in labor to take chloroform with relative impunity. These facts should make us cautious in the use of vascular

relaxants, unless we are certain that the high tension is useless and actually harmful. If Cushing is right, how many persons have been hurried to their end by bleeding in apoplexy.

Prolonged High Tension.—Aside from those types of high tension which are solely due to increased functional activity of the vasomotor and vascular systems, there is the high tension of the persistent type which often tells of the habits of the patient in the past and present, and directs us to the study of his heart, his kidneys, and his retinal vessels in a way that must not be ignored. In some instances there can be no doubt that a large part of the hypertension is functional in the sense that it is not entirely due to actual organic changes in the vessels; in other instances it is almost entirely due to the presence of fibroid change and the ability of a strong heart to pump vigorously. In some the spasm is due to great nervous strain and the use of stimulants, in others it is due to poisons in the blood, and in the functional type the very existence of these factors for long periods of time results in actual lesions. Brunton has recently advanced the view that not only does high arterial tension do harm by interfering with the nutrition of the tissues, but also by interfering with the nutrition of the vessels themselves. Thus he points out in his usual lucid way that the high tension compresses the vasovasorum between the inner coats and the fibrous coat, which is fixed, because it has reached the point of fixation by distention. Again, he advances the view that the normal constant expansion and contraction of vessels in health, like massage, maintains and aids the blood flow in the vasovasorum.

What is the significance of high tension as to the heart? If its sounds are approximately normal we learn that it is still a fairly healthy organ, able to stand up to its work, but we must bear in mind that in many men past middle life a state of increased tension exists which is not appreciated because the heart has gradually become accustomed to the strain. The stress and strength are so nearly balanced that when a sudden increase in exertion is made, as in running for a car, or taking any form of violent exercise, dilatation due to overstrain at once develops. Abnormal increase in tension means increased work for the heart muscle, and increased strain upon its valves, particularly the aortic and mitral leaflets. The result of this strain is speedily manifested in a previously normal heart by hypertrophy with associated dilatation, in a previously feeble or diseased heart by dilatation with increase in its feebleness, in failure of the mitral valves to stand the great pressure brought to bear on them with each systole, a failure increased in effect by the fact that the mitral rim is feeble also, so that mitral regurgitation takes place, the result being that the blood finds it as easy to slip back into the auricle

as to pass out into the aorta. The mere high tension due to narrowing of the arterioles is not alone responsible for this cardiac disaster; the very fixation of the vessels increases the work of the heart, and with fixation come elongation and tortuosity, which demand increased cardiac effort. The heart now fails not alone from overwork, but in addition it may begin to develop degenerative changes in its fibers, for the same factors that act deleteriously on the muscular fiber of the vessel wall also act on the more specialized muscular fibers of this organ.

Another question of importance is whether an abnormally constant high tension is always evil and ought always to be reduced. Are we to regard it as an evil to be attacked, or recognize that it has become a necessary and unavoidable evil? The present attitude of the profession in regard to high tension is tending to the abuse of vascular relaxants in many cases for the following reasons: It is a question whether high tension may not be designed by Nature to drive blood through narrowed vessels to distant parts for their proper nutrition. If we lower pressure by relaxants of the larger arterioles and arteries we starve distant tissues. Again, the heart in many cases of high tension has undergone compensatory hypertrophy, and this increased power and the high tension help to feed the heart muscle itself through the coronary vessels and the vessels of Thebesius. Again, the normal heart is designed to beat against a pressure of from 100 to 140 mm. of mercury, and nothing exhausts a heart so rapidly as to beat excessively because of low pressure. Very often the hypertrophied heart of high tension may be considered to have established for itself a new standard of pressure, say of 150 to 170, and if this is reduced a state is developed which may be considered as abnormal as is a pressure below the true normal. In other words, in studying high pressure, it is not sufficient to study the pressure alone. We must study the whole cardiovascular apparatus. We must endeavor to prevent an increase in tension, but we must not reduce tension simply because it is high unless we find that the heart cannot stand the stress, or that the pressure is so high and vessels so fragile that rupture is threatened.

Low Tension.—Having considered some of the facts which concern hypertension in the arterial system, we have still before us the study of hypotension. Such a condition, as a chronic state, is rare as compared to hypertension, and is most frequently met with as a part of some suddenly developed condition in the course of an acute illness, or as a result of accident.

When the hypotension is chronic it depends, in the great majority of instances, upon feebleness of the heart muscle, but in one particular malady at least we have a persistent low tension not so much as a result of cardiac feebleness as of vascular relexation, namely, exoph-

thalmic goitre, in which disease the thyroid secretion acts as a powerful vascular sedative.

Not rarely in neurasthenic persons, or persons who are suffering from the after-effects of an acute illness, hypotension is an important factor. Thus, persistent diurnal somnolence may exist while the patient is up and about, replaced by marked nocturnal wakefulness as soon as he lies down. This state is due to a low tension which prevents an adequate supply of blood to the brain, which organ immediately becomes active as soon as the recumbent posture is assumed.

But after all the state of hypotension is met with most frequently in acute illness, and it is usually of far greater gravity, when it is marked, than is hypertension, because it indicates lack of power, nervous and circulatory, and because we have no remedies upon which we can rely as all-powerful vascular stimulants as we can rely on the nitrites as all-powerful relaxants.

This condition of hypotension in acute illness may be considered under two headings: The first type is met with at the critical period of acute infections, of which the most noteworthy is, perhaps, croupous pneumonia. It is not uncommon to find in these patients at this time a state bordering on collapse: the face is anxious, the forehead, wrists, and the trunk bedewed with sweat, the pulse very full, but the arteries relaxed, and the heart's action excessive, as it actively endeavors to fill the leaking vessels, which do not offer the normal resistance to its action. This state depends chiefly, if not entirely, upon vascular hypotonus. The second type is met with in the course of prolonged fevers, such as typhoid fever, in which partly from toxemia and partly from lowered nervous force the vessels relax.

The Estimation of Arterial Tension.—Efforts have been made by numerous investigators to devise apparatus which would be capable of indicating the condition of arterial tension, but all of them have been more or less unsatisfactory. Probably the best at present is Stanton's modification of the sphygmomanometer of Riva-Rocci, which is well described in Fig. 103.

In applying the apparatus it is essential that the muscles of the arm shall be relaxed, and for this reason the limb should be supported in an easy position. Usually it is best to have the patient recumbent and to use the left arm as the manipulations by the physician are more easily carried out by the right hand. The cuff and armlet (*F* in Fig. 103) are applied together, the ends of the rubber being smoothly overlapped. The straps are now buckled, the cuff fitting snugly but without compression.

Before connecting with the manometer the latter should be placed on a firm, level surface and the mercury should stand at zero on the

scale. This is readily accomplished by placing the scale up or down on the glass tube. The connection having been made, the valve *B* is screwed tight and the valve *A* opened (arm parallel to the horizontal arm of the *T*). With the finger of the left hand on the pulse the right hand works the syringe. Compression of the bulb forces air into the closed system—distending the rubber armlet and with the same

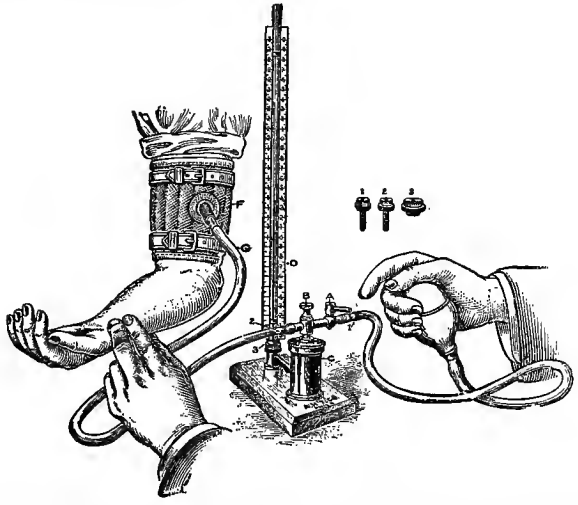


FIG. 103.—The systolic pressure is determined by noting the point at which the pulse reappears after obliteration, while the diastolic pressure is estimated by recording the point at which the greatest oscillations occur in the mercury column of the manometer. The pressure is applied to the arm by a rubber armlet which is 10 cm. wide. This armlet is prevented from expanding outward by a cuff *F* of double thick canvass with inserted strips of tin, which is held in place by two straps which completely encircle the cuff. On the rigidity of this depends, to a large extent, the transmission of pulsation. The rubber armlet is connected with a stiff-walled rubber tube *G*, which in turn connects with the manometer. The chamber *C* communicates by means of a metal tube with the glass column *D*, which is connected by a screw thread at 3, the caliber of *C* being approximately 100 times that of *D*. The cap of the chamber which screws on is provided with a metal *T*, which is connected at 2 with the rubber armlet and at 1 with the bulb used as an air pump. At *A* is a stopcock shutting the rubber bulb completely from the rest of the apparatus, while at *B* is a screw valve which allows the air to escape from the closed system. When desired the manometer can be made portable (without removing the mercury) by screwing the caps 1 and 2 into either end of the *T* at 1 and 2. Then tilt the manometer away from the glass column *D* until all the mercury has run into the chamber: unscrew the glass and screw in cap 3. Before removing cap 3 the manometer must always be tilted, else the mercury will be lost. Pressure is established in the apparatus by a double bulb syringe similar to those used with the thermocautery.

degree of force, displacing the mercury in *C*, driving it up the glass column *D*. When the pulse is no longer felt, the arm of the valve *A* is turned until it is at right angles with the thumb and finger. The valve *B* is now slowly unscrewed until the mercury column begins to fall. With the eye on the scale, the *point at which the pulse reappears is mentally noted as the systolic pressure.* Any pulsation noted

in the mercury column before the pulse beat reappears at the wrist is to be disregarded. As the mercury column falls the oscillations in the mercury increase in size until they reach a maximum and then decrease. The *base line of the greatest oscillation (the line from which it starts) is the diastolic pressure.*

Stanton emphasizes the necessity of regarding the following points with care:

In nearly all cases the first estimation will be found 10 to 20 mm. higher than subsequent estimations. This is probably due to excitement arising from fear that the examination will cause pain. Several estimations should be made until the level normal to the individual is obtained.

In cases with a very rapid pulse rate the diastolic pressure is hard to determine because of the inertia of the mercury. Repeated observations may be necessary.

With a very slow, strong pulse the oscillations may be so large that it is hard to distinguish the largest ones. In these cases by leaving the valve *A* open some of the oscillation is absorbed by the elastic rubber bulb and the reading becomes easier.

In cases showing threatened circulatory failure, especially in cases of high pressure, it will be found almost impossible to get a clear-cut high or low pressure. That is, in spite of repeated estimations, the high pressures will vary from 5 to 15 mm. These cases may at times show a condition in which an occasional beat comes through at a much higher level than all the beats can be detected. Often this is due to the action of respiration. This should be noted in the estimation thus: High pressure, occasional beat at 170; all other beats at 155.

Where the diastolic level is hard to obtain it is of help to get the greatest oscillation with increasing pressure as well as with decreasing pressure. With the valve *A* at right angles (shut off from the syringe) blow up the syringe until a good pressure is established in the second bulb. Now open *A* very slowly and the air can be made to enter at any desired rate of speed. As the mercury column rises the oscillations begin, and gradually increase in size until the maximum, and then diminish. By shutting off *A* completely the behavior of the oscillations under diminishing pressure can be noted.

Where it is desirable to compare the point at which the pulse disappears with the point at which it reappears this can readily be accomplished as follows: Inflate the apparatus until the pulse is nearly gone, then, dropping the syringe portion, gently compress the second bulb until the pulse completely disappears; relaxing the bulb allows it to reappear. The normal arterial pressure is recorded by the sphygmomanometer varies, of course, with age and disease. In normal youths at rest the diastolic pressure is about 100 and the

systolic pressure 130 mm. of mercury. In older persons the systolic pressure is 100 to 145 and in children from 90 to 110. Excitement may raise the pressure, particularly the systolic pressure, as much as 40 mm., but the error in estimating the pressure produced by thickened vessel walls can be ignored, as it represents only 5 to 10 mm.

The fallacies underlying the use of this apparatus are the facts that it is only approximately accurate even in well-trained hands and that the interest and excitement of the patient, who watches its application and often fears the result, sends his tension to an abnormally high point which does not represent by many millimeters of mercury the real state of the arterial pressure. On the other hand it gives the physician a standard which he can use to control the estimates which he makes with his finger tips and one of which he can have a record in his case-book and so compare the pressure from time to time.

The Pulse.—The pulse itself varies as to volume, character, rapidity, and force, and does so within normal limits, and still more so under the effects of disease. It varies greatly according to age. Thus, the pulse of the newborn child is usually about 135 to 140, at one year 120 to 130, at two years 105, at four years 97, at ten years about 90, at fifteen 78, and from twenty to fifty years about 70 per minute. At eighty years of age it is usually about 80 beats per minute. The rate is also increased by taking food, by exercise, by nervousness, and by pain and fever, as will be stated again later.

The volume of the pulse wave depends chiefly upon the quantity of blood expelled from the heart at each systole, and also upon the condition of the aortic valves of the heart, in so far as their ability to prevent regurgitation is concerned. The stimulation of the vagus nerves usually results in a large pulse wave, as does also cardiac hypertrophy with dilatation. If, on the other hand, part of the blood thrown out of the heart into the aorta falls back into the ventricle, we have a pulse of small actual volume, and this is called, because of the peculiar sensation which it gives to the finger, "trip-hammer," "water-hammer," or "Corrigan's pulse." In such a case, because of the power of the ventricle, the blood is forced out into the aorta with great force, but as the last part of the wave regurgitates the pulse is found to be short and sharp. In mitral regurgitation or in mitral stenosis the pulse is usually small in volume, because the left ventricle has not, or cannot get, enough blood at each beat to send out a voluminous wave. (See Figs. 105 and 106.)

So far as the character of the pulse is concerned, we recognize one which is slow and full, as that seen after digitalis is used; that which is short and sharp, as in aortic regurgitation; that which is small and hard, as is often seen in aortic obstruction, and the small, wiry pulse of acute peritonitis.

Various names are applied to a pulse possessing certain peculiarities. Thus, we have under the name *pulsus paradoxus* a pulse which disappears with each deep inspiration. It is usually due to indurative mediastinopericarditis, whereby inflammatory bands press on the bloodvessels or the heart or suction ensues when large vessels are drawn upon. If the beats of the heart are irregular in force but regular in rhythm, we have developed what is called a *pulsus alterans*.

A *dicrotic pulse* is one which is characterized by a reduplication, which feels like a second beat following the first before the latter is over. It is found in many cases of exhausting fever, and depends upon an undue elasticity of the bloodvessels, with relaxation of the

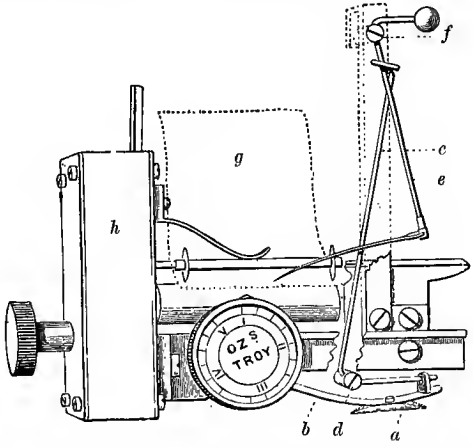


FIG. 104.—A sphygmograph. Certain supporting parts are omitted so that the multiplying levers may be displayed. *a* is a small metal plate which is kept pressed on the artery by the spring *b*. The vertical movements of *a* cause to-and-fro movements of the lever *c* about the fixed point *d*. These are communicated to and magnified by the lever *e*, which moves around the fixed point *f*. The free end of this lever carries a light steel marker which rests on a strip of smoked paper, *g*. The paper is placed beneath two small wheels and rests on a roller which can be rotated by means of clock-work contained in the box *h*. The paper is thus caused to travel at a uniform rate. The screw graduated in ounces (Troy) is brought to bear on the spring *b* by means of a cam, and by this the pressure put on the artery can be regulated. The levers magnify the pulse movements fifty times. (Dudgeon.)

arterioles, so that the blood first unduly distends the arteries, which then contract upon it, and thus produce the second wave or apex to the pulse curve.

We can study the pulse either by the touch or by the sphygmograph. If by the latter means, the instrument of Dudgeon is the best (Fig. 104). The normal pulse wave is shown in Fig. 105.

It will be seen that there is a distinct upstroke produced, which is called the line of ascent. This is due to the distention of the artery produced by the ventricle forcing blood out into the aorta. There is after this a line of descent interrupted by two separate secondary

waves, which are called catacrotic waves. The second or lower of these is called the dicrotic wave, and is the one which becomes marked enough to be felt in some cases of disease. The duration of the period of descent corresponds to the time the blood is flowing out of the arteries into the capillaries, and, if this flow is rendered difficult by vascular spasm, the line of descent will be gradual; if easy from vascular relaxation, it will be short. If the drop is very sudden, it is a pulse of "empty arteries," so called, as after severe hemorrhage or in cases of aortic regurgitation.

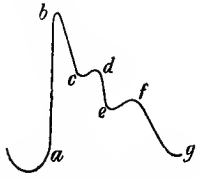


FIG. 105.—*a b*. Percussion upstroke. *a b e*. Percussion wave. *c d e*. Tidal wave. *e f g*. Dicrotic wave. *d e f*. Aortic notch. *f g*. Diastolic period.

Very small irregularities of the line of descent are due to the elastic bloodvessels being thrown into vibrations by a forcible pulse wave.

In Fig. 106 is shown the typical pulse wave of aortic regurgitation; and in Fig. 107 that of mitral stenosis, which is irregular in time and volume. (See page 273.)

The force and rapidity of the pulse also depend largely on the condition of the bloodvessel walls, particularly the rapidity. The

FIG. 106.—A tracing taken from a case of aortic regurgitation. Corrigan's pulse. Note the sharp upstroke and the sudden fall due to the blood falling back into the ventricle.

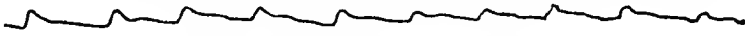


FIG. 107.—The small pulse of mitral stenosis.

latter also is influenced by the activity of the pneumogastric nerves in regulating the speed of the heart. Thus, if the arterial pressure be very high, through spasm of the arterioles, the difficulty experienced by the heart in forcing blood into the arteries will be so great that pulsation may be very slow, whereas if the normal resistance to the action of the heart be removed by vascular relaxation, the beat will be rapid, just as the wheels of a locomotive fly around on a slippery track when the friction or resistance is removed. If the

vessels are relaxed, the impetus communicated to the column of blood in the vessels by the heart is lost, and so the pulse is not forcible; or if the resistance is excessive, the force is dissipated.

The vagus or pneumogastric nerves are continually holding the heart in check, and by causing full diastole enable it to send out a large wave of blood at each contraction. If they are greatly stimulated, we have a very slow pulse and a full wave of blood with each heart beat; but as the heart now beats very slowly the blood pressure may fall for lack of blood in the vessels, unless there is an increased force of the heart at each contraction to make up for the number of beats in the minute which have been lost, or unless there is also a

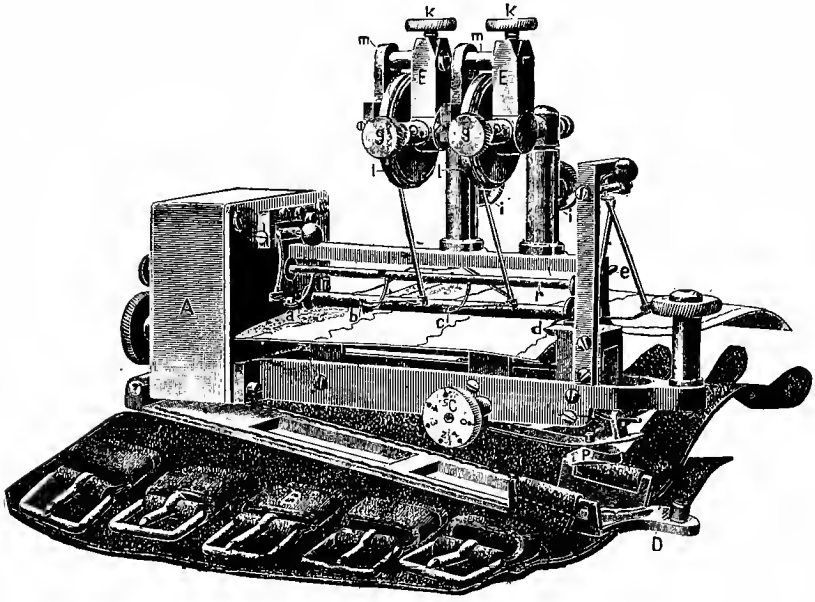


Fig. 108.—Jacquet's sphygmocardiograph.

great increase in arterial tension by contraction of the arterioles. A very slow pulse depends in the great majority of cases upon a high arterial tension from vascular spasm—*i. e.*, resistance to the flow of blood; more rarely it is due to irritability of the vagus nerves, produced by pressure or disease, or by drugs, such as digitalis.

The term "bradycardia" is applied to a very slow pulse, which may be as slow as twelve beats a minute.

A rapid pulse is seen most commonly as the result of stimulation of the heart by drugs, by fever, or by fear. Fear causes the vagus to lose control of the heart, and fever acts by reason of the stimulant effect of heat upon this viscus and its depressant effect upon the

vagus. In other words, the quick pulse of fever is not a mere coincident symptom of fever, but the result of it.

When the heart's action becomes exceedingly rapid it is called "tachycardia." It is due in the majority of instances to relaxation of the bloodvessels, and more rarely to depression of the pneumogastric nerves. As a symptom of organic disease it is a frequent manifestation of exophthalmic goitre. Often in this condition the pulse becomes so fast that it cannot be counted.

Great force of the pulse is due to hypertrophy, or overaction of the heart because of stimulation; and great feebleness is generally caused by marked dilatation not associated with hypertrophy, by chronic myocardial degeneration, and in acute disease by exhaustion of the heart muscle, or changes in the muscle fibers of the heart.

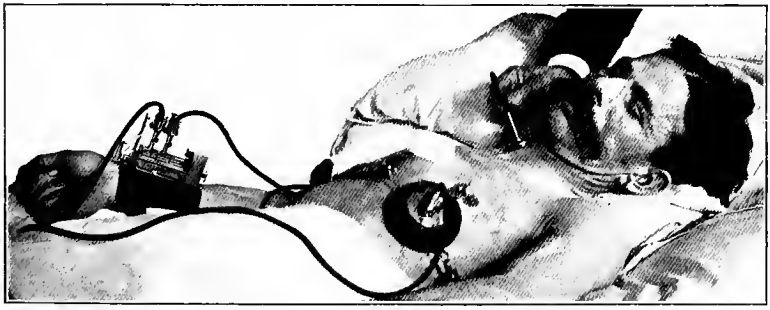
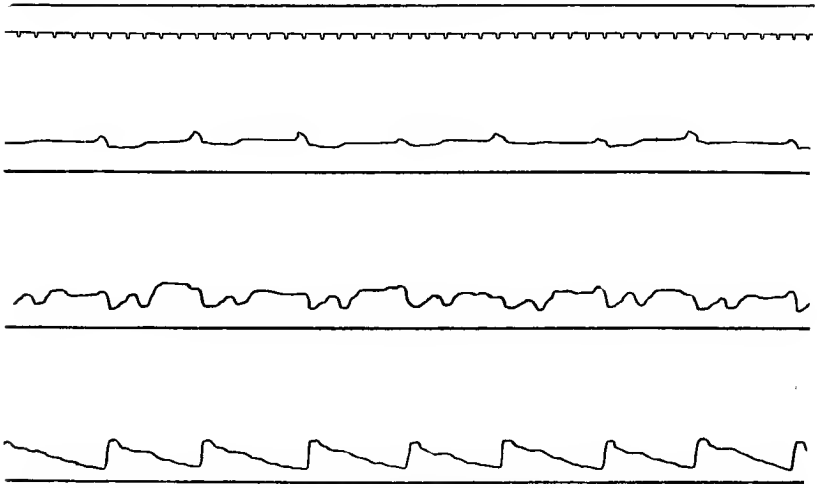


FIG. 109.—Application of the sphygmocardiograph of Jacquet to a case of aneurysm with systolic retraction at the apex. The recording apparatus is bound on the wrist as is the ordinary sphygmograph of Dudgeon. The radial pulse moves the lever nearest the arm, the jugular pulse is recorded by the lever attached by a tube to a tambour which is placed over the jugular vein and the apex beat by the lever nearest the wrist, which is connected by a tube to the tambour placed over the heart.

There still remains for consideration those states in which circulatory disturbances ensue as a result of incoördination between the auricles and the ventricles. Such a state may develop as the result of the action of large doses of digitalis and also in mitral stenosis in which disease it is very common. The most important state, however, is that to which is given the name of the "Stokes-Adams syndrome," in which there is not only incoördination between the auricle and ventricle, but the auricle may beat many times faster than the ventricle because of disease of the auriculoventricular bundle of His. The general symptoms of this disease consist in a remarkably slow pulse, pulsation of the veins of the neck and sometimes attacks of vertigo, syncope and apoplectiform or epileptiform seizures. For the purpose of getting a graphic record of this state of the circulation the instrument called the sphygmocardiograph of Jacquet may be used (Figs. 108 and 109). By means of columns of air in tubes the impulse

of the jugular vein and the apex beat are recorded by levers as in Dudgeon's sphygmograph and a third lever by pressure from the



Shows the normal radial pulse on the lowest line, the apex beat on the middle line, the jugular pulse on the upper line.

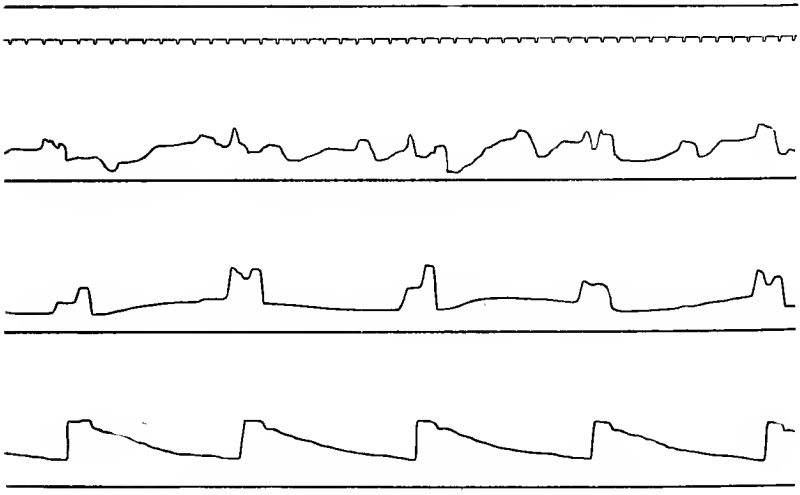


FIG. 110.—Tracings taken with the Jacquet apparatus.

A tracing from a case of "Stokes-Adams disease," which shows below the very slow pulse and in the middle the slow apex beat and above the rapid and abnormal jugular pulsation due to the regurgitation from the auricle which is beating far oftener than the ventricle.

radial artery produces a third tracing, each one being superimposed. (See chapter on the Thorax.)

CHAPTER IX.

THE ABDOMEN AND THE ABDOMINAL VISCERA.

The surface of the abdomen—Changes in the appearance and shape of the abdominal wall—The signs and symptoms of disease of the abdominal organs.

THE condition of the abdominal surface and abdominal contents is best studied by means of inspection, palpation, percussion, and

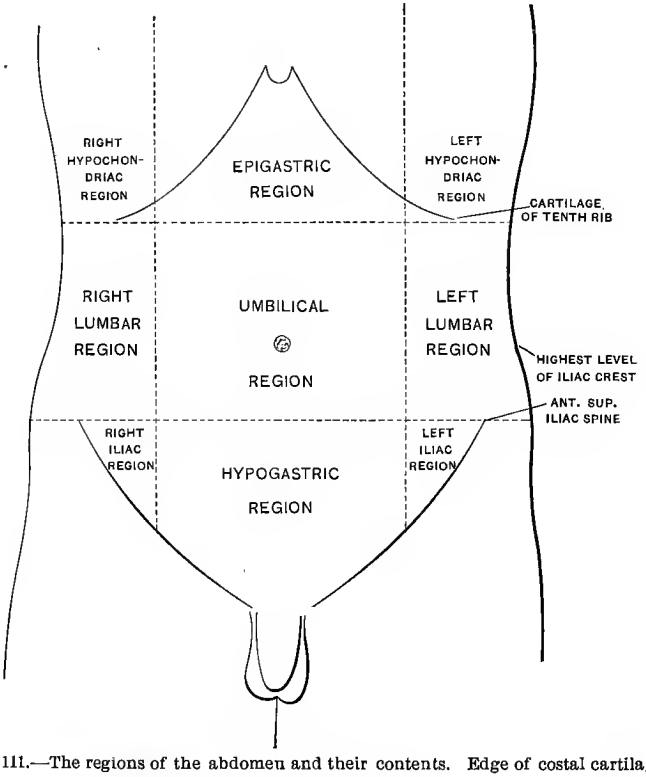


FIG. 111.—The regions of the abdomen and their contents. Edge of costal cartilages in curved outline. (Gray.)

auscultation. For the purposes of inspection the surface of the abdomen has been arbitrarily divided by diagnosticians into a number of spaces, which are best shown in the accompanying figure (Fig. 111), and which get their names from the regions in which

they are located, or from the organ immediately underneath the abdominal wall. By means of these arbitrary outlines we can readily describe the exact spot in which a physical sign or symptom is found.[1]

The following table, from Gray's *Anatomy*, clearly shows the viscera to be found under each of the areas named:

Right Hypochondriac.	*Epigastric Region.*	*Left Hypochondriac.*
The right lobe of the liver and the gall-bladder, hepatic flexure of the colon, and part of the right kidney.	The pyloric end of the stomach, left lobe of the liver, and lobulus Spigelli, the pancreas, the duodenum, parts of the kidneys and the suprarenal capsules.	The splenic end of the stomach, the spleen and extremity of the pancreas, the splenic flexure of the colon, and part of the left kidney.
Right Lumbar.	*Umbilical Region.*	*Left Lumbar.*
Ascending colon, part of the right kidney, and some convolutions of the small intestine.	The transverse colon, part of the great omentum and mesentery, transverse part of the duodenum, and some convolutions of the jejunum and ileum, part of both kidneys.	Descending colon, part of the omentum, part of the left kidney, and some convolutions of the small intestine.
Right Inguinal (Iliac).	*Hypogastric Region.*	*Left Inguinal (Iliac).*
The cæcum, appendix cæci.	Convolutions of the small intestine, the bladder in children, and in adults if distended, and the uterus during pregnancy.	Sigmoid flexure of the colon.

Inspection.—On inspecting the abdominal surface the physician should look for eruptions which may indicate some general disease, as typhoid fever; for localized swelling, which may be due to hernia; for striæ, indicating that the skin has been stretched by excessive fat, by great swelling from ascites, or by pregnancy. He should also in a case of suspected early pregnancy look for the dark streak in the median line. If the umbilicus is protruding and tense it may indicate distention due to grave abdominal disease, or it may be infiltrated by a morbid growth which has been primarily hepatic. If it be a secondary growth the navel will perhaps be "moored fast." Sometimes it is much swollen from chafing and eczema. Umbilical hernia may be found.

The general abdominal wall is protruded and retracted in normal respiration in both sexes, but more markedly so in males. It is pushed outward, or protruded, by many perfectly normal causes, such as an unusual amount of fat in the omentum, pregnancy, and an accumulation of liquid and food in the stomach after a heavy meal. It is also convex to an abnormal degree in cases in which ascites is present, when the stomach and bowels are overdistended with gas (tympanites), and when any of the organs found in the peritoneal cavity are the seat of swellings or tumors of large size. In children a protruding pot-belly, "the frog-belly" of the French,

[1] For changes in the skin of the abdomen, see chapter on the Skin.

is seen in cases of scrofula or tuberculosis of the mesenteric glands, and in those cases which suffer from chronic gastro-intestinal catarrh. It is claimed in a recently published paper by a French clinician that the intestinal canal is not only dilated, but of greater length than is normal in these cases.

If the belly wall is retracted, concave, or "scaphoid," as it is sometimes called, we look for the cause in abstinence from food, or remember the possibility that excessive vomiting or purging may have emptied the gastro-intestinal tract of its usual contents. We also find a retracted belly wall in nearly all cases of advanced wasting diseases, such as carcinoma, peritoneal tuberculosis of the cirrhotic type, or tuberculosis of the lungs; and if the retraction is associated with muscular rigidity of the belly wall and pain, we suspect the early stages of peritonitis or the presence of some acutely painful affection, such as renal or hepatic colic or lead colic. Marked concavity and retraction of the belly wall are also seen sometimes in cases of tuberculous meningitis.

Sometimes in thin patients with some atrophy of the abdominal muscles peristaltic waves are to be seen traversing the abdominal surface as the result of violent movements of the bowels. These waves are commonly seen in cases of intestinal obstruction, and in neurotic persons with mucomembranous enteritis, or, if in the epigastrium, may be due to a dilated stomach. If the waves are from below upward and in the right side, they are probably arising in the ascending colon; if from above downward and in the left side, in the descending colon and sigmoid flexure. Again, gastric waves pass from left to right, while those in the transverse colon pass from right to left.

Tympanites and Ascites.—The abdomen is distended very greatly by gas in many cases of peritonitis, typhoid fever, and in persons suffering from flatulent colic. If this be the cause of the distention, percussion of the anterior and lateral belly wall when the patient is lying on the back will give a tympanitic note. We separate, diagnostically, the swollen abdomen due to wind from that due to ascites by the fact that in the latter condition the epigastrium is moderately flat when the patient is lying down, while when tympanites is present it is more protruding. Again, in ascites the greatest bulging is generally to be found in the flanks, or, if the patient sits or stands erect, the hypogastric region bulges from the change in the position of the fluid. If the swelling be due to a moderate-sized ovarian cyst, this variation in form will not occur, as the cyst is not readily movable. If the ovarian tumor be large, the differential diagnosis may be most difficult and almost impossible, except by the history or by examining the liquid withdrawn by tapping.

In cases of ascites due to free liquid in the abdominal cavity per-

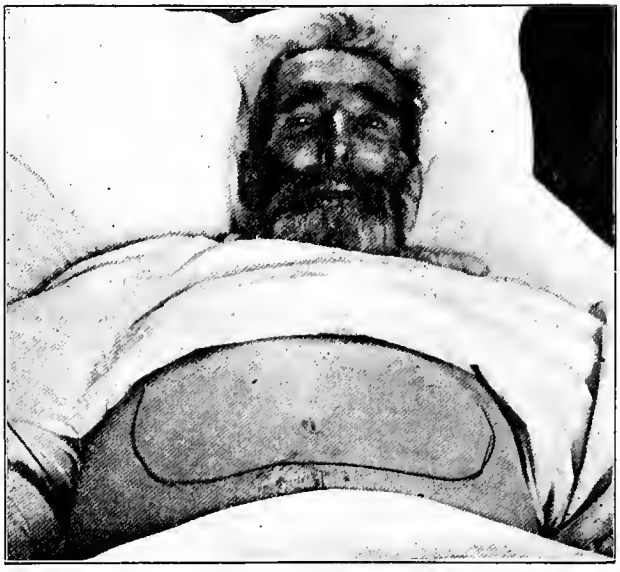


FIG. 112.—Enormous ascites. The area inside the line is that which was resonant on percussion (gut tympany). On either side and below the line there was absolute flatness due to the presence of fluid. (From the author's wards.)

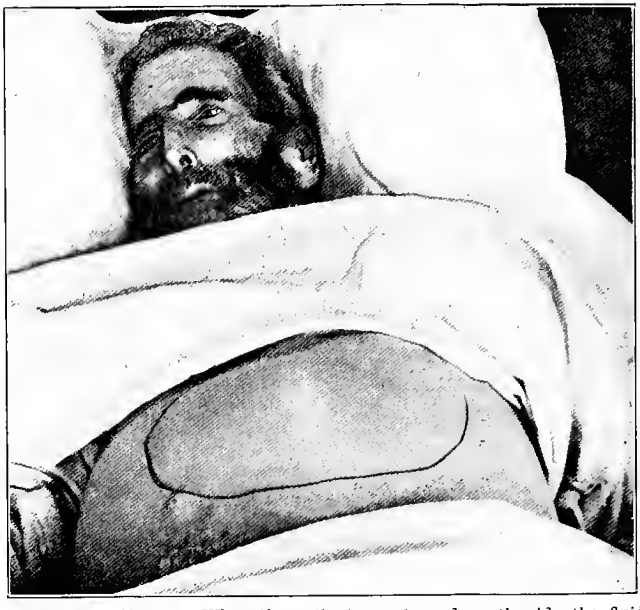


FIG. 113.—Same as Fig. 112. When the patient was turned on the side the fluid flowed in that direction, and so the area of resonance was altered, the area of flatness on the right increasing and on the left diminishing.

cussion will elicit flatness over the flanks and resonance where the intestines containing gas are floated up against the anterior belly wall above the effusion. Sometimes, however, if the large intestine be empty of fecal matter, percussion in the flank behind the mid-axillary line will reveal tympany, because the peritoneum walls off the liquid from the posterior surface of the bowel. If the patient is turned slightly to one side, the area of flatness on percussion is altered, as is shown in Fig. 113. Palpation will also reveal fluctuation in ascites, but none in tympanitic distention. To develop this

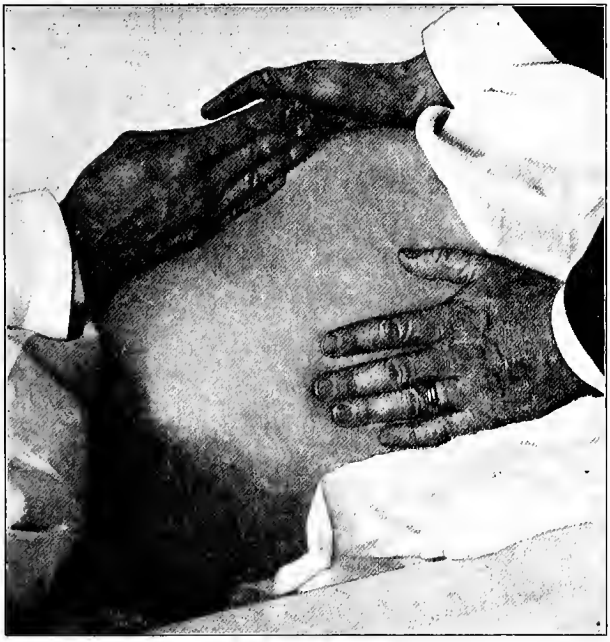


FIG. 114.—Showing method of determining the presence of fluid in abdomen by transmitted fluctuation. The hand of an assistant is placed on edge on the middle line, to prevent transmission of impulse by the belly wall. The right hand then taps the flank, and if fluid is present the impulse is felt by the left hand on the other side.

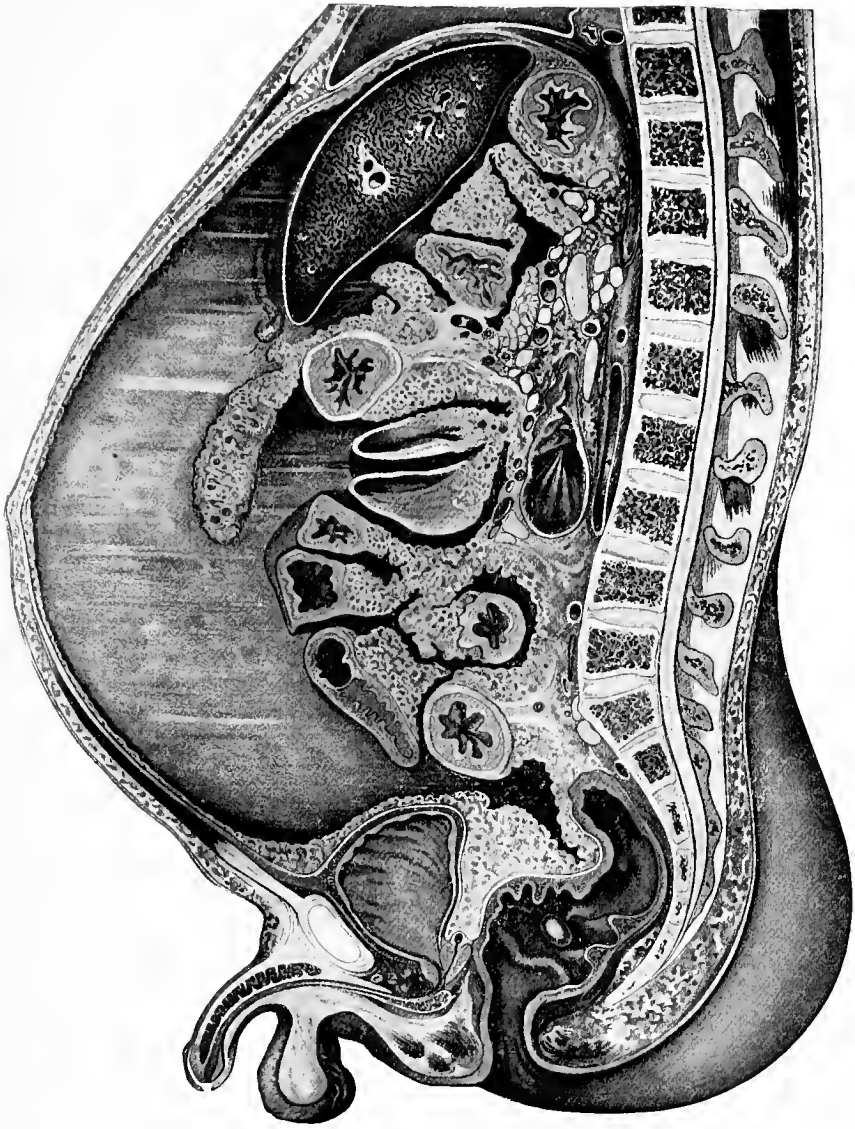
fluctuation, the patient is placed on his back and the finger tips of the left hand of the physician are placed against the skin of the flank. With the finger tips of the right hand the opposite flank of the patient is struck a blow as in performing ordinary percussion, when the impulse, if fluid is present, will be transmitted to the fingers of the left hand. To prevent a transmission of the impulse through the abdominal wall, an assistant may press with the edge of his hand over the linea alba (Fig. 114). As the result of gradually increasing intra-abdominal pressure the floating ribs become pushed outward,

the apex beat of the heart is often displaced upward and outward, and the umbilicus becomes protruded instead of retracted. The skin of the belly wall becomes thin and shining, and the recti muscles may become separated. After tapping in such cases the peristaltic movements of the bowels can be readily felt through the intervening skin.

Having decided that the distention is due to an accumulation of free fluid in the abdomen, it remains for the physician to determine what the cause of the ascites may be. Its most frequent cause is atrophic cirrhosis of the liver, which results in engorgement of the abdominal vessels with secondary transudation of fluid. (See Fig. 114.) If it be not due to cirrhosis, it may arise from an abdominal tumor, which by pressing on large vessels results in an effusion of liquid through their walls (see Plate IX), or be caused by tuberculous peritonitis, by obstruction of the thoracic duct, by valvular disease of the heart causing an obstruction to the flow of blood in the vena cava, or, finally, by chronic parenchymatous nephritis. If the last cause be present, there will be some edema of the lower extremities or general anemia with dyspnea and albuminuria. For the typical symptoms and physical signs of these various affections the reader is referred to those parts of this book in which they are discussed. (See Index.)

There is an additional source of information to be utilized as to the cause of the ascites, namely, the character of the effusion. If the fluid withdrawn on aspiration has a specific gravity of 1.008 and contains but a trace of albumin (about 0.97 per cent.), it is probably due to hepatic cirrhosis; whereas if due to the pressure of a tumor the specific gravity is usually about 1.012 and the albumin nearly 2 per cent. Such a specific gravity and proportion of albumin may also result when the ascites is due to heart disease or to pressure on the thoracic duct. When the effusion is the result of Bright's disease the specific gravity is apt to be only 1.006 and the proportion of albumin only a trace. In cases in which the ascites arises from some disease directly affecting the cells lining the peritoneal cavity, as carcinoma of the peritoneum or of the abdominal viscera, or tuberculous peritonitis with or without pus, the specific gravity is much higher than just stated, namely, from 1.018 to 1.027, as a rule, and the proportion of albumin ranges from 3.80 in the case of growths to 5.76 in the case of tubercle and 7.10 when there is pus. To express it otherwise, the percentage of albumin is in direct proportion to the inflammatory process, and an effusion due to hydremia would therefore contain only a trace of albumin. Further than this, it is asserted by Pohl and Rosenbach that the effusions due to venous engorgement, heart disease, and renal lesions can be separated from those due to disease in the peritoneum affecting this membrane directly

PLATE IX.



Median Vertical Section of Body of a Boy of Seventeen, who Died of Colloid Cancer of the Peritoneum and Iliac Flexure.

The gelatinous masses represent the growth and the ascitic fluid is readily seen. (Ponfick's Atlas.)

by a test following the administration of iodide of potassium. When this drug is given to the first class of cases it speedily appears in the effused fluid; but should effusion be due to the organic diseases of the peritoneum which have been named, it will not appear. The fluid to be tested is placed in a test tube and some nitric acid and chloroform added, when if iodine is present its characteristic color will appear. Should the cause of the ascites be a ruptured ovarian cyst the diagnosis of its cause, except from the history of a previous localized swelling, is exceedingly difficult. The fluid under these circumstances is usually of a specific gravity of 1.026, but its specific gravity may be much lower. It is asserted that in the instances in which the specific gravity of the fluid is very low the swelling is due to a cyst of the broad ligament.

Rotmann asserts that in all serous effusions, even in non-diabetic patients, there is present a trace of sugar; whereas in all seropurulent effusions sugar is absent.

Very often in cases of ascites, particularly when this condition arises from *hepatic cirrhosis*, there is developed on the anterior belly wall a more or less well-defined bunch of veins, which is some times called the *caput Medusæ*, as the result of an attempt at collateral circulation, to compensate for the obstructed flow caused by the changes in the liver. Sometimes a mediastinal growth will cause a somewhat similar development. When the obstruction is lower than the liver the superficial veins of the lower part of the abdomen (hypogastrium) will be found distended.

Localized bulging of the abdominal walls, chiefly on the right side, is found in cases in which the liver is enlarged by *hypertrophic cirrhosis*, or by cancer or other morbid growth, such as gumma or sarcoma, and by abscess. The swelling, if its origin be in the liver, will arise under the floating ribs on the right side, and will extend downward and forward toward the umbilical area. If the enlargement be great, it will extend far below the umbilicus and across the umbilical area to the opposite side of the abdomen. In enlargement of the spleen similar signs, springing from the floating ribs well over to the left side, may be developed (see Fig. 115), and large cystic kidney on either side may cause abdominal bulging, particularly if the kidneys be floating. (See Floating Kidney and Spleen.)

Marked swelling of the epigastrium indicates distention of the stomach by gas or food, or that this organ is the seat of morbid growth. Sometimes a similar distention results from enlargement of the posterior mediastinal and retroperitoneal glands. Again, distention of the epigastrium is apt to be caused by enlargement of the left lobe of the liver. In ovarian tumors the growth often gradually distends the entire belly equally; but, as already stated, the

history is usually that of swelling, low down, and of its being chiefly unilateral at first.

It should be remembered that the discovery of a pyriform swelling in the hypogastrium may possibly be due to a pregnant uterus, or to retention of urine, with consequent distention of the bladder.

Cases of dilatation of the stomach often show very great bulging of the umbilical area of the abdominal wall when that viscus is distended by liquid and gas. (See Percussion in this chapter.)

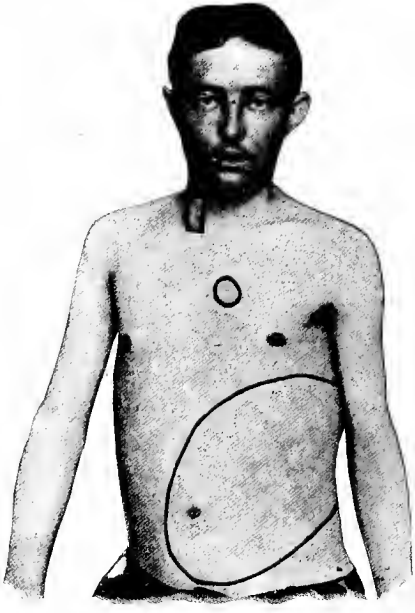


FIG. 115.—A case of splenic anemia with great enlargement of the spleen, as shown in the large outlined area. The smaller outlines indicate the areas of anemic murmurs near the base of the heart and in the carotid artery. (From the author's wards in the Jefferson Medical College Hospital.)

In inspecting abdominal swellings the physician should watch to see if they move up and down with respiration. If they do, they are probably connected with the diaphragm and depend upon disease of the liver and spleen, as tumors of the pancreas, stomach, and kidney are usually not attached to the diaphragm, and therefore generally do not move. Inspection of the abdominal wall will also show possible venereal infection if the glands in the groin are enlarged, or if in suppurating they have left puckered scars. If silvery lines extend across the belly, they may indicate pregnancy past or present, or any state of the abdominal tissues causing great stretching of the skin. Great bulging in the neighborhood of the umbilicus

will naturally suggest umbilical hernia, and swelling in the groin, adenitis, inguinal hernia, or perhaps an appendicular abscess.

Palpation and Percussion.—More important than any other external method of studying the condition of the abdominal contents is the use of gentle *palpation*, the fingers being gradually worked down into the abdominal cavity in such a way as not to cause pain or excite the muscles of the abdominal wall to resistance. The hand should always be carefully warmed before palpation is attempted, and the object of the examiner is to discover, first, the hardness or resistance to pressure; secondly, the consistency and form of the organs which he can touch; and, thirdly, whether any swellings which he feels are movable, bound down and immovable, pulsating, soft or hard, nodular or smooth. The patient whose abdomen is to be palpated must be placed flat on his back, with the knees drawn up to relax the abdominal muscles, the head and neck should be raised, and, if possible, the attention of the patient should be diverted by conversation about some symptom which exists elsewhere than in the belly, while the examination is made, as in this way voluntary muscular resistance is removed to some extent. He should be made to breathe easily through his opened mouth; and if the belly wall remains so rigid that a perfect examination is impossible, and yet the results of such an examination are very important, ether or chloroform should be given to relax the muscles. In other instances in which it seems inadvisable to give an anesthetic, the patient may be placed in a bath as hot as he can bear. He should be entirely submerged up to the neck. The hot bath often relaxes the abdominal wall sufficient to aid diagnosis very greatly.

After the abdominal contents have been carefully examined, the patient being on his back, he should be placed first upon his left side and then upon his right, and the abdominal contents again palpated. This is particularly necessary when examining the belly for growths or when enlargement or displacement of the liver, spleen, or kidneys is suspected.

It must be remembered, however, that the anterior abdominal wall, particularly that of nervous persons, is often very sensitive or "ticklish," and the mere exposure of the skin to the air of the room, coupled with the fear of examination, may cause great rigidity of the belly wall without there being any abnormal condition present. This can be generally overcome by gentleness in palpation and by resting the palm of the hand on the belly and partly flexing the fingers, rather than by attempting to insert the finger tips between the abdominal muscles. The writer has seen a case of rhythmical hysterical spasm of the recti muscles in a male, which at first gave the sensation of an enormous diffuse pulsating aneurysm of the abdominal aorta.

Peritonitis.—Great resistance of the rigid abdominal muscles is found whenever peritonitis is present in an acute form, in some cases of renal and hepatic colic, and more commonly in lead colic and in hysteria. In peritonitis great tenderness to the slightest touch is also present. Another symptom of acute peritonitis, aside from the exquisite tenderness of the abdomen, the drawn lip, the thirst, and the distention or rigidity of the belly wall, is pain of a severe character; unless it be septic peritonitis, when pain may be absent. There are

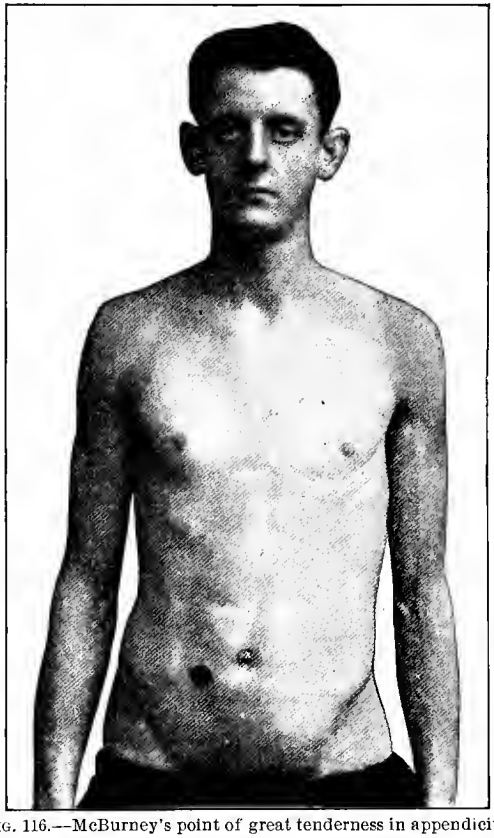


FIG. 116.—McBurney's point of great tenderness in appendicitis.

also the drawing up of the limbs to relieve abdominal tension, obstinate constipation, moderate fever, and a very rapid, quick pulse. The tongue speedily becomes dry and parched, and collapse may soon ensue in severe cases. It is not to be forgotten that localized peritonitis may result from many causes, usually from disease of the appendix vermiformis or the genito-urinary tract in women, and that the local symptoms and lesions may be limited by a wall of lymph to a very small area of the abdominal cavity.

Appendicitis.—In appendicitis rigidity may be general if the inflammation is widespread, or localized if the original area is limited or walled off from the rest of the abdominal cavity. If the rigidity be due to peritonitis, secondary to appendicitis, the following symptoms will point to an inflammation of the appendix as the cause:

There is usually a rapid pulse, which becomes more and more speedy as the gravity of the case progresses. Indeed, a very rapid pulse is a sign of great importance as indicative of the severity of the malady. There is marked tenderness at McBurney's point (Fig. 116) on pressure with the finger tip. The pain may be referred to the epigastrium and the sigmoid flexure as well, but is not so severe on deep pressure in these parts. There is usually an increased leukocytosis (see chapter on the Blood), an anxious face, and sometimes very great pain. (See chapter on Pain.) The febrile movement is usually moderate, and it may be absent. It is to be constantly borne in mind that the early stages of typhoid fever often so closely simulate appendicitis of the subacute type, that a differential diagnosis can only be reached after a most careful study of the case.

Abdominal Tumor.—Let us suppose that on placing the hand upon the epigastrium and the upper part of the umbilical area we find a swelling. In the first place, we must decide as to whether it is in the abdominal wall or in the abdominal cavity. If it is in the wall, it will be movable with the tissues of the wall and readily grasped by deep palpation; but if in the abdominal cavity the abdominal wall may be made to move over it unless it be attached to the parietal peritoneum.

Let us suppose it is in the wall of the abdomen, What can the swelling be? It may be a fatty tumor; in which case its surface will be dimpled and resistant, probably not painful, unless the part has been inflamed by rubbing or an injury, and it will not fluctuate. There will generally be a history that the person has exercised constant pressure on the part, as in leaning against a bench or table. Again, it may be an abscess; but aside from the rarity of this condition, we can exclude such a possibility by the absence of pain and fluctuation, and the absence of a history of a severe injury.

Very much more commonly a swelling in the epigastrium, or upper umbilical area, is due to an intra-abdominal cause. In adults the most common cause is probably a growth (generally a carcinoma) of the pyloric end of the stomach or an indurated gastric ulcer may be present. In other instances it is due, particularly in children, to enlarged lymphatic glands, as in tuberculous disease of the mesentery. This is also sometimes seen in adults. Sometimes by reason of tuberculous peritonitis a nodular mass is not only felt in this area, but an abscess containing tuberculous pus may be formed and become surrounded by walls formed by the gluing together of the

organs by lymph. Carcinoma of the pancreas may also cause a swelling in this neighborhood, or a cyst of the pancreas may be present. Aneurysm of the abdominal aorta is also not to be forgotten. Sometimes, too, a distended or carcinomatous gall-bladder may project into this area. (See below.)

Gastric Carcinoma.—If the growth be gastric carcinoma, the patient will be in or past middle life (probably between the fortieth and seventieth years, although cases may occur as early as thirty years); will have a history of constantly increasing discomfort in the stomach; there will have been much sour belching, and perhaps vomiting of coffee-ground-looking material; marked loss of flesh and some cachexia will be present. The disease occurs twice as frequently in men as in women. According to Welch's statistics, out of 1300 cases of gastric cancer, 791 were in the pylorus, 148 in the lesser curvature, 104 in the cardia, 68 in the posterior wall, and 61 involved the whole stomach. The remainder were in the fundus, the greater curvature, or the anterior wall. The growth, if in the pylorus, is usually freely movable, and for this reason can be readily felt, and then is often momentarily lost to palpation. Its position is apt to change with the posture of the patient and the presence or absence of food in the stomach. Pain is usually elicited on deep pressure, and, if the growth be large and at the pylorus, the symptoms of dilatation of the stomach may be present, because that viscus is dilated through obstruction of the pyloric opening, which results in retention of the gastric contents.

Similar symptoms may, however, be produced by a deposit of inflammatory lymph around a *pyloric ulcer*, which being changed into fibrous tissue, causes great thickening of the gastric wall with matting of the omentum around it, closely simulating the mass produced by a malignant growth or simple hypertrophic pyloric stenosis. When cancer involves the cardiac area of the stomach, this organ, instead of becoming dilated, becomes greatly diminished in size, and feels like a narrow band in the neighborhood of the left floating ribs and epigastrium. The diagnosis will be aided by discovering that the capacity of the stomach is very small and hydrochloric acid absent.

But the presence of a tumor which can be palpated in the neighborhood usually occupied by the stomach does not, of necessity, indicate that this viscus is diseased. Not very infrequently an abnormal position of the colon or of other portions of the viscera may result in the physician's mistaking a growth in these parts for a gastric cancer. Even carcinomatous lymph glands may lead to this error. It is in these cases that the testing of the stomach contents is most useful in making a differential diagnosis. Thus, if the normal gastric acidity be present we can exclude gastric carcinoma; even if the

growth be carcinoma of the duodenum, the gastric juice usually remains normal. Not only does the examination of the gastric contents aid us in this manner, but if a growth in the duodenum causes stricture, it may cause the duodenal contents to regurgitate into the stomach, so that we find pancreatic digestion going on there. Boas asserts that the presence of some liquid brown fecal fluid in the stomach indicates jejunal stricture.

In considering the differential diagnosis of carcinoma of the stomach and bowel, it is to be remembered that the former is far more common than the latter. Thus Heimann found that out of 20,054 cases dying of carcinoma in the hospitals of Prussia, 10,537 involved the digestive tract, and of these, 4288 affected the stomach, whereas only 20 involved the small intestine and 224 the large intestine.

Gastric Ulcer.—Sometimes in cases of chronic gastric ulcer the area involved becomes so indurated as to be felt as a hard mass through the abdominal wall. In such instances the points which aid us in separating the condition from gastric cancer are the fact that the patient is young and usually a woman; that the vomiting occurs immediately after taking food, for in gastric cancer it is only seen in most cases several hours after food has been taken; that there is no sign of gastric obstruction; that there is an excess of hydrochloric acid in the gastric contents in cases of ulcer, and an absence of this acid in cases of cancer. It is, however, a noteworthy fact that if the cancer involves only the pylorus, the acid may be present, because this part of the stomach is not that which secretes acid; and it is also to be remembered that a single test of the gastric contents is not to be depended upon, but that several should be made. (See tests for HCl in this chapter.) Any disease which produces atrophy of the gastric tubules may cause an absence of hydrochloric acid, as, for example, grave pulmonary tuberculosis and renal or cardiac disease. Even catarrhal jaundice may temporarily arrest its secretion, and as jaundice is often present in gastric carcinoma, because of pressure or hepatic involvement, this fact should not be forgotten. In other words, the absence of hydrochloric acid is not pathognomonic of cancer; neither is its presence a guarantee of the absence of the growth. The result of an examination for this acid simply gives us additional evidence in making a diagnosis. As a rule there is no cachexia in cases of gastric ulcer, though there may be marked anemia. There is usually in cases of ulcer no great loss of weight unless the symptoms have been present a long time. In gastric ulcer vomiting of blood may occur, whereas in gastric cancer if blood is present it is broken-down blood and resembles coffee grounds. (For the examination for occult hemorrhage, see chapter on the Bowels and Feces.)

In cases of gastric ulcer great pain is often produced by deep or

even superficial pressure over the epigastrium, and a painful spot can generally be found on the back, about the angle of the right scapula.

These painful spots are, however, as a rule quite localized, and the fact that they are very painful at one particular spot, and yet the surrounding parts are comparatively insensitive, points to ulcer as a cause.

It is worth remembering, however, that *duodenal ulcer* may cause identical symptoms on palpation. Diffuse tenderness in this area is more apt to be due to colitis or even to appendicitis or sigmoiditis, which often results in pain in this area or in tenderness on percussion.

Disease of the Gall-bladder.—A very more frequent lesion in the epigastric area, or so near to it as to lead the physician to consider it epigastric, is disease of the gall-bladder. Theoretically the area of the gall-bladder is distinctly to the right of the median line, but in women whose lower ribs have been compressed by corsets the gall-bladder is pushed inward, and, it may be, distinctly downward. Because of these facts, and because carcinomatous disease of the stomach and gall-bladder have a direct etiological relationship, the possibility of an epigastric mass being a gall-bladder growth must always be considered. As a rule, however, disease of the gall-bladder is discovered while palpating the lower border of the liver. If the mass is of good size and the abdominal wall thin, it will be felt as a pyriform body and may be slightly fluctuating. Such a mass is probably a distended gall-bladder, and it may be associated with enlargement of the liver and jaundice. The causes of enlargement of the gall-bladder are several, viz., distention from the accumulation of bile produced by obstruction in the cystic or common duct, or the presence of a large number of calculi in the gall-bladder, and often to the accumulation of thin mucus, which may or may not be bile-stained, the so-called "hydrops of the gall-bladder." The causes of acute obstruction of the ducts are gallstones, new-growths, such as carcinoma and rarely gumma, and inflammatory processes in the ducts themselves, with inflammation in surrounding tissues and organs. Often the inflammatory processes just named will have been produced by the presence of gallstones, which may, by causing ulceration of the mucous membrane, permit severe infections to take place. Rarely is it due to ascarides or echinococcus. (Courvoisier states the proportion to be in 74 cases, 57 due to stone, 7 to ascarides, and 3 to echinococcus.) The question then arises as to the cause of the enlargement in the particular case at hand. Obstruction of the bile ducts is, as already stated, due, in the vast majority of cases, to stone, aside, of course, from that due to an acute catarrh which is but temporary in its effects, and therefore the probable cause in every case is stone. If this is the cause, the

patient may give the history that at some time in the near or remote past, after a period of more or less distress in the hypochondrium, she has been seized by a pain which was paroxysmal in type and extended backward and upward to the region of the scapula or shoulder. This pain was also peculiar in that it was apt to occur at night rather than after taking food, as in gastric cancer or ulcer, because the bile is flowing to the gall-bladder at this time, instead of directly into the bowel. Examination at such times may have revealed a swelling of the gall-bladder. There may be, but often there is not, a history of jaundice. Manifestly this is a history of an attack or attacks of gallstone colic, and should lead us to the belief that the enlargement of the gall-bladder is due to stone, but it may be due to a malignant growth. In this connection it is well to take into consideration the importance of the presence or absence of jaundice at this time, and this is well expressed by Mayo Robson in the words: "Jaundice with distended gall-bladder is presumptive evidence of malignant disease, but jaundice without distended gall-bladder favors the diagnosis of cholelithiasis." This is sometimes called "Courvoisier's law." In other instances there may be no history of attacks of gallstone colic, the patient simply complaining of pain and discomfort in the hepatic area. Not rarely these attacks of pain are regarded by patient and physician as being due to indigestion, gastric or intestinal.

It is important to remember that in some cases of enlargement of the gall-bladder due to obstruction of its duct the enlargement may be so gradual and so great that the distended viscus may be felt far from its normal site in the middle line or even under the left ribs.

An additional symptom favoring gallstone as a cause is fever, which is due to infection produced by the stone injuring the mucous membrane. Sometimes it is simply an indication of inflammation, at others it indicates anything from mild purulent infection of the bile passages to empyema of the gall-bladder or a general cholangitis. If the infection be with a benign organism, the symptoms may be mild; but if it be due to the more malignant forms it is often virulent, and the febrile movement most severe. Much depends, too, upon the freedom of drainage. It the gall ducts permit the escape of the pus into the gut, the symptoms may become greatly reduced; but if it be retained they are apt to become more pressing. (See Cholangitis, in chapter on Fever.)

When the gall-bladder is distended with gallstones there is often a history of colic; there may be gallstone crepitus on careful palpation, and symptoms of fever due to a cholecystitis set up by the stones. It is not to be forgotten that gallstones are present in about 10 per cent. of all adults, although, as Kehr has well pointed out

fully 95 per cent. of such persons come to autopsy from other causes without even suffering from any manifestation of their presence. It is only when the calculus produces irritation or mechanically blocks the ducts that symptoms develop.

A new-growth by pressing on the gall ducts may obstruct the flow of bile and produce jaundice, and a mass; but there is usually no paroxysmal pain, and the growth is usually secondary to a growth elsewhere in neighboring parts, which may be demonstrable. Rare as is primary malignant new-growth of the gall-bladder, it is almost unknown in the liver, but it may be secondary to a gastric carcinoma which is small in size whereas that in the liver may be a large growth.

The general cachexia, rapid loss of weight, and the age of the patient may aid the diagnosis of the tumor, and in this respect Courvoisier's law is to be recalled.

Pancreatic Carcinoma.—The presence of a resisting mass, deeply situated in the epigastrium, or the upper part of the umbilical area, and felt only on deep palpation, and then often indistinctly, should bring before the mind the possibility of the presence of *carcinoma of the pancreas*, a diagnosis which will be largely confirmed if cachexia be asserting itself, if there be great pain in this neighborhood, and if there are oily stools after fats are taken, as a result of the absence of pancreatic juice. Still further confirmation of this diagnosis will be present if diabetes mellitus develops (pancreatic diabetes). Such a growth in the pancreas is usually a scirrhous cancer, and may be primary or secondary. Segré found that of 627 cases of carcinoma of the upper abdominal organs, cancer of the pancreas occurred in 127, but in only 12 of these primarily. Stiller asserts that the following symptoms are fairly sure signs of pancreatic cancer, namely, marked dyspepsia, rapid emaciation and cachexia, subnormal temperature, persistent and progressive jaundice without hepatic enlargement, but often with swelling of the gall-bladder from obstruction to its duct. These signs are, of course, only of value if the evidence of malignant growth elsewhere can be excluded.

Not rarely a mass, or masses, are felt in the abdomen which may be due to tuberculosis of the omentum, which causes a drawing up or puckering of the membrane, so that there can be felt a firm mass extending across the upper abdominal zone. Sometimes it can be felt to the right or left of the middle line. In other cases, but rarely, we find retracted and thickened coils of intestine which feel like tumor masses, and these are apt to be drawn against the spine, so that the belly is very scaphoid and empty. We also meet with cases in which large tuberculous growths of the mesenteric glands occur. In the last group of cases and in those of the omental type, named above, there may be some ascites.

Finally before dismissing the important subject of swelling and

tumor in the upper abdominal zone it is necessary to recall the fact because of the close juxtaposition of many organs in this area an exact diagnosis may be impossible, so far as a diagnosis of the actual lesion is concerned and yet a sufficiently accurate diagnosis may be made to direct treatment. Thus a chronic inflammatory process above the gall-bladder may by involving the pylorus in its adhesions and exudations cause signs of gastric dilatation and obstruction, or again a history which seems characteristic of gall-stone colic is really due to ulcer of the pylorus. Further than this it is to be recalled that all these facts are in health quite movable and in disease are often found far from their normal sites. Thus the pylorus may be anchored high up in the belly by adhesions or dragged into the umbilical area by the weight of a growth.

Gastric Dilatation.—Gastric dilatation results from obstruction of the pylorus or from inherent feebleness of the stomach wall, and when it is present the entire upper part of the abdomen may be found distended, and tense but yielding. The history will show that the patient is attacked now and again by vomiting, during which a most extraordinary quantity of food and liquid, which has gradually accumulated, will be expelled.

We discover the condition of the stomach as to its size and shape by means of washing it clean with the stomach tube and then filling it with a known quantity of water, which can be siphoned out and measured. Or instead we first wash out the stomach by means of a stomach tube and then fill it with gas by giving the patient to drink, first, a half-glass of water with 1 dr. of sodium bicarbonate in it, and then another half-glass with 30 gr. of tartaric acid dissolved in it, so that gas will distend the viscus. One is able with ease in a thin person, by means of percussion, to outline the stomach by the area in which a high-pitched tympanitic note is heard. It is best to mark the edge of gastric resonance by means of a blue pencil, and thus map out the gastric area. Instead of this, we may distend the stomach with air by attaching a Davidson syringe to a stomach tube or by using an atomizer bulb for the pumping purposes.

Even before the stomach is artifically distended with gas percussion may give us valuable information, for if obstruction of the pylorus exists, there may be found either a large area of gastric tympany through the accumulation of gas from fermentation, or, if no vomiting has taken place for some time, an equally great area of gastric dulness due to an accumulation of food and liquid. If there is a growth at the pylorus causing obstruction, there will be impairment of resonance wherever the pylorus may be situated. While this is a somewhat indefinite statement, it is to be remembered that a more definite one is liable to mislead the student, for even in health

the position of the pylorus changes greatly when the stomach is empty or is filled with food. Thus, when empty the viscus hangs with the pylorus very low, but when it is filled the pylorus is raised. Fig. 117 shows the normal gastric area when the stomach is distended with gas.

In other instances we can use the so-called gastrodiaphane of Einhorn, which consists of a small electric lamp, protected by strong glass, and attached to a rubber tube which contains the necessary

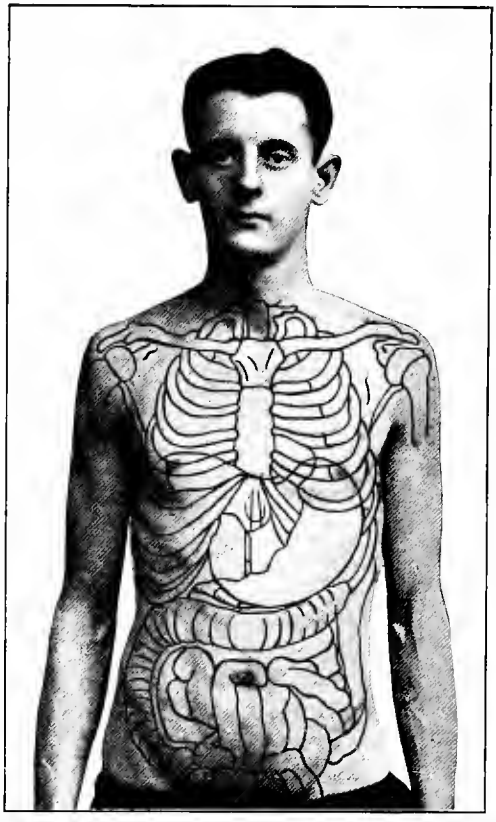


FIG. 117.—Outline of normal position and size of an adult stomach when distended with gas.

wiring for the electric current, and which is swallowed just as is the ordinary stomach-tube. The stomach should first be thoroughly cleansed by lavage, then filled with pure water and the lamp swallowed. If the patient be moderately thin and the inspection is made in a dark room, the outline of the lighted stomach can be seen through the abdominal wall and some idea of its dimensions obtained. Normally, the greater part of the stomach will be found to the left of the

middle line and about one to two inches above the umbilicus. (See Percussion.)

An improved and more thorough method of carrying out this plan of examination is by the use of fluorescin. The patient is given 2 gr. of quinine bisulphate three times a day for twenty-four hours before the examination is made. He then swallows a tumblerful of a solution of sodium bicarbonate in distilled water (35 gr. to the pint)

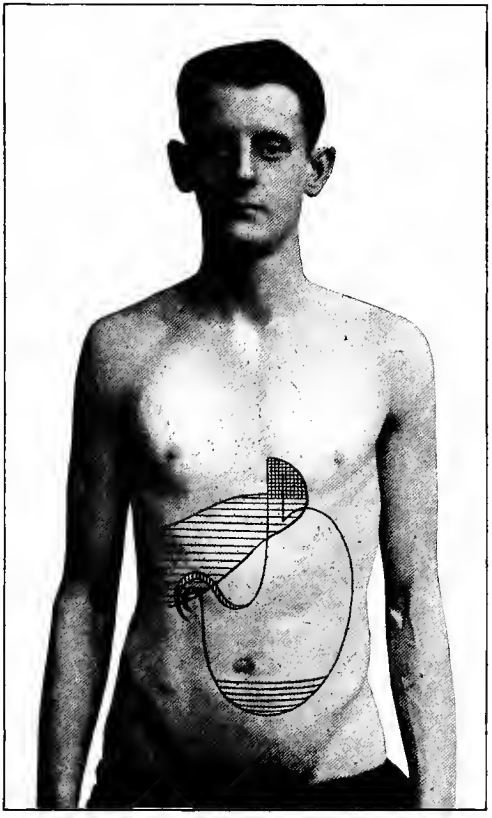


FIG. 118.—Showing the change in the position of the stomach in dilatation, and how difficult it is for this organ to empty itself of fluids.

and follows this by another tumblerful of another solution made by adding 2 fl. dr. of glycerin, $\frac{1}{4}$ gr. of fluorescin and 35 gr. of sodium bicarbonate to a pint of distilled water. If after this is done the gastrodiaphane is introduced it is said to greatly increase the degree of illumination. In a limited use of the gastrodiaphane, with and without fluorescin, I have not been impressed with its value, but a larger experience may give better results.

Another very valuable method of determining the presence of gastric dilatation and gastroptosis is by the employment of the *x*-ray and large doses of bismuth, which substance is used because it renders the gastric walls opaque. The dose of bismuth must be massive, as much as 2 oz. mixed with mashed potato or held in suspension in a pint of mucilage of acacia. After the examination the bismuth must be withdrawn by a stomach tube to prevent bismuth poisoning. The best results are naturally obtained when the patient is in the erect position. Either the fluoroscope or the radiographic plate may be employed. Usually it is best to use both methods.

FIG. 119.—Gastroptosis and enteroptosis due to relaxation and atrophy of belly wall. Hornet's nest belly.

In many cases of dilatation of the stomach, or of enteroptosis, the use of "Glenard's belt sign" may be resorted to. This consists in standing behind the patient, placing the hands upon the lower part of the abdomen and lifting upward and backward, when if gastroptosis or enteroptosis is present relief from the sense of dragging may be felt.

I have found this method of diagnosis very serviceable.

Gastric dilatation is often associated with atrophy of the gastric

tubules, or at least an absence of any secretion of normal gastric juice. The matters vomited or washed out of the stomach are often devoid of hydrochloric acid, but loaded with an excess of lactic acid.

Test for Lactic Acid.—Lactic acid is tested for as follows, the hydrochloric-acid test being given below: a few drops of neutral ferric chloride solution are mixed with one or two drops of pure carbolic acid, 10 c.c. of a 5 per cent. solution of carbolic acid, and water added until an amethyst hue develops. A few drops of the filtrate derived from the stomach contents are now added, and if lactic acid or lactates are present the amethyst hue will become yellow in color. This is a very delicate test for lactic acid.

Test for HCl.—The presence or absence of hydrochloric acid is determined in the following manner: The patient is directed to take no food for at least twelve hours before presenting himself to the physician. On his arrival for examination he is given what is known as "Ewald's test breakfast,' which consists of an ordinary dry roll and a little over a half pint of water which has been warmed, and he is directed, after swallowing these materials, to wait an hour. The stomach is now emptied by the introduction of the bulbed stomach tube, and the gastric contents filtered. A few minims of a solution of phloroglucin and vanillin are next placed in a porcelain dish and a few drops of the gastric liquid are allowed to trickle down to the edge of the solution. The dish is gently heated over a spirit lamp or Bunsen burner, and if hydrochloric acid is present there will appear a red tinge. This is an absolute proof of the presence of hydrochloric acid.

The solution of phloroglucin and vanillin is made as follows:

Phloroglucin	gr. xxx.
Vanillin	gr. xv.
Absolute alcohol	f ℥j

This solution is pale yellow in hue. It must be kept in dark bottles, as on exposure to the air and light it becomes brown and worthless.

In some cases symptoms of gastric dilatation may arise from the presence of "*hour-glass stomach*" a state in which a stricture divides this organ more or less completely into two parts. The presence of this state may be determined by filling the stomach with water or gas from the halves of a Seidlitz powder taken separately. On percussion it will be found that the cardiac area is distended but the pyloric area is empty, and some minutes later the pyloric portion becomes distended. During this period the use of a stethoscope over the middle area of the stomach may reveal the sounds made by the passing of the fluid or gas through the stricture. If the stomach is

washed out by lavage until the liquid is clear and then after a short time washed again and the returned liquid is found to be foul, this is a sign of hour-glass stomach because the cardiac area has been filled by retained contents on the pyloric side of the stricture. So too the gastrodiaphane may reveal light only in the cardiac area.

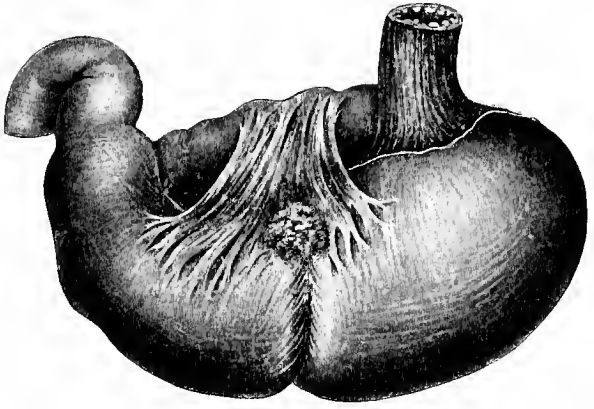


FIG. 120.—Hour-glass stomach.

The Liver.—Normally, in the adult, this gland cannot be felt below the ribs, except part of the left lobe in the epigastrium occasionally. Sometimes, on deep inspiration, the diaphragm pushes the liver low enough to be felt. In children the liver is naturally large enough to be felt below the ribs.

When the normal liver is percussed we find that it lies in the area shown in Fig. 115, and that as we percuss above it on the ribs in the mammary line we first get pulmonary resonance; then a little below this, impaired resonance, due to the fact that the lower edge of the lung is interposed between the chest wall and the liver; and still lower we find absolute dulness or flatness, due to the solid liver itself. Below this area, which ceases just below the lowest rib, we usually find tympany on percussion, due to the gas-distended bowel. If we percuss in the midsternal line, we get the same signs; but they begin as high as the nipple, or above it, and then cease at a line drawn across the abdomen about midway between the ensiform cartilage and umbilicus. To the left of the middle line of the sternum the liver dulness merges into the cardiac dulness (Fig. 121). In the mammary line liver dulness begins at the fifth rib, laterally it begins at the seventh and eighth, posteriorly at the tenth rib, owing to the sloping of the diaphragm.

When a hard and firm mass with a smooth surface can be felt in the right hypochondrium or right umbilical area, which is movable, and

which has an edge which can be readily felt on deep palpation, particularly when the patient takes a long-drawn, deep breath, the mass is probably an enlarged liver or a liver pushed down into the abdominal cavity by a large pleural effusion, pneumothorax, a subphrenic abscess, or sometimes by an emphysematous lung. The causes of enlargement are lymphadenoma and amyloid degeneration, congestion, hypertrophic cirrhosis, abscess, carcinoma, sarcoma. When the surface is found to be smooth, the condition is probably amyloid or fatty degeneration, or congestion. If the surface is

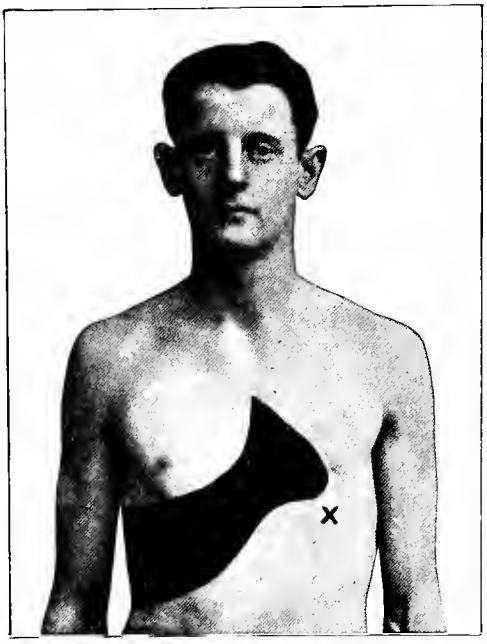


FIG. 121.—Showing percussion dulness of liver and heart. The outside line shows the area of partial dulness of heart and liver, modified by lung. The solid area is that of true hepatic dulness. At *x*, Traube's semilunar space.

rough, it will probably be due to cirrhosis, which gives a granular sensation to the hand when the abdominal wall is moved over the organ. In malignant growth large and small nodules may often be found, and depressions or umbilications of its surface may be marked, but it must be remembered that cancer of the liver is not necessarily associated with the presence of nodular masses. On the contrary, the growth or growths may be large, yet project so slightly above the hepatic surface that they cannot be felt. In such cases there may be pain, marked emaciation, cachexia, and the organ be found much enlarged.

The physician who feels distinct nodules on the surface of the liver should not immediately conclude that these are necessarily carcinomatous, for syphilis often produces a very extraordinary nodulation of the surface of this organ. So great is this, that when nodulation is excessive the possibility of syphilis being the cause is to be considered. This form of disease is, however, rarely accompanied by as great hepatic enlargement as is that due to cancer with marked and multiple nodules.

The consistency of the liver is usually very hard in cases of cirrhosis, carcinoma, and amyloid degeneration. In cirrhosis there will be some ascites in many cases, some swelling of the legs perhaps, and dull pain in the hepatic region. The digestion will be disordered, there will be marked loss of flesh, and often hematemesis. Sometimes coma comes on. In the case of amyloid liver there will be a history of prolonged suppuration elsewhere, and there will be present disordered digestion, irregular bowel movements, and little pain.

Marked tenderness of the hypochondrium is usually found in congestion of the liver, in inflammation of its tissues, such as that caused by an infection or by gallstones in its substance, and in malignant growth. Tenderness is practically absent in amyloid liver and in fatty degeneration.

In cases of *cirrhosis of the liver*, whether it be in the hypertrophic or atrophic form, the patient rarely complains of the organ, and no symptoms which seem to him hepatic in origin may be present, save that in the hypertrophic state its size is increased, so that it can be felt below the ribs, whereas in the atrophic state it cannot be felt except by pushing the fingers well up under the ribs. The symptoms accompanying cirrhosis are chiefly connected with disorders of the alimentary canal, either through direct failure in the digestion and assimilation of food, or from changes in the blood supply of the abdominal contents. The following excellent diagram, from Seymour Taylor's *Index of Medicine*, shows what these symptoms are, and discovers their cause at a glance, the cirrhotic process, of course, obstructing the flow of blood in the liver (Fig. 122). It is a noteworthy fact that in the atrophic form jaundice is rare even in the very last stages of the disease, whereas in the hypertrophic form it is commonly met with. Ascites is common in the atrophic form, rare in the hypertrophic variety.

Sometimes enlargement of the liver and ascites are due to *adhesive pericarditis*. The diagnostic signs of adhesive pericarditis consist in systolic retraction of the intercostal spaces in the anterior axillary line, and posteriorly at about the fifth or sixth rib on the left side, which retraction is followed by a diastolic rebound. If the patient is told to forcibly inspire or expire, the natural change in the position

of the apex beat of the heart in relation to the chest wall does not occur, neither does the edge of the lung on full inspiration diminish the area of cardiac dulness as it does in health. Pericarditis with ascites is often associated with hyperplastic perihepatitis, the "Iced Liver" of Curschmann.

The physician who finds the lower margin of the liver abnormally low down in the abdominal cavity should not make a diagnosis of enlargement of this organ until he has assured himself that the extension of the margin of the liver is not due to an effusion in the right pleural cavity which presses upon this organ. So, too, if the patient is a woman, the lower border of the liver may have been pushed down by tight lacing, and careful palpation may reveal a furrow across its surface produced by the corset. The displaced lower

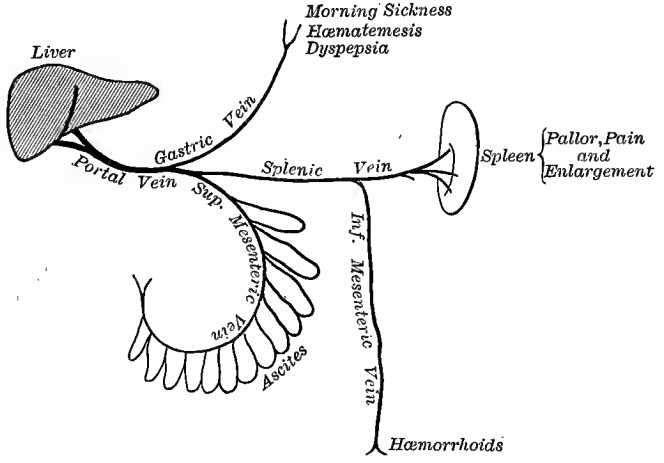


FIG. 122.—To illustrate symptoms of cirrhosis of liver. (Seymour Taylor.)

border of the gland gives rise to the diagnosis of enlargement of the liver if careful percussion shows that the upper border of liver dulness is in its normal place. If the upper border of the liver is depressed, and pleural effusion is absent, it is a sign of floating liver.

Hepatic Abscess.—When, on palpating the liver, we find marked tenderness and some swelling, and, associated with these symptoms, fever, rigors, sweats, and sometimes vomiting, and, in addition, a history that the patient has had dysentery or has had exposure to tropical heat or has swallowed bad water, the presence of an abscess of the liver is indicated. This may be single or multiple. If the latter, it is probably due to pyemia, and no spot of fluctuation will be found, as a rule; whereas, if it is large and single, fluctuation is sometimes felt. Further, the enlargement of the liver in the pyemic form is uniform, whereas in the single abscess there is

often one spot which is swollen or enlarged. The history of the case will usually separate the conditions, one from the other, for diagnostic purposes, for in the case of abscess the history will probably be that of a person exposed to tropical heat or one who has had an injury, an acute infection, or an amebic dysentery. More rarely a single hepatic swelling may be due to hydatid cyst, but the history and presence of fluctuation, combined with the result of examining the fluid aspirated from the swelling, will decide the diagnosis. Further than this, hydatid cyst yields on percussion a peculiar vibratory thrill called the hydatid thrill. Three fingers are placed over the area, the middle one being pressed firmly upon the growth and the lateral ones but lightly. The middle finger is now percussed with the other hand and allowed to remain *in situ*, when an after-thrill may be felt in the other fingers.

Pancreatitis.—The appearance of sudden swelling in the neighborhood of the pancreas, associated with intense pain, nausea, and vomiting, may be due either to acute hemorrhagic pancreatitis, to hemorrhagic infarction of the intestine, to intestinal obstruction, acute cholecystitis, or to acute peritonitis resulting from perforation of the stomach. (See chapter on Pain.) The last three are the more common. An exploratory operation is the only way of deciding the diagnosis positively, although the history of the patient may aid us in deciding the cause of the illness. Thus, if there is a history of gallstone colic, this may indicate that a stone has become impacted in the common duct near the papilla. Such an accident, if it dams back the secretion into the pancreas, causes hemorrhagic pancreatitis with fat necrosis. Sometimes, however, the symptoms of hemorrhagic pancreatitis are more prolonged, and life lasts for several weeks, local swelling, jaundice, and pain being present, with symptoms of suppuration.

Pancreatic Cysts.—Very rarely swelling in the epigastric region, either rapid or slow in onset, follows upon the formation of cysts in the pancreas, as a result of obstruction of a duct of the gland. When they occur, these cysts may quite fill the abdominal cavity, although, as a rule, they are small.

Hemorrhage into the Lesser Peritoneum.—As pointed out, however, by Jordan, the cause of a swelling in the pancreatic region may be hemorrhage into the lesser peritoneal cavity. He summarizes some of his views in regard to this matter as follows:

"Contusions of the upper part of the abdomen may be followed by the development of a tumor in the epigastric, umbilical, and left hypochondriac regions. Such tumors may be due to fluid accumulating in the lesser peritoneal cavity, and when the contents are found (on aspiration) to have the power of converting starch into sugar we may assume that the pancreas has been injured." Finally,

Jordan states that "many such tumors have been regarded as true retention cysts of the pancreas."

Pyopneumothorax Subphrenicus.—In other instances a swelling in this neighborhood may be due to what is called *pyopneumothorax subphrenicus*, a condition of abscess in the peritoneal cavity below the diaphragm, produced by perforation of the stomach or transverse colon or appendicitis. The abscess so produced may contain gas, and for this reason the swelling may be quite resonant on percussion (Fig. 123). Abscess in this region also follows abscess of the pancreas or fat necrosis of this organ in rare instances.

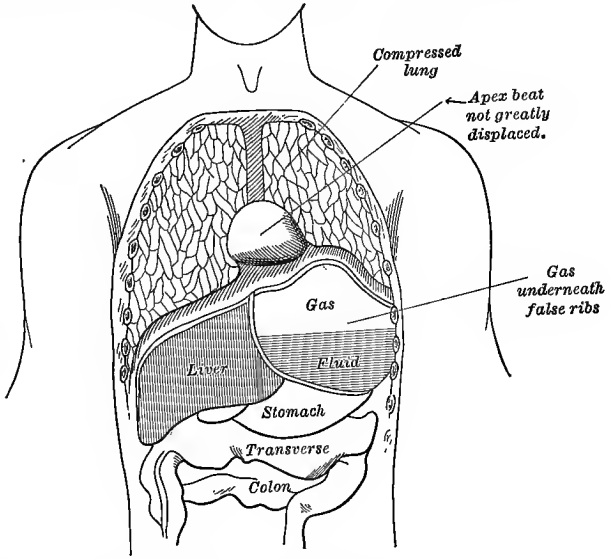


FIG. 123.—Left pyopneumothorax subphrenicus. (After Maydl.)

Epigastric Pulsation.—Either palpation or inspection may reveal pulsation in the epigastric area. This may be due to distention or enlargement of the right ventricle or to excessive aortic pulsation or to venous pulsation in the liver. If due to the action of the ventricle there will be additional signs of cardiac disturbance on examining the heart, and in hepatic pulsation there will not only be found tricuspid regurgitation, but a pulsation below the floating ribs at the lower border of the liver. An excessive aortic pulsation is often met with in hysterical or neurasthenic persons without any abdominal lesion. Epigastric pulsation is also often transmitted from the aorta to the hand by enlarged abdominal glands or tumor masses. If the pulsation of the aorta is not transmitted by glands or tumors, impulse may be due to aneurysm of the abdominal aorta, the diagnosis of which is established if, in addition to a pulsating

sensation, we also find on palpation a marked thrill, an expansile movement of the tumor, and on auscultation we hear a bruit. Pain due to pressure of the aneurysmal sac upon some of the nerves of the abdominal cavity may also be a prominent symptom, but it should be remembered that aneurysm of the abdominal aorta is so rare that the law of probabilities is always against its presence. Sometimes a horseshoe kidney extending across the vertebral column will mislead one into a diagnosis of an intra-abdominal tumor, for horseshoe kidney is not very rare, being found as often as once in 1650 autopsies.

Tumors of the Bowel.—Tumors or foreign bodies in the bowel can nearly always be moved about unless bound down by inflammatory adhesions, so differing from growths which involve the immovable parts, such as the retroperitoneal glands. Very rarely we find a cancerous tumor of the omentum, but when it is present it usually becomes retracted and indurated so that its hardened edges can be felt extending across the abdominal cavity. More commonly when multiple nodules are found in the omentum or studded over the surface of the bowels, they are due to peritoneal tuberculosis. Not rarely these nodular masses are also found studded over the mesentery.

Localized masses due to other causes than those already discussed are due to impaction of feces, volvulus and intestinal obstruction from other causes, as, for example, cancer of the bowel. Such a growth occurs most frequently in the cecum, when the tumor will be found in the right groin, or in the sigmoid flexure, when it will be found in the left groin.

Floating Kidney.—Floating kidney may also cause a marked movable swelling or tumor-like mass in the upper zone of the abdomen. It may be mistaken for a tumor of the uterus, or of the liver, omentum, ovary, or spleen, or even for a much distended gall-bladder. If the belly walls are thin, the peculiar shape of the kidney can sometimes be outlined by palpation, and even the pulsation of the renal artery can be felt; but, as a rule, this cannot be done, and the dilatation of the pelvis of the kidney by the obstruction of the ureter, which has become twisted, may distort the shape of the organ. Deep palpation of the flank, if the kidney has floated away from its normal seat, may reveal lessened resistance in this area, and bimanual palpation, one hand being placed at the back and the other in front, may reveal the presence of the organ elsewhere. Further, if the patient be made to lie on the side, the dislocated kidney may sometimes be clearly outlined by bimanual palpation. In other instances, the patient lying on the back with the thighs flexed, the physician grasps the side between the rib and the iliac spine and directs the patient to take a full breath, when the kidney, if movable from its normal resting place, can be

felt passing down between the thumb in front and the fingers behind. The fingers should be in the flank and the thumb over the side of the abdomen (Fig. 124). If the kidney be pressed upon gently, it can be slipped back into place on expiration. In other instances the patient stands erect, and the physician places the finger tips of the left hand in the lumbar region, and pressure is made toward the front of the body. The fingers of the right hand are placed anteriorly, and pressure is made backward and upward so as to engage the kidney between the finger tips of the two hands. The vertical posture of the body aids in displacing the kidney, which may stay in place in the dorsal decubitus. The kidney may slip with a jerk, if already displaced, back into its normal position; or, if already in place, it may be felt to escape downward into the abdominal cavity. Pressure on a floating kidney causes a peculiar nauseat-

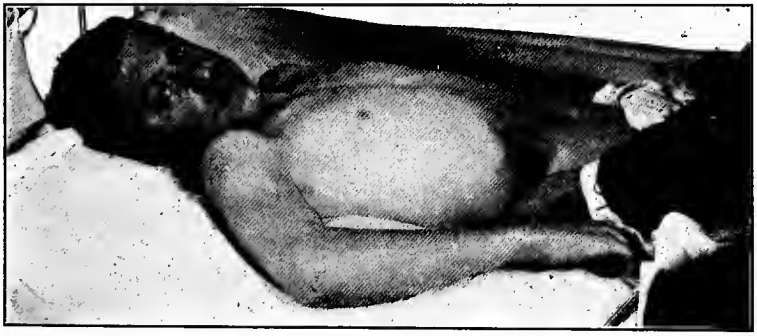


FIG. 124.—Method of examining for floating kidney. The physician grasps the side with his hand in such a way that the fingers and thumb are approximated, and when the patient takes a full breath the kidney can be felt slipping through between the fingers and thumb.

ing pain somewhat resembling that produced by squeezing a testicle, and when the organ is caught between the hands it slips from the pressure with a sensation resembling that felt by the fingers when an orange seed is pressed between the finger and thumb and escapes the pressure. The condition of floating kidney is more common in women than in men, but it occurs in both sexes. In 667 cases, 583 occurred in females and 84 in males. (Kuttner.) It is generally the right kidney which is displaced, although dislocation of the left kidney is not very rare. In Kuttner's 727 cases it occurred on the right side in 553, on the left in 81, and both sides in 93. Sometimes violent attacks of renal pain occur in cases of floating kidney. These have been called Dietl's crises. (See chapter on Pain.)

Cystic Kidney.—When the kidney is enlarged from cystic degeneration, from ordinary hydronephrosis, and from echinococcus cysts, it may be readily felt in the umbilical area in many instances.

Hydronephrosis.—Hydronephrosis has been mistaken, in children particularly, for sarcoma of the kidney, and in adult females for ovarian tumor. The diagnosis in some of these cases can be made only by tapping. The fluid obtained in hydronephrosis will usually be somewhat turbid and contain epithelial cells. It should not be forgotten that the condition of hydronephrosis may be intermittent, for, if this is not remembered, the physician may be misled into thinking that the disappearance of the swelling is due to a floating

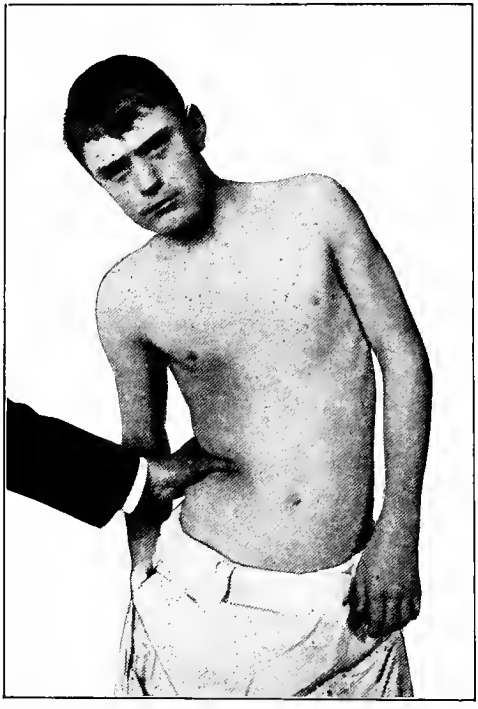


FIG. 125.—Method of examining for floating kidney. The physician grasps the side with his hand in such a way that the fingers and thumb are approximated, and when the patient takes a full breath the kidney can be felt slipping through between the fingers and thumb.

kidney slipping back into its place. This intermittence in the size of the tumor may be of considerable diagnostic aid, for sudden decrease in size would indicate the escape of fluid through a temporarily patulous ureter, and its redevelopment would indicate that this pathway of escape was again closed. Should the fluid escape into the bladder free urination would naturally take place shortly after the tumor decreased in size. Hydronephrosis may be bilateral. In 13 out of 20 cases collected by Roberts this was the case. Severe pain is often a symptom of intermittent hydronephrosis.

Pyelonephritis and pyelonephrosis may closely simulate hydronephrosis, but fever and the presence of pus in the urine will aid in making the differential diagnosis.

Bulging of the flank, with pain, fever, and perhaps fluctuation, indicates perinephritic abscess or caries of the spine with cold abscess.

Mesenteric Cyst.—A fluctuating swelling in the epigastrium or flank may also arise from cysts of the mesentery. These may grow to a very large size. In other cases a cystic hydroma of the tissues near the kidney may be present. Hawkins has recorded a case in which a large cyst, with an atrophic third kidney attached to it, filled nearly the entire right side of the belly, and from which after death five pints of clear fluid, devoid of albumin and casts, were removed. As already indicated, much diagnostic aid can often be given by tapping an obscure abdominal cyst.

J. G. Clark has recommended a method of separating solid from fluid tumors, which is called trimanual percussion. This consists in fixing the mass between two hands, one below it if possible, the other above it. An assistant now percusses, striking the finger of the physician. By this means a thrill can be felt which would otherwise be lost.

Phantom Tumor.—"Phantom tumor" is generally found in hysterical women, and often leads to ludicrous errors in diagnosis. It is due to persistent dilatation of a knuckle of intestine by gas, thereby forming a moderately hard and more or less constant mass, which may resemble a real tumor. Examination of the patient under ether will usually reveal its true character. This state is to be differentiated from what Nothnagel has called "intestinal rigidity." In this condition a knob projects through a thin anterior abdominal wall and gradually grows larger until in the course of a few moments it sinks out of sight and touch. It is a disorder of peristalsis. Localized superficial and inconstant tumors may arise through spasmodic but localized contractions of the recti muscles.

Umbilical Hernia.—Finally, a swelling in the neighborhood of the umbilicus should always arouse the suspicion of an umbilical hernia. The situation of the swelling at the umbilicus, the fact that percussion over it gives a highly tympanitic note, owing to the gas in the prolapsed gut, and the possibility of reducing its size by taxis in some cases, will render a diagnosis of umbilical hernia possible.

The Spleen.—In the left hypochondrium the spleen can be very readily outlined by percussion in persons not inordinately fat. Its normal position is best shown in the accompanying figure. (See Fig. 126.)

The upper border of the spleen is on a level with the tenth dorsal vertebra and the lower border on a level with the end of the eleventh

rib. Its upper edge or limit is on a level with the ninth rib. In percussing the spleen heavy percussion is to be avoided, since this may develop the resonance of the stomach or bowels. The spleen cannot be palpated unless greatly enlarged, but it may be found bulging from beneath the lowest rib in typhoid fever; in scarlet fever; as the result of acute or chronic malarial fever; in leukocythemia of the splenomedullary variety (see Fig. 128) (see chapter on the Blood); in amyloid disease, as that after long suppuration; in early syphilitic infection; and in any disease which causes venous

FIG. 126.—Normal position of the spleen.

engorgement of the abdominal viscera, such as cardiac disease or hepatic cirrhosis. The spleen sometimes reaches a very large size in the disease which has been called "splenic anemia." In this condition we have a state resembling Hodgkin's disease, save that there is not the involvement of the lymphatic glands. The blood, unlike that in true leukemia, does not show great changes in the white cells, any changes present consisting in an increase in the relative number of the lymphocytes, which may, in some cases, make up nearly all of the leukocytes. The red cells are greatly decreased in number, and there are present poikilocytes, megalocytes, and microcytes. Closely allied to this condition of the spleen is that which consists in the so-called "Banti's disease," in which the spleen is also greatly enlarged. This disease is divided by Banti into three stages: a stage of anemia

characterized by enlargement of the spleen, and lasting from three to ten years; a transitional stage; and a third stage of marked ascites, which usually terminates in a few months. In some cases of so-called Banti's disease, hemorrhages take place, such as vomiting of blood; nose-bleed, hemoptysis, and hematuria. Banti's disease differs from splenomedullary leukemia in the absence of an excess of white cells, and it is supposed to differ from splenic anemia in that there is not the same increase of lymphocytes as is characteristic of that condi-

FIG. 127.—A case of chronic enlargement of the spleen following typhoid fever. The dark line shows the margin of the organ on palpation, while the retraction in the line and the dotted line indicate the position of the splenic notch. (From the author's wards in the Jefferson Medical College Hospital.)

tion. It is to be remembered that splenic anemia and Banti's disease are scarcely regarded as pathological entities by many clinicians. These terms cover obscure conditions about which we know little. (For splenomegaly with polycythemia, see chapter on the Blood.)

Sometimes displacement of the spleen downward arises from emphysema of the lungs or left-sided pleural effusion.

Acute splenic swelling sometimes comes on in cases of general septicemia.

Nearly always the splenic surface is smooth, except for the notch in its surface, unless the disease be the rare condition of hydatid disease or carcinoma.

In connection with the subject of abdominal tumors, we should not forget the possibility of a floating spleen, a rare condition, but one more common than is generally thought. The shape of the organ, if it can be palpated, will aid the diagnosis, and the pres-

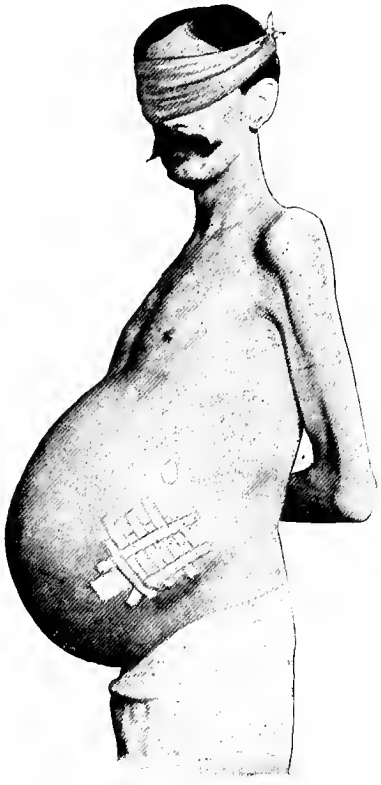


FIG. 128.—A case of enormous enlargement of the spleen in splenic leukemia. (From a private patient.)

ence of resonance on percussion over the area of normal splenic dulness will confirm the diagnosis that the spleen has become displaced. As the spleen in this condition may fall as low as the virgin uterus, it may simulate any growth from a uterine myoma to a tumor of the bowel or pancreas. By reason of twisting of its pedicle and secondary engorgement, its size may be enormous; but if this condition continues, atrophy finally takes place. As such a dislocated spleen drags on the stomach and pancreas, it may cause a long train of

curious symptoms, and even intestinal obstruction. Sutton asserts that by pressure it may cause displacements of the uterus.

In cases in which it is difficult to determine the location of an abdominal growth it may sometimes be localized by distending the colon with air, which is gently injected into the bowel by an ordinary bulb syringe.

The Groins.—There yet remains for discussion the significance of increased resistance on palpation, and percussion dulness, in the groins.

In the right iliac region the presence of swelling, increased resistance, impaired resonance, or tympanites, particularly if pain and tenderness are present, point strongly to appendicitis or to inflammation about the *caput coli*. Sometimes, however, the presence of a distinct lump in this region in a person advanced in life may mean a malignant growth, for carcinoma of the caput coli is not rare.

If the left groin is affected in a person well advanced in years, carcinoma is also to be regarded as possible, for the sigmoid flexure is a frequent seat of such growths. In a young person or a child impaction of feces, a foreign body, or intestinal obstruction is also to be considered. (See chapters on Vomiting and on the Bowels.)

For the diagnosis of renal disease reference is to be made to the chapters on the Bladder and Urine, the Blood, the Bloodvessels and the Pulse, and upon the Thorax (that part on the Heart), to the chapters on Vomiting and on Headache, and to those on Coma and Unconsciousness, and Convulsions and Spasms.

For further information in regard to the diagnosis of diseases of the abdominal viscera, the reader is referred to the chapter on the Skin (that part on Jaundice), the chapter on Vomiting (that part on Intestinal Obstruction), to that on the Bladder and Urine, and to that on the Bowels and Feces.

CHAPTER X.

THE BOWELS AND FECES.[1]

Constipation and diarrhea—The cause of these two symptoms and their diagnosis—The diseases in which these symptoms occur—Choleraic diarrhea—Dysentery—The color of the feces—Intestinal parasites.

THE consideration of the condition of the bowels and feces as indicative of disease affecting the intestines themselves and other organs closely associated with their functions, can be best divided into several parts, namely, the functional disorders of the intestines and the organic diseases from which they may suffer, on the one hand, and the appearance of the feces in both functional and organic diseases of the abdominal viscera in general, on the other. The most common forms of intestinal disturbance are constipation and diarrhea.

Constipation.—Constipation may be due to mere sluggishness of bowel movement because of both nervous and muscular atony, or to deficient secretion of the intestinal juices, or, again, to the too rapid absorption of the liquids from the fecal matter while it is passing through the colon. It is also associated with all those conditions which prevent the proper flow of bile, which liquid very materially increases peristalsis. Thus, we see obstinate constipation in most cases of true jaundice; in cases of *hepatic disease,* producing a deficient biliary flow; and in phosphorus poisoning, in which the fatty degeneration and hepatitis prevent biliary secretion. Further than this, the constant ingestion of foods which are absorbed nearly *in toto,* or, in other words, leave little residue, particularly raw or boiled milk, produces constipation. Again, the use of wines containing large amounts of tannic acid may produce similar results because of the astringency of this substance, and chronic constipation from the use of large quantities of badly infused or boiled tea made with hard water is frequently met with.

When the feces are very dry, the cause may be lack of liquid ingested, and the remedy be full draughts of pure water; or, again, constipation occurs as a manifestation of *diabetes insipidus* or *diabetes mellitus,* because the polyuria characteristic of these affections drains the body of liquid. Obstinate constipation should, therefore, always call the physician's attention to these affections and to two other possibilities, namely, that the con-

[1] For intestinal obstruction in its various forms, see chapter on Vomiting.

dition depends upon wilful disregard by the patient of the calls of nature, so that the bowel is forced to retain fecal matter until it becomes hard and dry; or, quite as important, that the constipation may be due to some reflex cause, which, as the result of irritation, results in an arrest of peristaltic movement. Thus, a woman with ovarian and other *pelvic trouble* may have obstinate constipation which yields little, if at all, to purgatives, but readily to nervous sedatives or even to an opiate. Or, again, in *chronic lead poisoning* the inhibitory fibers of the splanchnic nerves and the intestinal muscularis may be so irritated that peristalsis is impossible. Here a hypodermic injection of morphine may make a movement possible.

The organic diseases of the bowel producing constipation are many and of great importance. They consist in *intestinal obstruction* in all its forms, as by bands, growths, by the process of intussusception, by volvulus, by cicatricial contractions, and by impacted foreign bodies or fecal matter. The presence of a sudden attack of constipation, or the presence of this condition in a degree which fails to yield to mild laxatives, should always put the physician on his guard lest some grave condition is present. As severe and, finally, stercoraceous vomiting is a fairly constant and more marked symptom of intestinal obstruction than is constipation, a discussion of the various symptoms of intestinal obstruction will be found in the chapter on Vomiting, and the diagnosis of growths of the intestine will be found in the chapter on the Abdomen.

Aside from these causes, it is manifestly impossible to discuss all the conditions of the system in which constipation may be present. The physician must always bear in mind that constipation often results in the absorption of poisonous materials from the bowels, which in turn may produce all sorts of symptoms, nervous or otherwise, from epileptiform attacks, in rare cases, to severe headache and vertigo, with vomiting, in others.

Diarrhea.—Diarrhea of an acute type depends, as a rule, upon one of four causes, namely, the presence of *irritant material* in the bowel, which the intestines attempt to get rid of by increased secretion and excessive peristalsis; relaxation of the bloodvessels of the intestine, with profuse serous leakage and consequent watery purging; *acute inflammation*, with excessive secretion of mucus; and the endeavor of the system to eliminate poisons in this manner, as in cases of sudden profuse diarrhea, in cases of *chronic renal disease*, in which the purging is an effort at elimination. The last-named forms of diarrhea are usually sudden in onset and speedily get well of themselves, and it is a mistake to check them too suddenly.

It is impossible to speak of all the possible causes of diarrhea, or of all the diseases in which it is met with. Only those in which it is a prominent symptom, or one of importance, can be discussed.

One of these is *cholera morbus*, a disease which manifests itself in profuse watery purging, accompanied by violent pain in the belly, and, after several stools have passed, in a considerable amount of tenesmus. Mucus is almost entirely absent from the dejecta, but particles of undigested food may be found in them. Vomiting is often a severe and simultaneous manifestation of the gastro-intestinal disorder which exists, and, if the attack be very severe, it is practically impossible to separate it from true cholera Asiatica if an epidemic of that disease is present. The patient speedily becomes cold and pinched-looking, exceedingly weak, and finally may pass into collapse. The pulse becomes feeble, rapid, and running; the face livid, and finally the patient may develop the *facies Hippocratica*. The urine is greatly decreased or entirely suppressed, because of the watery purging, and possibly by reason of the effects of certain poisons upon the kidneys. In the great majority of cases the symptoms are not so severe as this, and complete recovery ensues as soon as the offending materials are passed out of the bowels and the patient has time to convalesce.

When an attack of diarrhea, such as has just been described, comes on in a young child it is usually called *cholera infantum*, or "summer complaint," and it is nearly always due to improper feeding or to the unintentional use of bad food or bad milk. The stools of the child are usually at first filled with curds of milk and green masses, looking as if the curds had been stained with grass juice or spinach. The child often passes with extraordinary rapidity into a state of collapse, and may die in a few hours or days. The tenesmus often becomes constant and is a distressing symptom, and the tissues become shrunken to a marked degree. The child manifests not only the evidences of the results of profuse purgation, but, in addition, is evidently intoxicated by the toxins absorbed from the bowel, so that it lies on the lap of the nurse in a relaxed and torpid state. The surface of its body is often abnormally cold, and its extremities may be pinched and blue; but the temperature of the internal organs is generally abnormally high, so that while the axillary temperature may be below normal, the thermometer will reveal a temperature of from 102° to 103° in the rectum. Sometimes the head becomes retracted, as if meningitis was present. The respirations may be sighing or of the Cheyne-Stokes type.

If the child or adult is seized with symptoms such as those described under cholera morbus or cholera infantum, and a suspicion of the presence of *true cholera* is raised, Are there any facts which will point to the correct decision in a case, even if, as already stated, a positive differential diagnosis cannot be made? In the first place, a train of symptoms of a malignant type points to the true cholera, rather than cholera morbus, or cholera nostras, as it is sometimes called. Again,

the evidences of infection or general systemic disease indicate the epidemic malady rather than does a profuse diarrhea alone. Thus, the systemic signs of infection may be so great that death from infection in true cholera occurs before diarrhea even begins. Again, it would be possible to determine the presence of true cholera if the comma bacillus could be demonstrated; but this requires the examination of the fecal matter to be made by an expert who is familiar with the technique of examining fecal matter for the germs and with the necessary measures for their artificial culture.

Symptoms identical with the more violent forms of cholera nostras or true cholera may be produced by *acute poisoning by antimony*, except that in this case we often have profuse sweating and salivation early in the attack. The same symptoms of vomiting, purging of rice-water stools, collapse, cramps in the calves of the legs, and violent pain in the abdomen may be present. A differential diagnosis without the history of the patient having taken poison is impossible, except by a chemical analysis of the vomited matter, which will contain antimony, as will the stools and the urine. The utmost care should be used that the vessels which receive these materials are chemically clean, that they are hermetically sealed until ready for the expert analysis, and that they are in the hands of thoroughly responsible parties up to the date of analysis.

While *arsenic* may cause somewhat similar symptoms to those due to antimony, the stools are generally bloody. Rarely certain poisonous toadstools produce somewhat similar symptoms.

If an adult who has not eaten anything which could have produced a diarrhea, as, for example, bad food, is seized with profuse watery purging, with very little or no pain, and without nausea and vomiting, it is probable that he is suffering from the *acute nervous diarrhea* which sometimes results from exposure to severe nervous strain. To illustrate the character of these cases the author may mention the fact that it is quite common for him to see medical students, exhausted by a long winter's work and anxious about their examinations, seized by an attack of profuse watery purging in the middle of the night preceding the examination of which they stand most in dread.

In other cases profuse purging develops suddenly in hot weather as a form of *heat prostration*.

Care must be taken by the physician in all cases of sudden and profuse diarrhea to which he is called to exclude the presence of *renal disease*, for purging may be an effort at elimination of effete materials, and its sudden arrest by drugs may induce uremic convulsions or coma.

Sudden attacks of profuse watery diarrhea in which the patient passes great quantities of liquid from the bowel, with or without pain

in association therewith, may be due to *locomotor ataxia,* manifesting itself in an "intestinal crisis."

In cases of persistent or obstinate diarrhea, serous or catarrhal, in which there is an excessive peristalsis which hurries the intestinal contents along so fast that the food cannot be properly digested, the physician should remember that *fissure of the anus* or some other source of irritation may be present in the lower bowel which produces reflex excitability of the nerves governing the bowel movements. In other cases a stricture in a feeble, dilated rectum will cause retention of feces until irritation, tenesmus, and even loose mucous movements are produced.

If, instead of watery or serous movements, the patient is attacked by a more or less acute diarrhea, accompanied by great pain and distention of the belly, and if there is marked tenderness on pressure over the transverse colon and mucus in the feces, which are not in very large quantities after the first few movements, there is probably present the condition known as *enterocolitis,* or inflammation of the ileum and colon. It is met with in both children and adults, and differs in its course from cholera morbus and cholera infantum very markedly. The pain is usually more constant, more aching, and less griping in character. Vomiting is not a constant feature, as it is in the watery choleraic diarrheas, and the course is more subacute, the duration of the illness usually being from one to three weeks. If food which is difficult of digestion has been eaten, it is passed, still undigested, from the bowel, and is apt to be coated with mucus. Such a diarrhea is called *lienteric diarrhea.*

Not far removed from this type of cases are those of a more chronic character depending upon more grave and lasting alterations in the gastro-intestinal mucosa. As a rule, the greater part of the trouble exists in the colon, and more or less griping pain in the neighborhood, namely in the upper umbilical area and left groin, may be present before each movement. The abdomen is apt to be distended and quite tender on pressure, particularly in varying spots, and considerable loss of bodily weight is apt to ensue, chiefly from failure on the part of the digestive tube to absorb the food that is eaten. The movements are not markedly watery, but are usually unformed and about the consistency of oatmeal gruel or a little thicker. Flakes of mucus are often found in large amounts in the fecal matter, and the feces may be frothy or flaky as the result of fermentation. Blood and pus are very rarely seen in the movements of these cases, unless the blood escapes from an inflamed hemorrhoid. Sometimes, when these cases are very severe in character, the mucus takes the shape of long cord-like or worm-like strings, or even seems to be membranous in character, the so-called *mucomembranous enteritis.* In other instances the feces, when formed, are passed in ribbon-

shaped masses, due either to spasm of the muscular fibers of part of the lower bowel or to cicatricial contractions from the healing of old ulcerations. In very severe cases the condition of the intestines gradually advances from a *mild follicular enterocolitis* to one of actual deep ulceration, and under these circumstances blood and pus may be present in the movements. At such times the pain produced by the patient having a movement of the bowels, or by the passage of fecal matter over the ulcerated surface, may be intense, and the invalid will often state that the pain feels as if one spot in the gut were made more painful by the feces rubbing over it. Such cases often continue for years, while some of them ultimately get well, others become chronic invalids from the slow changes in the intestinal walls. In this connection the diarrhea of *tuberculosis* is not to be forgotten, depending, as it does, either upon the general infection or upon the development of ulcerations in the intestinal canal.

In some cases in which the patient after exposure to cold or wet is seized with violent pain in the epigastrium and a feeling of weight in the rectum, a few loose movements and then intense tenesmus and bearing-down, with only a few drops of mucus in the way of a movement, the condition is one of acute rectal catarrh or *proctitis.*

The cases just named in the preceding paragraphs are to be separated from those in which there is *true dysentery.* Dysentery is a term very loosely applied, by the laity in particular, to any form of severe diarrhea, particularly if there are blood and mucus in the movement. In reality the term dysentery should be limited to cases due to an infection, and very apt to occur in epidemics.

Let us suppose that a patient is seized with diarrhea and some pain in the belly, and with only a slight chill, or this symptom may not be present. The pain soon becomes more and more colicky, and the stools are passed with ever-increasing bearing-down or tenesmus. The effort to empty the bowel, after it is in reality thoroughly emptied, results in agonizing bearing-down pains. Fever to the extent of from one to three degrees may be present. Thirst is excessive, the stomach is usually retentive, and the stools are first the ordinary bowel contents, and then mucus, which may be blood-streaked. Soon the mucus becomes jelly-like in appearance and more thick and tenacious, and, finally, after several days it begins to look mucopurulent, and the stools are less frequent. Sometimes small, bullet-like, hard pieces of fecal matter are shot out of the rectum after severe straining. Recovery usually begins at from seven to ten days. The entire trouble seems to be in the large bowel, and particularly in the sigmoid flexure and rectum. Such are the symptoms of *ordinary mild dysentery* of hot climates or of summer weather in the temperate zone.

The severity of the disease is much greater in hot weather, and the prognosis is not good in severe cases coming on during an epidemic.

On the other hand, if the patient has an irregular diarrhea after or during a residence in tropical parts, which may or may not have a sudden onset, with moderate fever and considerable loss of flesh, and has moderate bellyache, which soon becomes much less, and if the stools as just described above become more and more fluid, and the diarrhea intermits, the physician should think of the case being probably one of so-called *amebic dysentery*, a condition of infection by the so-called ameba coli. The course of the disease is slow, lasting from six to twelve weeks, and the death rate is high. Convalescence is always very slow, and liver abscess due to an hepatic infection by the ameba coli is very frequent. Sometimes secondary abscess of the lung develops.

A positive diagnosis of this variety of dysentery is made by the discovery of the amebæ in stools. These microörganisms possess active ameboid movements and are found in greater number when the diarrhea is severe. They are to be sought for in the small gelatinous masses which are found in the feces. Sometimes the entire stool seems loaded with amebæ; at other times only a most careful search will discover them. They are more refractive than the cells found in the feces, and contain numerous vacuoles, so numerous in some cases that the cells look very granular. These must not be mistaken for the compound granular bodies found in the feces. When they are active a division into an endosarc and an ectosarc can be discovered. Often red blood cells will be found in the amebæ.

Epidemic dysentery is usually due to Shiga's bacillus.

Sometimes a diphtheritic or *pseudomembranous dysentery* is developed in persons having chronic heart disease, and it has been seen as a sequel of acute croupous pneumonia. This is called *secondary diphtheritic dysentery*, and death generally results from exhaustion, only a suspicion of the intestinal condition having existed during life. Such a state is sometimes a complication of Bright's disease, probably owing to the irritation of the intestinal mucous membrane produced by the urea decomposing into the carbonate of ammonium. In *acute primary dysentery of a diphtheritic character* the patient may rapidly pass into a typhoid state, and the case be diagnosticated as one of typhoid fever with profuse diarrhea. The discharges are the only means of separating the two conditions (enteric fever and diphtheritic dysentery), as they often are filled with blood and mucus in dysentery, a condition rarely seen in typhoid fever.

Dysentery may be confused with the diarrhea sometimes produced by a *malignant and ulcerating growth* in the sigmoid flexure or rectum, but a physical examination will usually reveal the tumor, and the

cachexia will aid in pointing to it as the cause. *Syphilitic ulceration* of these parts may cause a somewhat similar train of symptoms.

Again, it is by no means rare to meet with the passage of several mucopurulent movements each day in persons who have pulmonary gangrene or pulmonary tuberculosis, partly due to the swallowing of fetid sputum or *tuberculous ulceration* of the bowels.

Diarrhea is also a symptom of *septicemia*. Distantly allied to this form of diarrhea is that seen in persons who have dissected a putrid body ("dissecting-room diarrhea," so called).

Finally, it is interesting to note that paroxysmal attacks of seromucous or bloody diarrhea sometimes come on in cases of *exophthalmic goitre*. Diarrhea of a more or less severe type may come on in cases of hysteria, often associated with tremendous eructations of gas and rumbling in the stomach and bowels.

Fatty diarrhea may ensue if feeble persons already suffering from irritable bowels take an excess of cod-liver oil, and in some cases it possesses great diagnostic importance. If associated with diabetes, it gives us reason to believe that there is disease of the pancreas producing both the glycosuria and the lack of digestion of the fats. Sometimes in jaundice, however, fat is found in the stools owing to the lack of bile to emulsify it in the intestine.

The Feces.—In this connection we naturally pass on to a discussion of the diagnostic indications of the feces. In the first place, it must be remembered that the *quantity* of the feces depends upon the quantity of the food, and again that the quantity varies with the character of the food, for if the food be such as to be bulky, yet contain little nutritive material, there will be a large residue to be passed out in the feces; whereas if the food be almost entirely composed of materials which can be assimilated very little residue is left, and the feces are consequently smaller in bulk. Thus, the cow eats a large bulk of food and passes large amounts of fecal matter, while the dog eats meat and passes very small amounts of fecal matter.

Again, it is not to be forgotten that many foods actually increase intestinal peristalsis, and so produce large and loose movements, as oatmeal and wheaten grits or apples, while other foods, such as cheese, do the opposite. If the stools are large and copious and the food which the patient has taken is not of a kind leaving a large residue in the bowel, the indication is that there is non-absorption of nutritive materials, with probable wasting of the patient.

The *consistency* of the feces in health varies from a formed "stool" to a mushy condition; but in disease we have a liquid watery stool if the trouble be serous diarrhea, and a pasty or slimy stool if it be due to a catarrhal state of the bowels. The passage of hard scybalous masses mixed with liquid indicates that the feces have become dried and hard in the sacculations of the colon, and are passed only

when they cause so much irritation as to produce diarrhea. If the feces are in narrow bands or flattened ribbon-shapes, there is probably a stricture of the rectum, offering an obstruction to their passage. A mushy or semi-watery stool is often seen in typhoid fever.

The *odor* of the stools depends very largely upon the food which is taken and upon the degree of fermentation present in the bowels. In nursing children the stools often have a faintly sour odor, and in the diarrhea of nurslings with acid fermentation there is an odor of the fatty acids. If the process is marked, this odor becomes actually foul, and in cholera infantum the stools have a musty, mouldy odor. If malignant growth of the bowel is present, the odor is fetid, as it is also in gangrene of the intestine. Sulphur when taken internally sometimes causes a very offensive stool, owing to the sulphuretted hydrogen gas which is developed in the bowel.

The *color* of the stools is of great diagnostic importance in several conditions. In health the feces should be brown or brownish black, the color being partly due to the food, but chiefly to the bile (hydrobilirubin). Certain fruits render the stools dark in color, and some drugs, such as iron and bismuth, make them black, and hematoxylon often makes them look red.

In the stools of persons living on a pure milk diet we usually find little color comparatively. Again, in cases of jaundice, phosphorus poisoning, and acute yellow atrophy of the liver, the stools are very light in color, owing to their lack of biliary coloring. They are also apt to be very light in chronic lead poisoning.

Bilious stools are either golden yellow, greenish, or reddish in hue, and if the flow of bile is profuse, they are apt to be watery. Greenish stools looking as if they contained chopped spinach are, however, a peculiarity of the diarrhea of fermentation, particularly in infants, the color being due to color-forming microörganisms; but a greenish stool may also be produced in an infant by the persistent administration of sodium bicarbonate.

If the stools are well mixed with mucus, the catarrhal process probably exists in the ileum; but if they consist of hard masses of feces coated with mucus, the disease is probably a colitis.

Bloody stools are most commonly due to hemorrhoids which are eroded. The blood may be bright if the hemorrhoid be a small arterial bunch, or more dark and grumous if slow oozing has gone on for some time prior to the movement. As a rule, the brighter the blood in the stool the nearer its source is to the anus, and the darker the blood the higher is its source in the bowel. Thus, if the stools are tarry-looking, the blood is almost certainly from the small intestine, and probably arises from a duodenal or other ulcer or from carcinoma of the stomach or bowel; while if it is only somewhat changed in appearance, it may be due to an ulcer or ulcerated morbid

growth in the colon. Sometimes, however, when the hemorrhage from the ileum is very profuse, as in typhoid fever, the blood comes from the anus only slightly changed in appearance.

Occult Blood.—Blood in the feces in such quantities as not to be recognized except by careful tests forms a very important diagnostic aid in cases of suspected gastric or duodenal ulcer or cancer, in cirrhosis of the liver, and even in typhoid fever when it is feared that deep ulceration of the bowel will result in free hemorrhage. The best test for "occult blood" is the so-called aloin-turpentine test. Before this is used rare meat must be excluded from the patient's diet and the test should not be regarded as negative in its results so far as determining a leakage of blood is concerned until several tests have been made covering a period of a number of days, as oozing hemorrhage is often inconstant. Blood from hemorrhoids or fissure of the anus must be excluded of course. The test as employed by J. Dutton Steele is as follows:

If the stools are not in a semiliquid condition they must be made so by thoroughly mixing them with distilled water. 5 gm. of fecal matter are used in every test. After the material has been completely softened the feces must then be thoroughly mixed with at least its own bulk of ether, and the whole well shaken. This is a very necessary part of the procedure, as it removes the fat, which otherwise produces a thick emulsion when the stools are extracted with acetic acid and ether, and renders it almost impossible to obtain a satisfactory ethereal extract. After being thoroughly shaken the mixture of feces and ether should be allowed to stand for fifteen minutes or longer and the supernatant liquor is then poured off. The remaining fecal matter is then mixed with one-third its volume of glacial acetic acid and 10 c.c. of ether. The mixture is again shaken and allowed to stand for at least fifteen minutes. The ethereal extract will rise to the top in a clear layer and can be readily poured off.

The solution of aloin used is made by dissolving as much aloin as will go on the end of a spatula in one-third of a test tube of 70 per cent. alcohol; 2 or 3 c.c. of the clear yellow aloin solution are then mixed in the test tube with about the same amount of the ethereal acetic acid extract; 2 or 3 c.c. of ozonized turpentine are next added and the whole is gently shaken. If blood is present the reaction may appear in one of several ways: either the whole mixture turns a pink which gradually deepens to a cherry red, or the solution of aloin sinks to the bottom and forms a layer beneath the mixture of ether and turpentine, and this lower layer of aloin in positive tests gradually becomes a deep cherry red. Sometimes if the ether and turpentine are first mixed and then the aloin allowed to flow gently down the side of the tube, the two sets of fluids will remain separate and a deep red ring will form at their junction. Not more than fifteen

minutes should be allowed for the red color to show itself, for after this the aloin will gradually turn red, even if blood is not present. It is extremely important to make up the solution of aloin freshly, for when it stands exposed to light it changes into about the color that it attains in the reaction when blood is present. When the test is negative the color remains a light yellow, which becomes a red after standing for some length of time. Hydrogen peroxide does not work satisfactorily as a substitute for turpentine in the aloin test.

The ozonized oil of turpentine should be prepared by allowing a chemically pure oil of turpentine, such as that prepared by Merck, to stand exposed to the air for at least three weeks.

Stools containing *pus* may receive this material from the surfaces of ulcers, but usually the source of the purulent matter, if it is present in large amount, is an abscess which has ruptured into the bowel, as, for example, in perirectal, or even subphrenic abscess.

Finally, we may find *gallstones* in the stools, which, if they are passed soon after their escape into the bowel, are found to be faceted. Stools which are being searched for gallstones should be washed through a sieve in such a way as to catch the stone and let the fecal matter through. The intrahepatic gallstone is not faceted and crumbles easily. This stone rarely escapes, because it is embedded, and if it does get into the bowel is usually broken up. All stones or concretions found in the feces are not to be considered as gallstones. They may be pancreatic calculi in rare instances, or they may be fecal stones (coproliths) or intestinal stones (enteroliths). Fecal stones are simply hard inspissated masses of feces, which may attain a very large size, whereas intestinal stones are composed of heavy, brown concentric layers of phosphates of calcium and magnesium around some nidus, as a seed or piece of bone. Sometimes they are concretions of insoluble drugs, such as salol or magnesium carbonate.

Intestinal sand appears to be a characteristic symptom of certain types of neurasthenia which is often provocative simultaneously of mucomembranous colitis. The chief constituent of intestinal sand is said to be calcium sulphate. Care must be taken to separate true intestinal sand from small seeds or the small sand-like bodies found about the seed core of pears.

Very rarely a portion of the *bowel sloughs away*, and yet recovery takes place. This is seen sometimes in intussusception.

INTESTINAL PARASITES.

Aside from the character of the stools themselves, we often search for the cause of an ailment in the passages, either for foreign bodies, such as pebbles or pins, or for *intestinal parasites* (worms). Some-

times worms may exist for long periods of time in the bowel without causing any symptoms, and, again, in children in particular, they

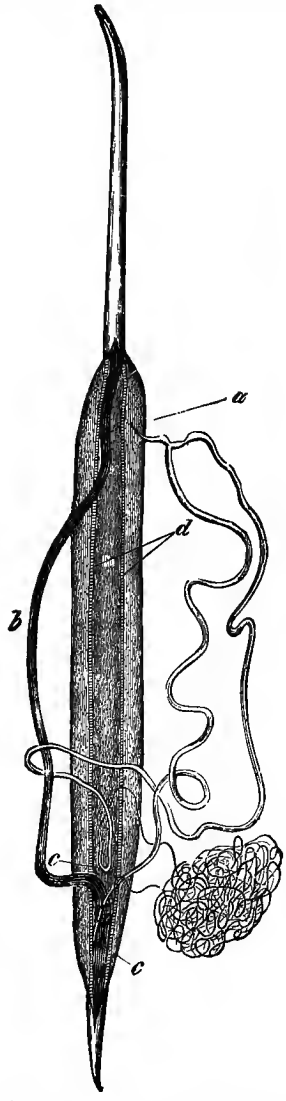


FIG. 129.—Ascaris lumbricoides dissected and walls thrown back. *a*. Genital orifice. *b*. Intestine. *c*. Oviducts. *d*. Longitudinal band. *c*. Ovaries. (Heller.)

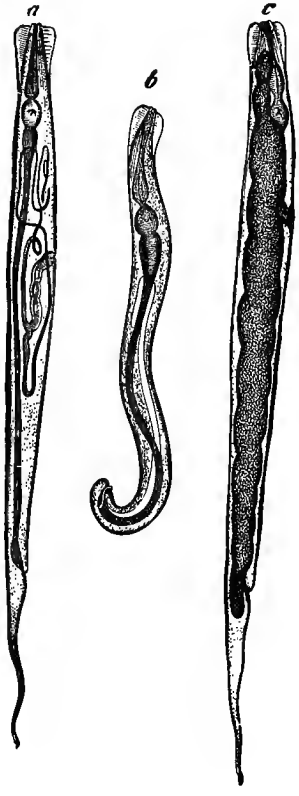


FIG. 130.—Oxyuris vermicularis magnified. *a*. Young female. *b*. Male. *c*. Mature female, full of eggs. (Payne.)

cause great systemic disturbance by producing disorder of the digestion or reflex irritation.

Under the name of *tape-worm* or cestodes we find in the intestine, and often in the stools, a parasite occurring in segments which are flat and ribbon-like, and usually from a quarter to one-half inch in length. The worm itself may be several yards long. Its head is small, and it maintains its hold on the bowel by its head. The segments are usually broken off one by one and escape in the stools, and the stools also contain the ova or eggs of the parasite, which

FIG. 131.—Trichocephalus dispar, natural size. *a*. Female. *b*. Male. (Payne.)

are developed in each segment, which also possesses male and female organs.

According to the shape of the head and the size of the worm and the source of infection, we divide tape-worms into three classes: the Tenia solium, the Tenia mediocanellata, and the Bothriocephalus latus.

If the patient passes a worm of from one to three yards in length, the head of which is about the size of a pin-head and glistening gray in appearance, the rest of the worm being yellowish white, and if upon the head can be seen four pigmented suckers surrounded by a crown of hooks, that worm is a *Tenia solium*, and is probably derived by the patient from raw or uncooked pork. The eggs of the tenia solium must be sought for by a microscope. They are round and covered by a hard shell, which upon pressure breaks into small fragments. In the shells may be found a few hooklets. These eggs are passed out in the feces by the host, and are then swallowed by the pig, in whose muscles the hooklets migrate and form cysts. In these cysts the hooklets develop, and when a man eats the meat raw they enter his intestine, attach themselves, and from them a tape-worm is developed.

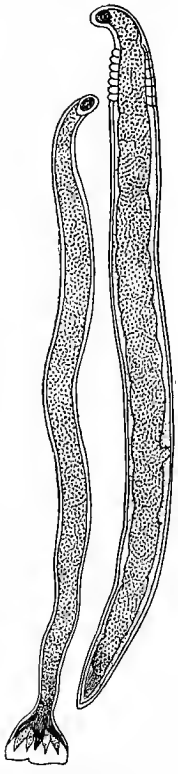


FIG. 132.—Ankylostomum duodenale magnified. *a*. Male. *b*. Female. (Bristowe.)

If the worm is from four to five yards long and the segments after leaving the anus have motile powers, and if the head is larger than that of tenia solium and devoid of hooklets about the suckers on its head, it is probably the *Tenia mediocanellata* or *saginata*. The egg

is slightly larger than that of the solium. This worm usually comes from eating raw beef. The *Bothriocephalus latus* is the largest of all tape-worms, often reaching seven to eight yards in length. It has a long head with two long, narrow suckers. The eggs are oval, very large, and the shell is light brown in color, and very easily broken. This parasite is not common in America, but is a very frequent cause of profound anemia in the persons whom it infects. Its joints are only rarely thrown off, so its presence is often overlooked, and this renders the search for the eggs very important in severe anemia with no ascribable cause. This worm is usually derived from fish. A worm which is comparatively rare is the *tenia cucumerina*, which has a head with sixty hooks. It infects dogs, cats, and sometimes children.

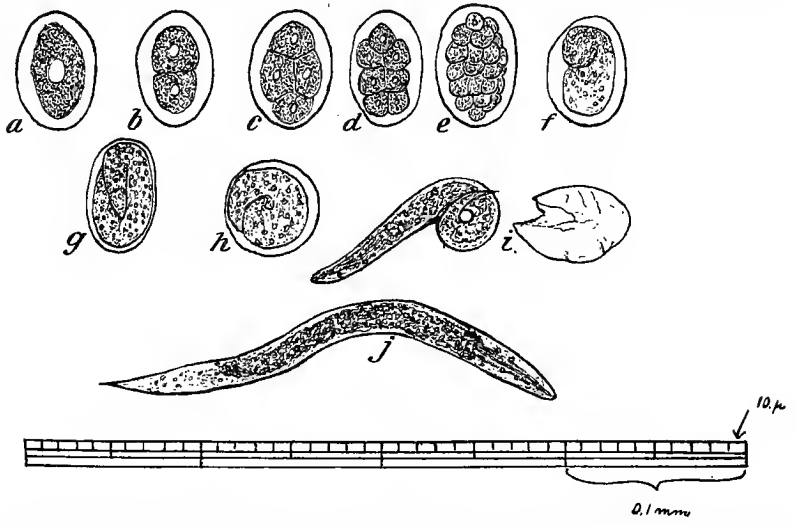


FIG. 133.—Ova and embryo of Uncinaria americana: *a*, unicellular ovum; *b*, *c*, *d*, *e*, ova showing various stages of segmentation; *f*, *g*, ova containing larval uncinariæ; *h*, peculiarly shaped ovum; *i*, larval worm just emerged from shell; *j*, larva extended after emergence. (Stiles.)

A round-worm, looking like an ordinary earth-worm, appears sometimes in the stools, and is called *Ascaris lumbricoides*. It is sometimes vomited, and rarely causes trouble by crawling into and blocking the common biliary duct.

Fine thread-like worms inhabiting the rectum are the *Oxyuris vermicularis*.

A very important diagnostic find in the feces is a worm looking very much like the thread-worm, but somewhat larger, which inhabits the duodenum. It occurs as the *Ankylostomum duodenale* sometimes called the *Uncinaria duodenale* and as the *Uncinaria americana* or *Necator americana*. These parasites belong to the nematodes. The

Ankylostomum duodenale is possessed of two pairs of hook-shaped ventral teeth and one pair of dorsal teeth projected forward. The male parasite is 8 to 10 mm. long, the female is from 10 to 18 mm. long. The *Uncinaria americana* has no hook teeth but a ventral pair of slightly developed lips and a dorsal pair of semilunar plates or lips. The male is 7 to 9 mm. long and the female 9 to 11 mm. long. The eggs are elipsoid and contains a well-developed embryo or are segmented. If the stools are set aside in a warm place the embryos can be seen to develop under the microscope if a small part is spread on a slide. The worms themselves are often of a red hue. A rough test proposed by Stiles is to place a small part of the stool on white blotting paper for an hour. If it is now removed and the paper is stained red the worm is present. The importance of finding this parasite lies in the fact that it produces the most profound and acute anemia by sucking blood from the intestinal wall, although some assert that the anemia is not due to loss of blood but to a poison formed by the parasite. The worms are usually only found after a vermifuge is taken, but the eggs are always present in the feces as unsymmetrical, thickly covered, segmented globules.

The so-called whip-worm, or *Trichocephalus dispar*, is a fine thread-worm without any medical interest.

CHAPTER XI.

THE URINARY BLADDER AND THE URINE.

Disorders and diseases of the urinary bladder—Retention of urine—Incontinence of urine—The characteristics of normal and abnormal urine—The normal and abnormal contents of the urine—Their significance—Tests for the contents of the urine.

THE BLADDER.

The objective symptoms of bladder difficulties are generally local, unless they are very chronic, when the face may appear worn and

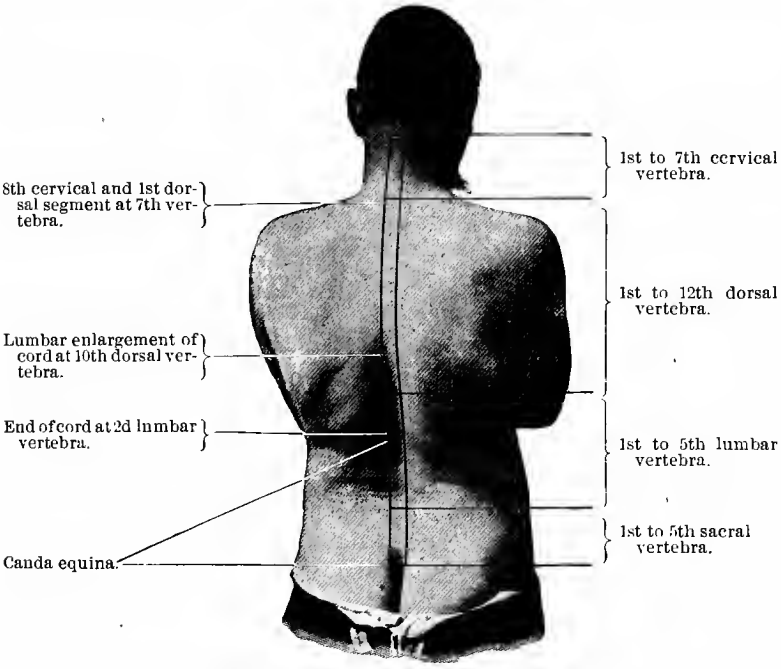


FIG. 134.—Showing the surface areas of the back corresponding approximately to the areas of the spinal cord supplying the trunk, limbs, and bladder.

weary, and, if a purulent cystitis be present, septic fever may occur. The subjective symptoms are tenderness, tenesmus, pain (see chapters on Pain and on the Abdomen), and retention or incontinence of urine.

Retention and Incontinence of Urine.—Retention and incontinence of urine usually depend upon causes arising outside this viscus. It may arise from disease or injury which destroys or temporarily impairs the function of the cells in the spinal cord which govern the contraction of the muscles involved in expelling urine from the bladder. These centres are situated at or about the level at which are given off the second, third, and fourth sacral nerves. (See Fig. 134.) (See also page 82.)

Paralysis of the bladder with retention may, therefore, follow *severe injuries to the spinal cord*, produced by a fall, blows, or other traumatisms, or be due to a *myelitis* which destroys such centres. (See chapter on the Legs and Feet, part on Paraplegia.) The bladder symptoms seen in myelitis—transverse, traumatic, or otherwise—usually come on in the acute form within a few hours after the sensory and motor disturbances have been noticed by the patient, and either incontinence or retention, or both, may occur.

If, however, the myelitis is not complete, the bladder may escape. On the other hand, if the portion of the cord which is involved happens to be that part governing the bladder, vesical symptoms may develop before the motor symptoms are clearly marked. Again, it is a noteworthy fact that when recovery takes place vesical control may be regained before any marked improvement can be found elsewhere. Often the loss of control of the bladder is such that the patient cannot voluntarily expel the urine and cannot retain it, and it dribbles away without his knowledge. Under such circumstances there is probably a myelitis involving the lower part of the dorsal cord and the upper and lower parts of the lumbar cord; in other words, all that portion in which the vesical centres are situated. If the dribbling of urine takes place without distention of the bladder, the fluid passing directly from the ureters through the urethra, the lower part of the lumbar enlargement of the cord is affected, owing to paralysis of the sphincter. On the other hand, distention of the bladder, due to retention of urine, occurs when the myelitis is in the lower dorsal and upper lumbar cord, and is due to paralysis of the detrusor muscles, which make no effort to expel the urine, while the sphincter, the centres of which are intact, maintains a tightly closed orifice. Such cases may empty the bladder spasmodically at long intervals (overflow incontinence)—that is, sphincter paralysis from distention may ensue. In such a condition the bladder should be emptied by the catheter to avoid paralysis and vesical disease. To put the case in another way, we can say that the spinal centre for the control of the walls of the bladder is situated at a higher point in the cord than is that for control of the sphincter, and, therefore, retention of urine indicates a lesion higher up in the cord than does incontinence without reten-

tion. Precisely similar vesical symptoms occur in cases of spinal tumor producing transverse lesions of the cord (see chapter on the Feet and Legs, Paraplegia), or may arise from spinal apoplexy.

The bladder symptoms of *locomotor ataxia* are often quite characteristic, and are to be separated from those of myelitis, spinal tumor, and the vesical troubles due to traumatisms of the cord. The disorder depends entirely upon interference with the reflexes of the viscus, and so presents varying symptoms which are motor and sensory. The patient sometimes complains of the fact that he has to strain for a long time before he can start a stream, which, even after it is started, is often jerking or interrupted; or, again, he must sit down and bend over in order to have the aid of his abdominal muscles before he can evacuate the bladder. As a result of this, residual urine in excess is always present, and cystitis or milder degrees of vesical irritability develop. In other instances the desire to urinate comes upon the patient so suddenly and forcibly that the urine is voided before he can, with his impaired gait, reach a place to pass it in a proper manner; on the other hand, it may be retained and can only be removed by a catheter. Still others find that urine escapes on laughing, coughing, or sneezing, owing to lack of complete control of the bladder and its sphincter; or, again, after many attempts to urinate, the patient gives up the effort, only to be humiliated by an involuntary passage of urine immediately after his penis has been withdrawn into his clothes.

These symptoms differ so materially from those present in myelitis as to make a diagnosis as to their cause nearly always possible.

In obscure cases of ataxia the vesical symptoms may aid the diagnosis quite markedly; thus the presence of bladder symptoms would confirm a diagnosis of ataxia as against pseudotabes due to peripheral neuritis. Again, in myelitis the presence of vesical symptoms points to that disease, and excludes from the diagnosis such affections as poliomyelitis and lateral sclerosis, affections in which vesical paralysis rarely, if ever, occurs. Precisely similar vesical symptoms are sometimes seen in cases of general paralysis of the insane, but the delusions of grandeur or of persecution and other characteristic signs of this disease separate it at once from ataxia.

Retention sometimes comes on in locomotor ataxia, in which disease the impulses from the bladder are not recognized, or are perverted, so that the sphincter which closes the bladder does not relax to permit the escape of urine, or the cord or brain fails to recognize that the bladder is full, and so sends no impulse for its relief. Again, retention of urine may arise from paralysis of the muscular part of the vesical walls by pressure produced in severe labor (childbirth). Finally, we see cases in which the bladder cannot be

emptied, because its walls have been paralyzed by overdistention with urine.

Incontinence results from loss of power in the sphincter, due to injury or disease in the cord at the level of the second, third, and fourth sacral nerves; and this, by the way, is a far more frequent occurrence than is absolute retention. The real condition under these circumstances is that the expelling muscles and retention muscles are both paralyzed, so that the urine accumulates in the bladder and then dribbles through the unguarded neck of the bladder. Sometimes, too, this incontinence is caused by the urethra being so insensitive that it fails to recognize the presence of the urine, and so does not send an impulse to the sphincter to tighten its hold. Incontinence also results from excessive reflex irritability of the walls of the bladder, so that the urine no sooner trickles into this viscus than an impulse is sent to the spinal centres which send a motor impulse to the muscles of expulsion. This is often the condition in the nocturnal incontinence of children, for as soon as the child sleeps its will-power over the bladder ceases, and reflex activity is alone in control. Irritating, concentrated urine may pervert the reflexes of the bladder and so cause incontinence.

Sensory Disorders.—The sensory disturbances of the bladder will be found discussed in the chapter on Pain, but it is worth noting here that accompanying the symptoms already named as characteristic of locomotor ataxia vesical crises of spasm and pain frequently occur.

When there is pain in the bladder, made worse by the attempted act of micturition, and tenesmus, with pain darting into the urethra, there is probably present a cystitis; but the physician should remember that cystitis may be present with almost no painful manifestations, even when in its acute form. In other cases this condition arises from concentration of urine, which produces irritation of the viscus. In children this concentration of the urine is the most common cause of nocturnal urinary incontinence.

Involuntary passage of the urine sometimes occurs in idiots, in some cases of insanity, in attacks of apoplexy, or any condition of abnormal unconsciousness, and sometimes in very severe infectious diseases, such, for example, as diphtheria. Oftentimes it results in children from irritation of the foreskin or vagina, or from rectal irritation produced by seat-worms, since all these causes disturb the reflex activity of the spinal centres.

Obstruction to Urinary Flow.—Interference with the passage of urine may also arise from two causes which are surgical in character, namely, stone in the bladder and tumors of the bladder, which are often situated near its neck and so produce obstruction. Finally, in old men, that most commonly met with cause of

difficult micturition, enlargement of the prostate, is to be remembered.

Aside from these causes of interference with the passage of urine, we must not forget the possibility of its obstruction by stricture of the urethra, nor should the physician ignore the fact that some persons have "nervous bladders," which will not respond to an effort of the will if another person is near by, although the urine is instantly passed as soon as the patient is alone.

THE URINE.

Changes from the normal in the urine, as already stated, are determined, first, by its general appearance, quantity, odor, specific gravity; second, by its microscopic appearance; and, third, by its chemical reaction and responses to tests. Any changes in this fluid of an abnormal character are solely symptomatic, and point with more or less distinctness to disorders of bodily metabolism, disease or disorder of the kidneys, ureters, bladder, or urethra, and sometimes of the prostate, testicles, vagina, or uterus.

The urinary secretion is one which is too frequently ignored by the student and physician in studying the diagnosis of disease. In many instances it will, if properly tested, give such positive evidence in regard to obscure affections that a correct diagnosis is at once possible, and in other cases its examination, as a matter of routine, will discover important facts, the existence of which has been unsuspected. Again and again will a diagnosis prove erroneous if the importance of urinary examinations is ignored, and costly errors for the patient and the reputation of the physician ensue.

In asking questions about the character of the urine passed and its quantity, the physician should be sure that the patient clearly understands his question. Often he is told that much urine is passed, when, in reality, it is really in small amount, but passed often; or that it is blood red, when red because of the presence of high-colored urates and uric acid. In inquiring about its color, we should remember that if large amounts of liquid have been swallowed it will probably be light in hue, or if small amounts of drink are taken, dark in hue. So, too, active exercise in warm weather may produce a somewhat concentrated urine, because much liquid has been lost by the skin in sweat, and the muscular exertion produces large quantities of nitrogenous material indicative of tissue waste. In winter the urinary flow is more apt to be free, because the skin is inactive.

The urine which is to be tested should always be passed directly into the vessel in which it is brought to the physician, and this bottle

should be scrupulously clean; or, if the urine is passed into any other vessel, care must be taken that it is perfectly clean. When it is thought that urethral disease may obscure the investigation a catheter should be passed, all urine in the bladder drawn off, and then the catheter allowed to remain in place, so that the urine will trickle directly from the ureters to the catheter, and so to a receiving vessel. This is very important when the urine is voided involuntarily. If the condition of the bladder is bad, this viscus should be washed out by boric-acid injections, in order to prevent it from contaminating the urine which is to be tested.

Quantity of Urine.—The quantity of urine passed by a healthy adult varies from two to four pints in the twenty-four hours, according to the amount of liquid ingested, the freedom of perspiration, and the amount of exercise.

The significance of any great and constant increase in the amount of urine passed in a given case is various. Thus, we find it greatly increased in diabetes mellitus, in diabetes insipidus, in some cases of neurasthenia, and in some cases of hysteria. It is also increased in many cerebral lesions. Hypertrophy of the heart, *particularly if associated with chronic contracted kidney*, causes an increase in the urine; and, therefore, if a patient has to urinate frequently or has to arise at night to empty the bladder, we suspect this trouble if diabetes and cystitis are excluded.

A copious flow of urine of a low specific gravity and of a pale, clear appearance, containing fatty, hyaline, and finely granular casts, is often seen in cases of *amyloid disease* of the kidney, and the presence of syphilis, of prolonged suppuration, or extensive bone disease, due, it may be, to tuberculosis, with concomitant enlargement of the liver and spleen, separates it from any other ailment. Albuminuria may be a marked symptom or be entirely absent.

Polyuria also ensues if the heart and kidneys are stimulated to increased effort by the action of drugs, such as digitalis, caffeine, or alcohol. We also find an increase in urinary secretion, without its possessing any grave significance, in convalescence from such diseases as typhoid fever and pneumonia.

The quantity of the urine is diminished in cases in which the heart fails to do its proper amount of work, with resulting stasis of the blood in the kidneys, and whenever any large amount of liquid is taken away from the body, as in diarrhea. It is also decreased by fevers and by the sweats following febrile movement. Persistent vomiting also has a similar effect. Parenchymatous nephritis, both acute and chronic, greatly diminishes the urine, and in grave, fatal illnesses urinary suppression may occur.

The Odor.—The odor of freshly passed urine is faint, but characteristic. What is often called a "urine odor" is really due to the

development of ammonia in urine which has decomposed. The odor is altered by many drugs and foods, notably by copaiba, turpentine, eucalyptus, valerian, musk, asafetida, and by asparagus. Diabetic urine possesses a heavy, sweet odor.

The Color.—The naked-eye examination of the urine often gives very important information, if its clearness, opacity, and color are observed. Its clearness and color are modified by the presence of pigments derived from outside sources, such as the educts of carbolic acid or salicylic acid, of senna or hematoxylon, and from urobilin, and many other substances coming from inside sources, such as blood and bile. Many of these causes may render it opaque, but there are two conditions, above all others, which make the urine cloudy even when freshly passed, namely, cystitis with phosphaturia and chyluria. After urine has stood for some hours it often becomes opaque, because it has undergone decomposition changes.

When urine is dark red in color and somewhat opaque the discoloration may be due to blood, hemoglobin, santonin, rhubarb, senna, logwood, or the presence of an excess of urates. Again, it may be rendered almost black, instead of red, by an excess of biliary coloring matter.

A black urine is sometimes seen in cases of melanotic sarcoma, ochronosis, and alkaptonuria, or it may turn black after the brownish urine produced by carbolic acid or uva ursi has been exposed to the air.

Hematuria.—If the color be due to blood or hematuria, the urine will be of a more or less bright red, according to the freshness of the sample brought to the physician and the seat of the hemorrhage. If the urine has been voided several hours, it will be of a dingy red or smoky hue, and on standing will deposit a coffee-ground or reddish sediment of a somewhat flocculent appearance. If, on the other hand, the urine is seen as soon as passed, it may be a bright red or a dingy red, according to the seat of the hemorrhage and the time which has elapsed since the bleeding began; if it has arisen in the kidney or ureter or bladder, and has been gradual, the mixture of blood and urine will be so intimate that changes in the blood will have taken place, whereas if the hemorrhage has occurred, simultaneously with urination, from the neck of the bladder or the urethra, the blood will be almost unchanged when it escapes from the urethra. The presence of clots in recently passed urine indicates a not very recent hemorrhage, and yet one of such size that the urine could not by dilution completely prevent clotting.

When the blood comes from the kidney some of the possible causes are acute parenchymatous nephritis, resulting from any one of the severe infectious diseases, such as scarlet fever or malarial fever;

embolism, resulting from ulcerative or other forms of endocarditis, producing renal infarction, sepsis of the kidney, the ingestion of irritating drugs, such as cantharides or turpentine; and injuries of the back, producing rupture or other disorganization of the kidney. All these conditions produce what may be called acute hematuria. If the cause be acute nephritis from the presence of an infectious malady, such as scarlet fever, pain in the loins, the presence of albumin in the urine, and the eruption will render the diagnosis easy.

Hematuria due to *malarial poisoning* may appear with the first malarial paroxysm, of the intermittent type, which the patient has ever had, and at a time when the history of the case renders it certain that an old malarial condition could not have previously damaged the renal tissues or those of other organs in the body. In other words, there are cases in which a free hemorrhage from the kidney takes place, by reason of the chill, in much the same manner in which hemorrhage takes place in acute nephritis due to exposure to cold or to irritants. Under these circumstances there may or may not be developed a true organic lesion of the kidney in the sense of permanent disease.

Secondly, we have cases in which bloody urine appears, not in the first malarial paroxysm of the intermittent type, but in association with the later attacks, which may have followed the first either rapidly or slowly. In these cases there may be no further cause for the hemorrhage than excessive congestion, but in all probability the vast majority of such patients present distinct renal changes, which permit such a symptom to develop when the paroxysm asserts itself.

Thirdly, we pass from those cases of bloody urine due to intermittent forms to those due to remittent attack. In these patients the process by which a bloody colored urine is developed may be very complicated, since it may be due to renal disease, functional or organic, or to a true hemoglobinuria, arising from dissolution of the red blood cells in the bloodvessels or blood-making organs.

Finally, there is a type of malarial hematuria which is brought on by the administration of quinine (Karamitsas and others).

All these forms of hematuria can be diagnosticated by the presence of the malarial parasite in the blood (see Chapter on the Blood) and the characteristic malarial symptoms, except that which occurs in persons who have a dyscrasia from old malarial poisoning when no organisms are found.

If the hematuria be due to *embolic infarction* of the kidney, an examination of the heart will probably reveal signs of valvular disease, from which source the embolus will have resulted, or in other cases the physical signs, combined with the history, will show malignant endocarditis with renal sepsis therefrom. Sometimes thrombosis of a renal vein occurs in feeble, wasted infants, and so

causes hematuria. If embolism is not the cause of the hematuria, the history of the ingestion of an irritating drug will be the diagnostic guide, or, if injuries be the cause, a history of trauma will elucidate the case.

The causes of chronic or persistent hemorrhage from the kidney are chronic diffuse nephritis, cancer of the kidney, calculus in the pelvis of the kidney producing ulceration, injury of the kidney by jarring of a stone, tuberculosis of the kidney, and cystic degeneration. It is often an early symptom of hypernephroma.

If the chronic hematuria arise from *chronic diffuse nephritis*, which is rarely the case, the diagnosis may be aided by pallor of the skin, anorexia, nausea, headache, decreased amount of urine, and albuminuria. The cause of the nephritis and the source of the bleeding demands careful investigation in such cases. It has been pointed out by Chute and others that it is quite possible for an acute infection to involve only one kidney or only part of one kidney, whereas a toxic nephritis due to a systemic poison naturally involves both kidneys. The history of the case and the use of the cystoscope, or the ureteral catheter, will determine whether the blood comes from one kidney, and if this be the case and the bleeding is excessive a nephrectomy may be needful to save life. On the other hand, if the history is one which indicates a toxic nephritis in which state both the kidneys are probably affected and if it is found that the blood comes from both kidneys then nephrectomy is contra-indicated, since the remaining kidney would be unable to maintain life and the hemorrhage would not be arrested. A bacteriological examination of the urine from one ureter which reveals infection, while the urine from the other ureter is sterile may also aid the diagnosis.

If the cause be *renal cancer*, the cachexia, pain, and the mixture of pus, blood, and disorganized renal tissue in the urine will render the diagnosis possible. If due to *calculus*, there may be a previous history of attacks of renal colic or of violent pain in the kidney; and if ulceration of the renal pelvis has occurred, there will be disturbances of the body temperature, pain in the lumbar area, and pus in the urine. The presence of tubercle bacilli in the urine decides the presence of *renal tuberculosis*. If *cystic degeneration* is present, it can only be determined when the cyst is large enough to be felt. A sudden profuse hemorrhage in the urine, sufficiently large to endanger life, may come from such a cystic tumor of the kidney.

Blood from the kidney usually possesses the following characteristics: it is well mixed with the urine, and is generally altered in appearance, to the naked eye and under the microscope, both as to color and the shape of the corpuscles which may appear as the pale,

almost invisible bodies known as "shadow corpuscles." The cells and casts which may be present are changed in color by the hemoglobin which is free in the urine. Again, blood casts or red blood corpuscles clinging to casts indicate renal hemorrhage. When the blood comes from the pelvis of the kidney it may appear in the urine in long, worm-like clots (moulds of the ureter), and their extrusion from the ureter produces symptoms of colic. Under such circumstances there may be alternations of hematuria and normal urine, due to the blocking of the ureter on the diseased side by a clot, so that all the voided urine comes from the healthy kidney.

Blood in the urine may arise from the *bladder walls* and be due to an acute cystitis, to papilloma, malignant growth, or tuberculosis of this viscus, or to injury. These vesical causes should, if possible, be determined by the use of the cystoscope.

It must not be forgotten also that blood in the urine may be due to *menstrual discharge*, to blood from uterine fibroids or malignant uterine ulceration, and that it is possible for a malingerer to place blood in the urine, with the object of deception. Rarely in certain cases of *locomotor ataxia*, hematuria develops after the vesical crises which have already been described (see Bladder in this chapter). This is due to capillary hemorrhage from the bladder walls.

FIG. 135.—Distoma hematobium, male and female. The two small bodies are the eggs.

There are other varieties of hematuria which must not be forgotten, although comparatively rare, namely, that due to the presence in the blood of the *Filaria sanguinis hominis*, which is a condition in which the presence of chyle in the urine so masks that of the blood that the urine has the appearance of pinkish cream or milk, but microscopic examination will show blood corpuscles and fat globules, as well as the embryos of the filaria. (See Chyluria in this chapter.) Another still more rare cause of hematuria is the *Distoma hematobium* of Egypt and Abyssinia (Fig. 135). This produces what has been called tropical hematuria. The third cause is even more rare in man, namely, the *Strongylus gigas*, which also causes pyelitis and renal colic. A fourth form of hematuria is that seen in some cases of *scurvy*, particularly of the infantile type, and, lastly, hematuria may also appear as a symptom of *purpura hemorrhagica*, *hemophilia*, and very rarely in *leukemia*.

Hemoglobinuria.—The urine, when not discolored by blood, may be discolored by the presence of the coloring matter of the blood. This is called hemoglobinuria.

The following table, based on Purdy's well-known work on Urinary Analysis, sums up these conditions and their significance:

Color.	Cause of Coloration.	Pathological Condition.
Nearly colorless.	Dilution, or diminution of normal pigments.	Nervous conditions: hydruria, diabetes insipidus, contracted kidney.
Dark yellow to brown red.	Increase of normal, or occurrence of pathological, pigments.	Acute infectious diseases.
Milky.	Fat-globules.	Chyluria.
	Pus-corpuscles.	Suppurative diseases of the urinary tract.
Orange.	Excreted drugs.	Santonin, chrysophanic acid, senna.
Red or reddish.	Unchanged hæmoglobin.	Hæmorrhages, or hæmoglobinuria.
	Pigments in food (logwood, madder, bilberries, fuchsin).	
Brown to brown black.	Hæmatin.	Small hemorrhages.
	Methæmoglobin.	Methæmoglobinuria.
	Melanin.	Melanotic sarcoma.
	Hydrochinon and catechin.	Carbolic-acid poisoning.
Greenish yellow, greenish brown, approaching black.	Bile-pigments.	Jaundice.
Dirty green or blue.	A dark-blue scum on surface, with a blue deposit, due to an excess of indigo-forming substances. Indol. Methylene blue.	Cholera, typhus; seen especially when the urine is putrefying.
Brown yellow to red brown, becoming blood red upon adding alkalies.	Substances which are introduced into the system with senna, rhubarb, and chelidonium.	

Hemoglobinuria arises from a number of causes, such as infectious disease, poisoning by mushrooms, and excessive doses of certain coal-tar derivatives, or of chlorate of potassium, or glycerin. Malarial poisoning sometimes causes it instead of hematuria. One form of malarial hemoglobinuria is intermittent, the urine being at one hour limpid, the next hour bloody, and the third hour again clear.

The possibility of confusing the hemoglobinuria of idiosyncrasy, when in a severe form, with that due to severe malarial poisoning, is very great, for the history of *paroxysmal hemoglobinuria* teems with reports of cases in which the chief manifestations of a malarial attack were present, such as chills, fever, and sweats. Probably many cases of so-called malarial hemoglobinuria are due to another parasite not as yet isolated. Hemoglobinuria also follows severe burns and the transfusion of human blood, and occurs in paroxysmal hemoglobinuria, a condition which seems to be produced by mere chilling of the surface of the body or even by immersing the hands of a susceptible person in iced water. It may also be produced either by exposure to cold and damp or to the chill of the milder forms of malarial paroxysm. Hemoglobinuria may also be a symptom of that curious vasomotor affection called *Raynaud's disease.*

Microscopic examination of the urine in such cases will show no corpuscles, although the urine will be coagulated by the nitric-acid test; but the coagulum does not settle in flakes, as it usually does in albuminous urine, but floats on the surface in a brownish mass. The naked-eye appearance of the urine is that of clear port wine. If a few drops of this urine be placed on a watch-glass and a drop of strong acetic acid be added, the blood crystals of Teichmann will be found by the aid of the microscope, showing that the coloring matter is hemoglobin.

If the discoloration of the urine be due to blood rather than to hemoglobin, a microscopic examination will reveal red blood corpuscles, white blood corpuscles, and perhaps fine filaments of clots; but the corpuscles will not be found in rouleaux, as in ordinary blood outside the body, and they may be crenated and distorted in shape, particularly if the urine is alkaline. (For clinical tests, see later text.)

Dark Urine not Due to Blood.—If the urine be red from other causes than blood, this may be due to the ingestion of logwood. The history of the ingestion of this substance will clear up the diagnosis. If it be due to senna, it will be carmine, due to the chrysophan in this drug; but this discoloration only appears if the urine is alkaline. Precisely similar changes are due to the taking of rhubarb. So in santonin poisoning a blood-red urine is sometimes seen, but it usually attains this appearance after being at first yellow, then saffron, and then purple red. One of the condi-

tions of the urine, due to a poison, which can be readily confused with hemoglobinuria or hematuria, is that produced by carbolic acid. This color is not due to blood, but to oxidized educts of the acid. The same educts produce a similar discoloration after naphthalin, creosote, and uva ursi have been taken in overdose.

Red urine, due to none of the causes which have been enumerated, may be due to an excess of urates (except urate of sodium, which is usually white). If on the addition of nitric acid the urine becomes brown where the fluids join, the coloration is due to urates; but if all the fluid is brown, the patient has probably been taking freely iodine or compounds of iodine.

Finally, the urine is often dark reddish brown or porter colored in jaundice, owing to the presence in it of biliary coloring matters. Under these circumstances it may be clear or opaque, and the fluid is apt to be frothy on shaking and to have a decreased surface tension, so that powdered sulphur rapidly sinks to the bottom of the vessel, when the sulphur is dropped on the urine. These biliary colors are at once recognized by the reaction with nitric acid in Gmelin's test, for if a little of the urine be placed on a white plate and nitric acid be allowed to touch the margin of the wet place, a play of colors from green to blue, blue to violet, and violet to red occurs. The same test can be used by wetting bibulous paper with urine, and the acid, if brought to the edge, will stain the paper in the colors named. Green is the only characteristic of the biliary reaction, for indican gives with nitric acid the other colors. (For the symptoms of jaundice, see chapter on the Skin.)

A *greenish-colored urine* is seen in cases of poisoning by salicylic acid, due to the indican and pyrocatechin, and after the use of saffron. Not rarely a greenish or blue urine is due to the ingestion of methylene blue, either as a medicine, as in the treatment of gonorrhea, or in candies colored by this dye.

Indicanuria is present in intestinal obstruction, intestinal putrefaction, cholera, cancer of the liver or stomach, and pernicious anemia. It may, however, be present in health as a result of constipation. (See Chemical Tests.) When through disease processes indican is formed and excreted in the urine, it may by oxidation be transformed into a blue color (indigotin) or into a red hue (indirubin). If urine containing indican be treated with two or three times its volume of hydrochloric acid, it will turn a violet hue.

White or Milky-looking Urine is seen in that condition called *chyluria*, due to the presence of the filaria sanguinis hominis in the blood. This urine on standing forms a creamy layer on its surface, and, if it is shaken with ether, some of the fat can be removed, rendering the urine clear. The diagnosis can only be confused by urine becoming mixed with milk or cream, and can always be made

if the embryos of the filaria be found in the urine. They lie in very delicate sheaths, and show a constant vibratory movement. The diagnosis is still further confirmed if filaria are found in the blood, where they are present in large numbers at night. (See chapter on the Blood.)

Urine may have a somewhat milky white appearance from an excess of phosphates, mixed with more or less mucus, as in catarrh of the bladder. A similar appearance may be due to the presence of pus.

Pus.—Should much pus be present in the urine, it is probably derived from a *pyelitis* or a suppurative inflammation of the pelvis of the kidney. The symptoms of this state are, briefly, a constant or intermittent pyuria, usually an acid reaction of the urine, chills and fever, which may mislead the physician into a diagnosis of malarial poisoning, or, in other cases, if the pyelitis be tuberculous, hectic fever may be present. Sometimes violent attacks of pain resembling renal colic occur, and not uncommonly anemia and loss of strength are notable. There is often pain in the back, which is made worse by pressure with the hand, and, rarely, if the suppurative process be marked, typhoid symptoms may be present.

If *tuberculosis of the kidney* is present, tubercle bacilli may be found in the urine in addition to pus and blood. The blood at times may be present in large quantities. The use of a segregator, or ureteral catheter, or the cystoscope may show nearly pure pus flowing from one ureter. The discovery of tuberculosis of the kidney is of the greatest importance because it is unilateral in 90 per cent. of the cases according to Bevan and usually primary so far as the rest of the genito-urinary tract is concerned. The removal of such a kidney, before the general health is greatly impaired, may save the patient's life.

If pyuria is due to a *calculus*, there may be a history of gravel and renal colic. The purulent urine of pyelitis is to be separated from that of cystitis by the fact that in the former the urine is acid, in cystitis it is ammoniacal. Additional aids to the diagnosis are the pain in the renal region, often unilateral; and the use of the cystoscope to exclude vesical disease. The prognosis varies. If due to an infectious fever, recovery usually occurs.

Specific Gravity.—After having examined the urine with the naked eye and by suitable tests, the physician proceeds to study it by the aid of the urinometer or specific gravity apparatus. This consists of a tall, straight glass into which the urine is poured at the temperature of 60° F. The specific gravity flask is now gently lowered into this fluid and allowed to float in it freely, the container being exactly filled, care being taken that no bubbles of air adhere to the bulb or to the upper end of the flask, since these will float it and help

to raise it out of the urine, thereby giving a fictitious reading of the scale on its stem. The urine used in the urinometer should be, if possible, a sample from the total amount passed in twenty-four hours, since the specific gravity of this secretion varies at different times of the day, and a single specimen may give an incorrect idea of the real specific gravity of the total amount of the fluid. When the physician is desirous of determining the total amount of solids excreted in twenty-four hours from the specific gravity the precaution of taking the specific gravity of the total quantity of urine is particularly important (see below). The normal specific gravity varies from 1015 to 1025.

The specific gravity of abnormal urine varies from 1005 to 1040 at 60° F.; but a persistently low specific gravity indicates chronic contracted kidney if no dietetic cause can be found, while a persistently high specific gravity either shows concentration of the urine as the result of fever or parenchymatous nephritis, or, if the urine is very light in color, the cause is perhaps diabetes mellitus, the high specific gravity being due to the sugar which it contains.

The Total Quantity of Solids excreted by the kidneys in twenty-four hours can be roughly estimated by taking the specific gravity of the total quantity of urine passed during that time, and then multiplying the last two figures of this specific gravity by 2. The number obtained represents the total amount of solids in 1000 c.c. of urine. Thus, supposing the urine to have a specific gravity of 1025, we would find that $25 \times 2 = 50$ gm. Instead of this means, we can also resort to what is known as Haine's modification of Häser's method, although if the solids are much decreased, more accurate methods of testing should be resorted to. The method just referred to is carried out as follows: the last two figures of the specific gravity of the urine are multiplied by the number of ounces voided in twenty-four hours, and the product is multiplied by 1.1. Thus, if a patient passes 32 ounces, and the specific gravity of the urine is 1012, we multiply 32 by 12, which equals 384, and this is multiplied by 1.1, which equals 422, which would be much less than the normal for a person of, say, 150 pounds, who should pass about 945 grains of solids in twenty-four hours.

This estimation of the total quantity of solids eliminated in the twenty-four hours is of great diagnostic and therapeutic value. It has been found as the result of an immense amount of research that a healthy person of about 145 pounds' weight, and from twenty to thirty years of age, taking an ordinary diet and ordinary exercise, should eliminate 61.14 grams or 945 grains of solids in twenty-four hours. In the patient between forty and fifty years of age, we must deduct 10 per cent. from this. If between fifty and sixty, 20 per cent.; if between sixty and seventy, 30 per cent. In patients over

seventy, 50 per cent. If a patient is on a very light diet, we must deduct 15 per cent. from the average solids, and if at rest and free from exercise, 10 per cent. If it be found that a patient is persistently eliminating much less solids than normal, we should consider that he has renal inactivity, and it is our duty to give diuretics. This is particularly the case in fevers, in which there is great tissue waste, so that the solids ought to be greatly increased above the normal. If they are not eliminated, danger is present from their retention. (For accurate estimation of urea, see Chemical Tests).

As a further guide to determine the activity of the kidney, we may employ the so-called methylene-blue test, which consists in injecting into one of the muscles of the thigh 1 grain of methylene blue in 10 minims of water. Ordinarily the blue appears in the urine within half an hour, and disappears within thirty-six to forty-eight hours; whereas in disease of the kidney, its primary appearance is delayed, and it remains present for several days because the kidney is not competent speedily to eliminate it from the system.

In chronic contracted kidney the color may be present in the urine after fifteen days.

If too large a dose is given the color may appear very quickly even in cases of disease.

This is not a positive test, because one part of the kidney concerned with the elimination of one substance may be diseased while another is in health. The rapidity with which absorption takes place from the tissues must also be considered. This is seriously impaired in anasarca and when the circulation is very feeble.

MICROSCOPIC APPEARANCE OF THE URINE AND ITS CONTENTS.

Having considered the macroscopic appearance of the urine, we may turn to its microscopic appearance, and this part of the subject is of even greater importance than the study of the gross appearance of this secretion, for, very commonly, a sample of urine which looks quite normal to the naked eye is loaded with microscopic objects of the greatest pathological significance. The most important of these objects are what are called "casts"—that is, moulds of the uriniferous tubules, formed as a result of the disease process present in the kidney. These casts consist of epithelial cells, blood corpuscles, and pus corpuscles, masses of microörganisms, or of broken-down organic matter, as in fatty casts, and in hyaline or transparent bodies, or moulds which are made up of unknown material, but often covered by corpuscles, pus corpuscles, or epithelial cells In addition to these bodies we find a large number of

organic bodies or derivatives of organic matter, and inorganic substances derived from the tissues or from food.

The physician who desires to examine urine successfully by the aid of the microscope must bear in mind that it can be examined satisfactorily after it has stood still in a glass or other vessel for a long enough time to allow sedimentation to take place—that is, until the objects floating in the fluid have had time to settle, or employ a more rapid method of obtaining the sediment by the use of the centrifuge, an apparatus by means of which the solids in a fluid are separated by centrifugal force. By the use of this apparatus a sediment can be obtained in a few minutes after the urine is passed. (See Fig. 136, and for a description of the principle of the apparatus, see chapter on the Blood.)

The sediment is to be drawn up into a pipette which has been introduced into the urine and a drop placed upon a glass slide, covered by a cover-glass and the slide placed under the microscope.

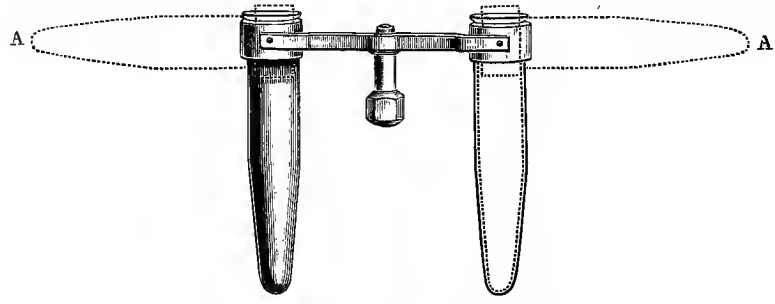


FIG. 136.—Holder for urine tube in centrifuge.

Casts composed of epithelial cells present an appearance similar to that seen in Fig. 137, and are due to proliferation or exfoliation of the epithelium lining the uriniferous tubules. The cells look swollen and granular and may contain globules of fat. These epithelial casts occur in three forms: first, they may appear as hollow casts of the tubule when the epithelium has exfoliated *en masse* (that is, the lining of the tube is cast off in one piece); second, they appear as casts made up of epithelial cells glued to one another; and, third, the cells are attached to the surface of a clear, transparent basis, looking like a hyaline cast. All these varieties are highly refractive of light and are not altered by chemical substances as easily as are the other casts about to be described.

Having found bodies of this sort in the urinary sediment, what is their significance? At one time they were considered a positive sign of an inflammatory process in the parenchyma of the kidney, or, in other words, of parenchymatous nephritis, but at present we

know that almost every sample of urine if centrifuged for some minutes will yield a few hyaline casts.

Blood casts consist of more or less well-preserved blood corpuscles attached to one another in a mould of the tube in which they have escaped. They are rarely seen and are masked by freely floating cells. The significance of these blood casts is great, as they indicate an acute inflammation of the kidney, acute congestion of this organ, or a renal infarction. They are of importance, too, in separating hematuria arising from other sources than the kidney from hemorrhage from this organ, because they are not found unless the escape of blood has been into the uriniferous tubules.

Casts composed of pus corpuscles are still more rarely seen, but, if constantly present, may indicate multiple abscess of the kidneys.

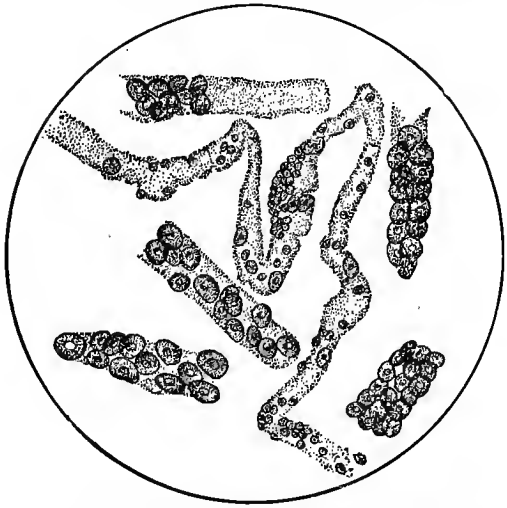


FIG. 137.—Casts containing epithelial cells. (Peyer.)

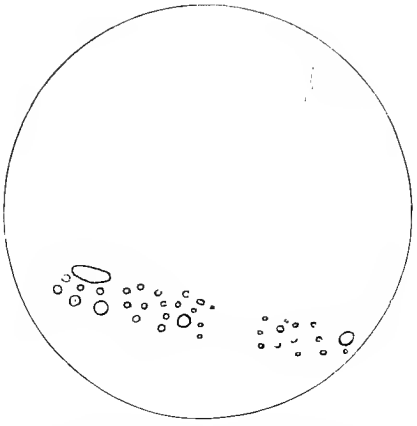
When masses of micrococci become grouped together in the tubules they may be expelled in casts, and under a low power look somewhat like granular casts (see below). They can be seen to consist of micrococci if a higher power is used, and they are not quickly changed by acids, as are casts composed of other materials.

The significance of their discovery is that septic infection of the kidney is present, as the result, it may be, of septic embolus brought from a distant infected part. They are seen in suppurative renal inflammation and in cases of pyelonephritis in which the true renal tissues are being involved by an extension of the disease.

Casts, composed of broken-down organic matter, are found as granular and fatty bodies; that is, they represent broken-down blood corpuscles and epithelial cells, and their appearance varies

PLATE X.

Fig. 1.

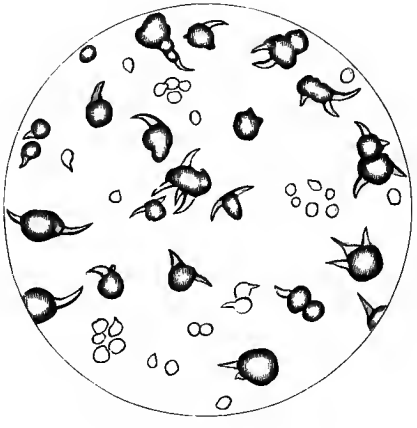


Fatty, Waxy, Hyaline, and Granular Casts.

Fig. 2.

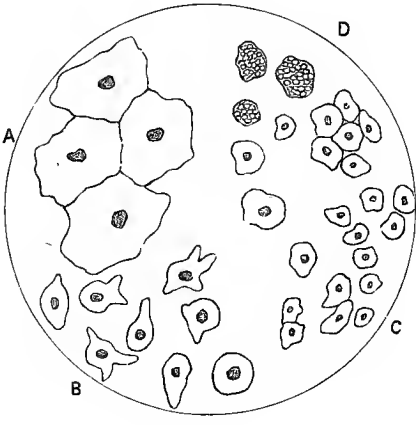
Uric Acid Crystals.

Fig. 3.



Ammonium Urate Crystals.

Fig. 4.



Epithelial Cells.

A, squamous epithelium ; B, bladder epithelium ; C, kidney epithelium ; D, kidney epithelium (fatty).

greatly according to the stage of the process and the origin of the materials composing them. Thus, the granular appearance may be very fine, as shown in Fig. 138, or light and refractive, dark or opaque. Very often the edges of these casts are irregular and the ends frayed and uneven. The color of these bodies may be yellow, brown, or grayish.

The significance of granular casts is grave. They are usually present in large numbers in chronic parenchymatous nephritis. If they are not very granular and are only found in small numbers in centrifuged urine they possess less importance.

Fatty casts, composed of minute globules of oil, cohering to one another or attached to a central core of epithelium, or fat crystals,

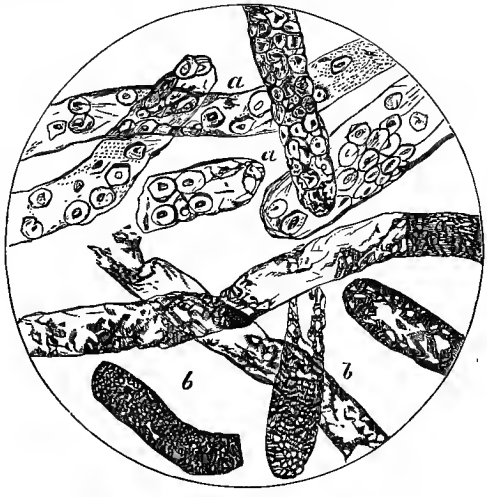


FIG. 138.—*a*, *a*. Epithelial casts. *b*, *b*. Opaque granular casts from a case of acute Bright's disease. (Roberts.)

are found in cases of widespread fatty degeneration, as the result of disease or poisoning, as in the case of large white kidney, on the one hand, or phosphoru , arsenical, antimonial, or iodoform poisoning, on the other. They show the presence of a very slow process if due to disease, but have not the same significance if caused by poison. (See Plate X, Fig. 1.)

Hyaline casts are long, worm-like, transparent bodies, sometimes with very fine granulation, particularly along the edges, and because they are transparent they are often hard to find. These bodies are supposed to be composed of albumin which has been exuded into the tubules. They may be found in the urine in any type of nephritis or even when nephritis is not present, but when they are the only cast found and the urine possesses certain other characters,

their significance is exceedingly grave, particularly if they are present in great numbers, as they point very strongly to that incurable malady, chronic interstitial nephritis. If these casts are very large, they may show amyloid degeneration of the kidney. They have often been wrongly called "waxy" casts. (See Plate X, Fig. 1.)

Casts are not to be confused with cylindroids or streamers. These cylindroids appear in several forms. Most commonly they look like threads or filaments which are transparent and often somewhat striated or hyaline in appearance. They are often long enough to extend completely across the microscopic field, and if followed out to the end will be found to taper off or gradually become more and

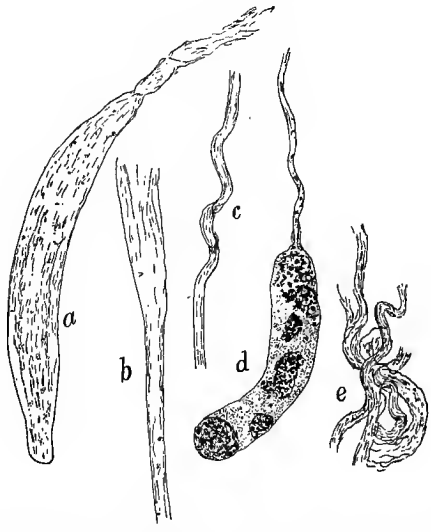


FIG. 139.—Cylindroids from albuminous urine. *a*, *b*, and *c*. Ribbon-like forms. *d*. Cast-like form, with cells upon its surface. *e*. Filamentous forms in a clump.

more transparent until they cannot be outlined. For this reason too much light should not be used in searching for them, nor should a lens of too high a power be used. These cylindroids often are grouped in bunches. In other instances we find cylindroids in the form of ribbons, or, in other words, they are wider than the thread-like masses just described. In still other instances the resemblances to true tube casts are so marked that a differentiation is scarcely possible, except that they are sometimes found to have a filiform tail-like ending (Figs. 139, 140, 141 and 142). The significance of cylindroids is not definitely known, but they may be taken as an indication of irritation of the kidneys, even if albumin and true casts cannot be found in the urine. They are often seen in the renal

irritation following or, rather, accompanying the conditions called lithemia or uricemia, and in that condition in which we find oxaluria.

According to Bramwell, the following is the best method of staining and mounting tube casts and other urinary deposits. He uses picrocarmine.

"1. An ordinary conical urine glass is filled with equal parts of urine and an aqueous solution of boric acid, and set aside until the deposit settles.

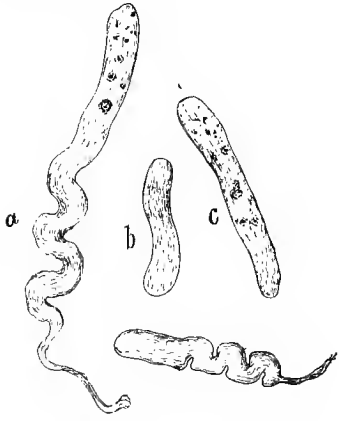


FIG. 140.—Non-albuminous urine. Cast-like forms with deposit of urates.

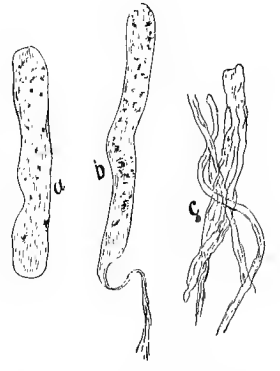


FIG. 141.—Non albuminous urine. *a* and *b*. Cast-like forms. *c*. Filamentous.

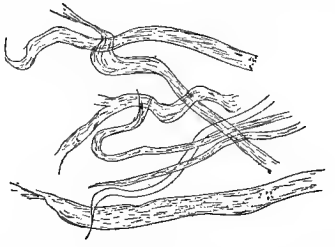


FIG. 142.—Filamentous and ribbon-like cylindroids.

"2. The deposit is then drawn off by means of a pipette, and transferred to an ordinary test-tube, in which a small quantity ($\frac{1}{2}$ dr. is quite sufficient) of picrocarmine solution has been previously placed.

"3. The urine and staining fluids are then thoroughly mixed by inverting the test tube two or three times, the end being closed, of course, by the thumb.

"4. The test tube containing the urine and staining fluid is then set aside to stand for twenty-four hours.

"5. The deposit, which has by that time settled at the bottom of

the test tube, is then drawn off by a fine-mouthed pipette, placed on a slide, covered, and examined under a low power.

"If any tube casts are present, they are very easily detected by this method.

"When a cast is detected, it should be carefully brought to the centre of the field and examined with a higher power. If amyloid degeneration is suspected, methyl violet may be used, for in some cases of waxy disease of the kidney the tube casts give the characteristic rose-pink reaction with methyl violet. For permanent preparation the deposit is drawn off as in No. 5, above, and transferred to a small tube of Farrant's medium,[1] in which it remains until the organic deposit has settled, when it is again drawn off and transferred to clear Farrant's solution, whence it is mounted in the usual manner. All organic deposits are thus stained and mounted in a perfectly clear medium. Their minute characters can be studied with the highest powers of the microscope."

The most important sedimentary substances for diagnostic purposes, other than casts, are the products of tissue changes, or are derived from articles of food. These substances are chiefly the acid urates of sodium and potassium, the alkaline urates of ammonium and potassium, uric acid, oxalate of lime, the phosphate, carbonate, and sulphate of lime, and the so-called triple phosphate (ammoniomagnesic phosphate).

The discovery in a urinary sediment of fine shapeless granules, which occasionally may be crystalline and shaped like a fan, which are generally brown or pinkish in hue, indicates acid sodium urate. Urine containing such deposits is found to become acid on standing, and will form a brick-dust deposit as soon as it is cooled. Acid potassium urate and acid calcium urate, which occur in an amorphous form, are mixed with it in smaller quantities.

The urates themselves have no particular importance except that they are often present in excess in fever, wasting diseases, gastric disorders, and in attacks of gout.

When in a highly acid urine the student finds rhombic or diamond-shaped plates (see Plate X, Fig. 2), or plates of a similar shape with the lateral angles rounded off, or quadrate crystals or square plates, or plates like double-headed arrows, or rosettes of crystals, or bundles of crystals like bundles of kindling wood, these forms are uric acid. Usually they are slightly yellow but they may be colorless. Any urine will deposit such crystals if it stands for many hours (say ten hours), as its acidity increases, and, therefore, the discovery of these

[1] Farrant's solution is made as follows: Dissolve 1 gm. of arsenous acid in 200 c.c. of distilled water. In this dissolve 130 gm. of gum acacia with frequent stirring, and add 100 c.c. of glycerin. Filter the solution through fine Swedish paper upon which has been deposited a thin layer of talc.

crystals only possesses significance if they are found in from four to six hours, as this shows an excess of uric acid, which in turn is found in gouty or rheumatic persons or in those who eat to excess and take no exercise. Often an excess of uric acid in the urine antedates the development of chronic contracted kidney. Uric acid also appears in excess in cases of fever and acute inflammations. It is also eliminated in excess in leukemia, splenic enlargement, hepatic cirrhosis, and gastro-intestinal catarrh. The rosette crystals just named are often found in diabetic urine.

Small, square, brilliant octahedral crystals which are perfectly transparent and refract light strongly, looking somewhat like the back of a square envelope at times, are those of oxalate of lime (Fig. 143). The significance of oxaluria is quite important, for it

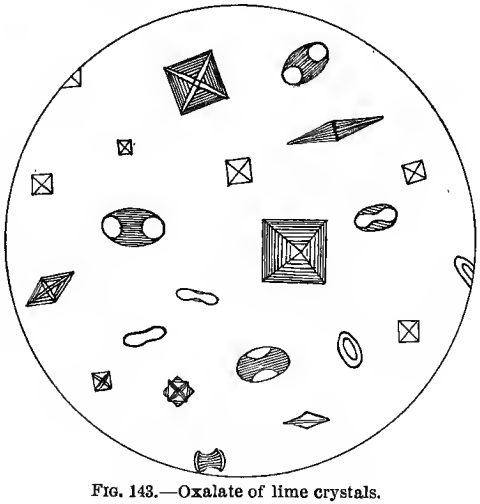


FIG. 143.—Oxalate of lime crystals.

is often a concomitant symptom of melancholia depending upon defective metabolism. The finding of oxaluria separates this class of cases from those of the true disease melancholia, and indicates the use of nitrohydrochloric acid. These crystals are, however, found in the urine of persons who have eaten pears, cabbage, or tomatoes, and in that of persons suffering from spermatorrhea. If not due to the ingestion of the foods named, oxaluria indicates deficient oxidation of nitrogenous tissues.

Creatin in the urine occurs in very brilliant prisms of a rhomboid form, the end of which is often split or frayed. (See Fig. 144, *a*.) It is not present in normal urine.

Creatinin exists in normal urine in small amounts in prismatic, colorless, brilliant crystals of the shape shown in Fig. 144, *b*.

When dark-brown spherical masses covered with thorn-like crystals or sharp spicules are formed in alkaline urine, they are composed of ammonium urate (see Plate X, Fig. 3), and they will be found associated with crystals which are flat or shaped like coffin lids, or more rarely are feathery, star-shaped masses which are large

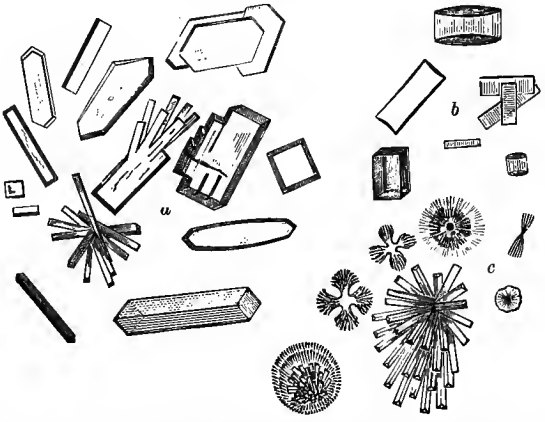


FIG. 144.—Crystals of creatin and creatinin. *a*. Crystals of creatin. *b*. Crystals of creatinin. *c*. Crystals of chloride of zinc and creatin. (Charles.)

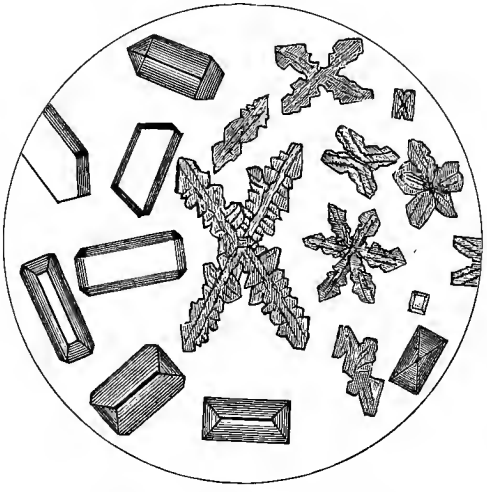


FIG. 145.—Triple phosphate crystals.

in size. These are the crystals of the triple phosphate (Fig. 145). In addition, such urine will contain amorphous calcic phosphate.

The crystals of the triple phosphate are of some diagnostic importance, as they do not exist in the normal urine, but are formed when

ammonia is set free by the decomposition of the urea. If such crystals are found in freshly passed urine, they indicate that ammoniacal fermentation is taking place in the bladder, a condition often seen in chronic cystitis and in some cases of paraplegia arising from injury to the cord or myelitis. A deposit of the triple phosphate and amorphous calcium phosphate, making a sediment like that of purulent urine, is sometimes seen in persons suffering from overwork of the nervous system and in cases of general debility.

In addition to these amorphous and crystalline bodies found in the urine there are a number of others derived from the body, or due to extraneous contamination. These are epithelial cells derived from the kidneys, ureters, bladder, or urethra. (See Plate X, Fig. 4.)

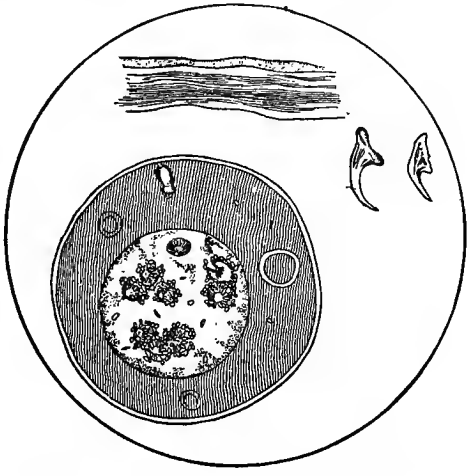


FIG. 146.—Echinococcus, with two hooklets, and section of cystic membrane, greatly magnified. (Peyer.)

Eggs or bodies of several parasites, tubercle bacilli, gonococci and streptococci, or staphylococci are also sometimes seen under the microscope. In addition, we find spermatozoa in certain cases. (See Fig. 148.)

Thus, we may find the embryos of filaria, echinococcus hooklets (Fig. 146), and the eggs of distoma hematobium, which are very rarely seen.

Tubercle bacilli are to be found by the same staining processes as when they are sought for in the sputum (see chapter on Cough and Expectoration), and, if found in the urine, indicate renal or vesical tuberculous infection, provided that the patient has contaminated the vessel containing the urine by sputum infected with the organism. They are not to be confused with the bacilli found in preputial

smegma, which look like tubercle bacilli and take the same stains, but they are differentiated by decolorizing methods.

Gonococci indicate the presence of a specific urethritis or vaginitis, and are found by staining and using a $\frac{1}{12}$ homogeneous immersion lens with a No. 2 eye piece. The process of staining is by the use of eosin and methylene blue. The material on the cover-glass is stained for a few seconds in an alcoholic solution of eosin, then the excess of stain is washed off, and the slide is placed for ten

FIG. 147.—Streptococci. (Abbott.)

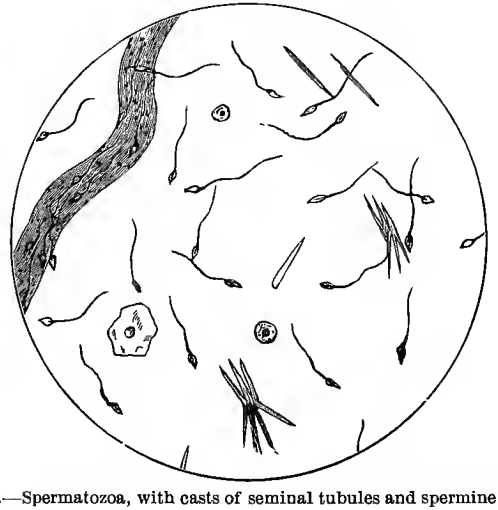


FIG. 148.—Spermatozoa, with casts of seminal tubules and spermine crystals.

minutes in an aqueous solution of methylene blue. Streptococci appear in chains and are stained by the same process. They show infection from pus and are found in cases of erysipelas (Fig. 147). Staphylococci also indicate pus formation in the urinary tract.

The presence of spermatozoa is not so common as is generally thought. They may be in the urine either as the result of a true spermatorrhea, which is rare, or from some of the semen remaining in the urethra after an ejaculation in coitus, or from an emission at night without intercourse. They appear as small, transparent

bodies having a head and tail, and, if alive, possess very active movements (Fig. 148).

Fermentation resulting from the presence of a number of special fungi takes place in both healthy and diseased urines after they are passed. In normal urines the acidity, which is generally present to a slight degree, becomes still more acid through the growth of a special fungus. This process is accompanied by the deposition of uric acid, acid sodium urate, and calcium oxalate, and also amorphous urates. After the urine is exposed still longer it undergoes an alkaline fermentation, and there develop in the fluid the *Micrococcus ureæ* and *Bacterium ureæ*. As a result, the urea takes up water and decomposes with the development of CO_2 and ammonia. No sooner is a positive alkaline reaction established than those ingredients of the urine which are insoluble in an alkaline solution are precipitated, namely, amorphous calcic phosphate, ammonium urate, and ammoniomagnesic phosphate. The first is amorphous, but the ammonium urate appears under the microscope in the form of small granules of a dark color which are covered with spines. The crystals of ammoniomagnesic phosphate are shaped like a coffin lid and are large.

The third form of fermentation taking place in the urine is that which occurs in diabetic urine, and is due to saccharomyces albicans, the microörganism which produces fermentation in ordinary solutions of glucose.

CHEMICAL TESTS.

The chemical tests of the urine give us much important information. We commonly test it for *albumin* and for *sugar*, and if we wish still further information, we examine it for its percentage of *urea*, *peptones*, *chlorides*, *blood*, and *albumoses*.

Urine to be subjected to chemical examination should always be filtered if strictly accurate results are sought.

Tests for Albumin.—There are several forms of albumin found in the urine, of which four are possessed of considerable diagnostic significance, namely, serum albumin and serum globulin, nucleo-albumin and Bence-Jones albumin or albumose. Serum albumin and serum globulin are the forms found in the urine in cases of nephritis, whereas, nucleo-albumin arises from pus or other causes. Bence-Jones albumin is sometimes indicative of bone tumor (see below).

The tests most commonly used are the boiling test, with acidified urine, and Heller's, or the nitric acid test. If either of these tests show albumin to be present in large amounts such albumin is almost certainly serum albumin and serum globulin. If, however, the reac-

tion is very faint it may possibly be due to nucleo-albumin, and this must be excluded by additional tests (see below).

The *boiling test* consists in taking filtered urine and pouring enough of it into a perfectly clean test tube to fill it about two-thirds. If the urine be turbid by reason of an excess of urates, the fluid can be rendered clear by gently heating all of it. To this urine are now added a few drops of acetic acid to render it acid; for if neutral, the albumin will not be coagulated by heat. The upper part of this urine is now boiled by holding it over an alcohol lamp, and if albumin is present a fine cloud will appear in the boiled part of the urine, while the lower part remains clear. This cloud may be due to albumin or to earthy phosphates. If a drop or two of nitric acid is allowed to trickle down the side of the tube, the cloud is dissipated if due to phosphates but not changed if due to albumin.

If acetic acid is employed in place of nitric acid for the purpose of acidifying the urine, care must be taken that it is not used in excess, as under these circumstances the albumin will be dissolved. Indeed, this possibility of the solution of acid albumin is one of the fallacies in the boiling test which may be avoided if, after acidifying the urine until it is distinctly acid, one-sixth of the urinary volume of a saturated solution of common salt, sodium sulphate, or magnesium sulphate is added to it, when, upon boiling, the albumin is precipitated. This test, performed with these modifications, discovers any albumin which may be present, even if its amount be very small. If upon the addition of the saturated solution of common salt a precipitate is formed, this may be due to the presence of albumoses, but the albumose precipitated is redissolved on boiling.

Another useful, rapid, and moderately accurate test for albumin is the so-called *nitric acid test:* 8 to 12 c.c. of urine are placed in a test tube, and then a small quantity of pure nitric acid is allowed to trickle down the side of the tube, in which, by reason of its greater specific gravity, the acid passes to the bottom, the urine forming a supernatant layer. If serum albumin is present, a distinct white cloud will appear in the form of a ring at the point of contact between the two liquids. A much more effective way of performing this test is to use a conical glass, somewhat like a sherry glass, placing in it 20 c.c. of urine, and then by means of a pipette, which is carried to the bottom of the vessel, allow half that quantity of nitric acid to escape. This modification of Heller's test makes a line of albumin quite readily seen. Where only a small quantity is present, it may be necessary to allow the two liquids to remain in contact for several minutes. If an excess of uric acid be present, five or ten minutes after the addition of the

nitric acid a sharply defined ring, somewhat similar to that due to albumin, will be found a short distance above the line of contact.

Another way is to place the acid in the tube first and allow the urine to flow on it. Boston has suggested for bedside use that the physician dip a glass tube in the urine. The finger is placed on the top of the tube to prevent escape of the fluid as it is lifted from the vessel. The outer surface is wiped with a towel and the tube is then immersed in a bottle of nitric acid. The finger is removed and the acid pushes the urine up the tube. At the point where the two fluids meet in the tube the ring of albumin may be seen.

The *potassium ferrocyanide* test for albumin consists in strongly acidifying a few cubic centimeters of urine with acetic acid, and then adding a few drops of a 10 per cent. solution of potassium ferrocyanide. If albumin is present in quantity, a flaky precipitate occurs. If present in minute quantity, the solution becomes turbid. If the urine is very concentrated, it should be diluted with water before this test is employed. In this test, also, the precipitate may be due to albumose, and it will disappear upon boiling. If the precipitate only partially disappears, it is fair to assume that both albumose and albumin are present. The development of a cloud in the urine after the acetic acid is added and before the potassium ferrocyanide is used, does not indicate either albumose or albumin.

The *trichloracetic acid* test for albumin is more delicate than any of those which have been described. Indeed, it is so delicate that it frequently discovers albumin in urine which is certainly normal. By means of a pipette, one to two cubic centimeters of a watery solution of trichloracetic acid, specific gravity 1147, are conveyed to the bottom of a test tube holding several cubic centimeters of urine which has been carefully filtered. If albumin is present, a white ring will be seen to form at the line of contact between the trichloracetic acid below and the urine above. This line may be due to albumose, but is again to be differentiated by the fact that it disappears on boiling if due to this substance. Sometimes uric acid produces a somewhat similar line to that caused by albumin, but it also disappears on heating, and its appearance can be prevented by using a urine which is fairly well diluted.

It is to be borne in mind that one test is not exclusive in proving the absence of albuminuria. Several tests of different samples of urine are essential before the correct result is arrived at because it not infrequently happens that one sample may be free from albumin even if it is usually present.

When nitric acid is added to the urine after it has been boiled, the urine sometimes becomes distinctly yellow, and, upon cooling, a white precipitate appears. Under these circumstances the presence of albumose is indicated. This precipitate is to be separated from

the dark-brown coloration sometimes due to uric acid, which develops after a considerable length of time in urine which has been treated in the manner described.

FIG. 149. Albuminometer.

The presence of a very faint reaction for albumin may be due to the presence of *nucleo-albumin*, which is not indicative of renal disease. This substance is not precipitated by heat and acid in urine having a high content of salt. If, therefore, after adding to the urine an amount of saturated sodium chloride solution equal to one-fifth its volume, heating and adding 2 or 3 drops of 50 per cent. solution of acetic acid and then heating again the cloud persists, this proves the presence of serum albumin and excludes nucleo-albumin.

It is to be remembered that albumoses are precipitated by heat, but redissolved when the temperature approaches the boiling point.

The Quantitative Tests for albumin are many of them impractical for the busy doctor. The best method is by means of percentage tubes placed in a centrifuge machine. By this means all the albumin is thrown down. The tubes are filled to the 10 c.c. mark with urine and $2\frac{1}{2}$ c.c. of potassium ferrocyanide solution (one part to ten) are added. Next we add $1\frac{1}{2}$ c.c. acetic acid and thoroughly mix all these liquids, and the tube being placed in the centrifuge the machine is worked until all the albumin has settled. Each $\frac{1}{10}$ c.c. mark on the tube represents 1 per cent. by bulk of albumin; that is, if the albumin extends up to the $3\frac{1}{2}$ c.c. mark the albumin amounts to 35 per cent.

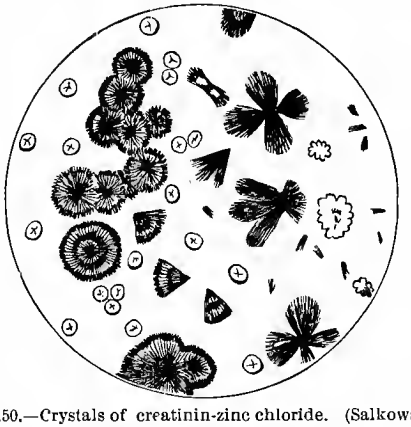


FIG. 150.—Crystals of creatinin-zinc chloride. (Salkowski.)

Another useful method is that known as Esbach's. A special test tube, known as an albuminometer, is used, on which is marked

a letter U and a letter R (Fig. 149). The urine, which has been carefully filtered, is poured in the tube until it reaches the letter U, this portion of the tube being also provided with a scale reading from 1 to 7. The reagent employed is then poured in until the letter R is reached, when the tube is closed with a stopper, and it is turned up and down a number of times, so that the fluids may become thoroughly mixed. It is then set aside for twenty-four hours, when the serum albumin, serum globulin, albumose, uric acid, and creatinin will have been precipitated. The amount of these solids in grams per thousand may be then directly read off from the scale. The reaction of the urine to be tested should be acid, and, if necessary, acetic acid must be added to it for the purpose of acidifying it. Further, its specific gravity should not be over 1006 or 1008. If the specific gravity is higher than this, it must be reduced by the addition of water. The temperature should be in the neighborhood of 70° F. Esbach's reagent is composed of 10 gr. of picric acid and 20 gr. of citric acid dissolved in a liter of distilled water.

The Significance of Albuminuria.—The significance of albuminuria is not as grave in all cases as it was considered at one time, nor is its quantity necessarily of great import, for in some of the gravest cases of *renal disease*, as chronic contracted kidney, it is excreted in very small amount, and it occurs in the urine sometimes in large quantities without any kidney lesion being present. As a rule, however, it indicates renal disease in one of its forms, provided it is associated with other renal symptoms. It may depend on *changes in the blood* in which the diffusibility of its albumin is increased (Semmola), and when its coagulability is diminished. Wright and Ross have shown that the use of full doses of calcium chloride or lactate will arrest this form of albuminuria in some cases. We see albuminuria in cases of *anemia* and in convalescence from protracted illness or from the effects of poisons. Again, *circulatory changes* may cause albuminuria by causing congestion of the kidney, as in cases of failing heart from its various causes. There is an intermittent, little understood form of albuminuria, called *cyclic albuminuria*, orthostatic albuminuria, or the albuminuria of adolescence, in which the albumin is absent on rising from bed in the morning, but appears if exercise is taken. An *excess of albumin in the diet* may cause albuminuria, which is not necessarily indicative of renal disease. Traces of albumin may be due to the presence of a little *pus* arising from cystitis or urethritis, or from a vaginitis. In the presence of albuminuria the real test to determine the presence of actual kidney disease rests upon the discovery of casts on microscopic examination, but even the absence of casts does not negative the possibility of nephritis, for they may be absent, yet renal disease be present.

Tests for Blood.—A test which can be most easily applied to determine the presence of blood, if the microscope cannot be used, is Heller's test, which consists in adding to a few cubic centimeters of urine a little caustic soda, so as to render the liquid strongly alkaline. The urine is now heated to boiling, and if blood is present a bottle-green color is produced, and the phosphates fall to the bottom of the test tube in fine flakes, tinged brownish red by the coloring matter of the blood.

Still another test, and perhaps the best known to determine the presence of hemoglobin in the urine, is the so-called guaiacum test. The urine having been placed in a test tube, a mixture in equal parts of tincture of guaiacum and oil of turpentine, which has been ozonized by exposure to the air, is allowed to flow gently along the side of the tube, so as to rest upon the urine. If blood pigment is present, a white ring, which gradually becomes blue, forms at the point of contact between the two liquids.

The best test for *blood* is Holland's modified guaiac test: A solution is made of freshly broken pieces of guaiac resin by boiling them with alcohol in a test tube for a few minutes until the tincture is yellow. The suspected material, which may be a drop or two of blood or of bloody urine or of water in which a blood-stained fabric has been steeped, is cautiously mixed with a drop or two of guaiac solution to make a milky mixture. This is brought in contact with a fragment of sodium perborate on a white plate.

If the proportion of blood is large the white perborate turns blue in a few minutes and remains blue until the drying of the guaiac leaves a yellow residue which changes the blue to green. This blue-green color persists on and about the perborate and is well shown on the white background for at least a week. If the proportion of blood is small the white perborate takes on a pale blue hue which turns green as the guaiac dries. The next day a distinct green stain is left on the white plate. The test is simple and delicate, though it must necessarily be open to the fallacies that belong to the guaiac test in any form. A distinct reaction was obtained from a small five-year-old blood-stain on linen.

Tests for Albumose.—Albumoses in the urine may be discovered by the use of certain tests, but even if they are found their presence has no definite significance.

Harris asserts that albumosuria is simply a manifestation of the action of microörganisms, and is thus only an indication of an infective process; but Fitz holds that its persistent presence nearly always points to a fatal ending. It ought not to be forgotten that albumosuria occurs in the normal puerperium.

Under the name of myelopathic albumosuria, Bradshawe has described a very rare condition, multiple myeloma, in which, with

thinning of the bones and overgrowth of gelatinous masses in the cancellous tissue, there develops an extraordinary degree of albumosuria. The patient becomes exceedingly weak and feeble, and finally dies from exhaustion or intercurrent maladies. The peculiarity of albumosuria is that the albumose coagulates below the boiling point and re-dissolves on boiling. Mineral acids precipitate it in the cold, but boiling dissolves the precipitate.

Tests for Peptone.—Peptonuria, at one time considered pathologically identical with albumosuria, has been separated from the latter state by Chittenden's researches. It is discovered by saturating the urine, which has been first slightly acidified with acetic acid whilst boiling, with ammonium sulphate, and filtering out any precipitate. If the filtrate contains a substance which is precipitated by potassiomercuric iodide or picric acid, it is peptone. Peptone is not precipitated by saturation with ammonium sulphate, and albumose is precipitated by it. Peptonuria is present in croupous pneumonia, all suppurative processes, empyema, tuberculosis, smallpox, mumps, erysipelas, cancer of the viscera, jaundice, and apoplexy, and in typhoid fever and phosphorus poisoning. Von Jaksch asserts that it is present in epidemic cerebrospinal meningitis and absent in tuberculous meningitis, and that it is a positive differential sign of the former disease if no ulceration of the lungs is present. In the presence of acute inflammations the development of peptonuria indicates suppuration.

Tests for Sugar in the Urine.—The presence of sugar is determined by a large number of qualitative and quantitative tests, of which the simplest and most reliable are Haines' test and the test of Whitney. *Haines'* test consists in making a solution as follows: pure copper sulphate, 30 gr.; distilled water, ½ oz.; thoroughly dissolve the copper salt in the water; add pure glycerin, ½ oz., which is to be thoroughly mixed; and then add liquor potassæ, 5 oz. One dram of this is to be placed in a test tube and gently boiled, and to this are added 6 to 8 drops of the urine, and the liquid again gently boiled. If sugar is present, a copious yellow precipitate is formed. This is better than Fehling's test, because it is a permanent fluid.

Whitney's test is a solution of ammoniocupric sulphate, of which 1 dr. is decolorized by $\frac{1}{30}$ gr. of glucose. The solution of the amount of 1 dr. is placed in a test tube and heated to the boiling point. The urine is now added drop by drop. If no sugar is present, no change will occur; but if it is, the blue color will begin to fade, and finally the liquid will become perfectly colorless. As the fading process begins the urine should be added more slowly, three to five seconds of boiling intervening between each drop. If there is any shade of blue or green left in the solution, reduction

has not taken place. The following table shows how this test may be used for the quantitative estimation of sugar:

If reduced by	*It contains to the ounce.*	*Percentage.*
1 drop	16 or more grains.	3.33
2 drops	8	1.67
3 "	5.33	1.11
4 "	4	0.83
5 "	3.20	0.67
6 "	2.67	0.56
7 "	2.29	0.48
8 "	2	0.32
9 "	1.78	0.37
10 "	1.60	0.33

If the urine contains more than 3.33 per cent. of sugar, it is to be diluted by from 1 to 10 parts of pure water, and the amount found in the table multiplied by the amount of dilution. Usually diabetic urine contains not less than ½ of 1 per cent. and rarely more than 1 per cent. This test is not reliable if the solution is not freshly prepared.

As Fehling's test is so widely used it must be mentioned. Wickham Legge thus describes it:

This solution may be prepared in the following way: 665½ gr. of crystallized potassiotartrate of sodium are dissolved in 5 fl. oz. of a solution of caustic potash, sp. gr. 1120. Into this alkaline solution is poured a fluid prepared by dissolving 133½ gr. of sulphate of copper in 10 fl. dr. of water. The solution is exceedingly apt to decompose, and must always be kept in stoppered bottles and in a cool place. It is usually, therefore, more convenient not to mix the alkali and copper until the solution is wanted for use. In this case 1 fl. dr. of the sulphate of copper solution may be added to ½ fl. oz. of the alkaline solution prepared as above.

About 2 drams of this test solution are poured into an ordinary test tube, and the fluid boiled over a lamp and set aside for twelve hours. If no deposit forms, the solution may be used for analysis; but if a red precipitate be thrown down, the liquid has decomposed, and a fresh supply must be had.

While the solution is boiling in the test tube the urine must be added to it drop by drop, and the effect watched. A few drops of a sample of urine which contains a large percentage of sugar will at once give a precipitate of yellow or red suboxide of copper; but if no precipitate occur, the urine should be added to the fluid drop by drop, any deposit being carefully looked for, until a quantity equal to that of Fehling's solution employed has been added. If on precipitate be found after setting the test tube aside for an hour, the urine may be considered free from sugar.

Cautions: 1. The test solution should never be used without boil-

ing beforehand for a few seconds, the tartrate being exceedingly apt to decompose, and the solution then reduces copper as effectually as would grape sugar.

2. The quantity of urine used in the test should never be greater than the quantity of test solution employed.

3. After boiling urine in volume equal to Fehling's solution, the boiling of the mixture must be discontinued, as other bodies present in the urine, besides sugar, will reduce copper at a high temperature.

If the examination for sugar is to be made with the greatest care, the urine should always be filtered, at least three times, through animal charcoal. This removes all urates and uric acid, which often partly reduce the Fehling's solution, but the sugar goes through the filter.

Sir William Roberts directed that Fehling's solution be placed in a test tube to the depth of about one-quarter inch and the filtered urine added to the depth of two inches, and the two fluids well mixed. The flame of the lamp is then applied to the upper part of the urine, as in testing for albumin, and this is briskly boiled for a few seconds. The test tube is now held up to the light, and, if sugar is present, the upper part has a yellowish tinge, while the earthy phosphates are thrown down in golden-colored flocculi.

Trommer's test for sugar consists in strongly alkalinizing a few cubic centimeters of urine with a solution of sodium hydrate, and then adding drop by drop a 5 per cent. solution of sulphate of copper until the cupric oxide formed is no longer dissolved. The mixture is then carefully heated, and if sugar is present, a yellow precipitate of cuprous oxide is formed which gradually settles to the bottom of the test tube as a red sediment.

The phenylhydrazin test consists in adding to 6 or 8 c.c. of urine two small pinches of phenylhydrazin hydrochlorate equalling about 5 to 7 gr., and 15 gr. of acetate of sodium. This mixture is then warmed until the salts have been dissolved, a little water being added, if necessary, to aid their solution. The test tube is then placed in boiling water for twenty to thirty minutes, and then transferred to a vessel containing cold water, when, if sugar is present in considerable quantity, a bright-yellow crystalline deposit will at once be thrown down and partly adhere to the tube. These crystals are composed of phenylglucosazon and are insoluble in water. This is less open to fallacy than any other test for sugar.

The *quantitative estimation of sugar* is best made by the Whitney test, already described, or by the fermentation method of Roberts, which depends upon the principle that grape sugar is decomposed into alcohol, carbon dioxide, etc., by the fermentation set up by yeast. As a result of this the urine loses its specific gravity, and each degree of specific gravity has been found to equal 1 grain of sugar in the

fluidounce. In other words, if the specific gravity before the test was 1035 and after the test 1015, the amount of sugar present would be 20 gr. per ounce. Four ounces of urine are placed in a twelve-ounce bottle and a lump of German yeast added. The bottle is then corked with a perforated cork to permit the gas to escape, and kept in a warm place for twenty-four hours. By its side is placed a tightly corked bottle of the same size, holding 4 oz. of urine and no yeast. The specific gravity of both specimens is taken simultaneously, and the difference in degrees represents the number of grains of sugar in each ounce. The loss in degrees of specific gravity multiplied by 0.23 will give the percentage of sugar.

The Significance of Sugar in the Urine.—The significance of sugar in the urine is various. If it is persistent and accompanied by wasting, polydipsia, and polyphagia, it is a sign of *diabetes mellitus*. If diabetes mellitus occurs in a young person, the prognosis as to life is nearly always fatal; if in middle age, it is hopeful; if in persons after fifty, it is quite favorable.

Sugar is sometimes found in small amounts in the urine of very obese persons, and its presence under these circumstances does not necessarily indicate a grave prognosis; but, on the other hand, there are cases of so-called diabetogenous obesity in which the prognosis is very grave. True diabetes mellitus is to be separated from cases of glycosuria by the fact that the systemic symptoms of wasting, depraved nutrition, itching, furunculosis, and profuse diuresis are present. Diabetes occurring in old age, or after sixty years of age, has not the grave prognosis attached to it that exists in connection with the disease in earlier life, as just stated. The younger the patient the graver the malady. (See also the end of this chapter.)

The indications of glycosuria, aside from diabetes mellitus, are of little importance. Glycosuria occurs in the course of convalescence from many infectious diseases, particularly typhoid fever, measles, scarlet fever, diphtheria, influenza, and malarial disease; after cerebral hemorrhage and nervous injuries, and after the ingestion of some poisons, notably phloridzin, chloral, arsenic, alcohol, and curare. It also sometimes occurs as a result of the ingestion of large amounts of sugar and starchy foods in persons who are unable to digest and assimilate carbohydrate foods in excess. Unless the glycosuria is associated with the other symptoms of diabetes mellitus, it is not a positive sign of the disease, for glycosuria is a symptom of a number of states other than diabetes mellitus, as has just been pointed out. *Pentosuria*, or the presence in the urine of an optically inactive sugar which does not ferment with yeast, is a rather rare condition which in nearly every case has been mistaken for diabetes. This urine reduces Fehling's solution, but in an unusual way, the fluid remaining clear a short time and then suddenly

changing color throughout. The orcin test practically identifies the pentoses, but complex chemical methods are needed absolutely to verify the diagnosis. In the cases studied the condition appears to have been relatively harmless, though its true significance is not as yet fully understood. The dietetic treatment of glycosuria has no effect upon it, hence the importance of differentiating between it and glycosuria.

Finally, it is not to be forgotten that a condition known as *alkaptonuria* may exist. In this state the urine reduces alkaline solutions of copper, and on exposure to the air absorbs oxygen in the presence of an alkali and becomes of a dark-brown or black hue. The specific gravity of the urine is low, 1014 to 1020, and there is no marked polyuria. While such a urine reduces Fehling's solution, it will not give the reaction with the bismuth, the phenylhydrazin, the fermentation, or polariscope tests for sugar. This condition of alkaptonuria has no direct pathological significance so far as is known. It is often found in several members of the same family.

Tests for Acetonuria.—The presence of an excess of acetone in the urine in association with glycosuria is diagnostic of true diabetes mellitus, and if the acetone is present in large quantity, the condition may be considered as grave.

The best test for acetone, in that it is the simplest, is Oppenheimer's modification of Dennigés' test. It is carried out as follows: The reagent named below is added drop by drop to about 3 c.c. of urine until the precipitate which is formed no longer disappears on stirring. A few more drops are now added, and after several minutes the precipitate is filtered off. The clear filtrate now has added to it 2 c.c. of the reagent and 4 c.c. of a 30 per cent. solution of sulphuric acid, and is boiled for a minute or placed in a water-bath of boiling water. If acetone is present in large quantity, a heavy white precipitate forms at once; while if it is present in minute quantity, a slight cloud develops on standing after several minutes. The precipitate is almost completely dissolved by adding an excess of hydrochloric acid. It is claimed that this test will reveal acetone in the presence of 1 to 5000. If albumin is present in the urine, large amounts of the reagent must be added. The reagent consists of 20 grams of concentrated H_2SO_4 added to 100 c.c. of distilled water, to which are also added 5 grams of freshly prepared yellow oxide of mercury.

Tests for Indican.—An excess of indican in the urine possesses considerable clinical significance. It nearly always indicates putrefactive processes going on in the intestine, probably resulting from the putrefaction of albuminous foods. A certain amount of indican is always present in the urine, particularly if large quantities of animal food have been taken, and in the presence of intestinal

obstruction it often appears in the urine in very large quantities, so that its presence in excess has been considered as a valuable aid to the diagnosis of this condition, since in ordinary constipation it does not appear in great excess.

The test for indican is performed by means of the method of Jaffé, as modified by Stokvis, as follows: Equal parts of urine and concentrated hydrochloric acid are placed in a test tube, and to this mixture are added 2 or 3 drops of a strong solution of sodium hypochlorite. To this are also added 1 to 2 c.c. of chloroform, and the mixture is then thoroughly shaken. The indigo which is set free is taken up by the chloroform, which becomes blue to a greater or less extent according to the quantity of indican which is present. The fallacies connected with this test are: First, that bile pigment interferes with it. If bile pigments are present in considerable quantity, they must be removed by the previous addition to the urine of a solution of subacetate of lead in moderate quantity. The presence of iodine, due to the use of potassium iodide, will also color the chloroform red instead of blue.

My colleague, Dr. Holland, has suggested the following test as more reliable: To a test-tube filled one-fourth with urine an equal quantity of concentrated hydrochloric acid is added to liberate the indoxylsulphuric acid or urinary indican and then as oxidizer a piece of sodium perborate as large as a full-sized pea. The mixture, which immediately effervesces briskly, is gently agitated to dissolve the perborate. The urine promptly deepens in color, and if the amount of indican be large, turns faintly blue. To concentrate the color, 1 c.c. (16 minims) of chloroform is added, the tube is closed with the thumb and the contents *gently shaken for at least two minutes*. The chloroform separates at the bottom as a layer varying in depth of blueness with the proportion of indican. Some pathological urines yield a layer almost black in color. If the shaking is too vigorous the chloroform is emulsified and remains milky, though with a decided blue tinge.

Test for Bile.—The best test for *bile* in the urine is Gmelin's test, as modified by Rosenbach. After the urine has been filtered through thick filter paper, the paper, still wet, is removed from the funnel, and a drop of concentrated nitric acid is allowed to touch its inner surface, when if bilirubin is present, a play of colors will take place at the line of contact, the green color being most typical.

The Urea in the Urine.—The amount of urea is to be estimated by the process of Lyons, as follows (Fig. 151):[1]

1. A bottle is provided with a perforated rubber cork and delivery tube; in this the decomposition of the urea is effected.

[1] This apparatus, with full directions for use, can be obtained from Parke, Davis & Company, Detroit, Mich., for one dollar.

2. A small test tube to contain the urine, graduated to hold 4 c.c., the quantity employed in each experiment.

3. A graduated jar for measuring the gas evolved. The jar is provided at the bottom with an "overflow" tube, and at the top with a vent tube closed with a rubber cap, to secure accurate adjustment of the level of the fluid in the jar at the commencement of the experiment.

The process is as follows: put into the square bottle 20 c.c. of a special solution of chlorinated soda (for formula see below), and add 5 c.c. of a 20 per cent. solution of potassium bromide; fill the test tube exactly to the mark (4 c.c.) with the urine to be examined, and lower it into the bottle by means of a thread or by the aid of a pair of dressing forceps, taking care that none of its contents is spilled in the operation. Fill the graduated jar with water, which must be of the same temperature as the air of the room, to a point a little above the 0° of the scale, supporting the extremity of the overflow

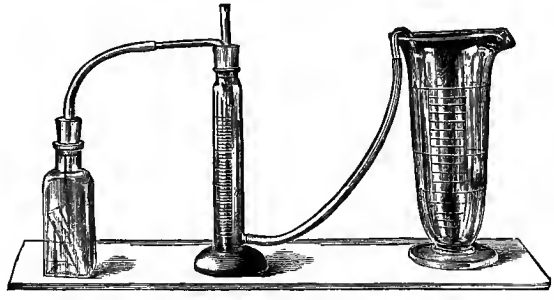


FIG. 151.—Ureometer.

tube so that no water can escape. Remove the rubber cap from the vent tube and connect the apparatus, pressing in the rubber stoppers firmly so as to make the joints air-tight. Finally, put on the rubber cap, drawing it down so as to force a little water out of the overflow tube, and bring the level of the water remaining exactly to the 0° mark, the orifice of the overflow tube being on the same level. A little practice will make this easy.

To make sure that the connections are all perfectly air-tight, lower the end of the overflow tube a few inches; a few drops of water will escape from diminished pressure, but if the joints are perfect there will be no further dropping. If there is any leakage, the defective joint must be found and the difficulty corrected before proceeding farther with the experiment. Having made sure that the connections are perfect, catch the curved end of the overflow tube over the edge of a measuring graduate, as shown in the illustration (an ordinary bottle or any other receiver may be used in place of the graduate). Now, by canting the bottle, cause the urine to flow out of

the test tube and mix the test solution. Effervescence is at once produced, and the gas evolved forces a corresponding volume of water out of the overflow tube. Shake the bottle occasionally to promote the escape of the gas. When the action appears to be at an end pour into the measuring graduate enough water to reach above the opening of the overflow tube, in order that cooling of the gas evolved, which at first is quite warm, may not draw air into the apparatus. Let the apparatus stand fifteen or twenty minutes to cool, then shake the bottle containing the urine once more and proceed to read off the result. To do this, it is necessary to bring the opening at the end of the overflow tube just to the same level as that of the fluid remaining in the graduated cylinder, since raising or lowering the tube slightly affects the volume of the gas to be measured. The percentage of urea is read off without need of any calculation from the scale of the instrument. The accompanying table will enable the physician to ascertain from the percentage amount of urea in the specimen examined what is the absolute amount of that compound excreted during the day, provided, of course, the whole of the urine passed during the twenty-four hours has been collected and carefully measured.

Per cent. of urea of ureometer.	Quantity of urea in grains in 1 fluidounce.	Per cent. of urea by ureometer.	Quantity of urea in grains in 1 fluidounce.
0.1	0.456	1.9	8.658
0.2	0.911	2.0	9.114
0.3	1 367	2.1	9.570
0.4	1.823	2.2	10.025
0.5	2.279	2.3	10.481
0.6	2.734	2.4	10.937
0.7	3.190	2.5	11.393
0.8	3.646	2.6	11.848
0.9	4.101	2.7	12.304
1.0	4.557	2.8	12.760
1.1	5.013	2.9	13.215
1.2	5.468	3.0	13.671
1.3	5.924	3 1	14.127
1.4	6.380	3.2	14.582
1.5	6.836	3.3	15.038
1.6	7.291	3.4	15.494
1.7	7.747	3.5	15.950
1.8	8.203		

EXAMPLE.—The patient has passed 24 fluidounces of urine, found to contain 2.4 per cent. of urea. The total urea excreted will therefore be 10.937 (from the table) × 24 = 262.488 grains.

For exact estimations the temperature of the room in which the experiment is made must be about 70° F. (21° C.). A variation from this temperature of 20° will, however, made a difference in the result of only about 0.2 per cent., so that the temperature correction may be regarded as unimportant.

In the process given for the manufacture of the test solution the hypochlorite is changed into hypobromite.

This mixture gives more uniform and trustworthy results than those obtained with the chlorinated soda alone, which is recommended by Dr. Squibb. It is, in fact, identical in its action with the hypobromite solution, without the great inconvenience of handling bromine. A few minutes must be allowed to elapse after the mixture is made before mixing the urine with it; but this need occasion no delay, since the mixture can be put into the bottle before filling the cylinder and making the connections.

The activity of the solution of chlorinated soda can be easily tested by adding to a little of it in a test tube a few drops of the solution of potassium bromide, and then a little muriate of ammonium, which should cause brisk effervescence. If this is not the case, it is too much deteriorated for use.

In some rare instances it will happen that the urine contains a larger proportion of urea than the ureometer is capable of indicating. When this is the case, and in general when the specific gravity of the urine exceeds 1.030, sugar being absent, it will be best to dilute the urine with an equal volume of water before making the test. 4 c.c. of the diluted urine will then be used as usual in the experiment, but the percentage given by the reading of the instrument must be multiplied by 2.

It will be found in practice that an estimation of urea by this apparatus consumes very little time, and the results for all practical purposes are as accurate as could be wished.

Formula for Special Solution of Chlorinated Soda.—Shake chlorinated lime (best quality) 12 gm. with water 100 c.c.; let settle and filter into a 250 c.c. bottle. Wash the residue with enough water to obtain 130 c.c. of clear filtrate.

Dissolve sodium carbonate 24 gm. in water 45 c.c. Add this solution to the above filtrate, mix thoroughly, and, when reaction is complete, filter, passing, if necessary, enough water through residue on filter to obtain 165 c.c. of filtrate.

The estimation of the daily excretion of urea is of some value as an index of the activity of the kidney in the various forms of nephritis, in diabetes mellitus, during pregnancy or in the puerperium, and before surgical operations. The results, however, are not considered of as great importance as formerly for it has been found that actual renal disease may be well advanced, and yet a high percentage of urea may be present because the portion of the kidney which is affected may not be that which is concerned in the elimination of this substance. The quantity of urea excreted in twenty-four hours is increased in nearly all fevers and inflammations, and is decreased in many cachectic states in which the metabolic changes in the tissues are impaired. It is also decreased in diseases which greatly modify the activity of the liver, the gland which forms urea.

The finding of a low percentage of urea indicates that the kidneys are probably made to eliminate other substances which are toxic and conversely, the finding of a percentage which is approximately normal does not necessarily prove that the function of these organs is perfect so far as the elimination of toxic materials is concerned. (For the estimation of urea, see early part of this chapter.) A persistent scanty urea elimination is, however, of considerable indicative value.

Although the quantity of urea varies very greatly in perfect health, the mean amount excreted in twenty-four hours by a healthy man of twenty to forty years is about 512 grains. Women excrete a little less than men, and children still less in actual quantity, but more in proportion to their weight.

It is absolutely necessary in estimating the amount of urea excreted in twenty-four hours to test a sample of the urine obtained from all the quantity passed in that time, as a test of the urine passed on one occasion is no guide for the total daily quantity.

Chlorides in the Urine.—The urine in health contains chlorides of sodium and potassium, and these are to be discovered by placing a fluidrachm of urine in a test tube and then adding a drop of nitric acid, and finally a few drops of a solution of nitrate of silver. If chlorides are present in considerable quantity, a white precipitate of chloride of silver is thrown down, which can easily be distinguished from albumin; but if some doubt is felt as to its character, the addition of a little caustic ammonia will redissolve it if composed of chlorides, and it will be reprecipitated if nitric acid is again added. If the same quantities of urine and reagents are taken daily and placed in a test tube of equal dimensions and the precipitate allowed to settle for twenty-four hours, we can gain an approximate estimate of the relative quantity of the chlorides. The amount ordinarily passed in twenty-four hours by a healthy man is 250 grains.

The clinical significance of a decrease in the chlorides is not great. They are decreased in the acute stages of croupous pneumonia, acute articular rheumatism, and some other fevers; and if they gradually increase, they indicate the development of convalescence.

Diazo-reaction.—Ehrlich has claimed that a distinct aid to the diagnosis of enteric fever can be obtained by the so-called diazo-reaction of the urine, although it is to be remembered that this takes place in several other conditions of the body, notably pulmonary tuberculosis, measles, diphtheria, croupous pneumonia, malaria, pyemia, scarlet fever, and erysipelas. It is usually present only in severe cases of these ailments. Further than this, Ehrlich asserts that the reaction is usually to be obtained from the fourth to the seventh day of the disease. So far as constancy is concerned it appears more frequently than the Widal reaction. A faint reaction is indicative of a mild attack.

The test is as follows:

(1) 2 gm. (30 gr.) of sulphanilic acid, 50 c.c. of hydrochloric acid, and 1000 c.c. of distilled water. (2) A solution of sodium nitrite in water of the strength of 0.5 per cent. Fifty parts of No. 1 and one part of No. 2 solution are now placed in a test tube and an equal amount of urine added, and this mixture is then rendered strongly alkaline by strong ammonia-water. If the diazo-reaction is present, the liquid becomes pink in color; and if the test tube is shaken, this color is seen in the foam. Indeed, it is this color of the foam which is the determining factor, for this coloration is the point in the reaction. If the liquid only is red, the test is not positive. After standing a day a green precipitate will form in the tube, and this is very confirmatory of the presence of typhoid fever.

Greene has shown that the diazo-reaction will take place in typhoid fever when the dilution is 1 to 100 or 1 to 150, and that this rarely, if ever, occurs in tuberculosis. By means of this dilution, therefore, additional points in differential diagnosis can be obtained.

The diazo-reaction can also be used as a differential agent in separating measles from rötheln, for it does not occur in the urine in the latter malady. It is of value as a prognostic agent in pulmonary tuberculosis, because it does not appear, as a rule, until the disease is far advanced, and life rarely lasts more than six months after it appears.

THE GENERAL SYMPTOMS ASSOCIATED WITH URINARY DISORDERS.

Having considered the pathological changes found in the urine and their significance, we now pass on to a consideration of the general symptoms which will usually be found associated with these variations from the normal functional activity of the urinary organs.

Let us suppose that a patient presents himself complaining that he has been seized with pain in the small of the back, and perhaps by nausea and chilly sensations, followed by a marked decrease in the quantity of urine secreted, which decrease may actually amount to suppression of the urine. The urine that is passed is high-colored or smoky in hue, sometimes looks like porter, and forms a very heavy sediment on standing. If it is filtered and tested for albumin, it will be found to contain this abnormal ingredient in large amount, and a microscopic examination of the sediment will reveal a large number of blood corpuscles, epithelial cells, and casts made up of blood cells, epithelium, and albumin. Scarcely will these signs have been noted when the patient will be seen to be anemic and

puffiness of the face about the eyes will be evident. This puffiness may then pass on to a general anasarca, but it is to be remembered that the most violent *acute diffuse nephritis* may exist without developing anasarca. If the disease be in a child and it is due to scarlet fever, anasarca is common, as is also uremia. The pulse in patients with this form of nephritis is usually hard and tense, and the sharp and clear sound of the aortic valves, as heard at the second right costal cartilage, will indicate the high arterial tension. The skin is generally dry, and, it may be, harsh to the touch. Should the symptoms persist for over a month the possibility of the disease becoming chronic renders the prognosis doubtful; but, as a rule, particularly in young persons, the prognosis of acute diffuse nephritis is favorable. In the acute diffuse nephritis of pregnancy the prognosis is, of course, grave if the pregnancy continues. The history of a case prior to the attack of acute diffuse nephritis will usually be that the patient has been exposed to cold or wet, has been or is a sufferer from an acute infectious disease, has swallowed or inhaled some irritant poison, or has suffered from some severe burn of the surface of the body.

If, instead of an acute attack of illness, the symptoms just described come on gradually and insidiously, and the tendency to anasarca is marked and persistent, we have before us a case of *chronic parenchymatous nephritis*, in which the prognosis is most grave. Uremia, vomiting, and coma may occur in this class of patients. (See chapter on Vomiting.) Blood cells are also found in the sediment of the urine in these cases, but are not so numerous as in acute diffuse nephritis.

A group of symptoms which differ very markedly from those just described occurs in cases of *chronic contracted kidney* (chronic interstitial nephritis). The following description of the symptoms may be taken as representing a typical case: the patient, who is usually past middle life, finds that he or she urinates more frequently and passes a greater amount of urine than heretofore. Often the sleep is disturbed by the necessity of arising to urinate. Instead of the urine being heavy and clouded, it is unusually clear and limpid; and in place of the high specific gravity of diffuse parenchymatous nephritis, we find it unusually low (only 1.010 to 1.015). Albumin is found only inconstantly and in traces, and is generally to be sought for in the urine passed by the patient when first arising from bed. The pulse is usually much increased in tension, and atheroma of the bloodvessels is more or less marked. This high-tension pulse is a valuable diagnostic sign. The heart, which in acute diffuse nephritis may be slightly dilated, or in chronic parenchymatous nephritis somewhat hypertrophied, is in this disease usually markedly hypertrophied, and the second sound at the second right costal cartilage is

commonly accentuated. In addition to these symptoms we find that chronic bronchitis is not rare, and that pulmonary edema and attacks of shortness of breath, which may be called asthmatic, are often present, the latter being most marked at night. Uremic symptoms are more commonly seen in this class of cases than in any other, and violent vomiting, difficult of control, should always make the physician test the urine to discover renal mischief. Unlike parenchymatous nephritis, dropsy is a rare complication of chronic contracted kidney. Microscopic examination of the urine will reveal only a few hyaline and granular casts. The prognosis as to cure is bad, but life may be prolonged indefinitely.

Let us suppose, however, that a patient comes to us with a history of exceedingly copious urination, of great thirst, of loss of flesh, and has a dry, harsh skin, we immediately recognize that a test of the urine will probably reveal the case to be one of *diabetes mellitus.* This will be pointed to if a high specific gravity is found present in a clear limpid urine, and confirmed if the tests for sugar already given produce a reaction. The other prominent symptoms of diabetes mellitus are furunculosis, intense itching and erythema (see chapter on the Skin), an excessive appetite, and, in some severe cases, gangrene of the extremities or diabetic coma (see chapter on Coma and Unconsciousness). If the urine has a constant low specific gravity and contains no albumin or sugar, the case is probably one of *diabetes insipidus.*

CHAPTER XII.

THE BLOOD.

The various forms of red and white corpuscles—Their proportionate number in health and disease—Alterations in their form and character—The hemoglobin of the blood in health and disease—The various forms of anemia—Leukemia and pseudoleukemia—Parasites of the blood—The blood in typhoid fever and diabetes.

As already pointed out in the chapter devoted to the skin, marked changes in the blood speedily produce manifest alterations in the appearance of the patient. The present chapter will be devoted to a consideration of the changes in the blood seen by the naked eye or by the aid of various forms of delicate apparatus designed to furnish accurate information. Before studying the conditions of the blood which are found in disease, it is well briefly to rehearse the characteristics of normal blood when it is examined outside the body.

The blood consists of a liquid basis or plasma, in which are found two varieties of cells—the red blood cells, or erythrocytes, and the white blood cells, or leukocytes. The red cells are biconcave disks, dark at the edges, and with a clear or bright spot in the centre, due to their biconcavity. They are non-nucleated. The red color of the blood is due to the aggregation of immense numbers of these bodies, the coloring matter of which is called hemoglobin, though the individual corpuscle when placed in a bright light on the stage of the microscope appears a pale greenish yellow. The number of red blood cells is about 5,000,000 to the cubic millimeter of blood in a healthy adult male, though counts above this, even to 5,500,000, are not infrequent, and about 4,500,000 are present in the healthy female. If this number is exceeded the condition is called polycythemia; if decreased, oligocythemia.

The proportion of the white to the red cells in health is about 1 to 500 or 600, but very great variations occur. Thus, after meals the white corpuscles are always increased, so that the proportion may be 1 to 350 of the red cells. Hirt found before breakfast the proportion to be 1 to 716; one hour after breakfast, 1 to 347; three hours after breakfast, 1 to 1514; ten minutes after dinner, 1 to 1592; half an hour after dinner, 1 to 429; two and a half hours after, 1 to 1481; half an hour before supper, 1 to 544; and two hours after supper, 1 to 1227. It is important to remember this influence of food on the leukocyte count. Not only may this cause produce great altera-

tions in the results obtained in a blood examination, but many others must be considered, it being always borne in mind that there are pronounced variations in the blood within healthy limits, both in quantity, quality, and corpuscular elements. For these reasons slight variations should not be given much weight unless by repeated tests they are found constant. It is usually unsafe to rest upon a single test. Thus sweating, if profuse, may cause such a concentration of the blood that a normal number of red cells to a cubic millimeter may be shown when in reality there is marked anemia. So, too, tapping an effusion, or the use of an active hydragogue cathartic, or profuse diarrhea may cause great concentration. Blood obtained from a cold ear or a cold finger tip, or from these parts when made unduly warm by friction or heat, gives erroneous ideas of the state of the blood in general.

Proceeding, then, to the study of the blood for diagnostic purposes, we examine it by the microscope, and by color tests. The object of the microscopic examination is to determine the quality and character of the red and the white corpuscles, their number, and the presence of parasites. The color tests are for the purpose of determining the proportion of hemoglobin, or, in other words, the ability of the corpuscles to carry oxygen to the tissues.

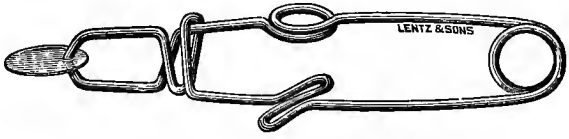


FIG. 152.—Stewart's forceps for holding cover-glass.

Microscopic Examination of the Blood.—To study the blood microscopically we need, for ordinary corpuscular work, a quarter-inch or, better, a fifth- or sixth-inch objective, or, as they say on the Continent of Europe, a Number 7 Hartnack or a D Zeiss; and for examinations for parasites a $\frac{1}{12}$ oil-immersion lens for use with a condenser. The eye pieces used are generally Nos. 1, 2, and 4.

Perfectly clean cover-glasses and slides, such as are used in ordinary microscopic work, are also needed. It is of the greatest importance that these glasses shall not only be free from dust and dirt, but also that they shall not be finger-marked. After ordinary washing they should be washed in ether and alcohol, or acid alcohol, and then carefully dried by gauze or lint-free tissue paper. They are further improved by being passed a time or two through an alcohol or gas flame. The operator should use a pair of fine forceps provided with a clip, so that he can pick up a cover-glass without touching it with his fingers and lay the glass down without releasing it from the forceps (Fig. 152), or handle them by the edge only.

The tip of the finger or the lobe of the ear is now washed clean with alcohol and dried very gently, so as not to cause an artificial hyperemia, and the skin of the part is then punctured by a tenotome or a small spear point made for the purpose (Fig. 153). The first drop of blood which escapes is wiped off, and the centre of a cover-glass is touched to the second drop, so that a small amount adheres to

FIG. 153.—Lance for stabbing skin of tip of finger or lobe of ear to obtain drop of blood.

the surface. It is then placed with the blood side down on a clean glass slide, which has been slightly warmed, and examined under the microscope. This procedure is invaluable in the study of parasites, but gives so crude an idea of the true state and number of the corpuscles, that for such purposes it is usual to employ more delicate technique according to the object sought by the physician.

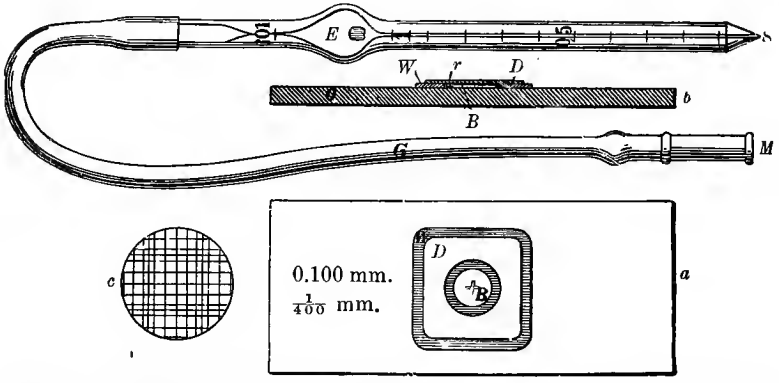


FIG. 154.—Thoma-Zeiss blood-counting apparatus. A heavy glass slide (*a*), in the middle of which is a cell (*B*) exactly $^1/_{10}$ millimeter in depth. The cell is limited at the periphery by a circular gutter to prevent fluid placed upon the cell from flowing beyond it between the slip and cover-glass. The floor of the cell is ruled into squares whose sides are $^1/_{20}$ mm. Dark lines mark out large squares containing twenty-five small squares. Thick, carefully ground cover-glasses (*D*) are provided in the case. The ordinary Potain *Melangeur* (*S*) is used to measure and mix the blood. It consists of a capillary tube the upper portion of which is blown into a chamber (*E*) holding 100 c. mm. The stem of the tube is graduated at 0.5 and 1 c.mm.

Red Blood Cell Counting.—If he wishes to know the number of the red cells, he resorts to what is called a hematocytometer, of which the best is that called the Thoma-Zeiss apparatus. This is composed of two parts. One part is a glass capillary tube, about 10 cm. long, with an expansion near the middle, which contains a small movable glass ball. On the tube are three marks. Part way up it is marked

0.5, just below the expansion 1, and above the expansion 101. The second piece of apparatus is a heavy glass slide, upon which is cemented a square of glass with a central circular opening. In the centre of this opening is a small disk of glass, which is not as thick as that forming the surrounding square, and the surface of which is ruled in 400 minute squares, every fifth one being marked by an extra line (Fig. 155). If the special cover-glass is now placed over the square, it will not touch the surface of the centre disk, but leave the space of 0.1 mm. Looking from above downward there is seen a cover-glass, and under it a disk which it does not touch; between the disk and the boundary of the circular opening is a narrow moat. It is upon the upper surface of the ruled disk that the drop of diluted blood, about to be described, is placed.

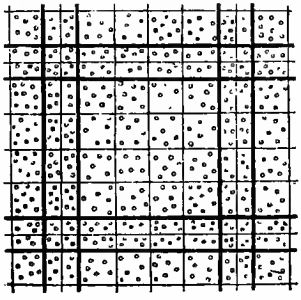


FIG. 155.—Appearance of blood in the Thoma-Zeiss cell.

The finger or ear having been freshly pricked and the first drop wiped off, the blood is drawn up to the mark 0.5 or 1.0 in the capillary tube, and the tip of the tube is then wiped clean. A 0.6 per cent. solution of common salt is drawn up after it, until the tube and bulb are filled to the point marked 101. The tip of the tube is now wiped dry by means of a clean cloth. By shaking the glass ball in the tube the blood and salt solution become well mixed in the proportion of 1 to 100, or 1 to 200, depending upon whether the blood was drawn to the 1.0 or 0.5 mark. After the salt solution in the lower part of the capillary tube has been forced out by compressing the rubber tube of the pipette, and the blood mixture has reached its tip, a drop of this homogeneous fluid is forced out so that it rests on the surface of the disk, covering the central third. The cover-glass is now allowed to fall upon the drop in such a way that all air is excluded, and the drop distributed over the surface of the disk. The cell should now be allowed to stand for several minutes to allow the corpuscles to settle and become stationary, and care should be exercised that the stage is perfectly level, as otherwise the blood cells will gravitate to one margin. As we look through the micro-

scope, we now see a space of 16 squares surrounded by double lines, and if to these 16 squares we add the squares over which the heavy lines run, we have 36 squares under observation. The corpuscles are now counted in series, following the wavy line shown in Fig. 156, so that the same square may not be counted twice. After all the cells in this group of 36 squares are counted, 5 other groups of 36 are counted. The total number of red cells is now divided by 6, to find the average in each group of 36 squares. This in turn is divided by 36, to determine the number in 1 square, and this number is multiplied by 800,000 if the blood has been drawn up to 0.5 in the pipette, or 400,000 if it has been drawn up to 1. These figures, 800,000 or 400,000, are not arbitrary, but are based on the fact that each square equals 1 to 4000 of a millimeter, and the blood has

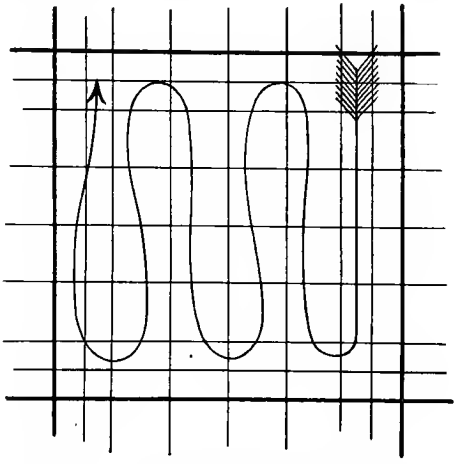


FIG. 156.—Method of counting red cells following the direction indicated, to avoid mistakes.

been diluted 200 times or 100 times. Thus we have the following approximate process as an illustration:

Total corpuscles in 216 squares = $1296 \div 6 = 216 \div 36 = 6 \times 800{,}000$ = 4,800,000 in a cubic millimeter.

Or if the blood has been diluted 100 times, then we have the following illustration:

Total corpuscles in 216 squares = $2592 \div 6 = 432 \div 36 = 12 \times 400{,}000 = 4{,}800{,}000$ in a cubic millimeter.

For the beginner the procedure is simplified by using only blocks of 16 within the double lines, the latter being regarded simply as landmarks to separate the different blocks.

In making the count, it will be found that some of the corpuscles overlap the line of a given square, and may lead to error by being

counted twice or left out altogether. For this reason it is customary to include those corpuscles which overlap the upper and left-hand borders. Further, it is best to put down the number of cells found in each square as they are counted, and not to attempt to carry the addition in the memory, since the loss of one corpuscle makes considerable difference in the ultimate result. The more squares included in the original count the more accurate the result.

Immediately after using the apparatus the glass slide should be washed in pure water, not alcohol, and the pipette washed out first with distilled water and then with alcohol and finally with ether.

White Blood Cell Counting.—When making the count of the red blood corpuscles care should also be taken to estimate the white corpuscles, since the proportion of white to red cells often gives us very valuable information in disease. This may be done in the same specimen by using as a diluent for the blood in the pipette what is called Toisson's solution instead of the solution of salt already named. This stains the white cells blue, and thus makes their counting possible. Toisson's solution is composed of:

Methyl violet	0.03 (½ grain)
Neutral glycerin	30.0 (1 ounce)
Distilled water	80.0 (2½ ounces)
Mix thoroughly and add	
Chloride of sodium	1.0 (15 grains)
Sulphate of sodium	8.0 (2 drachms)
Distilled water	80.0 (2½ ounces)

As the red cells are counted the number of the white cells can also be noted in the 400 squares of the field. By counting several fields, made possible in one specimen by the extra rulings of modern counting chambers, and taking the average of each the true ratio is obtained. Thus if the average is 8 in a dilution of 1 to 100, add three ciphers, and 8000 is the number of white cells present.

When we desire to count the white corpuscles alone, we employ a pipette (sometimes called the "white pipette"), which makes the dilution of the blood in the proportion of 1 to 10, and we use in place of salt solution as a diluent a 0.3 to 1 per cent. solution of glacial acetic acid in water. This acid solution dissolves the red corpuscles, but makes the white ones more readily seen. The method of calculating the number of white corpuscles in a cubic millimeter is the same as that given for the red corpuscles, except that as the dilution is 1 to 10, instead of 1 to 100, we multiply by 40,000 instead of 400,000.

When the large white pipette is used, it will be found that the blood is sucked into it much more readily than into the "red pipette," because the caliber of this tube is larger. Greater care must there-

fore be used that the blood or fluid does not quickly pass up above the marks on its surface. This fact also causes the blood and fluid to run out of the tube very easily if it is held vertically. For this reason the bottle holding the diluting fluid should be tipped a little, so as to keep the tube as nearly level as possible. Because of the large caliber, a greater amount of blood is needed than is usually required for blood examination, and, therefore, the puncture of the skin must be large enough to permit a free flow.

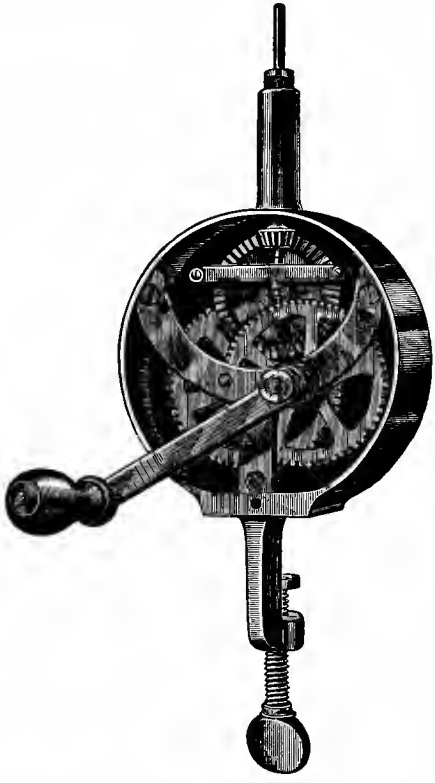


FIG. 157.—Centrifuge.

Several fields of white cells should be counted, because the number of white cells found in a single field are too few to enable us to get accurate results, for a variation of one or two cells may make a great difference when they are multiplied by so large a figure. This is the reason that we do not use the red-cell diluting pipette for this purpose. At least 100 white cells should be counted, to obtain correct results. The number of leukocytes per cubic millimeter of normal blood is from 6000 to 10,000, with a mean of about 7500.

The Hematocrit.—A method of obtaining an approximate estimate of the number of the red and white blood cells is by the use of the centrifuge, with the hematocrit attachment (Figs. 157 and 158).

This instrument is used for the volumetric estimation of the red and white blood corpuscles without previous dilution of the blood. The hematocrit attachment consists of a metallic frame, carrying two graduated capillary glass tubes, 50 mm. long and $\frac{1}{2}$ mm. bore, in which is placed the freshly drawn blood. These accurately graduated glass tubes, seated in rubber-cushioned cups at 1 and 2, are held in position securely by spring cups, *A A*, so that there is no

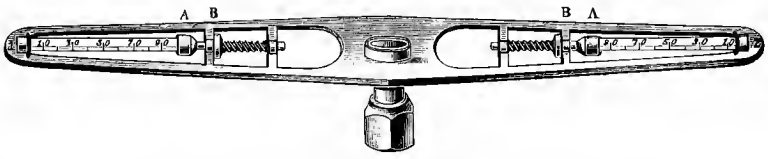


FIG. 158.—Hematocrit attachment for centrifuge.

possible danger of losing the tubes during rotation. By drawing back the milled heads, *B B*, the tubes are instantly released and as quickly clamped again into position (Fig. 158). This apparatus should be made of aluminum, in order that it be strong and light. The advantage gained by the use of this metal is that it is possible to greatly increase the length of the arms of the hematocrit, thereby taking advantage of the well-known law of mechanics that "the centrifugal forces of two equal bodies, moving with equal velocity at different distances from the centre, are inversely as their distance from the centre." In order, therefore, to obtain any desired amount

FIG. 159

of centrifugal force it is not necessary to increase the speed of the machine, but simply to increase the distance from the centre.

The finger of the patient is thoroughly cleansed with water, and then punctured by means of a spear-pointed needle. The first drop of blood is rejected, and a second drop is secured by very slight pressure. The blood is then drawn, by suction, by means of a constricted dropper (Fig. 159), with rubber-bulb connection, into the capillary tube, which is then placed in the hematocrit and rapidly revolved for at least one minute.

The rapidity and simplicity of this process are theoretically at once apparent. The blood does not have time to coagulate, and

by the centrifugal force the red corpuscles, having the greatest specific gravity, are thrown to the distal extremity of the tube, and will occupy about one-half of the tube, or to the mark 50.

The white corpuscles, next in specific gravity, will occupy a position between the red corpuscles and the liquor sanguinis, which is found in the proximal end of the tube, quite clear and free from corpuscles.

When the column of red blood corpuscles extends to mark 50 we have, as a rule, about 5,000,000 red corpuscles per cubic millimeter; but if the precipitated corpuscles reach only mark 30, there are only about 3,000,000 per cubic millimeter, or 60 volume per cent. If they reach only mark 20, there are about 2,000,000 per cubic millimeter, or 40 volume per cent.

This method of blood estimation was highly thought of when first introduced; it later largely lost its popularity, but now is again receiving some attention. For determining the number of red cells it must always be subject to grave error because of the change in size of these cells in many conditions. It furnishes valuable information regarding the total volume of the red cells.

Significance of Variations in Number of the Red Cells.—Changes in number of the red cells are of practical diagnostic importance. They are diminished, oligocythemia, in essentially all cases of anemia of whatever type, though in some this reduction does not form the most striking feature of the blood condition. The decrease is especially prominent in the secondary anemia of certain infections and parasitic diseases and in pernicious anemia. An increased number of red cells, polycythemia, is constant in the newborn and often attains an extraordinary degree in cases of congenital heart disease, in persons who remove from lower to higher altitudes, and in the subjects of that little-understood affection characterized also by chronic cyanosis and enlarged spleen, chronic polycythemia with splenomegaly. In the last-named condition counts as high as 12,000,000 have been recorded.

(For a discussion of the Blood Diseases, see the latter part of this chapter.)

Differentiation of the Leukocytes.—Continuing the examination of the blood for definite diagnostic purposes, we find that a very important part of this study consists in the differentiation of the various forms of white blood cells. These cells appear in the blood of healthy individuals in five forms:

1. They occur as white cells the size of, or smaller or larger than, the ordinary red cell. Each of these small white cells contains a nucleus so large that it almost completely fills the body of the cell, the protoplasm being a narrow rim or not discernible. They are called lymphocytes and form about 20 per cent. of the total number

of white cells in health. They are not phagocytic, nor actively ameboid.

2. They occur as what are called large mononuclear leukocytes, or hyaline cells, or large lymphocytes, much larger than the red cells, possessing a moderately large single nucleus, which is surrounded by a zone of pale non-granular protoplasm. Sometimes these cells show a change in the shape of the nucleus, and are then called transitional leukocytes. These mononuclear cells make about 5 to 8 per cent. of the white blood cells.

3. They occur as large white cells with a nucleus of irregular shape (polymorphous nucleus), or a nucleus split up into several smaller nuclei (polynuclear). They are often for this reason called polymorphonuclear or polynuclear leukocytes. Their protoplasm contains fine granules, which stain when brought in contact with neutral dyes, and for this reason these cells are often called neutrophiles. A neutrophile, a polymorphous leukocyte, and a polynuclear leukocyte are, therefore, one and the same thing. They equal about 70 per cent. of the white blood cells and are actively ameboid and phagocytic.

4. Polymorphonuclear cells, usually slightly smaller than the preceding, containing very coarse granules, which stain when brought in contact with acid dyes, of which the chief is eosin. They are called eosinophiles, and are limited in number to 2 or 3 per cent. These cells possess ameboid movement, but are not phagocytes.

5. Basophile cells. In size and in shape of nucleus they resemble the polymorphonuclear leukocyte, but the protoplasm contains basic-staining granules, variable in size. They are not above 0.5 per cent. in normal blood.

In disease we find variations from these types as to proportional and actual number, and in addition other white cells are present. The most important abnormal leukocyte is the myelocyte or marrow cell, never found in normal blood. It is a round or oval cell, often larger than the large mononuclear which it resembles except that the protoplasm contains acid, neutral, or basic-staining granules. The neutral or neutrophile type is the one most frequently found.

In order that the various forms of white cells that we have named may be readily separated from one another we have to resort to certain stains, it having been shown by Ehrlich and many others that the nuclei of these cells are susceptible to different stains, as are also the granules found in their protoplasm. These stains differ as to their color and reaction. We have basic stains and acid stains, but these terms are largely arbitrary and it is not to be thought that they are actually acid or basic in their chemical reactions. As a rule, the acid stains affect the protoplasm of a cell, and the basic stains the nuclei. The basic stains are methylene blue, methyl violet,

methyl green, hematoxylin, and gentian violet. The acid stains are picric acid, eosin, orange G, and acid fuchsin. The cells whose protoplasmic granules stain with basic stains are sometimes called basophiles, those whose protoplasm is stained by acid stains acidophiles (eosinophiles), and those cells which stain with both acid and basic stains neutrophiles. Further, these stains render the nucleus one color and the granules another, as in Plate XI, in several of the figures of which will be found cells with red granules and blue nuclei.

One of the best solutions for staining purposes is that of Ehrlich, which is called a triple stain. It is composed as follows: saturated watery solution of orange G, 6 c.c.; saturated hydro-alcoholic (20 cent. of alcohol) solution of acid fuchsin, 4 c.c. These ingredients having been mixed gradually and thoroughly shaken, the following constituents are added, the shaking being continued: saturated watery solution of methyl green, 6.6 c.c.; absolute alcohol, 10 c.c.; glycerin, 5 c.c.; water, 5 c.c. This solution should stand for twenty-four hours, to allow of sedimentation; it improves with age; and when it is used the supernatant liquid is to be drawn off by a pipette in order to avoid the sediment. It must not be filtered. This stain acts in a few minutes.

Cover-glass films, made as already described, are heated over an alcohol flame or on a hot stage made of sheet copper or in an oven, and kept at 120° to 140° C. for fifteen minutes to half an hour. After this they are placed in the staining fluid for from one to four minutes, then washed in pure water, dried, and mounted in Canada balsam or cedar oil. The Canada balsam should not be prepared with chloroform, as it will decolorize the specimen. The film is then ready for microscopic examination with a one-twelfth oil-immersion lens. The eosinophile granules in the corpuscles will be stained a reddish hue, the neutrophile granules lavender or purple, and the nuclei bluish green or blue. The greatest objection to this stain is that it does not stain basophile granules nor parasites. (See Plate XI.)

For routine blood work Ehrlich's stain is being largely superseded by the various polychrome methylene blue-eosin combinations, of which those bearing the names of Leishman, Wright, and Hastings are well known. These stains possess several advantages. Films need no previous fixation (the menstruum being pure methyl alcohol) and basophile granules and parasites are well stained. Neutrophile granules, however, are usually not so well stained as by Ehrlich's method. All these stains are made after complex formulas which need not here be given, as the practitioner had best obtain them already prepared. The technique for using them all is very similar. The dried, unfixed film is covered by a few drops of the stain to

Fig I

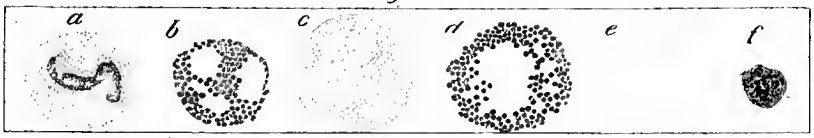
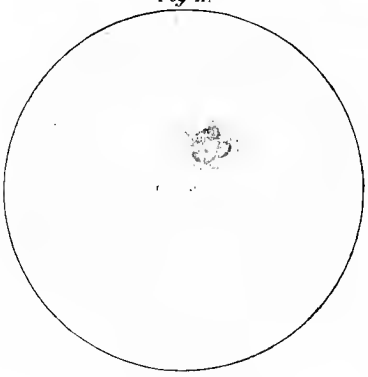


Fig II.



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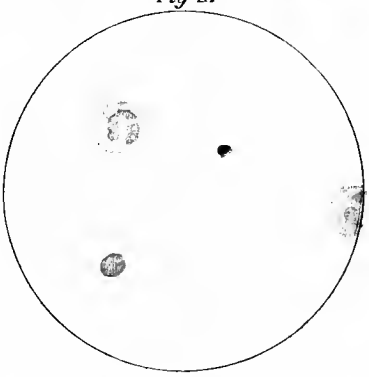


Fig IV.

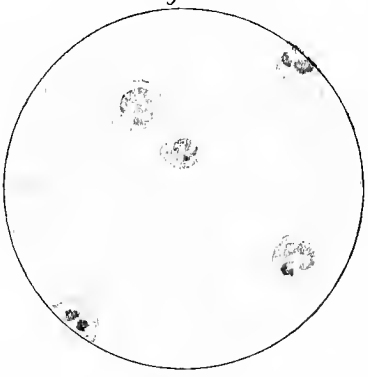


Fig.V.

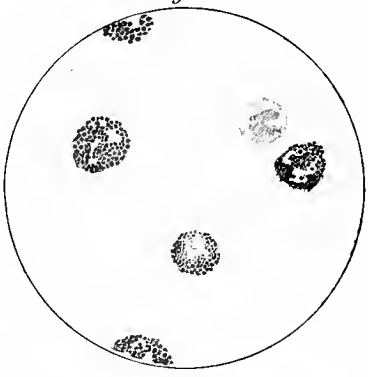


Fig VI.

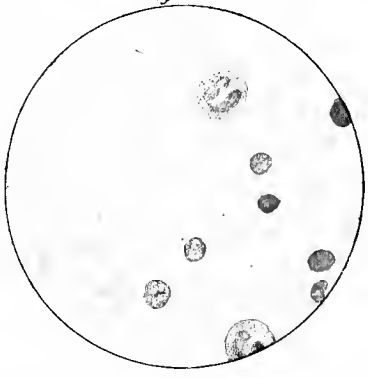


Fig.VII.

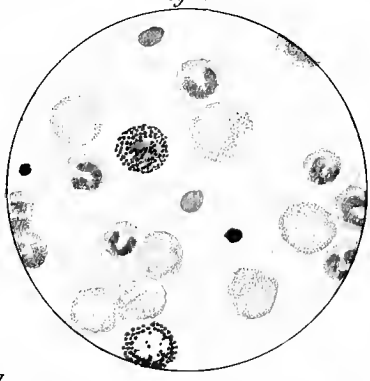


Fig.VIII

DRAWN BY J.N.Z. CHASE

PLATE XI.

BLOOD.

(Ehrlich triple stain.)

(Prepared by Dr. I. P. Lyon.)

Fig. I. TYPES OF LEUCOCYTES.

a. Polymorphonuclear Neutrophile. *b.* Polymorphonuclear Eosinophile. *c.* Myelocyte (Neutrophilic). *d.* Eosinophilic Myelocyte. *e.* Large Lymphocyte (large Mononuclear). *f.* Small Lymphocyte (small Mononuclear).

Fig. II. NORMAL BLOOD.

Field contains one neutrophile. Reds are normal.

Fig. III. ANÆMIA, POST-OPERATIVE (secondary).

The reds are fewer than normal, and are deficient in hæmoglobin and somewhat irregular in form. One normoblast is seen in the field, and two neutrophiles and one small lymphocyte, showing a marked post-hæmorrhagic anæmia, with leucocytosis.

Fig. IV. LEUCOCYTOSIS, INFLAMMATORY.

The reds are normal. A marked leucocytosis is shown, with five neutrophiles and one small lymphocyte. This illustration may also serve the purpose of showing the leucocytosis of malignant tumor.

Fig. V. TRICHINOSIS.

A marked leucocytosis is shown, consisting of an eosinophilia.

Fig. VI. LYMPHATIC LEUKÆMIA.

Slight anæmia. A large relative and absolute increase of the lymphocytes (chiefly the small lymphocytes) is shown.

Fig. VII. SPLENO-MYELOGENOUS LEUKÆMIA.

The reds show a secondary anæmia. Two normoblasts are shown. The leucocytosis is massive. Twenty leucocytes are shown, consisting of nine neutrophiles, seven myelocytes, two small lymphocytes, one eosinophile (polymorphonuclear) and one eosinophilic myelocyte. Note the polymorphous condition of the leucocytes, *i.e.*, their variations from the typical in size and form.

Fig. VIII. VARIETIES OF RED CORPUSCLES.

a. Normal Red Corpuscle (normocyte). *b, c.* Anæmic Red Corpuscles. *d-g.* Poikilocytes. *h.* Microcyte. *i.* Megalocyte. *j-n.* Nucleated Red Corpuscles. *j, k.* Normoblasts. *l.* Microblast. *m, n.* Megaloblasts.

which, after the lapse of one minute, is added drop by drop distilled water until a scum forms or until a predetermined amount has been added, it being necessary experimentally to determine the exact quantity for each lot of the stain. The diluted stain is allowed to act for three or four minutes, washed off with water, the film blotted dry and mounted in balsam. Nuclei are stained blue or lilac, neutrophile and eosinophile granules pink, basophile granules blue or purple, the protoplasm of the lymphocyte a deeper, of the large mononuclear a paler, blue than the nucleus.

Changes in form, size, and staining reaction of the red cells are determined by the examination of stained films made in the following manner: The centre of a clean cover-glass is touched to a small drop of freshly drawn blood and immediately dropped, blood side downward, upon a second cover. The instant the blood ceases spreading, the two covers should be rapidly drawn apart, without pressure or lifting, and the films allowed to dry in the air. They are then stained by one of the methods described in connection with the leukocytes. The properly made film is made up, except at the periphery, of a single layer of red cells.

Massive changes in staining reaction, polychromatophilia, and punctate basic areas, granular or basophilic degeneration, may in this way be detected. Basophilia is an almost constant feature in chronic lead poisoning.

In the anemias are found deformed red cells known as poikilocytes. Small red cells, microcytes, and large cells, macrocytes or megalocytes, may also be present; the former are occasionally found in normal blood. Nucleated erythrocytes, microblasts, normoblasts, or megaloblasts, according to their size, appear under certain circumstances. Cells may contain vacuoles or be so deficient in hemoglobin as properly to be called "shadow corpuscles." Some of the diseases in which these changes are found will be discussed farther on.

Estimation of Hemoglobin.—Having discovered the number and the quality of the red corpuscles, an equally important measure is to determine the quantity of hemoglobin which they contain. This is often done by the use of the hemoglobinometer of von Fleischl, although that of Gowers is sometimes used. Fleischl's apparatus consists of a small table, in the centre of which is a hole into which fits a cylindric chamber with a glass bottom, divided perpendicularly in the middle by a metal diaphragm, the space on one side of which is filled with pure water, that on the other with diluted blood. Under the stand is a frame in which is set a piece of colored glass as near the hue of diluted blood as possible, and tapered off gradually, so as to give a lighter shade of red at one end than at the other. The frame carrying this glass

wedge is marked by a graduated scale, and is moved from side to side under the half of the cylinder, which is to contain only pure water, by a thumb screw. Beneath the glass and cylinder is a white reflector to direct the rays of light through them (Fig. 160). The rest of the apparatus consists in a little capillary tube attached to a tiny metal handle. This tube will hold just enough pure and healthy blood to color the water on one side of the cylinder to the hue shown in the colored glass when it is opposite the normal mark "100." The finger being punctured, the end of the capillary tube is lightly touched to the drop of blood, which fills the tube. The blood is

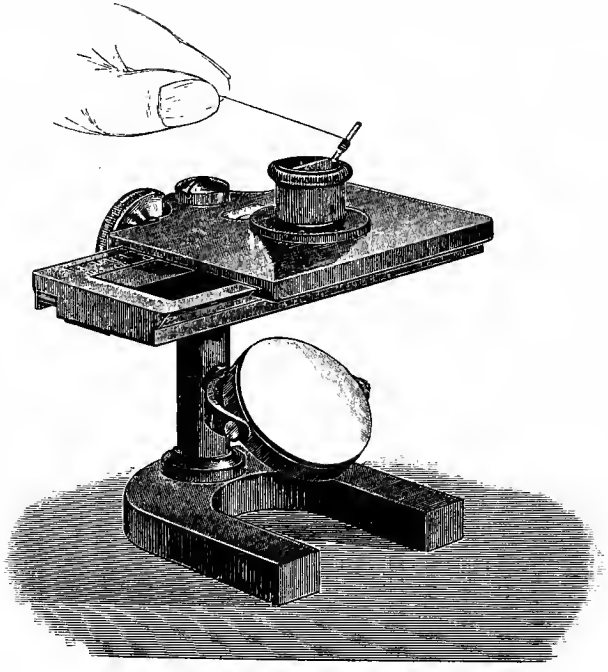


FIG. 160.—Von Fleischl's hemoglobinometer.

now washed from the capillary tube into one of the sections of the hemometer container, into which was previously placed a few drops of water, by directing through the tube a stream of distilled water from a fine-pointed pipette. After thoroughly mixing the blood and water with the handle of the pipette, both compartments are filled to the brim with distilled water. The inside of the capillary tube should be perfectly dry before filling it with blood, and, when filled, no blood should be allowed to cling to the outside. Neglect of these precautions invites serious inaccuracies in the results obtained. The apparatus is then exposed to candle- or lamplight, because with day-

light the hue of the glass does not match blood color, and the frame is moved under the side of the cylinder, which contains only pure water, until the glass seems to the eye to match the fluid containing the blood on the side through which the pure light streams. If the glass matches the blood color when the mark on the frame is at 50, it shows that the hemoglobin equals only 50 per cent. of normal; or if it is at 85, it signifies 85 per cent. As a matter of fact, an examination of perfectly healthy blood will often give not more than 85 to 90 per cent. of hemoglobin with this apparatus.

Care should be taken in regard to three points: first, to be sure that all the blood is washed out of the capillary tube into the water; second, that the two halves of the cylinder are filled to the brim with water, so that there is neither a positive nor a negative meniscus; third, to be careful to cleanse the entire apparatus thoroughly after each use of it before putting it away.

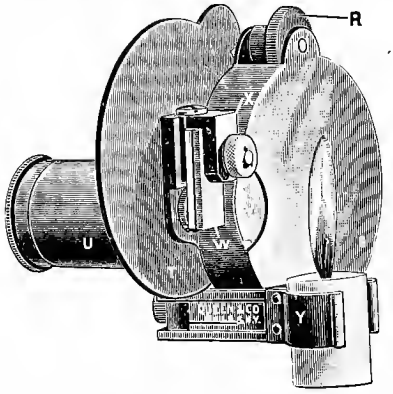


FIG. 161.—Dare's hemoglobinometer. *W.* Blood holder. *R.* Wheel to turn color glass. *V.* Color glass. *U.* Observation tube. *T.* Shade. *Y.* Candle.

Miescher has recently modified the apparatus of von Fleischl in a way that ensures greater accuracy, but the instrument is so complex as to be practicable only for laboratory use. It possesses the very desirable feature of giving the hemoglobin reading in grams per 100 c.c. of blood instead of in per cent., as do other instruments.

A better apparatus than that of von Fleischl has recently been introduced by Dr. Arthur Dare, formerly one of the assistants in my clinic at the Jefferson Medical College Hospital. It may be described as follows:

The application of this instrument depends upon the principle that the color of a thin film of undiluted blood, illuminated by candle-light, may be compared with a graduated color comparison. It has the advantage over other methods employed for a similar purpose, that while but a small drop of fluid is needed for its

application, this consists of pure blood undiluted with artificial serums.

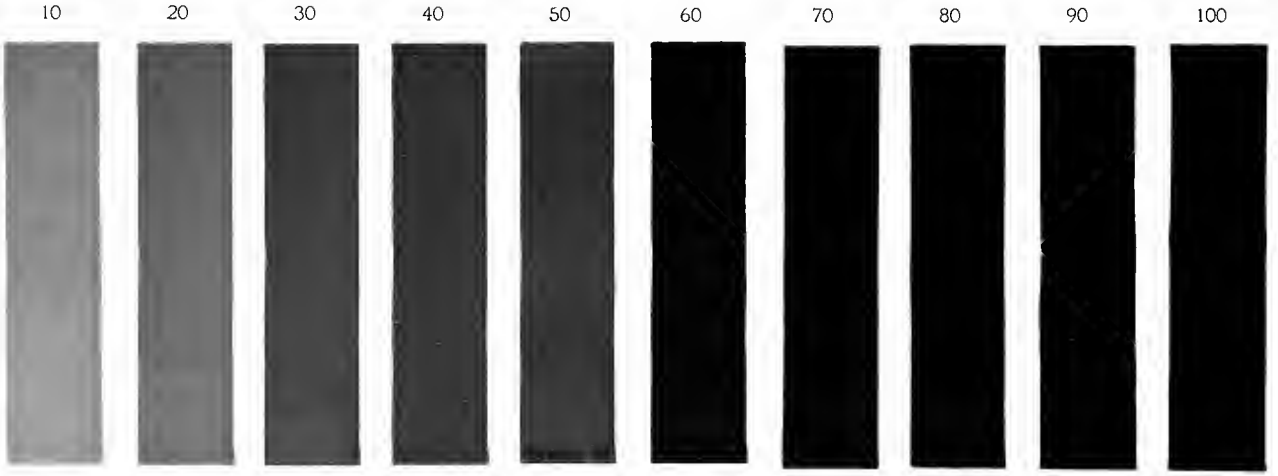
The technique and possible errors of dilution are thereby entirely eliminated, and leukocytosis, which is a frequent source of error in hemoglobin estimations, imparting as it does a turbidity to mixtures of blood and water, becomes imperceptible when viewed by transmitted light against an opaque background, the red color of the hemoglobin only becoming visible, while the opacity due to increase in the number of leukocytes is incalculable.

Furthermore, the shades of color to be compared are very decided, even in blood markedly deficient in hemoglobin, and thus facilitates accurate comparison. Besides the colorimetric principle, there is an element of density, due to concentration of coloring matter present in the blood film, that magnifies the shade of color, giving a wider range between decimal points, ensuring more uniform comparison and greatly reducing the "personal equation" of estimation existing in different individuals.

The examination, which consists of collecting the blood and the estimation of hemoglobin percentage by comparison with the graduated color scale, requires very little time for its accomplishment. The plane surfaces of the blood pipette are quickly cleansed and the whole procedure completed in a very brief period.

Still another method of estimating the hemoglobin, which does not require any costly apparatus, but for which considerable technical skill is necessary, is the so-called specific gravity method. This is Hammerschlag's modification of Roy's method. Chloroform and benzol are mixed in an ordinary urinometer tube, so that their specific gravity is 1059—in other words, that of normal blood. A drop of blood obtained by the ordinary method of puncture is dropped into this mixture in such a way that it does not touch the sides of the urinometer tube. The drop floats undissolved in the chloroform and benzol mixture. If it sinks to the bottom, its specific gravity is higher than normal, and we add a few drops of the heavier ingredient, chloroform, until the blood drop floats free in the mixture. If, on the other hand, it rises, we add the lighter benzol until it becomes suspended. The specific gravity of the fluid, which must be also that of the blood, is now taken, and if it is above normal we have reason to believe that the hemoglobin is above normal, or if it is below normal, that the hemoglobin is scanty. This is true in persons in ordinary disease, but in dropsy, when the red cells may be water-soaked and heavy, or in leukemia, when the white cells are present in excess, the specific gravity may be excessive and yet the hemoglobin below normal. Otherwise the test is fairly accurate, since the specific gravity of the blood cells depends largely on their content of hemoglobin. The various specific gravities equal the

PLATE XII



Tallquist's Color Scale for estimating Hemoglobin.

percentage of hemoglobin in the following proportions, according to Hammerschlag:

1033 to 1035 = 25 to 30 per cent.
1035 to 1038 = 30 to 35 " "
1038 to 1040 = 35 to 40 " "
1040 to 1045 = 40 to 45 " "
1045 to 1048 = 45 to 55 " "
1048 to 1050 = 55 to 65 per cent.
1050 to 1053 = 65 to 70 " "
1053 to 1055 = 70 to 75 " "
1055 to 1057 = 75 to 85 " "
1057 to 1060 = 85 to 95 " "

Another method, not so accurate but useful in lieu of those described, for estimating the hemoglobin, has been devised by Tallquist, and consists in taking up a drop of blood with pure white bibulous paper and promptly comparing the saturated area with a correct hemoglobin color scale. The test should be made in direct sunlight. This is, of course, a very simple and only approximately correct method. (See Plate XII.)

Estimation of the hemoglobin is one of the most valuable points in a blood examination. A fairly high hemoglobin content may be found in the blood of persons presenting the superficial appearances of well-marked anemia and conversely a low amount in the apparently healthy. The normal amount is regarded as 100 per cent., but often the reading is below this, to 95 or in females even 90, in persons with perfect health.

Reduction in hemoglobin, oligochromemia, occurs in all types of anemia, but is absolutely and also relatively to the red cells much more severe in some than in others. Da Costa finds these average readings: In pernicious anemia, 25 per cent.; in leukemia, 39 per cent.; chlorosis, 43 per cent.; secondary anemia, 55 per cent. An increased hemoglobin percentage may be present during temporary concentration of the blood and commonly accompanies polycythemia, in some types of which it has been found to reach 200 per cent.

In important cases the several methods of estimating hemoglobin should be used before a definite decision as to the exact percentage is reached.

Color Index.—Not only is it desirable to estimate the quantity of hemoglobin which may be present in the blood, but also to know the relative richness of hemoglobin in each corpuscle. Thus, in chlorosis the red cells may be nearly normal in number but sadly lacking in hemoglobin. In pernicious anemia the reverse condition is present. To determine this, we divide the percentage of hemoglobin by the percentage of red cells. For example, suppose we find that the hemoglobin is present in the normal amount of 100 per cent., and a blood count reveals in a man 5,000,000 corpuscles to a cubic millimeter, we know that the percentage of red cells is also 100. We, therefore, divide 100 per cent. of hemoglobin into 100 per cent. of red cells, and 1 is the result. This is said to be the *color index*, and indicates

that each cell is normally rich in coloring matter. The color index may, however, be apparently normal, and yet anemia be present, for if the red cells be reduced to 2,500,000, or 50 per cent., and the hemoglobin to 50 per cent., or one-half, the index would still be 1. In chlorosis, however, the cells might be 4,000,000, or 80 per cent., and the hemoglobin 50 per cent., in which case we follow the rule to divide the percentage of hemoglobin by the percentage of red cells, which would be $50 \div 80 = 0.6 +$, or if in pernicious anemia the red cells are 1,000,000, or 20 per cent., and the hemoglobin is 40 per cent., we find that $40 \div 20 = 2$. Each cell here contains twice its normal amount of hemoglobin, yet there is actually a great lack of it in the blood as a whole.

DISEASES IN WHICH THE CHIEF CHANGES ARE IN THE RED CELLS.

Anemia.—Having studied the methods of examining the blood, we come next to the consideration of the diagnostic value of the conditions which we find in it. We find, first, that anemia, or blood deficiency, is represented by two conditions, in one of which the pallor and other symptoms are due to a diminution in the number of red blood corpuscles, while in the other there is a decrease in the amount of hemoglobin in each corpuscle. In regard to the white corpuscles we find even more valuable diagnostic data, since their variation in number, form, and character is marked in some diseases. Practically all conditions of the blood which are pathological represent diseases in organs connected with the blood directly or indirectly, and do not depend upon primary changes in this liquid, except in rare instances.

Secondary Anemia.—A patient's blood having been found lacking in the proper number of red blood corpuscles, the question naturally arises as to what conditions underlie this variation from the normal. The most common causes of this decrease are the *infectious diseases*, all of which result in producing a degree of anemia most marked during early convalescence; the history of such an attack should always be sought, and, if found, regarded as an important point for consideration in reaching a diagnosis. If there be no history of acute illness, the most natural condition to be thought of is that known as simple anemia, produced by no apparent disease of the organs of the body, but due to lack of good food, pure air, proper hygienic surroundings, and exercise. If this be excluded from the diagnosis, we must not forget that if food is taken and not absorbed properly the corpuscular richness of the blood is decreased, and, therefore, chronic indigestion may be the cause of the

difficulty. Again, profound anemia, as to the number of the red blood corpuscles, may be present and seem inexplicable, until it is discovered that the patient suffers from *bleeding hemorrhoids*, and the daily loss of blood, even though it be small, is sufficient to produce anemia. Similarly repeated attacks of *nose-bleed* or of *excessive menstruation* may so result. Naturally the physician will have excluded the possibility of the anemia being due to a single profuse hemorrhage from any cause before searching as far as this for a diagnosis.

In addition to these causes of anemia we find anemia due to a decrease in both the corpuscles and hemoglobin. A large proportion of these cases have already been mentioned when speaking of the anemias of convalescence and hemorrhage, but a far more important cause of this condition, yet one often overlooked, to the great regret of the physician in later years, is *tuberculosis*. Still other causes of such anemia are *sarcoma*, *renal disease*, and *cancer*, particularly gastric cancer, in which condition the blood may resemble that of pernicious anemia, and *gastric ulcer*, in which the loss of corpuscles may also be extraordinary, even if no hemorrhage occurs. *Chronic lead poisoning*, *arsenical poisoning*, and *uremic poisoning* may cause it, and it arises from the presence of numerous forms of intestinal parasites, such as *tape-worm* and *Ankylostomum duodenale* or *Uncinaria americana*, and last, but by no means least, from *malarial infection*, either as manifested by acute attacks, frequently repeated, or by slow poisoning, with the development of cachexia. (See farther on in this chapter.)

In the foregoing group of anemias, broadly classed as *secondary anemias* because the blood condition is clearly a sequence of some recognizable affection primary in other tissues, the blood picture is in general as follows: The hemoglobin is diminished, usually more than are the erythrocytes, hence the color index is lowered. The red cells are moderately reduced in numbers except in extreme cases. Poikilocytes and erythroblasts are present in severe cases, the latter being normoblasts or possibly microblasts in type. The leukocytes are commonly increased, a quite well-defined polynuclear leukocytosis being the rule. Myelocytes in very small numbers may be present.

Estimations of hemoglobin and red cells are of value in certain acute conditions. If a patient is brought to the physician in a state of advanced anemia, acute or chronic, and needs an operative procedure for relief, and the hemoglobin is below 35 per cent., it is well to remember the rule of Mikulicz, which is, "Do not operate."

Again, a great decrease in hemoglobin and red cells would aid in separating the collapse of concealed hemorrhage, as in a case of rupture of the tube in extra-uterine pregnancy, from other conditions of collapse not dependent on loss of blood.

Primary Anemias.—There still remains to be considered the primary anemias, of which one is called *pernicious*, in that it progressively gets worse until death occurs in the majority of cases. At present we do not understand the pathogenesis of this disease. It is characterized by marked pallor without loss of flesh, or, to speak more correctly, the subcutaneous tissues are added to rather than robbed of fat. There are gradually increasing dyspnea, failure of strength, cardiac palpitation, venous murmurs, some vertigo, and roaring in the ears. The blood shows a most extraordinary and continually diminishing number of red blood corpuscles, the count in one recorded case being only 143,000 to the cubic millimeter. In addition, the following points of great diagnostic importance are to be noted: First, the individual red corpuscles are richer than normal in hemoglobin, the color index often being above normal; second, many of them are larger than normal (megalocytes), producing a high volume index; third, the red corpuscles are deformed, some being ovoid, others irregular in shape from projections and constrictions on their surfaces (poikilocytes); fourth, there are present microcytes, or red blood cells which are smaller than normal; fifth, nucleated red blood cells, quite constantly large cells named megaloblasts, which have a pale staining nucleus. These last are often larger than the megalocytes, and are sometimes called the "corpuscles of Ehrlich," since he regards them as pathognomonic of pernicious anemia. Normoblasts also may be present. The white blood corpuscles are normal in number, or decreased, with a relative increase of lymphocytes and possibly a few myelocytes, although the great diminution in the red cells renders the proportion of white to red greater than normal. Cases of pernicious anemia in which there are no regenerative changes in the bone-marrow are known as aplastic anemia. This type is rapidly fatal, the chief differences in the blood picture from that described being a low color index and the absence of megaloblasts.

Another form of anemia, which at one time was considered secondary but now classed as primary, is called *chlorosis*. In this state the corpuscular diminution is so slight that it may be ignored; but the decrease in hemoglobin is extraordinary, sometimes falling as low as 30 per cent. of the normal or below it, the color index often being as low as 0.5. The red corpuscles are, however, in severe cases irregular in form—that is, there is more or less poikilocytosis, and many of them may be undersized. The white corpuscles remain normal in number or are slightly increased. Normoblasts are occasionally found in chlorosis of a severe type, but the larger varieties of nucleated erythrocytes are seldom seen. The diagnostic points of chlorosis, in addition to those just named, are the facts that the patient is generally a girl of from fourteen to twenty-five years, that the skin

is peculiar in its pallor (see chapter on the Skin), and that there is often little if any menstrual flow, which is usually only faintly pink in hue. Dyspnea, cardiac irregularity, constipation, and a wayward appetite are often present. Auscultation of the neck on the right side over the jugular vein will reveal a peculiar murmur called a "humming-top" murmur. Febrile movement of slight degree may also be present.

Polycythemia.—Polycythemia, or that state in which there at least appears to be an actual increase in the number of red cells to an abnormally high degree, is met with in instances in which there is concentration of the blood by diarrhea or excessive diuresis. An actual or real polycythemia is also met with in certain cases of congenital heart disease, in those who live at high altitudes, and in cases of tracheal stenosis. The most interesting of all the instances of polycythemia is that called chronic splenomegalic polycythemia which is characterized by an increase of the red cells to as high a number as 7,500,000 or more and progressive asthenia, high arterial tension, cyanosis, and enlargement of the spleen. The white cells are not increased.

DISEASES IN WHICH THE CHIEF CHANGES ARE IN THE WHITE CELLS.

There yet remains to be considered those conditions in which we find not only some of the states already described, but, in addition, marked alterations in the white blood corpuscles as well as the red, alterations of such moment that they become the salient features of the blood when it is examined. These are of great diagnostic importance.

The points of importance in examining blood in regard to the white corpuscles are their number and their peculiarities and kinds. Before doing so, however, it must be borne in mind that the proportion of white cells in different individuals varies very greatly in the limits of health, being increased by the taking of food (digestion leukocytosis), by pregnancy (gravid leukocytosis), by violent exercise, and by massage. Starvation correspondingly decreases the white cells. Digestion leukocytosis usually lasts for two to four hours after a meal. In these changes the relative percentage of all forms of white cells except the eosinophiles may be increased, although in most cases the polymorphonuclear cells are in excess. Again, much depends upon the general state of the patient's health and nutrition. A shrivelled, old woman may have as low a proportion as 40 per cent., and a stalwart, athletic man have 80 per cent. of polynuclear leukocytes, and be in perfect health. Usually, it may

be considered that about 60 per cent. of these polymorphonuclear cells is normal, but the individual characteristics must be taken into account in determining the diagnostic value of the percentage.

Leukemia.—When the disease *splenomedullary leukemia* is present, the total leukocyte count usually ranges between 200,000 and 500,000 per cubic millimeter, though very much higher figures have been attained. The typical change in the blood is the presence of the so-called myelocyte of Ehrlich, the abnormal leukocyte previously described. This often forms 20 per cent. or more of the leukocytes.

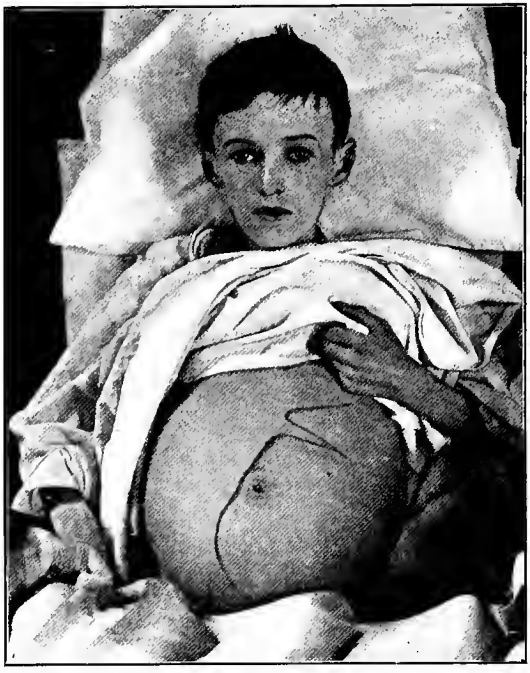


FIG. 162.—Splenomedullary leukemia, showing outline of enormous spleen in a boy of fourteen years. (Author's wards.)

Mast-cells are also a conspicuous feature. The ordinary large mononuclear cells are not greatly increased, and the polymorphous or polynuclear cells are decreased relatively though absolutely increased. Eosinophile cells are often both absolutely and relatively increased, though they may not be present. At one time they were supposed to be pathognomonic of this disease, but they are not so considered at this time.

The other changes found in leukemia are that the red blood cells are decreased in number, and a large number of nucleated forms, chiefly normoblasts, may be seen. The hemoglobin is also

decreased. The symptoms of this form of leukemia are pallor and puffiness of the face, dyspnea, and general feebleness, with great and gradual enlargement of the spleen and liver, and marked splenic tenderness. Auscultation over this organ may reveal a murmur and palpation a crepitus. Hemorrhage, generally from the nose, is common, and dyspnea and diarrhea are often present. Frequently retinitis develops, and slight fever may occur. This is by far the more common form of leukemia.

In advanced anemia, when the proportion of white to red cells is 1 to 10, and the lymphocytes (that is, the mononuclear, deeper-staining cells, with a rim of non-granular protoplasm), or the large mononuclear, are greatly increased in number, we suspect *lymphatic leukemia*. Myelocytes, so typical of the splenomedullary form, appear in this condition only in very small numbers if at all and splenic enlargement is absent, but in its place there is often enlargement of the superficial lymph nodes, though these never grow so large as in Hodgkin's disease, or pseudoleukemia. (See chapter on Fevers.) The acute form of this disease often presents the clinical features of a severe acute infection.

Pseudoleukemia, or Hodgkin's disease, is to be differentiated from true leukemia by the blood examination, it being recalled that in this malady there is usually only a slight decrease in the red cells and no other marked changes. Recently, however, Martin and Matthewson have emphasized the fact that in some cases of pseudoleukemia the lymphocytes have increased as greatly as in many instances of true lymphatic leukemia. As a general rule, however, the blood condition separates the affections. (For Banti's Disease, see page 310.)

Leukocytosis in Acute Diseases.—We study the white cells as to their number and character for other diagnostic purposes than those already mentioned, namely, for the discovery and differentiation of acute infectious diseases. Thus, in nearly all infections associated with acute inflammation we find an increase in the white cells, or *leukocytosis*. The particular white cell increased under these circumstances is the polymorphonuclear. Acute croupous pneumonia is particularly apt to produce this state unless infection is very virulent, when reaction fails to occur. By studying leukocytosis we can often decide as to the presence of deep-seated inflammations. Thus in doubtful cases of appendicitis a study of the leukocytes will aid the diagnosis and separate the condition from obscure non-inflammatory states. If an increase of the polymorphonuclear cells is present, we can exclude gallstone colic, renal colic, fecal colic, and ovarian neuralgia, unless there is associated with one of these affections an acute inflammation. The degree of leukocytosis in a case of appendicitis in the early stages is in direct ratio to the severity of the process. So, too, if leukocytosis gradually in-

creases, it is a sign of spreading inflammation. If it is absent after several days' illness with appendicular symptoms, its absence may indicate sepsis of a very grave form. It is important to recall in this connection the fact that a high leukocyte count is met with very early in intestinal obstruction. If it speedily falls, intestinal gangrene is to be suspected.

Often accompanying inflammatory leukocytosis is the condition known as iodophilia, or the presence in the polynuclear leukocytes or plasma of iodine-staining granules. This is specially prominent when suppurative products are being absorbed but is by no means limited to cases of pus formation.

Leukocytosis is usually absent in typhoid fever, malaria, severe septicemia, tuberculosis in all its forms, in influenza, and in measles, in many cases there being an actual diminution, *leukopenia*, in the number of white cells. Should some complicating focus of acute inflammation be developed, then leukocytosis appears. In suspected typhoid fever the absence of leukocytosis, with a diminution of the multinuclear cells and an increase in the mononuclear cells, is of importance in pointing to the presence of this malady. Should perforation of the intestine ensue, leukocytosis is apt to occur, unless a general peritonitis is produced, when the number of leukocytes in the peripheral circulation may be decreased by the outpouring of cells into the abdominal cavity.

An increase of the eosinophile cells, or *eosinophilia*, occurs in cases of skin disease and other conditions, but is of special diagnostic value in parasitic affections, in particular trichiniasis and ankylostomiasis.

A moderate leukocytosis occurs after childbirth, and is normal at this time, but does not persist during lactation.

Toward the end of prolonged fatal illness, or where the onset of death is gradual, an increase in white cells develops, called terminal leukocytosis.

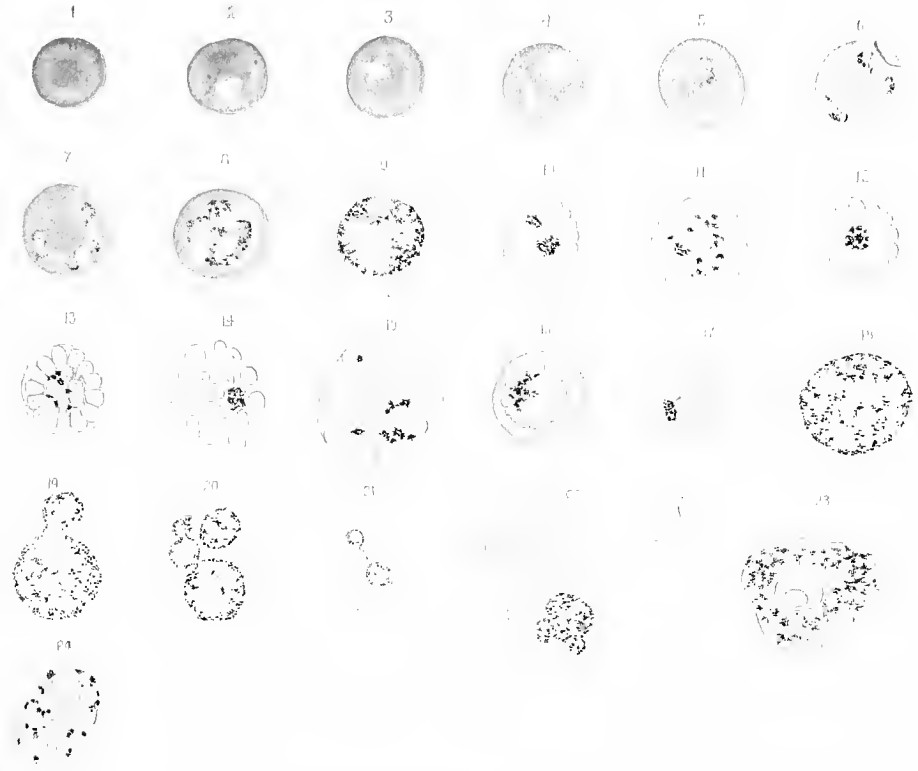
PARASITES OF THE BLOOD.

We still have for consideration the parasitic diseases of the blood. Of these the most important are due to the presence of the malarial germ of Laveran, or, as it is more properly called, the "hematozoon malaria" of Marchiafava and Celli, the Filaria sanguinis hominis, and the Trypanosoma.

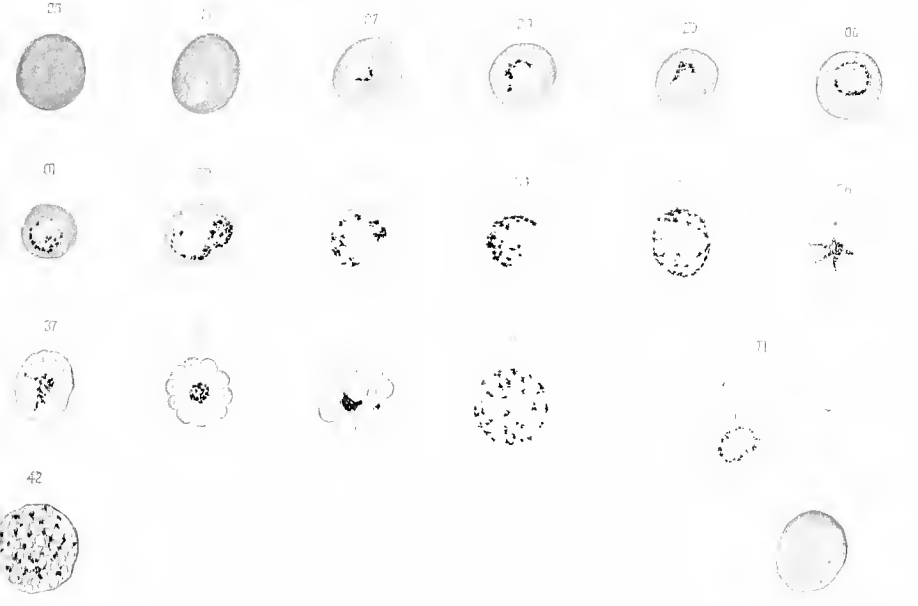
Malarial Organisms.—No more important addition to the study of disease from a diagnostic standpoint has been made than the discovery of the presence of a parasite in the blood of persons suffering from malarial fever, a parasite which is always present under these circumstances, and in all probability acts as the cause of all malarial

PLATE XIII

The Parasite of Tertian Fever



The Parasite of Quartan Fever



manifestations. These parasites are varieties of sporozoa, which develop inside the red blood corpuscles of the individual attacked.[1]

The parasite of malarial fever occurs in three forms, namely, as that of tertian fever, that of quartan fever, and as the parasite of the so-called estivo-autumnal fever.

The Tertian Parasite.—The intracorpuscular tertian parasite first appears as a small hyaline, colorless body, which occupies but a slight extent of the interior of the red blood corpuscles. (See Plate XIII, Figs. 2, 3 and 4.[2]) When quiet it is round, like the corpuscle in which it lies; but if the specimen examined be fresh, it may be seen to possess active ameboid movements, thereby changing its shape.

Soon this ameboid body grows in size and begins to develop within itself reddish-brown pigment granules. (See Plate XIII, Figs. 5, 6 and 7). These pigment granules are rapidly moving bodies, and, as they are often found in the projections of the parasite, it may look, until this fact is corrected by fine focusing, as if several parasites were in one corpuscle. As the pigment masses increase, the corpuscle which contains the parasite becomes more and more pale, and at the same time swells up or expands, and the ameboid movements of the parasite grow less and less, while the pigment tends to arrange itself toward the periphery. (See Plate XIII, Figs. 7 and 8.) Finally, only a shell of corpuscle is left (see Plate XIII, Fig. 9), the pigment after collecting in the centre becomes motionless, and then the parasite undergoes segmentation, there finally developing 10 to 20 segments, arranged about the central clump of pigment like a rosette. Each segment has a spot looking like a nucleus, and soon the spore-like bodies so formed break out of their host and attack new and previously healthy blood cells. Sometimes the parasite becomes so large that it entirely destroys the corpuscle and floats free in the blood, in which case the pigment granules quiet down and the mass becomes misshapen and apparently dead, breaking up into smaller masses, and giving rise to several small bodies, which, however, soon seem to lose life (see Plate XIII, Fig. 21), or it becomes filled with vacuoles (see Plate XIII, Figs. 23 and 24), or, finally, in drawn blood there springs from these extracellular bodies flagella or waving arms, extending from the margin of the parasite. (See Plate XIII, Fig. 33.) These flagella now and again break off and keep waving through the blood, looking like spirilla. The entire process just described consumes about forty-eight hours, and it is of interest to

[1] The chief American investigators into the life-history of the malarial parasites have been Osler, Councilman, Craig, and Thayer and Hewetson, from whose exhaustive and able monograph of "The Malarial Fevers of Baltimore" much of the information in the text of this book is derived.

[2] No 1 is a normal red corpuscle. Plates XIII and XIV are taken from Thayer and Hewetson's monograph.

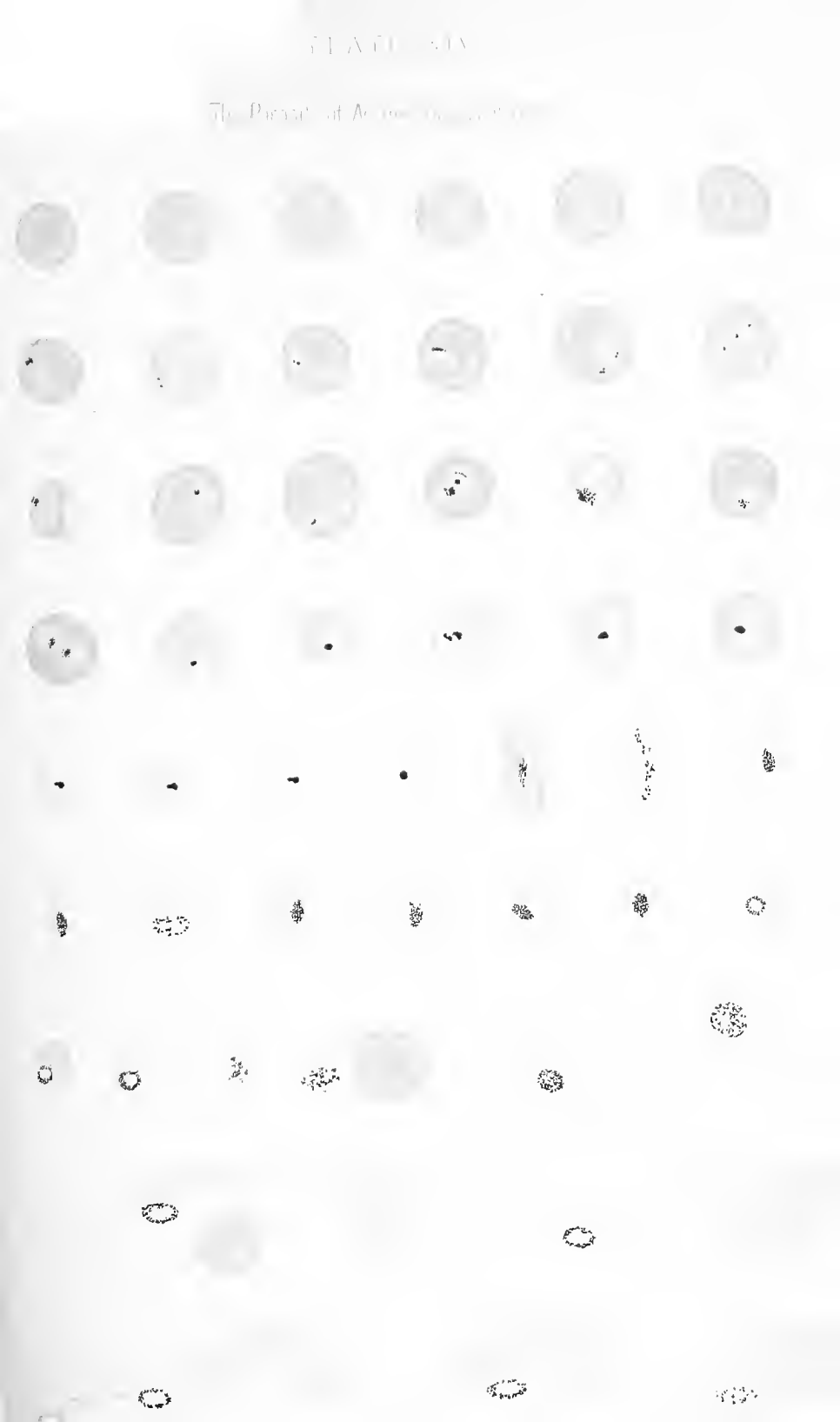
note that the acme of paroxysm or the disease occurs with the segmentation of the full-grown parasite, so that the presence of segmenting bodies indicates the near approach of an attack. If, on the other hand, there be a double tertian infection—that is, an attack daily—or a quotidian form, there are two sets of parasites, which reach their period of segmentation on alternate days, and so a daily attack is caused. In such blood during a paroxysm will be found two sets of parasites: one set segmenting or causing the paroxysm, and the other set half developed, which will produce the attack of the morrow.

The Quartan Parasite, or the one causing an attack every third day, in its earlier stages of development looks very much like that of the tertian form, for it occurs as a small hyaline ameboid body filling a fraction of the corpuscle. It soon, however, develops the following differences: First, it possesses a sharper outline; second, it is more refractive; third, the ameboid movements are slower (Plate XIII, Fig. 26); fourth, the pigment granules are coarser and darker (Plate XIII, Fig. 27), and, more important still, they lie very quietly around the edge of the parasite; fifth, the corpuscle acting as host does not increase in size, and finally disappear, as it does when affected by the tertian type, but grows smaller and darker, more refractive and metallic looking. (See Plate XIII, Figs. 28 to 34.) Reaching its complete development in about sixty-four to seventy-two hours, it appears as a small round body, taking up nearly all the space in the corpuscle in which it lives, or it appears free in the blood serum. (See Plate XIII, Fig. 35.) As the time for the paroxysm approaches, the pigment granules which have been scattered begin to collect at the centre (Plate XIII, Figs. 36 to 39) in a stellate form, and the protoplasm of the mass then divides by segmentation into from six to twelve small pear-like bodies, each of which has a refractive centre. These bodies become more and more separated from one another, and simultaneously we find new corpuscles infected by the original small round bodies which we first saw.

Sometimes these parasites expand and become very transparent, their pigment granules become very active, but finally become quiet, and the body of the parasite grows more and more indistinct. They become dead parasites. (See Plate XIII, Fig. 40.)

Again, the parasites may undergo a breaking up into smaller bodies, which are badly formed and indistinct; a degenerative form may also appear, and vacuoles may develop. (See Plate XIII, Fig. 42.) Finally, flagella may develop, as in the tertian organism (Plate XIII, Fig. 41), but they differ from the tertian form in being smaller and their granules coarser.

The Estivo-autumnal Parasite.—In the third form of infection (estivo-autumnal fever) we find at first the small hyaline bodies,



but they have a ringed appearance, and are sometimes very small. (See Plate XIV, Figs. 3 to 6.) Suddenly this body becomes larger and the ring is lost, the edge becoming wavy, and ameboid movements occur, the pseudopodia often joining to form a true ring. Pigment granules finally develop after a variable length of time, but they are few, rarely more than two in a parasite, are near the edge (Plate XIV, Figs. 7 to 12), and quite still. The corpuscles are not decolorized, but often are shrivelled and very brassy looking.

The peripheral circulation during the paroxysm of estivo-autumnal fever contains very few, if any, parasites, but blood drawn from the spleen may show intracorpuscular parasites, with blocks of pigment and some free parasites. As segmentation goes on the parasite may look like the tertian form, but it is far smaller. (See Plate XIV, Figs. 21 to 28.) After this parasite has been present for some days we find in the blood larger parasites, of an egg-shape or crescent-shape, the remains of the blood cell looking like a "small quarter of an apple glued to the side of the crescent," with central clumps of pigment granules. It is these bodies which are characteristic of estivo-autumnal fever. (See Plate XIV, Fig. 29.) Vacuolization and flagellation may develop in this form as in others, and the use of quinine in the first week may prevent the development of the crescents.

The following table separates each of these malarial forms from the others:

Tertian Parasite.	*Quartan Parasite.*	*Estivo-autumnal.*
Develops in 48 hours.	Develops in 72 hours.	Develops in 24 to 48 hours.
Pale and indistinct.	Sharply outlined and refractive.	Has a winged appearance.
Actively ameboid.	Slightly ameboid and later motionless.	Actively ameboid.
Pigment fine.	Pigment coarse.	Pigment granules are very few.
Pigment active in movement.	Pigment slow in movement.	Pigment granules quite still.
Pigment light.	Pigment dark.	
Corpuscle becomes colorless and swollen.	Corpuscle becomes brassy-looking and shrunken.	Corpuscle is shrivelled and very brassy, but not decolorized.
Full size of the corpuscle.	Smaller than the corpuscle.	Very much smaller than a corpuscle.
Degenerative forms twice as large as corpuscle.	Degenerative forms very much smaller than in tertian.	
Segments 16 to 20.	Segments 6 to 12.	The process of segmentation goes on in the internal organs, so segmenting form is not found in the peripheral blood.
Irregular segments often.	Beautiful rosettes.	Forms crescents.

Craig has directed special attention to a latent human cycle of the malarial parasite, the chief feature of which is intracorpuscular conjugation. Two of the merozoites or segments of the parasite enter a red cell, and, uniting, produce a resting stage resistant to

quinine and other injurious agents. In this stage it remains latent until favorable circumstances arise when the parasite segments and thus causes a recurrence of the disease. Craig regards this conjugation as the chief cause of the maintenance of malarial infection.

There is a great difference in the ease with which the various forms of the malarial parasite are found in the blood in the peripheral circulation. In infection with the tertian parasite, during the later stages of development the organism is much more frequently found in the blood of the spleen than in the peripheral circulation. In cases of quartan infection, the parasites appear in the peripheral blood as well as in the spleen, and in the internal organs in all stages of development; while in the estivo-autumnal form of the disease, only the young forms appear in the peripheral circulation, and when the parasites are arranged in groups no organisms may be found in the peripheral circulation during the paroxysm. This may also hold true in regard to the quartan and tertian parasites, and the entire cycle of development may take place in the internal organs, so that examination of the peripheral blood may reveal very little. In other words, while the discovery of the parasite in a suspected case confirms the diagnosis, the absence of the parasite does not absolutely negative the opinion that it is the cause of the malady. Of course, a very considerable number of slides should be carefully examined before it is stated that malarial parasites cannot be found. Given a case in which a considerable number of parasites are found in the peripheral circulation, we have a right to infer that the infection is severe. But this does not necessarily follow.

It has already been stated that the paroxysm of the malarial disease takes place at the time when the parasite is breaking up into segments. In other words, the attacks occur whenever the cycle of growth of a set of parasites is completed, which in tertian fever is every forty-eight hours and in quartan fever every seventy-two hours. If there be two sets of parasites in the blood, however, of the tertian type, the attacks may be daily, or quotidian, since each set matures on alternate days. This is often called double tertian. The tertian is the most common form of the disease in the United States. If there be a double quartan infection, the attacks come on two successive days, then a day of intermission ensues. If three sets of parasites of this type are present, the attacks may be daily for three days—triple quartan infection. (See chapter on Fever.)

The parasite of estivo-autumnal fever is often very irregular in its development, and is often the cause of the irregular malarial fever seen in the fall of the year. It yields less readily to quinine than the others, and is the usual cause of the pernicious types of the disease.

From what has been said it can readily be seen that the best time to

examine the blood for the parasite is a short time before the paroxysm is expected, as then the fully developed forms, easily recognized even by the novice, are present. Occasionally during or after the paroxysm is the better time. In some of the chronic or irregular infections, repeated examinations of the peripheral blood fail to reveal parasites which may be detected in blood obtained by splenic puncture.

Malarial infection differs from most infections, the symptoms of which resemble it, in that there is no increase in the leukocytes, whereas in sepsis a great increase is usually present. In fact, there is often a distinct decrease in the number of leukocytes with a relative increase of the large mononuclears. This gives us an important aid in differential diagnosis. (See chapter on Fever.) When the malarial organisms cannot be found the presence of leukocytes bearing pigment granules may indicate the breaking down of the red cells by the parasite, and so point to the probable presence of malaria.

Changes in the red cells and hemoglobin may be pronounced. In severe acute malarial infection, the red cells may within three or four weeks fall to 1,000,000 or less, the hemoglobin usually to a greater degree. In some cases the extensive hemolysis is evidenced by pronounced hemoglobinuria. In chronic cases anemia is often profound.

Staining the Parasites.—In examining the blood for the estivo-autumnal parasite, it will often be found that the best results are obtained if the blood is stained. For while, as a rule, the examination of a fresh specimen, prepared in the manner already described, is the best method of examining the blood for the malarial parasite, it is a fact that it is difficult to discover the small hyaline forms in a fresh specimen. Under these circumstances Thayer recommends Nocht's modification of Romanowsky's stain. This method is as follows:

Polychrome methylene blue is carefully neutralized. This is done by first adding dilute acetic acid until the solution is acid. When litmus paper is dipped into the solution, it is colored by the methylene blue, but on the margin of the moist portion the acidity causes a red line to appear. The solution is now brought back to the neutral point by the addition of more polychrome methylene blue until the red line fails to appear on the blue litmus paper. The polychrome methylene blue may be obtained already prepared It can, however, be readily made by heating for several hours, on a water-bath, a solution consisting of:

Methylene blue	1 part.
Caustic soda	1 part.
Water (distilled)	100 parts.

After cooling, the solution is to be filtered.

To this polychrome methylene blue an equal quantity of distilled water is added, and then a saturated solution of ordinary methylene blue until the red color is completely lost, the solution appearing simply blue. For this purpose, about 1 part of the saturated aqueous solution to 10 parts of the diluted polychrome solution will be used.

A solution of eosin is now prepared, according to Nocht, by adding 3 or 4 drops of a 1 per cent. aqueous solution of eosin to 1 or 2 c.c. of water. This is practically a 0.2 per cent. solution; it can be made up in quantity. To 3 or 4 c.c. of the eosin solution, Nocht adds the methylene-blue solution until the red color disappears. At this point a fine precipitate is thrown down, and a scum begins to form on the surface.

Thayer also advises that the eosin and methylene blue be mixed immediately before being used, and the mixture used only once, since when it is kept a day or two, though it gives the chromatin stain, it is but feebly. The best method of fixing the blood smears for this stain is to treat them with 0.25 per cent. solution of formalin in 95 per cent. alcohol for one or two minutes. When this stain is used, the hyaline forms of the parasites appear as delicate blue rings, the central part being occupied by a large colorless nucleus, at one side of which is a small spot of chromatin substance, which stains a deep carmine violet.

Any of the polychrome methylene blue-eosin combinations mentioned for general blood work answers well as a routine stain for the malarial parasite, though by some of them the chromatin is not sharply differentiated. For this the method of Giemsa is probably the best. Whatever method be employed, the stain is applied to thin films of blood prepared as described for the study of the corpuscles of the blood.

The Development in the Mosquito.—The bodies which are crescentic and those which are ovoid are not capable of sporulation, and are called gametocytes. While they are incapable of development in the human being, in the definitive host, the mosquito, or even on the slide of the microscope, the microgametocytes or male elements give off a number of long motile flagella (microgametes), which, breaking loose, penetrate and fecundate the female forms, or the macrogametes. This fecundated female form now enters the wall of the stomach of the mosquito, where it begins development within forty-eight hours; there then develops in the stomach wall of the mosquito small, round, refractive bodies of granular appearance, and which contain pigment granules. Five days later these bodies develop striæ, due to the development of sporoblasts, and finally the original organism or mother oöcyst bursts, and sets free a multitude of small sporoblasts, which find their way into the venenosalivary glands of the mosquito, and from there pass into the body of the bitten patient, where they enter red cells as

young parasites. The shapes other than those which are crescentic or ovoid have an asexual development and infect new blood cells without having to pass into the body of the mosquito. The *Anopheles* is the only mosquito which is known to act as a host in the way described. It occurs in several forms in the United States, the most common being the *Anopheles quadrimaculatus*, which is identical with the *Anopheles claviger* or *maculipennis* of Europe.

This mosquito is to be separated from the other mosquitoes by the following points: The wings are mottled, the posterior pair of legs is nearly parallel to the wall on which the insect rests, although they may be lifted over the back, in the same manner as does the *Culex*. The body, instead of being parallel with the resting surface, is at an angle of 45 degrees, whereas, the body of the non-malarial mosquito rests parallel with this surface (Fig. 163).

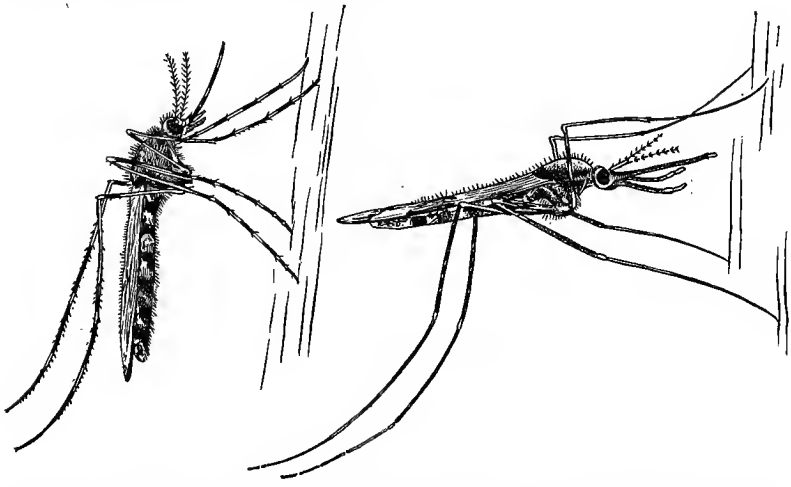


FIG. 163.—The picture to the left represents the Culex or non-malaria-bearing mosquito at rest. The figure to the right shows the resting attitude of the Anopheles quadrimaculatus. (Modified from drawing in the British Medical Journal.)

Filaria.—The *Filaria sanguinis hominis* appears in the blood in its embryonal form, and is found fully developed only in the lymphatics. It occurs in three forms. These forms are: (1) *Filaria diurna;* (2) *Filaria nocturna;* (3) *Filaria perstans.* These names are indicative of the habits of the animal, the *filaria diurna* being found in the superficial vessels solely or chiefly during the day; the *Filaria nocturna* solely or chiefly during the night; while the *Filaria perstans* is constantly present in the capillaries of the integument. The *Filaria diurna* and *Filaria perstans* are confined, thus far, to the west coast of Africa and adjoining districts, while the *Filaria nocturna*

is pandemic in the tropics and endemic in certain sections of the United States. The adults of *Filaria nocturna* have been frequently found; that of *Filaria perstans* never, so far as Henry has been able to ascertain. Manson has also described another parasite, the *Filaria demarquai*, which is less than half the size of the *Filaria nocturna*, and another form which he calls the *Filaria ozzardi*. In the opinion

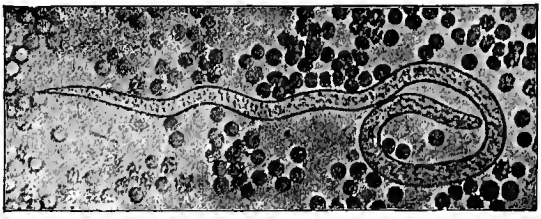


FIG. 164.—Filaria alive in the blood. Instantaneous photomicrograph. Four hundred diameters' magnification. Four millimeters Zeiss apochromatic. (Henry's case.)

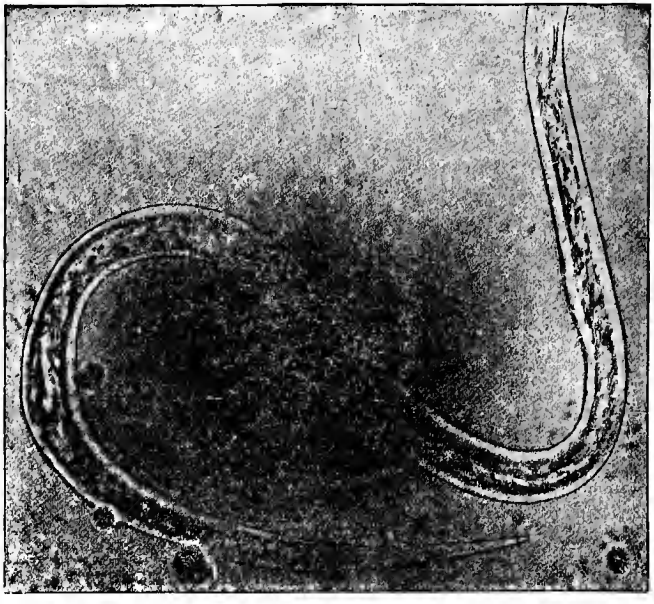


FIG. 165.—Filaria in the blood. Eight hundred diameters. (Henry's case.)

of Manson, the *Filaria loa*, found chiefly in the eye of the negro of Old Calabar, is probably the adult form of the *Filaria diurna*. If it is not, he argues, then there must be another blood worm yet to be discovered, for the embryos of the *loa* must escape from the body of their host through the medium of the circulation.

The second variety, or *nocturna*, is the one ordinarily seen in blood

obtained from the peripheral circulation during sleep or at night (Figs. 164 and 165). The adult male filaria measures 83 millimeters long by 0.407 millimeter broad, and the tail is twisted into a spiral form. The female measures 155 millimeters long by 0.715 millimeter wide, and the vulva is 2.56 micromillimeters from the anterior extremity. The adult parasite is found only in the lymph vessels. The embryo, which is found in the circulating blood, measures 270 to 340 micromillimeters long by 7 to 11 micromillimeters wide, and has a pointed tail. This embryo is in an almost imperceptible shell, which does not impede its movements, and as it is about the diameter of a red blood corpuscle it passes through the capillaries in extraordinary numbers. Its active movements and typical appearance render it readily seen in the blood. The discovery of this parasite in the blood renders a diagnosis certain, and it should always be sought for if chyluria or elephantiasis is present. If the patient remains awake at night and sleeps during the daytime the organism will be found in the blood during the sleeping period.

The *Filaria diurna* is found in the blood during waking hours. The embryos of the *Filaria perstans* are the only form of this parasite known.

Trypanosoma.—This parasite, like the filaria, does not enter the blood corpuscles. It is a flagellate, in length two or three times, in width one-third, the diameter of a red blood cell. It possesses an active, screw-like motion imparted by an undulating membrane continuous with one border of the parasite and a single flagellum projecting anteriorly. It can be readily recognized in fresh blood by the aid of low powers of the microscope and in films is well shown by polychrome methylene blue-eosin stains. Various species of this parasite are found in the blood of many small animals and also in horses and cattle. The *Trypanosoma gambiense* is pathogenic for man and infests great numbers of the natives in some of the West African States. A number of Europeans have also contracted the disease. The parasite is transmitted by a fly, the *Glossina palpalis*.

Clinically, the infection is chronic in type, first characterized by circulatory and respiratory disturbances, irregular fever, multiple areas of edema and erythema, enlargement of the spleen and lymph glands, emaciation, and progressive loss of strength. During this period the parasite is found in the blood, usually in small numbers, and in the fluid from the affected lymph glands. Later, the parasite enters the cerebrospinal fluid, giving rise to the well-known "sleeping sickness," which is uniformly fatal. The trypanosoma may be demonstrated in the sediment obtained by centrifugalizing spinal fluid obtained by lumbar puncture. The blood picture in this infection is that of a moderate anemia with normal or reduced

number of leukocytes, showing a relative increase of the large mononuclear forms.

Widal's Test for Typhoid or Enteric Fever depends upon the fact that the blood serum of a patient suffering from typhoid fever exercises an agglutinative effect upon the typhoid bacillus. The microscopic method is as follows: The lobe of the patient's ear having been pricked, the drop of blood is placed on a clean glass slide and allowed to dry. A loop of twenty-four-hour bouillon culture inoculated from a twenty-day agar growth of the typhoid bacillus is now placed on an absolutely clean cover-glass, and to this is added a large loopful of a watery solution of the dried blood. From the mixture of blood and typhoid bouillon a "hanging-drop" preparation is made, and examined with $\frac{1}{6}$ or $\frac{1}{8}$ dry objective, when it will be noticed, if the patient is suffering from typhoid fever, that the typhoid bacilli rapidly form clumps and become non-motile. If the patient has not typhoid fever, this clumping and entanglement of the bacilli with arrest of their movements does not take place, unless he has had the disease within several months, when the reaction may occur without signifying the onset of a new attack. Partial clumping is not to be considered indicative of a positive test. By this method accurate dilution cannot be made, hence if uniformity is to be secured the fresh blood should be at once diluted with distilled water, best in a hematocytometer pipette, 1 to 20, as a dilution of 1 to 40 with the bouillon culture is commonly employed. With this dilution, if the reaction be not completed or well advanced in thirty minutes, the test should be considered negative. Fairly satisfactory macroscopic methods for performing this test have also been devised.

This test has now been placed within the reach of practitioners who have not laboratory facilities by an apparatus marketed byParke, Davis & Co. By its use the necessity of the microscope and the fresh live culture of typhoid bacilli are obviated. A bottle of sterile permanent suspension of typhoid bacilli is provided together with four test tubes, a lancet and a tube for collecting blood. With this is one vial for diluting the blood serum, one small pipette for distributing the diluted serum, and one large pipette with two graduations (each corresponding to 10 drops of the size delivered by the small pipette) for filling the tubes with suspension. The three tubes labelled 50, 100, and 200 are to be used for the test. The fourth tube is employed as a control tube and no serum is added to it.

The blood is allowed to flow into the blood tube until the bottom is covered with a layer one-eighth to one-quarter inches thick. The blood will flow much more rapidly if the lobe of the ear is squeezed intermittently between the thumb and index finger.

Cork the tube and replace in an upright position.

In a short time (an hour) the serum will have separated, or may be readily made to do so by carefully loosening the edges of the clot with the lancet.

After the serum has separated, insert the pipette into the blood tube, the point resting in the lateral depression, and incline both slightly, when the serum will readily enter the pipette.

Add 1 drop of serum to 10 drops of clear water in the diluting tube, and shake well. If the diluted serum is cloudy, let it clear by standing a few minutes before distributing to the tubes of suspension.

By means of the large pipette put 20 drops (two graduations) of the suspension of typhoid bacilli in each of the four test tubes.

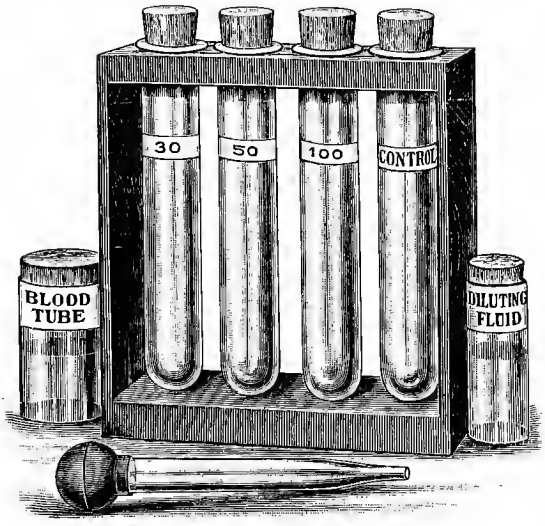


FIG. 166

Add the serum dilution to the typhoid suspension in the following amounts: Four drops added to the tube marked 50 give a dilution of 1 to 50; 2 drops added to the tube marked 100 give a dilution of 1 to 100; 1 drop added to the tube marked 200 gives a dilution of 1 to 200.

No serum should be added to the control tube.

After adding the serum dilution, cork the tubes and shake well. Put away in a warm place.

Examine the tubes at the end of one and four hours, and again on the following day. The rapidity of the reaction depends both upon the agglutinating power of the blood serum and the temperature at

which the tubes are kept. The reaction may be seen with the greatest distinctness when one stands near the middle of the room facing a window. The tubes should be held on a level with the eye and inclined slightly away from the observer.

When the reaction is positive, floccules appear in one or more of the tubes, depending upon the agglutinating power of the serum tested. These flakes are small at first and disseminated through the fluid. They gradually increase in size and settle to the bottom of the tube.

In a complete reaction the supernatant fluid is perfectly clear.

In the positive but incomplete reaction, floccules are seen in the still cloudy fluid.

In a negative reaction, the fluid in the tubes remains uniformly clouded, as in the control.

All apparatus and corks should be thoroughly washed before using a second time.

The Widal test varies in the constancy of its development with the stage of the disease, being most marked in the third and fourth weeks. In a large series of cases Park found it present in 20 per cent. of cases in the first week, 60 per cent. in the second week, 80 per cent. in the third week, 90 per cent. in the fourth week, and in 75 per cent. at the end of two months. The chief difficulty with this test, therefore, is that it fails to appear at a time when the diagnosis is often most difficult, namely, in the first few days of the illness.

Sugar in the Blood.—The blood in persons suffering from diabetes contains sugar in excess, and R. T. Williamson has suggested its discovery by the following process. The normal blood will not give this reaction. A small, narrow test-tube is well cleaned, and at the bottom of the tube are placed 40 c.mm. of water. To measure this the capillary tube of a Gowers hemoglobinometer is used, which is graduated for 20 c.mm. The tip of one of the patient's fingers is cleaned and dried, then pricked, and when a large drop of blood has escaped it is sucked up into the small capillary hemoglobinometer tube. 20 c.mm. of blood are taken up from the finger. The blood is then blown gently into the water at the bottom of the small test tube. If it should adhere to the side of the tube, it must be carefully shaken to the bottom. Then 1 c.c. of a 1 in 6000 watery solution of methylene blue is added. Finally, there is added to the mixture 40 c.mm. of liquor potassa. The contents of the tube are then well mixed by shaking. As a control experiment a second test tube of similar size is taken, and into this is placed the same quantity of non-diabetic blood, with the same proportion of water, methylene blue, and liquor potassa.

The fluid in each tube has a fairly deep-blue color. Both tubes are then placed in a beaker or capsule containing water. This is heated over a spirit lamp until the water boils; it is allowed to continue boiling for about four minutes. By the end of this time the fluid in the tube containing the diabetic blood has changed its color from fairly deep blue to a dirty pale yellow (almost the color of normal urine), while the fluid in the tube containing the non-diabetic blood remains blue, or occasionally becomes bluish green, sometimes pale violet, but is never decolorized—that is, it never entirely loses its blue color. The tubes should be kept quite still while in the water-bath, as by shaking the decolorized methylene blue is oxidized by the oxygen of the atmosphere, and a blue tint may then return to the fluid. This is the reason why it is necessary to use a water-bath, since if the test tubes be heated directly over the spirit lamp it is difficult to avoid shaking the fluid.

Opsonic Index.—A method of testing the blood for the purpose of determining its ability to aid the body in combating infection has been devised by Wright, and depends upon the presence of something in the blood serum which so influences invading organisms that the leukocytes can destroy them by phagocytosis. This something, which has not yet been isolated, is given the name of "opsonin;" the word "opsonin" being derived from the Greek, meaning "To cater for," or "to prepare food." In other words, an opsonin affects the microörganism in such a way that the leukocytes can devour them. In those individuals who are susceptible to infection, a condition has arisen which has impaired or destroyed the opsonic activity of the serum. The test to determine the resistance of an individual to bacterial invasion consists in comparing the activity of the leukocytes of the patient in devouring the bacteria, when these bacteria are exposed to his own serum, with the activity of the leukocytes when the microörganism is exposed to the influence of the serum of a normal individual. If each polymorphonuclear leukocyte exposed to the normal serum contains on the average 2 bacteria, the phagocytic index is said to be 2. If each leukocyte of the patient contains 1 bacterium, his phagocytic index is said to be 1. The phagocytic index of the blood to be tested divided by the phagocytic index of the normal serum is called the "opsonic index." This test has not been studied long enough to determine its value, but promises much both for diagnosis and therapeutics for two reasons. It is, however, valueless except in the hands of the most expert, and even then may give variable results. In diagnosis it aids us in determining not only the vital resistance of a patient, but, in addition, the exact nature of his infection, because if he has any vital resistance, in the course of time the opsonic index in his serum for a particular infecting germ

rises to meet the needs of the individual in resisting the invading microörganisms. So, too, it is possible by the introduction into the body of a carefully prepared product, or vaccine, of a definite microörganism to so stimulate the development of the opsonin as to enable him to still better combat infection.

In resorting to opsonic therapy it is important that the germ causing the particular condition to be treated be isolated and that the vaccines be prepared from cultures so obtained.

CHAPTER XIII.

THE EYE.

The general diagnostic indications afforded by the eye—Diplopia and disorder of the external ocular muscles—Strabismus and squint—Disorder of the internal ocular muscles—The pupil—Hemianopsia—The visual fields—Color vision—The optic nerve and its lesions—Retinitis—Amblyopia and blindness.

THE eye affords more information for diagnostic purposes concerning the condition of other organs of the body than any single part which can be examined. We gather from it not only a clear idea as to its own state, and the state of the nervous centres more or less intimately connected with the government of its movements and its special functions, but in addition we often gain positive information as to the condition of organs more remotely situated, as, for example, the kidneys. The very fact that so many different tissues are found in this organ renders it susceptible to the many diseases affecting similar tissues elsewhere in the body. The parts of the eye which give us the greatest amount of knowledge about changes in other tissues are the optic nerve and retina and its vessels and the ocular muscles. The crystalline lens, the conjunctiva, and the cornea often give additional evidence indicating the general systemic condition. Cataract should make the physician suspect diabetes, even if it appear in persons advanced in years. The eyelids, if puffy in appearance, may indicate renal disease, cardiac lesions, or the overuse of arsenic. (See chapter on the Face.) An examination of the inner side of the lids may reveal a pallor due to anemia. Slight conjunctival hemorrhage may result from violent coughing, and when it is recurrent it should arouse the suspicion of renal disease with secondary vascular troubles. In old persons such a hemorrhage, if not due to injury, may indicate degenerative vascular changes.

Prominence of the eyeball, or exophthalmos, is seen as an almost constant symptom of true goitre, which for this reason is called *exophthalmic goitre*. (See Fig. 8.) Associated with the bulging eyeball we find more or less enlargement of the thyroid gland, an irritable heart, and a very rapid pulse, throbbing carotid arteries, marked general nervousness, often mental depression, and insomnia. In well-marked or advanced cases of exophthalmic goitre we often

have a condition in which the upper eyelid does not follow the eyeball in its downward movement. This is sometimes called "Graefe's symptom." Again, the lids may so imperfectly cover the eye that the sclera can be seen above and below the cornea, "Stellwag's symptom." Or, again, there is insufficiency of convergence, so that a near point cannot be seen with both eyes at once (Moebius' sign).

On examining the exterior of the eyeball we often notice a grayish ring along the junction of the cornea and sclera. It possesses, when a complete ring, but little significance, except age; but if it is the segment of a ring or in two segments, at the upper and lower margins of the cornea, it is a true *arcus senilis*, and is said to indicate in some cases fatty degeneration of the tissues of the body. The one is an annulus senilis, the other an arcus senilis, and the arcus is the change worthy of note, although many clinicians, including the author, deny that either has much significance.

An examination of the pupil may reveal that it is immobile from an old plastic iritis, due to syphilis or rheumatism, but it is not to be forgotten that this condition may arise from iritis due to purely local causes. A *widely dilated pupil* may indicate the use of some mydriatic or the ingestion of atropine. Such a pupil is also seen in fright, in some hysterical seizures, and in glaucoma and whenever the vision is lost, unless the pupil be contracted by disease of the iris. A *contracted pupil* indicates the use of a myotic or the existence of central nervous disease, such as ataxia, which causes the Argyll-Robertson pupil as well. Sometimes corneal inflammation by causing photophobia may cause excessive myosis. Pin-point pupils may also result from the use of opium or its alkaloids, and serve to differentiate the condition from true coma, in which the pupils are usually dilated. If, however, the coma be due to cerebral inflammation or meningitis, the pupils may be contracted; or if it be due to intracranial pressure, they are usually dilated. (See Paralysis of the Intraocular Muscles.)

In addition to these objective symptoms we have also a very important set of signs connected with the ocular muscles, external and internal, as manifested by the various forms of strabismus or changes in the pupil and in the accommodation of the eye, by the ptosis already discussed in the chapter on the Face, and in nystagmus and ocular spasm. (See later pages.) Beyond this, too, we have two other ocular symptoms subjective in nature, namely, diplopia, or double vision, and partial or complete blindness.

Diplopia depends upon the fact that in an eye in which the muscles are abnormal in their function the image which falls upon the fovea, or visual acuity spot of the retina, in the well eye fails to fall upon the same spot in the weak eye. To the well eye the object appears to be in the direction in which the eye is turned, whereas to the weak

eye it appears to be in another direction. As a result, the mind gets the impression of two objects instead of one. The impression made on the well side is the "true image," as it is called, and that in the diseased eye is called the "false image." Any cause which interferes with the fixation of each eye on the same point produces diplopia, and, as the eyes are normally directed to the object fixed by the ocular muscles, paralysis of any one of these muscles produces diplopia when the axis of one eye is deviated from the point of fixation, because the eye on one side is not properly moved by reason of the fact that one muscle has failed. Diplopia is ordinarily a constant sign of ocular muscular paralysis; but if only weakness or insufficiency of a muscle is present, diplopia may never be a symptom recognized by the patient. The forms of diplopia—that is, the position of the false images in respect to the true images—vary with the muscles affected, and will be studied (see below) when paralysis of the muscles is tested for and their diagnosis discussed. It only remains at this place, therefore, to point out the probable significance if a patient with diplopia presents himself to a physician.

Thus, a patient with diplopia may be suffering from a lesion in the cerebral cortex, such as hemorrhage, sclerosis, or softening; or from a lesion in the cranial nerve nuclei, in the pons or corpora quadrigemina, or in the fascicular fibers. Again, diplopia may arise from lesions at the base of the brain, as meningitis, tuberculous or syphilitic, or from injury to the nerves in the orbit or in their peripheral ending. As a result, we find diplopia as a symptom of any disease which may affect these parts, and it is quite a common symptom in locomotor ataxia, in Friedreich's ataxia, and in paretic dementia. Probably it is seen most commonly in ataxia, and with it, as the oculomotor nerve in its branch supplying the levator palpebræ is particularly apt to be paralyzed in this disease, we may find ptosis.

Diplopia is also found in cases of ptomaïn poisoning, and in poisoning by belladonna, spigelia, conium, and gelsemium, owing to their effects on the ocular nerves.

The differential diagnosis between the various lesions producing diplopia is to be made by the other symptoms and the history of the case.

Paralysis of Extra-ocular Muscles.—As something has already been said in the chapter on the Face and Head of the diagnostic import of paralysis of the ocular muscles in connection with the subject of ptosis, a further consideration of the abnormal changes in their functions will be discussed first in the present chapter.[1] Before doing so, however, it is necessary to describe the methods resorted to for the purpose of demonstrating or determining departures from

[1] In the preparation of this chapter free use has been made of the article of my friend, Dr. de Schweinitz, on "Diseases of the Cranial Nerves, in Dercum's "Nervous Diseases."

the normal in these muscles. In the first place, it must be clearly understood that the function of the extrinsic muscles of the eyeball is to direct the ball toward the object at which the patient desires to look, and also evenly balance one another to keep the eye steady in its axis. Thus, the external and internal rectus muscles maintain the horizontal equilibrium of the eyeball. If the internal rectus is completely paralyzed in one eye, we have developed a unilateral external squint, the eye looking toward the outer side of the orbit; and if the external rectus fails, the eyeball is turned toward the nose. If these muscles are affected in both eyes, we have a divergent squint in the first case and a convergent squint in the second. Not only do the muscles of each eyeball govern the eye movements of that side, but by the nervous centres governing the eye muscles the two sets of eye muscles are coördinated, so that they move as one organ in health.

Just here it is well for the reader to make a clear distinction between *concomitant* and *paralytic squint*, for they are two very different things in origin, symptoms, course, and prognosis. A concomitant squint is a wrong relation in the visual axes, so that they do not intersect in the point looked at; but there is no marked limitation of the movements of either eye in any direction. Be the direction of the eyes what it may, the squint remains practically unchanged. Further, if the fixing eye is covered, the other eye promptly fixes, and the covered eye deviates without the patient altering the position of the eye. (Jackson.) On the other hand, paralytic squint is the deviation which takes place when the attempt is made to turn the eyes in certain directions by means of the muscles which are paralyzed in whole or in part. When the attempt is made, the eye with the sound muscles turns as it should, while the eye with a paralyzed muscle hangs back, beginning to deviate as the eyes are turned, so that this muscle is required to perform its function, and deviates more as greater effort is required. The degree of squint and of separation of the double images it causes varies with the direction in which the eyes are turned, there being none at all in certain directions.

We examine the functional activity of the ocular muscles by the following measures:

The patient is told to look at the tip of a pencil or the tip of the finger of the physician, held about three feet from his face. This object is then gradually brought nearer and nearer to him, and the eyes of the patient necessarily converge more and more as it approaches his nose. Normally the eyes will be coördinately converged when the object is only three and a half inches from them; but if any weakness or insufficiency of one internus is present, the eye on that side will deviate or fail to converge before this point is reached.

Again, a fine point like a pin-point is held at about eight or ten

inches from the eyes and below the horizontal, and one eye is covered by a card or hand. If the eye which is separated from the object by the card deviates inward, it indicates insufficiency of the external rectus. If, on the other hand, it deviates outward, it shows insufficiency of the internal rectus. On sudden removal of the card the eye at once springs back into place for the purpose of fixing upon the object, and "in general terms each millimeter of movement deviating from the fixation point corresponds to what is called 2° of insufficiency, as measured by prisms." (Randall.) If the internus is insufficient, and the covered eye moves in to fix in several distinct impulses, each impulse should be multiplied into the foregoing result.

A very useful, and the simplest, apparatus for testing the functional balance of the ocular muscles is the rod test of Maddox.

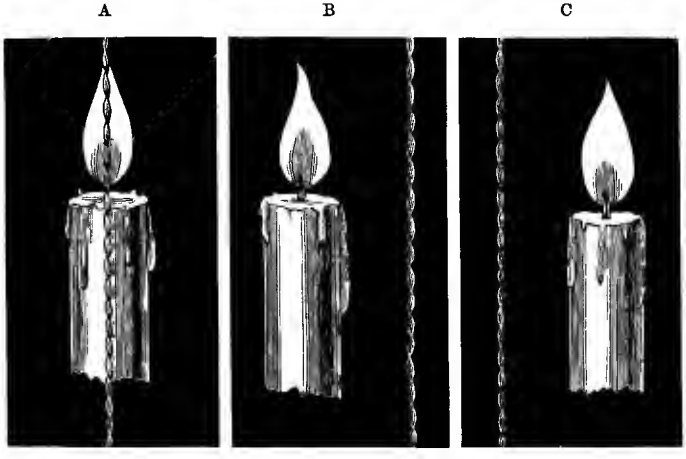


FIG. 167.—Maddox's rod test for horizontal deviation. The rod is before the right eye. A. The line passes through the flame—orthophoria. B. The line passes to the right of the flame—latent convergence, or esophoria. C. The line passes to the left of the flame—latent divergence, or exophoria. (de Schweinitz.)

A cell in which is mounted a transparent glass rod is placed in a trial frame, which is then placed in front of the eyes. If the horizontal deviation is to be determined the physician should "seat the patient at six meters from a small flame, and place the rod horizontally before one eye, a colored glass before the other. If the line passes (vertically) through the flame there is orthophoria (equipoise), as far as the horizontal movements of the eyes are concerned. Should the line lie to either side of the flame, as in most people it will, there is either latent convergence or latent divergence: the former if the line is the same side as the rod (homonymous diplopia), the latter if to the other side (crossed diplopia) (Fig. 167).

When the vertical deviation is to be estimated the rod is placed

vertically in the frame. If the patient states that the horizontal line of light passes directly through the flame, the vertical balance of the eyes is normal; if, on the other hand, the line is above the flame, there is a tendency to upward deviation of the naked eye; but if the line is below the flame, there is upward deviation of the eye covered by the rod test (Fig. 168). Testing of this kind refers to the insufficiencies and not to the palsies of the ocular muscles.

The importance of being able to demonstrate these minor failures in the ocular muscles by these means lies in the fact that in this manner headaches due to muscle eye-strain may be remedied by removing their cause by properly fitted glasses, or by gymnastic exercises with prisms, or in some cases by tenotomy.

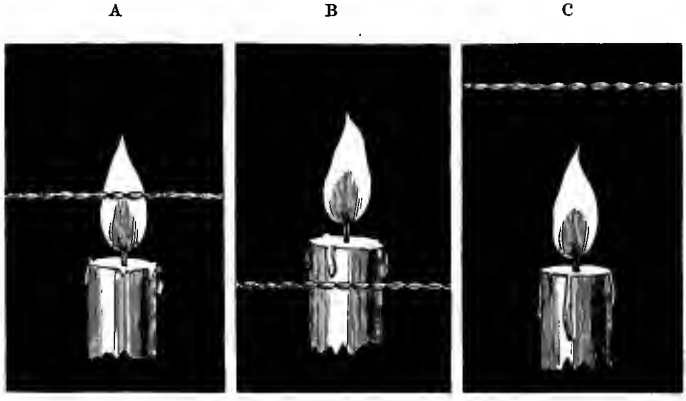


FIG. 168.—Maddox's rod test for vertical deviation. The rod is before the right eye. A. The line passes through the flame—orthophoria. B The line passes below the flame. The upper image belongs to the left eye—right hyperphoria. C. The line passes above the flame. The upper image belongs to the right eye—left hyperphoria. (de Schweinitz.)

Where there are marked *palsies* of the ocular muscles, there is usually some poison exercising its effects upon their nervous centres or the nerves themselves, or there is some central nervous lesion affecting the centres governing these muscles in the cortex, or there is a lesion in the nuclei or fasciculi, or, again, there may be lesions in the basal ganglia, or in the course of the fibers of the nerve between the nucleus and the eye, or in the orbit or nerve endings.

The signs of paralysis of the ocular muscles consist in the following symptoms: Diplopia, which is due to the failure of the images to fall on the corresponding points in each retina. This diplopia becomes more and more marked as the object moves toward the side on which the paralyzed muscle lies. Strabismus, which may or may not be constant, usually develops when the patient endeavors to turn his eyes in the direction of the paralyzed muscle. Vertigo, which

is due to the diplopia, or, if the well eye is closed, to an erroneous localization of the objects in the field of vision. Altered carriage of the head, due to the fact that the patient tries to turn his head in the direction in which he is least troubled by double images—that is, he obtains the natural fixation point of the weak eye, and then adjusts the well eye accordingly.

If the paralysis of the ocular muscle be complete, the squint and the loss of movement of the muscle which is paralyzed will usually enable the physician to find out the paralyzed muscle; but if there be only a partial paralysis or paresis of an ocular muscle, then squint is not necessarily present, and the diagnosis of the part affected must be made by a study of the double images. This is made by placing before the patient, at a distance of from three to five yards, a candle on the same level as his eyes. One eye is covered by a piece of red glass, so that the patient can readily distinguish between the images. The lighted candle is then moved from the middle of the patient to the right and left, and the relative positions of the red and white images are noted. Then the candle is moved up and down, and the results recorded. These operations having been recorded, it is to be remembered that diplopia is most marked and sometimes only appears when the patient turns his eyes in that direction which calls into play the affected muscles, no diplopia being present if other muscles are used. Again, the image which belongs to the affected eye is projected in the direction toward which the paralyzed muscle normally turns the eye, and, finally, the distance of the double image increases when the eyes are turned in the direction of the action of the paralyzed muscle, or, in other words, that image is false and belongs to the affected eye which in the region of diplopia moves faster than the moving test object—that is, the candle flame.

If there is divergent squint with failure of movement in all directions, except outward and slightly downward, and there are ptosis, moderate mydriasis, and paralysis of accommodation, there are oculomotor paralysis and crossed diplopia.

The following table of Hotz (*International Clinics*, vol. iii, fourth series) summarizes the facts as to the diagnosis of the conditions producing strabismus:

I. Lateral diplopia indicates paralysis of an internal or external rectus.

1. Homonymous diplopia indicates paralysis of an external rectus.

(*a*) Images separating to the right indicate paralysis of the externus of the right eye.

(*b*) Images separating to the left indicate paralysis of the externus of the left eye.

2. Crossed images indicate paralysis of an internus.

(*a*) Images separating to the right indicate paralysis of the internus of the left eye.

(*b*) Images separating to the left indicate paralysis of the internus of the right eye.

II. Vertical diplopia in the upper field indicates paralysis of the superior rectus or inferior oblique.

1. Homonymous images indicate paralysis of the inferior oblique.

(*a*) Image of right eye higher means paralysis of the inferior oblique of the right eye.

(*b*) Image of right eye lower means paralysis of the inferior oblique of the left eye.

2. Crossed images indicate paralysis of the superior rectus.

(*a*) Image of right eye higher means paralysis of the superior rectus of the right eye.

(*b*) Image of right eye lower means paralysis of the superior rectus of the left eye.

III. Vertical diplopia in the lower field indicates paralysis of the inferior rectus or superior oblique.

1. Homonymous images indicate paralysis of the superior oblique.

(*a*) Image of the right eye higher means paralysis of the superior oblique of the left eye.

2. Crossed images indicate paralysis of the inferior rectus.

(*a*) Image of the right eye lower means paralysis of the inferior rectus of the right eye.

(*b*) Image of the right eye higher means paralysis of the inferior rectus of the left eye.

It is exceedingly difficult, however, always to localize exactly the affected muscle, a difficulty which is much increased when more than one is paretic, the paresis being of different degrees.

Paralysis of the ocular muscles may be due to a lesion in one of several places. Thus it may arise from hemorrhage, sclerosis, and softening of the cerebral cortex, in which case the other symptoms of lesions in those parts will be present as in apoplexy, disseminated sclerosis, or meningeal disease. Or it may depend upon lesions in the fasciculi between the cortex and the nuclear origin of the nerves, as in the crus. This is rare. Or, again, it may be due to lesions in the nuclei. If this be the case, we have developed ophthalmoplegia,[1] or paralysis of all the ocular muscles supplied by the third, fourth, and sixth nerves. This nuclear paralysis is divisible into two classes, the acute and chronic. Sometimes it is called acute and chronic nuclear palsy. The acute form is sudden in its onset, all the ocular muscles losing power. With the onset of the attack there may

[1] Ophthalmoplegia is here applied in its strict sense. The word is often used to signify loss of power in individual eye muscles; and while its use in both ways is correct, it is better to confine its usage to nuclear and complete lesions.

be fever, vomiting, and even convulsions. Such an attack results from minute hemorrhages among the nuclei, or from an acute hemorrhagic polio-encephalitis in the fourth ventricle, arising from syphilis, tuberculosis, ptomain poisoning, alcoholic, and sulphuric acid poisoning. Such cases are usually rapidly fatal. A less fatal form follows injuries, and the effects of nicotine, lead, carbonic oxide, or such diseases as diabetes, syphilis, and epidemic influenza. Sometimes acute ophthalmoplegia comes on with acute poliomyelitis or acute bulbar paralysis.

Chronic nuclear paralysis is gradual in its onset, muscle after muscle failing, and even ptosis coming on. Sometimes after a certain degree of paralysis is reached the disease comes to a standstill. The trouble may be unilateral or bilateral, and is often unsymmetrical, and it occurs after acute ophthalmoplegia, as a congenital defect producing bilateral ptosis (see chapter on the Face), as an acquired disease in childhood and adult life, and in conjunction with locomotor ataxia, paretic dementia, disseminated sclerosis, progressive muscular atrophy, chronic bulbar paralysis, and in connection with paralysis of the frontalis and orbicularis palpebrarum, which are innervated by the facial nerve. The cause may be tuberculosis or syphilis, but in some cases no cause can be found.

If the cause of the paralysis of one or two muscles be basilar lesions, these may arise from hemorrhage, pachymeningitis, meningitis, both simple and tuberculous, chiefly the latter; purulent meningitis, abscess as the result of middle-ear disease, and anemia. It may also arise as the result of obliterating arteritis, particularly in syphilitics, and from tumors. The frequency with which palsy of the ocular muscles depends on syphilis is noteworthy. Alexander asserts that 75 per cent. of all cases of unilateral paralysis of the third nerve are due to syphilis, and Uhthoff states that only about 15 per cent. of the cases of cerebral syphilis escape some ocular palsy. In children sudden convergent strabismus and diplopia are often among the earliest symptoms of tuberculous meningitis at the base of the brain due to pressure on the roots of the sixth nerve.

If the cause be in the nerve trunks themselves, the lesion will probably be cellulitis, tenonitis, hemorrhages in the orbit, or fractures of the orbit; or, again, there may be disease of the frontal sinus. If the lesion is distinctly peripheral, it may be due to rheumatism (when the external rectus is commonly affected), neurasthenia, or it may arise from lithemia and gout. Further, such lesions may be due to influenza, diabetes, diphtheria, lead, and alcohol, or any one of the drugs which paralyze the ocular nerves.

So much for general statements as to the common and possible sites of the lesions producing paralysis of the ocular muscles. We can now go farther than this, and locate the lesion more accurately

from the knowledge we have gained as to particular muscles affected and the other symptoms presented by the case.

Let us suppose that a patient suffering from paralytic *internal squint* or a diplopia which indicates paralysis of the external rectus, presents himself to the physician, what diagnostic significance has this symptom?[1]

In the first place, it is to be remembered that the external rectus receives its nerve supply from the abducens, or sixth nerve (Fig. 169), which arises from the pyramidal body close to the pons. (See Plate II, and Fig. 6.) Its deep origin is a nucleus under the floor of the fourth ventricle. The nerve pierces the dura mater on the basilar surface of the sphenoid bone (see Plate XV), passes through the clinoid process, enters the cavernous sinus, and, finally, enters the orbit through the sphenoidal fissure between the heads of the external

FIG. 169.—Paralysis of left abducens in a case of hemiplegia of syphilitic origin. (Dercum.)

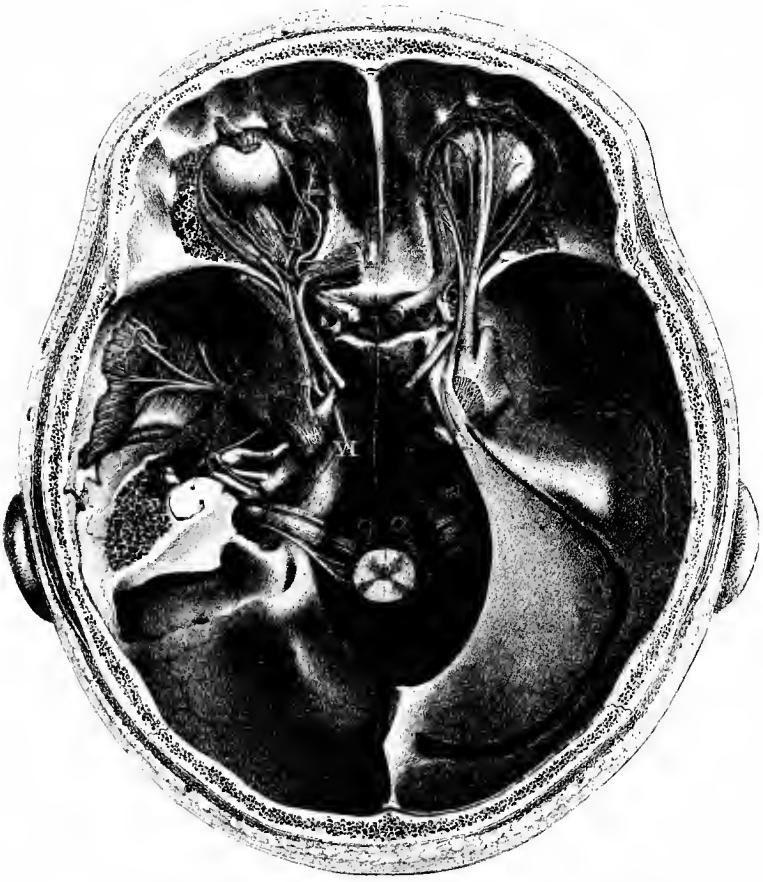
rectus. If this form of squint is associated with hemiplegia of the opposite side of the body, the lesion is in the pons on the same side as the affected eye and the opposite from the hemiplegia, because the eye fibers have crossed higher up, but the motor tracts for the limbs cross lower down.

On the other hand, if there is monoplegia and abducens palsy (internal squint) on the same side of the body, the lesion is in the point of origin of the abducens and arm centre in the cortex, or, in other words, the lesion has taken place above the point where the tracts cross. Such a paralysis is, therefore, cortical.

If, again, there is *complete unilateral paralysis of the abducens* (*internal squint*) with loss of the associated action of the internus, the lesion is in the nuclei under the floor of the fourth ventricle, because the nuclei of the third and sixth cranial nerves are closely

[1] This refers to paralytic and not to concomitant squint.

PLATE XV.



Showing the Sixth or Abducens Passing through the Clinoid Process.
(Modified from Arnold.)

connected, so that a lesion involving the sixth nucleus weakens the nucleus of the third nerve. Complete paralysis of the externus may, therefore, be due to a nuclear lesion; for if the lesion were above the nucleus, this nucleus might obtain collateral impulses, and, therefore, the paralysis would be only partial. It may also be due to a peripheral lesion and sometimes an inflammatory process pressing upon the basilar surface of the sphenoid, and thereby involving the nerve, may cause a similar effect. Loss of power of the external rectus may also arise from neurasthenia, gout, and rheumatism, and in tuberculous or syphilitic meningitis at the base, as already stated. It also comes on in some cases of diabetes, la grippe, and in chronic poisoning by lead and alcohol, or the acute poisoning of gelsemium, ptomain poisoning, conium and spigelia poisoning.

Again, let us suppose that the internal rectus is paralyzed, causing *external squint.* We remember that it is supplied by the oculomotor nerve, which springs from the inner side of the crus close to the upper border of the pons (see Fig. 6 and Plates I and II), very near the roots of the fourth and sixth nerves. It arises from several roots. The nerve itself pierces the dura mater below the posterior clinoid process, passes along the outer wall of the cavernous sinus, and after dividing into two branches enters the orbit through the sphenoidal fissure. (Plates I and XV.) The upper branch supplies the superior rectus and the levator palpebræ, and the lower one after dividing into three branches supplies the internal rectus, the inferior rectus, and the inferior oblique muscles. (See Plate VII.) The oculomotor nerve receives filaments from the cavernous plexus of the sympathetic, and from the first division of the fifth nerve. In addition to divergent squint there are, as already pointed out in the last few pages, in oculomotor paralysis, as additional symptoms, ptosis, mydriasis, and paralysis of accommodation.

The lesion producing *unilateral ptosis* may be found in the cerebral cortex on the opposite side from the affected eye in the angular gyrus just below the interparietal fissure. Again, tuberculous or other degenerative disease of the corpora quadrigemina may cause double ptosis.

If the patient has ptosis with preservation of the function of the intra-ocular muscles (that is, partial oculomotor paralysis), with hemiplegia of the opposite side of the body, the lesion, according to Mauthner, probably affects the fascicular fibers—that is, those between the cortex and the nuclei. There may he associated with this form of oculomotor paralysis loss of power in the hypoglossal and facial nerves. On the other hand, if the oculomotor paralysis is complete, the lesion is almost certainly at the base of the brain, and this diagnosis becomes practically certain if there is associated with it paralysis of other cranial nerves. Paralysis of the oculomotor

nerve on one side with hemiplegia on the opposite side of the body is not positively a crus or fascicular lesion unless the paralysis occurs simultaneously. (Hughlings Jackson.)

If, however, there be double oculomotor paralysis, the lesion is bilateral and probably due to a lesion at the base, as meningitis or arteritis (see Plate I), or to an inflammatory exudate involving both nuclei; or, again, to diphtheritic poison, or the lesions of tabes dorsalis.

If that very rare form of ocular muscle paralysis, namely, isolated *palsy of the fourth*, or trochlear, nerve, is present, we will probably find that the paralysis is due to a lesion at the base of the brain, due to meningitis, or the pressure of a growth.

Supposing, however, that a patient presents himself with swelling of the eyelids, exophthalmos, a contracted followed by a dilated pupil, anesthesia of the skin of the upper eyelid and of the temple, or the area supplied by the first division (ophthalmic) of the fifth nerve, and ophthalmoplegia—that is, *paralysis of the extrinsic ocular muscles on one side*—Where will be the lesion productive of this train of interesting symptoms? It will be seen at once that such a condition is the result of paralysis of the oculomotor (third), pathetic (fourth), and abducens (sixth) nerves, and that, as in all probability only one lesion has produced these symptoms, it must exist at some point where all these nerve fibers are so closely approximated that they are readily involved together. It will be recalled that the course of these nerves is as follows: the oculomotor nerve, having arisen from the nucleus in the corpora quadrigemina, pierces the dura mater below the posterior clinoid process, passes along the outer wall of the cavernous sinus, and there divides into two branches. The pathetic nerve passes near the clinoid process along the outer wall of the cavernous sinus, and with the oculomotor nerve enters the orbit through the sphenoidal fissure. The sixth nerve pierces the dura mater on the basilar surface of the sphenoidal bone, passes through the clinoid process, and enters the cavernous sinus, finally reaching the orbit through the sphenoidal fissure. It is thus seen that a lesion at the sphenoid fissure and pressure in the cavernous sinus would cause all the symptoms described above. (See Fig. 170 and Plates I and XV.) This occurs in cases of thrombosis of the cavernous sinus. Where there is an arteriovenous aneurysm of this sinus there will be pulsating exophthalmos. Injury or inflammation, if in this area, may also produce a series of symptoms.

The significance of *conjugate lateral paralysis* producing a deviation of both eyes to the right or left, as the case may be, is that some lesion exists in the cerebral cortex, the corona radiata, or the internal capsule, or in the pons before the fibers have crossed. The lesion, if in the cortex, however, does not have to be localized in one spot,

for any source of irritation in the cortex may apparently cause conjugate deviation. If the lesion is the result of an apoplexy, the eyes

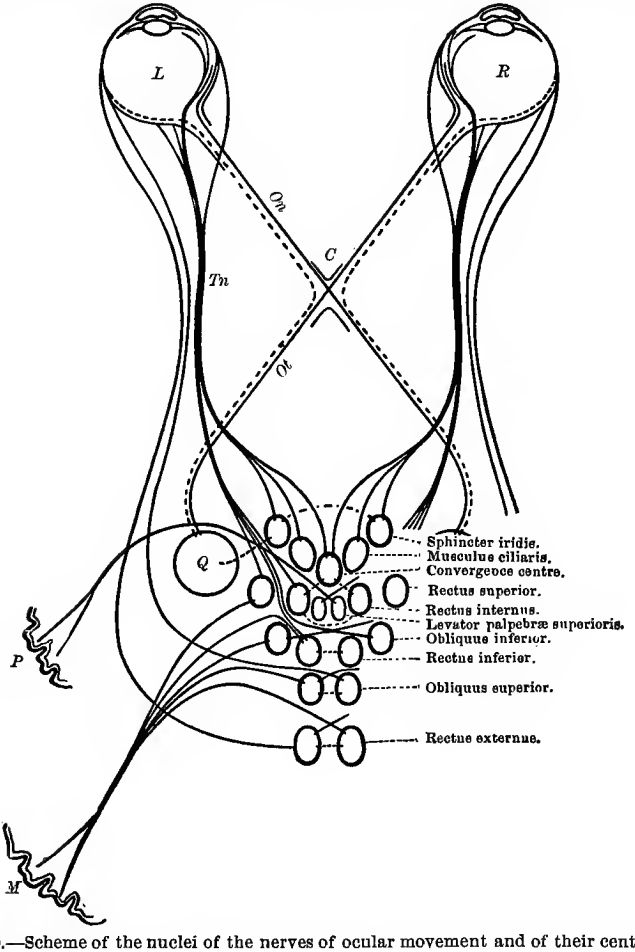


FIG. 170.—Scheme of the nuclei of the nerves of ocular movement and of their central and peripheral tracts: *R*, right eye; *L*, left eye; *C*, chissm, *On*, optic nerve; *Ot*, optic tract; *Q*, pregeminum (anterior quadrigeminal body); *P*, cortical centre for the movement of elevation of the upper eyelid; *M*, cortical centre for ocular movements; *Tn*, course of all ocular nerves in the cavernous sinus. The names of the different nuclei are printed on the diagram, and the nerve tracts going from these nuclei can be readily traced to where they converge in their course in the cavernous sinus and where they diverge to pass the various muscles of the eye. The dotted lines represent associating and commissural tracts, showing how the fibers of the third, fourth, and sixth nerves come together in one bundle in the cavernous sinus. (From Mills' Nervous Diseases.)

are turned toward the side opposite to the paralysis (Prevost's symptom)—that is, the "patient looks at his lesion." The reason that a unilateral lesion can cause a bilateral deviation is that the lateral

movements of the eye are governed by an impulse which passes down from the cortex to the sixth nerve nucleus, and thence across the posterior longitudinal fasciculus to the opposite side, where it passes to the nucleus of the third nerve. As conjugate lateral deviation is caused by contraction of the internal rectus on one side (third nerve) and the external rectus on the other (sixth nerve) the mechanism of the deviation is clear. Thus, if the lesion be a distinctive one on the left side of the brain, causing right hemiplegia, the eyes will be turned to the left by the action of the unaffected left external rectus and the right internal rectus; while if the lesion be on the right side of the brain, the reverse will occur. If, however, the lesion be irritative (as a tumor), this deviation is reversed, because in this case the centres are irritated and cause spasm of the muscles receiving their nerve supply from the affected side of the cerebrum. In other words, the eyes are turned toward the side of the body which is convulsed.

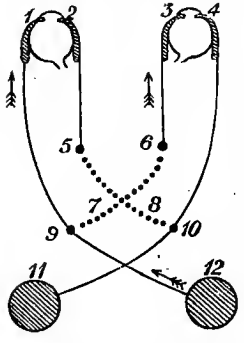


FIG. 171.—1. Left ext. rectus. 2. Left int. rectus. 3. Right int. rectus. 4. Right ext. rectus. 5. Nucleus left third nerve. 6. Nucleus right third nerve. 7 and 8. Post. longitudinal bands from sixth nerve to opposite third nerve. 9. Nucleus left sixth nerve. 10. Nucleus right sixth nerve. 11 and 12. Left and right cortical centres. An impulse starting from 12 would travel down to 9, and produce an associated movement of the eyes to the left.

In the first instance the eyes are turned away from the affected side because the muscles of the eyes on that side are also paralyzed, and the eyes are, therefore, turned by the muscles which remain intact. In the second instance the eyes are turned toward the convulsed side because the internal and external rectus are spasmodically contracted and so overcome the healthy muscles.

We find, however, that if the lesion be in the pons rather than in the cortex, these conditions are reversed, for now a destructive lesion causes the eyes to be turned to the paralyzed side, and an irritative lesion directs them away from the paralyzed side.

This is best explained by the following diagram and description from Swanzy's well-known book (Fig. 171).

A destructive lesion at twelve, the right cortical centre, involving also motor centres of the body, would cause left hemiplegia; and, since the external rectus of the left eye and internal rectus of the right eye would be paralyzed, the antagonists would turn the eyes to the right—*i. e.*, away from the paralyzed side. A destructive lesion

of the right side of the pons, also producing left hemiplegia, if it involves the sixth nucleus, will produce paralysis of the external rectus of the right eye and of the internal rectus of the left eye, and then the antagonists would turn the eyes to the left—*i. e.*, toward the paralyzed side. It is easy to see how irritative lesions would produce exactly the opposite effects.

Squint which is due to *hysteria* is always caused by spasmodic contraction of the eye muscle and is never due to paralysis, as it often is in organic disease. Very often there is a spasm of the eyelid or eyebrow associated with it. The administration of a relaxant, such as chloroform, will at once overcome such a squint.

Nystagmus, or the rapid oscillation of the eyes from side to side or in a vertical or rotary movement, is usually bilateral.[1] When not congenital it is a frequent symptom of disseminated sclerosis, Friedreich's ataxia, and advanced locomotor ataxia; and while it does not localize the lesion, it indicates very positively that one is present and that the case is not one of hysteria or functional disease. Nystagmus occurring in children is very often associated with imperfect vision of great degree or with blindness as a result of opacity of the cornea, congenital cataract, or atrophy of the nerve. In other instances, however, it arises from growths in the cerebellum or pons, and it is sometimes seen in hydrocephalus and very rarely in acute meningitis and in epilepsy. Very rarely lateral nystagmus is seen in children who seem otherwise normal, and it then possesses no particular diagnostic importance.

Paralysis or Disorder of the Intra-ocular Muscles.—Having discussed the diagnostic indications of changes in the functions of the extra-ocular muscles, we next proceed to a consideration of these facts in connection with the intra-ocular muscles. These consist, it will be remembered, in the muscular fibers of the iris, circular, and radiating, and the ciliary muscle. The nerve supply of the iris consists in fibers from the oculomotor or third nerve, the upper or ophthalmic division of the fifth, and the sympathetic. It will be remembered that in the posterior part of the orbit there is situated a ganglion called the ciliary or ophthalmic ganglion. By its short or motor root this ganglion is connected with the third nerve, by its sympathetic root with the cavernous sympathetic plexus and the cervical sympathetic plexus, while by its long or sensory root it is connected with the nasal branch of the ophthalmic or upper branch of the fifth nerve. From this ganglion extend forward two sets of nerves, one short (the short ciliary nerve), which supplies the iris and the ciliary muscle, and one set long (long ciliary nerves), which also go to the iris. The filaments which go to the ganglion by means

[1] The physician should remember that some occupations, such as mining, produce in some persons nystagmus without the presence of the disease about to be named.

of its short or motor root (from the oculomotor nerve) pass forward to the circular fibers of the iris, while those which have arisen in the sympathetic plexus pass forward to the radiating fibers. These last fibers are in part derived from the cervical sympathetic ganglion, run through the carotid plexus, and are controlled to some extent by the ciliospinal centre of Bunge in the spinal cord at about the seventh cervical or first dorsal vertebra.

The ciliary muscle is supplied by the fibers of the short ciliary nerves, which have arisen in the floor of the third ventricle and which is connected with the nucleus of the third nerve.

Testing the Pupil.—The normal pupil is about 4 mm. in diameter, but this varies according to the degree of light to which the patient is exposed. It ought always to be measured by a millimeter measure, which gives its approximate diameter.

The pupil to be tested must be free from any abnormal conditions produced by new or old inflammation of the iris, and the light used should not be excessively bright, but about that usual to the eye.

The patient is to be placed facing the light and told to look at some distant object. The hands of the physician are then placed one over each eye, the patient being told to keep his eyes open. One hand is now quickly removed from one eye and the pupil observed. This observation must be acute or the pupillary contraction will occur before it is seen. This reflex is due to the fact that we have an irritation of the optic nerve by light, and this sends a reflex wave to the centres governing the pupil and causes it to contract. Not only does the uncovered pupil react in this manner, but the covered one does the same thing. The first is called a direct reflex, the second is called the indirect or consensual reflex. The exact pathway of this reflex is unknown.

Not only does the pupil change its size by reason of the ordinary light reflex, but it also contracts or dilates in association with the other muscles governing accommodation and convergence, namely, the ciliary muscle and internal recti. This is the associated reaction of the pupils, and is tested by causing the patient to direct his eyes to a near object—for example, the point of a pencil. If the sight is intact, contraction of the pupil will occur.

The pupil-dilating centre is in the medulla and is very sensitive to reflex irritation.

Abnormal Pupillary Movement.—Supposing that the pupillary movement is abnormal, we should recollect before studying the case farther what the causes of its perversion may be. Thus, its size is altered by drugs, by local disease of the iris, by spinal disease of the sympathetic, by localized cerebral lesions, by abeyance of the cerebral functions, and by irritation of the brain. Let us suppose, however, that on testing the ocular reflexes in the manner

already described we find that the pupil of one eye when uncovered does not contract, and immediately does so as soon as the other eye is uncovered, What is the indication? It indicates that there is disease of the optic nerve of that eye which does not convey the impulse of light from the retina; whereas, if it contracts when the other eye is uncovered, it shows that the rest of the mechanism involved in the reflex is intact. Accommodative reaction of the pupil is intact also.

If the irides fail to react to light, but do to accommodation and convergence, we have the "Argyll-Robertson pupil," so called, which indicates that a lesion exists in the fibers back of those concerned with the ordinary light reflex.

This condition is seen in locomotor ataxia, general paralysis of the insane, sometimes in cerebral syphilis, and as the result of poisoning by the bisulphide of carbon. Grube has reported three cases in which the Argyll-Robertson pupil developed in the course of diabetes mellitus. Marinesco has reported an instance of Argyll-Robertson pupil in a patient suffering from amyotrophic lateral sclerosis. It is worthy of note, however, that late in all these affections the reaction to accommodation may also be lost. Rarely the reverse of the Argyll-Robertson pupil occurs as the result of a lesion in the second and third parts of the oculomotor nucleus. If the eyes fail to react to light and to accommodation, there is probably blindness due to optic-nerve disease.

If on throwing light into the right eye there is no reaction of the pupil of that eye, and on throwing it into the left eye there is still no reaction in the pupil of the right eye, there must be a lesion of the nucleus of the right oculomotor nerve or palsy of the conducting fibers of each optic nerve.

Sachs asserts that *immobility of the pupil* is very characteristic of syphilitic cerebrospinal disease, and if the diagnosis lies between multiple sclerosis on the one hand, and cerebrospinal syphilis on the other, the discovery of immobility of one or both pupils should decide in favor of its being a syphilitic case. He also asserts that persistent pupillary immobility in a case of hemiplegia indicates a syphilitic endarteritis. It is important in this connection to remember that the pupillary changes due to syphilis often suddenly improve, while those due to sclerosis are absolutely permanent.

Contraction of the pupil occurs in a large number of conditions and yet possesses considerable diagnostic significance. Thus it is generally found in the early stages of all acute inflammatory processes in the brain or its membranes. In cerebral hemorrhage it is usually contracted at first, thus serving to separate acute paralysis due to hemorrhage from that due to embolism, for in the latter Berthold states that the pupil is unaltered. In the early stages of

intracranial tumors which irritate the third-nerve nucleus it is also contracted. Finally, we find myosis as a result of chronic tobacco poisoning, from irritation of the pupil-contracting centre by nicotine, at the beginning of an attack of hysteria or epilepsy, and in watchmakers and jewellers. Such forms of myosis are called "irritative myosis." Myosis is also seen in disease of the apex of the lung. (See next page.)

Paralytic myosis is met with in lesions above the dorsal vertebra of a chronic type, as a rule. Its most interesting form is that seen in locomotor ataxia, when the disease has involved the ciliospinal centre.

Of course, pupillary contraction may result from the action of a myotic drug, as eserine or pilocarpine.

Dilatation of the pupil may also be due to irritation or paralysis. Thus, irritation of the pupil-dilating centre may cause mydriasis, and this is met with in congestion of the cervical spinal cord and in spinal meningitis, as the result of tumors in the cervical cord, in spinal irritation, in the anemia of convalescence, as an early sign of tabes dorsalis, and in acute mania. Certain cases of acute croupous pneumonia present dilatation of both pupils, the dilatation being most marked on the affected side, probably by irritation of the sympathetic fibers by pressure of the consolidated lung. Sometimes in progressive paralysis of the insane there may be irritative mydriasis in one eye and myosis in the other. Von Graefe asserted that *alternating unilateral mydriasis* is an early sign of mental derangement.

The states in which we find *paralytic mydriasis* are in the later stages of progressive paralysis, in lesions at the base affecting the oculomotor centre, late in thrombosis of the cavernous sinus, in orbital disease which causes pressure on the ciliary nerves, in glaucoma, and in intracranial growths of considerable size. Not only may paralytic mydriasis be due to an oculomotor lesion, but as the result of some blocking of the pathway from the retina to the centre.[1]

Under the name of "*hemiopic pupillary inaction*" or "Wernicke's pupil," we sometimes, though rarely, meet with a condition associated with hemianopsia, or blindness in one-half of the eye, which is demonstrated in the following manner: the patient is seated in a dark room and one eye is covered. The other eye is now illuminated by just sufficient light from a flat mirror to enable the physician to see the eye. By means of the concave mirror of an ophthalmoscope the physician now directs into the uncovered eye a bright beam of light, taking care that it falls upon one side of the retina, or, in other words, enters the eye obliquely and strikes on the side of the retina which is blind. If when the light falls on the blind side of the retina

[1] For a useful summary of these facts and for references, see "Diseases of the Eye," by Swanzy, sixth edition.

there is no pupillary reaction, it is considered that the lesion exists in the arc between the optic chiasm and the corpora quadrigemina; but if there is a pupillary reaction, the lesion must be farther back in the visual centres, back of the reflex arc. When the lesion is found back of the reflex arc it may indicate a lesion of the optic tract, the posterior segment of the thalamus, the posterior part of the chiasm, or rarely it may be caused by a lesion of the optic nerve if the hemianopsia be monocular, which is rarely the case.

Finally, a rhythmical contraction and dilatation of the pupil, called "*hippus*," is seen in health for a moment on sudden exposure to light; but when constant is a sign of disseminated sclerosis, epilepsy, or the early stages of acute meningitis. It is sometimes seen in hysteria.

The presence of a recurrent, unequal dilatation of the pupils of a transitory character is said by Rampoldi to be an early and almost constant sign of pulmonary tuberculosis. He believes that this is due to a reflex irritation of the nerves governing the pupil through the sympathetic system. Probably in these cases enlarged glands in the chest are the cause of the pupillary phenomenon. Destree claims that 97 per cent. of his cases of phthisis present this pupillary symptom. On the other hand Souques asserts that myosis is commonly present in tuberculous apical disease owing to the dilator fibers from the ciliospinal centre which pass through the first dorsal nerve to the cervical sympathetic being pressed upon by the apex of the pleura. Evidently it is an irritative reflex and results in mydriasis or myosis according to the degree of pressure.

Knies points out that pupillary contraction and dilatation take place in association with Cheyne-Stokes breathing. Dilatation usually exists with the inspiratory movements, and myosis occurs during the interval of apnea.

Changes in the Acuity of Vision.—Having discussed the diagnostic value of alterations from the normal in the function of the extra- and intra-ocular muscles of the eye, we can proceed to a consideration of the value of changes in the acuity of vision. The questions of the acuity of vision in relation to errors in the refractive media of the eye will not, of course, be included in this book.

Failure of vision in part or *in toto* depends upon a lesion which destroys the peripheral ocular sense organ (the eye), the optic nerves, the optic tracts, or the receptive and perceptive centres of sight. It also is dependent upon bilateral lesions in the crystalline lens, as in cataract, or in the cornea, as in severe keratitis.

Before we discuss these various causes of blindness it is necessary that we recall the nervous anatomy of the organs of sight. These nerve fibers starting with the rods and cones of the retina and the fibers from the macula pass back along the optic nerve until they

come to what is known as the chiasm, where the various fibers from the eye decussate, in that the fibers from the inner half of each eye

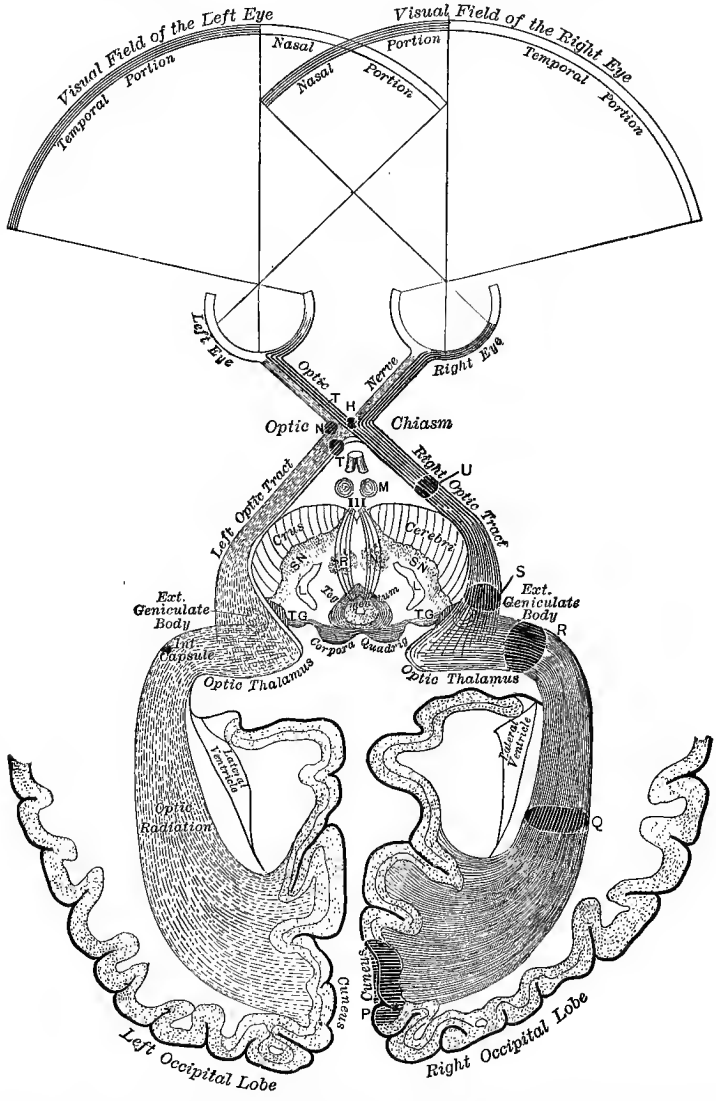


FIG. 172.—The visual tract. The result of a lesion anywhere between the chiasm and the cuneus is to produce homonymous hemianopsia. *H.* Lesion at chiasm causing bilateral temporal hemianopsia. *N.* Lesion at chiasm causing unilateral nasal hemianopsia. *T.* Lesion at chiasm causing unilateral temporal hemianopsia. *S N.* Substantia nigra of crus. *L.* Lemniscus in crus. *R N.* Red nucleus. III. Third nerves. *P, Q, R, S, U* Lesions in the occipital lobe and in front of it, producing left homonymous lateral hemianopsia.

cross to the opposite side, whereas those of the outer half of each pass to the same side, as is shown in Fig. 172. After the optic tracts have been formed by this (partial) decussation each one winds around the corresponding crus cerebri, and terminates in two roots upon the corpora geniculata externa and interna and upon the posterior part of the optic thalamus. These parts are known as the primary optic centres. After leaving them the fibers pass backward into the posterior part of the posterior limb of the internal capsule and thence to the cortex, rise in a fan shape, pass outside the tip of the lateral ventricle, and reach the secondary or true optical centre in the lower part of the median aspect of the occipital lobe (Fig. 172).

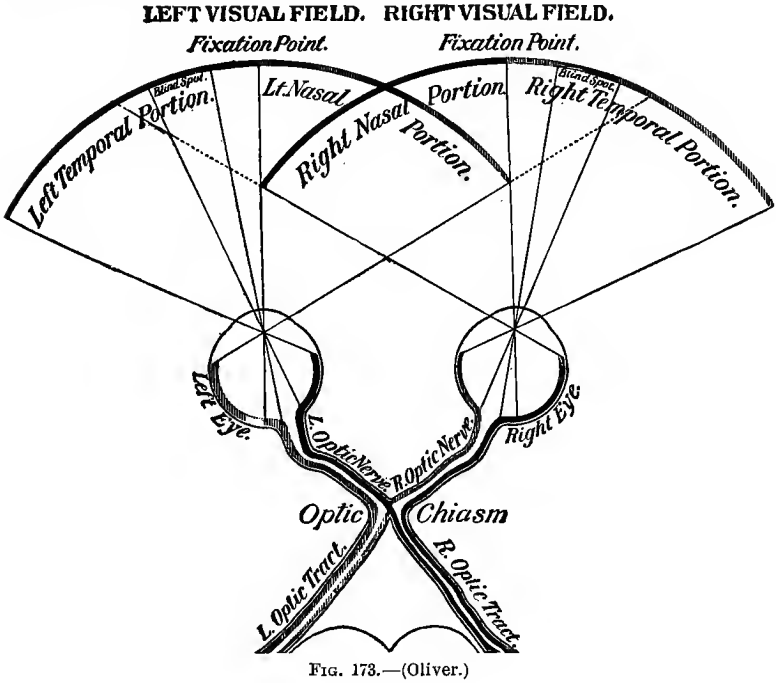


FIG. 173.—(Oliver.)

Hemianopsia.—As lesions of the nervous centres frequently produce partial or complete blindness, it is of importance, first, that the presence of partial blindness should be discovered, and, second, that the lesion causing it should be located. Aside from general failure of vision due to changes in the retina or optic nerve we have in many cases of nervous disease a condition called hemianopsia or partial or complete blindness of one-half of the retina. Usually hemianopsia is bilateral—that is, in both eyes; and it is usually homonymous—that is, on the same side of each eye; or, in other words, if it is in the

outer half of the left eye, it will be in the inner half of the right eye. If this is the case, it is called *left bilateral homonymous hemianopsia.* If, on the other hand, the outer half of each eye is blind, this is called *bitemporal hemianopsia;* if the blindness is found in the nasal side of both eyes, it is called *binasal hemianopsia.* It must be remembered, however, that the apparent blindness of the outer side of the eye is really due to disease of the fibers supplying the opposite side of the retina, as is shown in Fig. 173.

The presence of hemianopsia in any form is determined by the following method of examination: The patient is placed with the

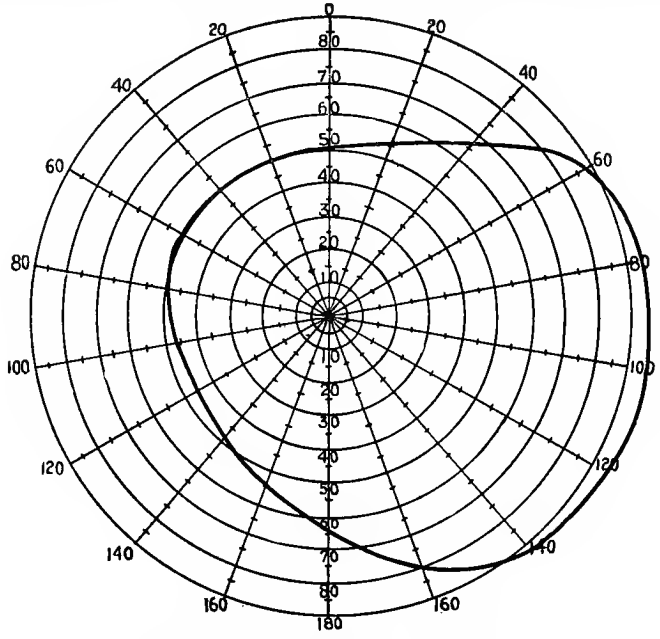


FIG. 174.—Chart of visual field of right eye.

back to the light and one eye is covered while the other is fixed upon the centre of the physician's face, which should be two feet away. The finger of the physician is now moved to the left and right as far as the patient can see it, the head and the eyeball of the patient remaining fixed. If the eye fails to see the finger when but a little distance to one side or the other of the fixation point, hemianopsia is present.

We measure the field of vision more accurately by means of what is known as a perimeter, which is a semicircular metal band which revolves upon its middle point, being capable therefore of describing a hemisphere in space. This arc is divided into degrees marked on

it from 0° to 90° and at the centre of it is placed the eye which is to be examined, which eye finds its fixation point in the centre of the semicircle. A small piece of white paper is now moved along the metal

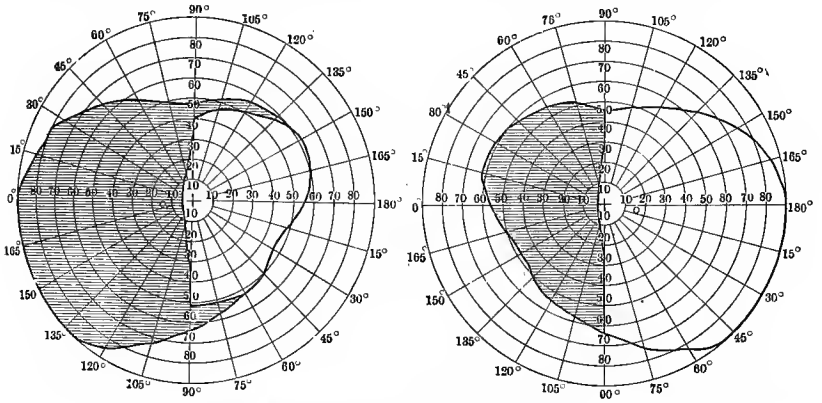


FIG. 175.—Left homonymous hemianopsia from a case of gunshot wound, with suspected lesion of the right cuneus. (de Schweinitz.)

arc on its inner surface, from the extremity and toward the centre, until it comes into view, when the physician notes the number of degrees at which the object is seen and marks it on a chart (Fig. 174). The area of the normal field is well seen in this figure.

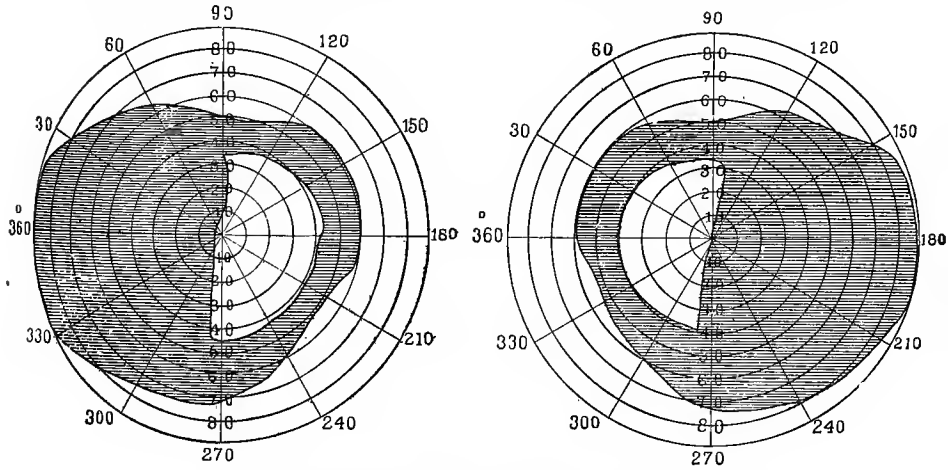


FIG. 176.—Bitemporal hemianopsia from a case of acromegaly. (de Schweinitz.)

Let us suppose that on using the tests just described we find *left lateral homonymous hemianopsia*—that is, blindness in the visual field, as shown in Fig. 175. This signifies that the patient has a

lesion somewhere in the visual tract back of the chiasm, either in the cuneus, in the occipital lobe, in the optic radiations, in the internal capsule, in the primary optic centres, or in the optic tract. Fig. 172 shows the sites of these lesions and why they cause left homonymous hemianopsia.

Supposing, on the other hand, that instead of left homonymous hemianopsia we find *bitemporal hemianopsia* (Fig. 176), this indicates that the patient has a lesion of the optic tracts in the crossing fibers in the middle of the chiasm (see *H* in Fig. 172); or if binasal

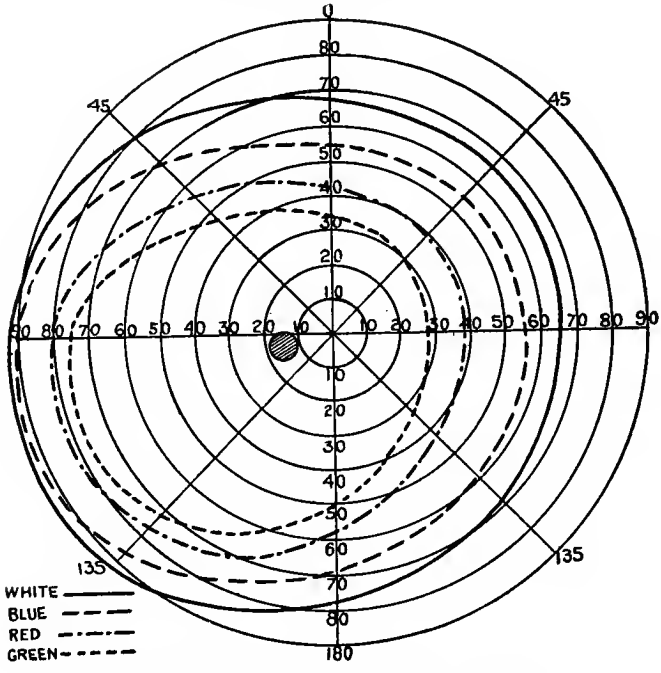


FIG. 177.—Chart of visual field of left eye. (Landolt.)

hemianopsia, that he has a lesion on both sides of the chiasm or one on the outer side of each optic nerve. This is a very rare lesion.

Hemianopsia of the homonymous form is very rarely found in hysteria, generally in association with hysterical hemianesthesia, in which condition the conjunctiva is usually anesthetic, thereby differing from the condition of the conjunctiva of persons suffering from hemianesthesia of an organic origin.

Visual Color Fields.—In some cases in place of hemianopsia we have simply an alteration in the visual fields for color. It will be remembered that the boundaries of the power of the clear perception of colors are not identical with the boundary for white light, nor are

they identical with one another. Passing from the periphery toward the centre of the visual field in ordinary daylight we find that blue is the color first seen, its boundary being almost as great as that of white. After blue come yellow, orange, red, and finally green. The blue, red, and green being the most important colors, their boundaries are shown in Fig. 177. These fields are determined by means of small pieces of colored paper passed around the perimeter in the manner already described.

The alteration of the visual field for colors is called, if so changed, *homonymous hemidyschromatopsia,* and the lesion producing it is situated in the cortex of the occipital lobe; while if the colors are indistinguishable, it is called hemiachromatopsia. This site of the lesion has recently been denied.

The transposition of the visual fields for color is usually a symptom of hysteria, and, as a rule, the red field takes the place of the blue, and *vice versa.* The fields for all the colors are also markedly narrowed in hysteria. This transposition, rather than loss of color sense, helps us sometimes to a distinction between the ocular symptoms of hysteria and those of true tabes dorsalis, a distinction which is of great importance, yet one which is often exceedingly difficult, save for these and two other symptoms, namely, that in hysteria the knee-jerks are usually preserved and the Argyll-Robertson pupil is not seen. The following table from Charcot's lectures for 1888–1889 summarizes these differential points:

	Tabes.	Hysteria.
Motor apparatus of the eye.	Paralysis from lesion of a motor nerve of the eye (bulbar or peripheral); consequent diplopia.	1. Sometimes associated paralysis. 2. Blepharospasm. 3. Monocular diplopia; micropsia and macropsia.
Pupillary disturbances.	Argyll-Robertson pupil.	
Optic disk.	Atrophy.	
Symptoms due to affections of the optic nerve or visual centres.	1. Irregular concentric contraction of the visual fields. 2. *Tabetic* achromatopsia or dyschromatopsia, affecting first green and red, yellow and blue being preserved to the last. 3. Progressive blindness.	1. Regular concentric contraction of the visual fields. 2. Dyschromatopsia from simple contraction of the visual fields for colors. Frequently perception of red alone persists. 3. Transitory amblyopia or amaurosis.

It must not be forgotten that patients often have, in distinction from distorted images, visions or flames of light or bright sparks before the eyes, or in their place dark spots called muscæ volitantes. Often the visions are the prodromes of an attack of migraine or of an epileptic seizure. In the case of spots of light or stars we usually

find them as a result of severe indigestion, and the dark spots may arise from the same causes. *Muscæ volitantes* may also be due to small particles of mucus floating over the cornea or to small floating bodies in the vitreous.

Partial or complete blindness is sometimes seen in cases which are under the influence of a drug, as, for example, quinine or other drugs; and sometimes partial or complete blindness results from uremia (uremic amaurosis). As a rule, it does not occur as a single symptom, but follows an attack of acute uremic manifestation—that is, it is found after a convulsion or period of coma has passed by. As a rule, nothing abnormal is found in the eye to account for it, and the pupillary reflexes are intact. The effect of the poison in the blood is, therefore, exercised upon the optical centres, probably in the occipital lobe. Sight is usually regained in these cases in a few days.

The Optic Nerve and the Ophthalmoscope.—There still remain to be considered the diagnostic indications afforded us by the optic nerve. Before taking up this subject mention must be made of the manner of using the ophthalmoscope.

The patient is to be seated in a darkened room, and by his side, at the level of the eye to be examined and far enough back of him for his face to be in shadow, should be placed a lamp, or, if gas can be had, an Argand burner. The physician now seats himself, if the right eye is to be observed, at the right side of his patient, and takes a chair slightly higher than that of the patient. The ophthalmoscope is now taken in the right hand and held in such a position that the concavity of the physician's brow fits over the convexity of the instrument. The eye of the physician is so placed that he can readily see through the aperture in the centre of the ophthalmoscope, and by means of the concave mirror on the face of the instrument he reflects the light into the eye through the pupil. The patient must not look into the ophthalmoscope, but to one side, and his vision should be directly distant and accommodation as far as possible relaxed. If the examiner is not skilled in the use of the ophthalmoscope and the result of the examination is of great importance in the diagnosis of the case, it is justifiable to use homatropine to dilate the pupil and prevent the alterations of accommodation by paralyzing this function. The ophthalmoscope and the head of the physician are now approached as closely as possible to the eye of the patient, the angle of the two heads being as nearly as possible identical, as shown in Fig. 178. If the light be now directed slightly toward the nasal side of the eye, the optic nerve will be seen, or in its stead a retinal blood-vessel will be seen across the field of vision, and this should be traced along its course to its origin in the papilla. If the patient or the physician is short-sighted (myopic), the ophthalmoscope must be adjusted to correct this error by placing over the aperture a concave

lens; but if ordinary degrees of far-sightedness (hypermetropia) are present, the use of a convex lens is not necessary, because the accommodation of the eye makes up for the error in refraction. If the hypermetropia is so great that accommodation cannot overcome it, then a convex lens must be used. The view of the eye which is obtained ordinarily by a beginner is clouded, not because of myopia or hypermetropia, but because the physician has not as yet learned to relax his accommodation in making the examination. A concave glass usually remedies this.

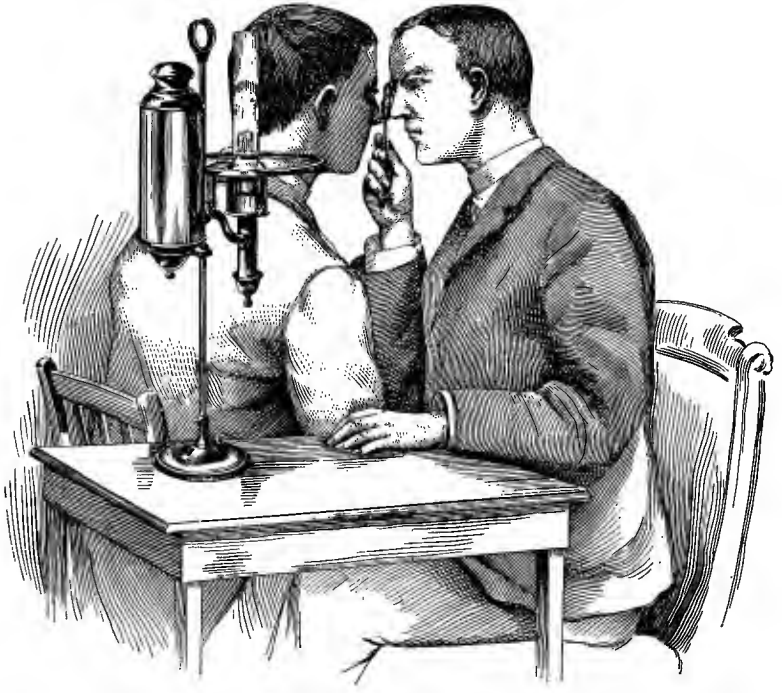


FIG. 178.—Relative position of physician and patient whilst employing the direct method. (Norris and Oliver.)

In health the optic nerve appears as a nearly round or slightly oval disk, situated somewhat to the nasal side of the eye, and varying in color from grayish pink to red, the centre being whiter and the nasal half the darkest part. Around the papilla are seen two rings, the outer one darker and generally incomplete or absent, while the inner one is a faint white stripe, which becomes more marked as the patient grows older. The first is called choroidal ring, and represents the edge of the choroidal coat of the eye where it is pierced by the nerve. The second is the scleral ring, which is the edge of the

sclerotic coat. The centre of the optic papilla may be even with the surface or cupped, and may be stippled or dotted in appearance. The retinal arteries emerge from this central spot and the chief venous trunks empty into it. Generally one arterial and one venous stream pass up and a similar one downward, and both soon bifurcate, afterward still further dividing. The arteries are distinguished from the veins by their bright-red hue, while the veins are darker in color. The veins are about one-third larger than the arteries. A bright stripe due to an optical delusion seems to divide each vessel longitudinally into two parts. The arteries of the normal eye do not pulsate, but pulsation of the veins is quite common. It must be remembered that the appearance of the papilla and of the bloodvessels as they leave it varies very greatly within perfectly physiological limits. As already stated, the cupping of the papilla may be quite deep or quite shallow, and the bloodvessels may divide as already described, or divide in the papilla into four branches. The veins are usually more tortuous than the arteries. The retina is practically transparent, so that the underlying choroid is seen. In persons with a dark skin the retina has a grayish hue in the neighborhood of the papilla, which is most marked on its nasal side and is slightly streaked.

To the outer side of the papilla, slightly below the horizontal meridian, is the macula lutea or yellow spot, which is about the size of the end of the optic nerve, but darker in color, somewhat granular, and devoid of any retinal vessel. It is the point of the eye-ground in which direct vision is best developed. In its centre is a bright spot, the fovea centralis. As a person grows older these clear distinctions vanish and the macula lutea is to be distinguished from the surrounding eye-ground only by its darker hue and the absence of vessels. The macula is difficult to see, because as the light falls on it the pupil at once contracts. If the eye is dilated by a mydriatic, however, and the patient looks directly into the ophthalmoscope, the macula is readily seen.

The red glare produced by throwing the light into the eye by the ophthalmoscope is due to reflection from the bloodvessels of the choroid coat.

The pathological significance of alterations in these normal appearances is very great.

Optic Neuritis.—Let us suppose that on examining the eye-ground we find the end of the optic nerve red and its edges irregular and obscure, or, if the morbid condition is further advanced, that the nerve head looks protruding or mound-like and the arteries going to it are smaller than normal and partly concealed, while the veins are enlarged and tortuous. Hemorrhages may be seen in the papillary region or near it, occurring in flame-like shapes. These are the signs of *optic neuritis*, and optic neuritis depends upon intra-orbital

or intracranial disease, although, if the process is not marked, it may be due to hypermetropic astigmatism. Vision is often unaffected, but if the lesion be in the cerebellum sudden blindness may come on.

As some differences of opinion exist as to the various forms of neuritis of the optic nerve, the term papillitis is often used to signify all the forms of optic neuritis which we meet with, or in other cases is spoken of as "*choked disk.*" Papillitis is more commonly the result of brain tumor than of any other intracranial lesion, and, again, it is much more common in lesions of the cerebellum than in tumors elsewhere in the brain. Another fairly common cause of papillitis

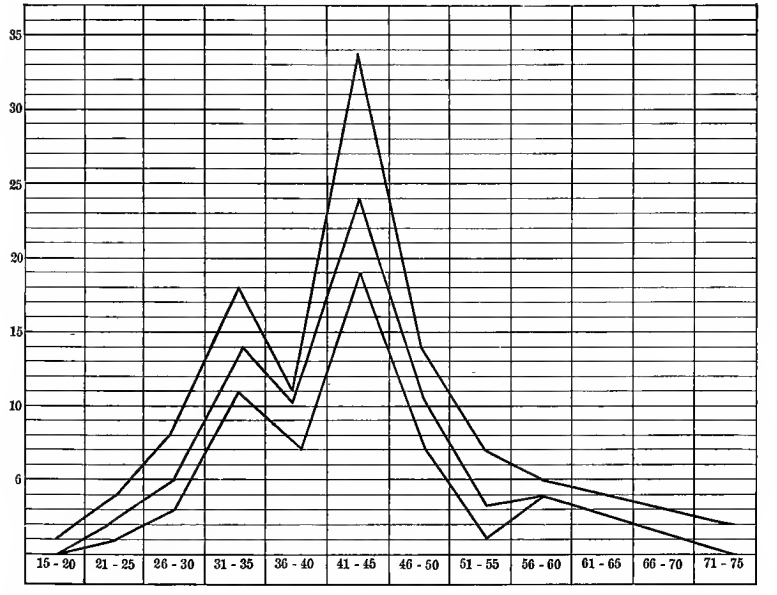


FIG. 179.—Upper curve, frequency of tabes. Middle curve, frequency of severe ocular symptoms. Lower curve, frequency of atrophy of the optic nerve. (Berger.)

is meningeal inflammation, particularly about the base of the brain, and tuberculous meningitis is very prone to produce it. Cerebral abscess may also cause this change in the optic nerve.

In addition to the cranial causes of papillitis we have acute febrile disorders, syphilis, toxemias from lead and alcohol, rheumatism, and anemia. Sometimes, however, they produce an acute or chronic retrobulbar neuritis. There is nearly always in such cases a large central scotoma, which causes a failure to recognize color, as, for example, green or red. Sometimes the patient realizes the failure of his vision, which may be impaired otherwise than by disorder of the color sense. In other cases he fails to do so until his eyes are

examined. The chronic form of retrobulbar neuritis is generally the result of the excessive use of tobacco and alcohol, and produces what is called tobacco amblyopia or toxic amblyopia, with failure of vision from these causes. In such cases there is a central scotoma between the macula and the optic nerve where the senses of red and green are lost. The ophthalmoscope may reveal in such cases discoloration of the disk and a triangular spot of atrophy in the outer and lower part of it. Supposing, however, on using the ophthalmoscope we find in place of a papillitis an atrophied state of the nerve, in which, if the disease be young, the nerve ending looks gray and the outline of the disk is sharp, or if it be well advanced the edges appear hazy, the arteries contracted, and the veins large and tortuous, while the disk is quite white. This primary or gray form of atrophy is most typically seen in the optic-nerve lesion of *locomotor ataxia*, and so is often called tabetic atrophy. About 34 per cent. of all tabetics suffer from this change. Again, it is seen in cases of paretic dementia somewhat less frequently. Optic atrophy is often seen in cases of *disseminated sclerosis*. Because of the fact that gray atrophy of the nerve is one of the earliest signs of locomotor ataxia, in some cases it is a valuable one in the diagnosis of this grave disorder, separating it from pseudotabes due to ordinary peripheral neuritis. The diagram (Fig. 179) on page 439, taken from de Schweinitz's article on this subject, shows the relation between age, severe ocular symptoms, and atrophy of the optic nerve.

The more advanced forms of optic atrophy with a hazy outline of the disk usually result from diseases in the optic centres or in the nerve itself. Thus there may be present a tumor pressing on the chiasm or optic tracts.

Retinitis.—If on the use of the ophthalmoscope we find that there is a faint haziness of the retina, that whitish streaks are seen in it which may be bluish-gray or yellowish in hue, that the bloodvessels are tortuous and minute vessels are easily seen because of their enlargement, that hemorrhagic exudations of a flame-like character are present, that dark pigmented spots show where previous hemorrhages have been, and, finally, that the head of the optic nerve is not clearly outlined, we have the picture of retinitis. Generally, in association with these signs, we find as subjective symptoms changes in the visual field, a distorted vision, so that straight lines appear bent inward or outward, and there are pain and fear of light. If in addition to these symptoms the vitreous humor is opaque, syphilis may be present, and the iris may give evidence of iritis. Where the hemorrhages are very manifest and profuse (hemorrhagic retinitis) the cause may be disease of the heart and bloodvessels.

By far the most important of these forms of retinitis from a diagnostic standpoint is what is known as *albuminuric retinitis*, or that

due to *parenchymatous nephritis*. Here, in addition to the flame-like hemorrhagic areas, we find irregular spatterings of white which may be star-shaped. If the retinitis be due to chronic Bright's disease the prognosis is very bad, death occurring in a year in 50 per cent. of the cases, whereas not 20 per cent. live more than two years. The importance of the discovery of such changes is that by it the first suspicion of renal trouble is aroused. This sign is of the greatest value in pregnancy, in which the ultimate prognosis is not so grave. Retinitis also sometimes results from diabetes.

Hemorrhages into the retina without retinitis are usually the result of septicemia, ulcerative endocarditis, hemophilia, diabetes, gout, and malarial fever of a severe type. They are also seen in cases of great cardiac hypertrophy with stenosis, and after suffocation.

Ophthalmoscopic evidence of general arterial disease and chronic contracted kidney not infrequently is manifested by edema of the retina and retinal hemorrhages; but an early sign is the influence of the arterial pressure on the venous blood streams of the retina, where artery and vein cross one another. There may be simply inequality in the caliber of artery and vein, or the vein may be somewhat displaced, where it lies beneath the artery, in the direction of the arterial circulation, and its flow obstructed. In advanced cases the vein is greatly narrowed where the artery crosses it and distended on its peripheral side. When these appearances are well studied they are exceedingly suggestive of early arterial changes. Changes of this character, as the author can testify from studies made with Dr. de Schweinitz, are of serious prognostic import and may be the forerunners of intracranial extravasations.

Iris.—The iris indicates disease in other organs more rarely than the retina and optic nerve and the muscles, but an irregular pupil indicating an old iritis should raise a question as to a history of injury or rheumatism or syphilis.

Finally, it should not be forgotten that cataract sometimes occurs as the result of diabetes mellitus and that corneal ulceration is often an evidence of scrofulous tendencies, while a distorted pupil due to an old iris should raise a suspicion of syphilis.

CHAPTER XIV.

CHILLS, FEVER, AND SUBNORMAL TEMPERATURES.

Chills—The methods of taking the temperature—The significance of fever—The febrile movements of various diseases.

CHILL.

A CHILL is of very considerable diagnostic importance when observed by the physician, or even when reported as having occurred in the immediate history of the patient. It may follow prolonged exposure to cold, without subsequent development of disease, or be a precursor of some acute malady. Often it is an early symptom of the onset of one of the acute infectious diseases, such as croupous pneumonia, erysipelas, or scarlet fever. In other instances it is a symptom of the development of a purulent or pyemic process. When chills recur repeatedly they may be due to malarial infection, in which case they may be controlled by using quinine; as a result of deep-seated abscesses and general pyemia; and finally, they may indicate tuberculosis or ulcerative endocarditis. In some cases of typhoid fever a chill ushers in the attack, and chills may repeatedly occur without apparent cause, so that the disease may be singularly like remittent malarial fever. (See Figs. 180 and 181.)

FEVER.

Fever is that state of the human body in which its temperature is raised above the normal limit, or 98.8° F., but variations from 97.8° to 99.5° may occur without indicating disease. From 99.5° to 100.4° the temperature is spoken of as subfebrile, from 100.4° to 101.3° as mildly febrile, while the term decidedly febrile is applied to temperatures varying from 103.1° to 105°. Hyperpyrexia is a term applied to a febrile movement in which the temperature rises as high as 105°. Cases are on record of a temperature of 115° or even more.

The method of taking the temperature consists in placing a self-registering clinical thermometer in the mouth under the edge of the tongue, the lips being then closed tightly about its stem; or of inserting it in the axilla, the hand and arm being then placed across the

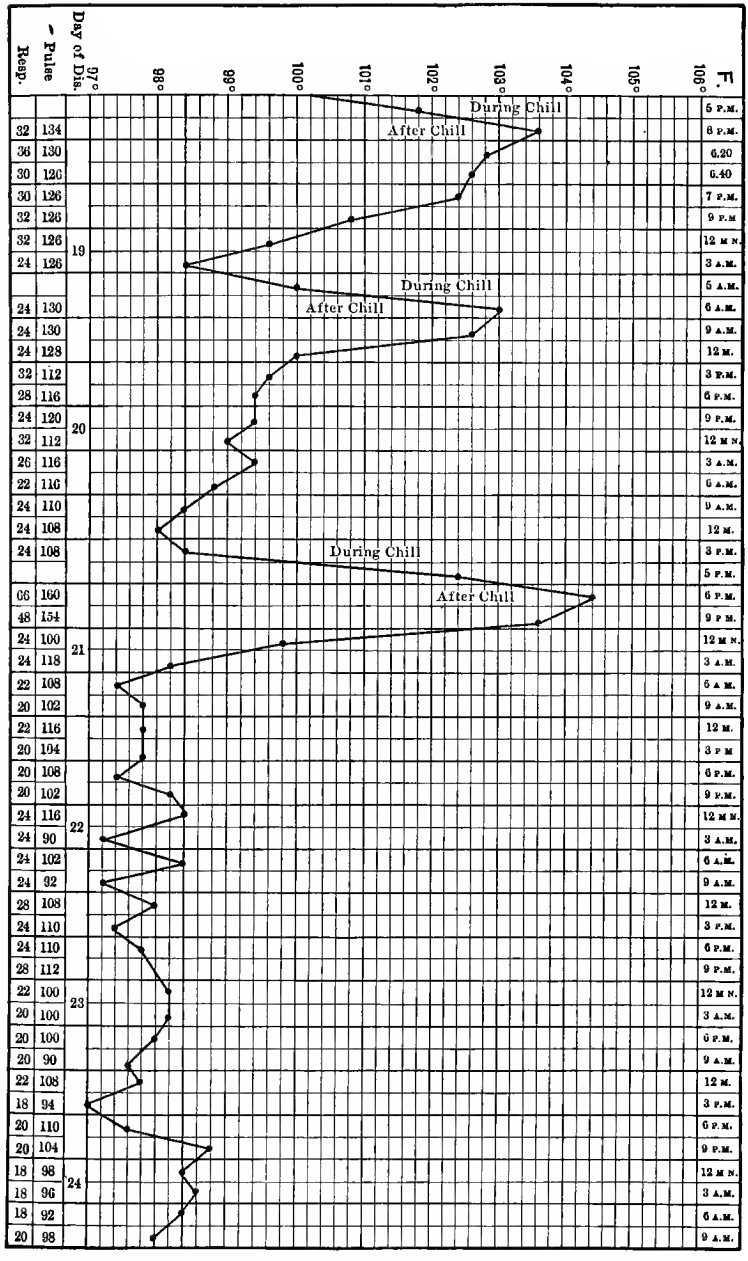


FIG. 180.—Toward the end of this case of typhoid fever severe chills developed on the eighteenth, nineteenth, and twentieth days, and the fever ended by crisis.

patient's chest or epigastrium, so as to cause the axillary tissues to be in close contact with the bulb of the thermometer. Before the thermometer is placed in the axilla this space should be carefully wiped dry, since if perspiration is present its evaporation will so chill the thermometer that a false record will be made by the index. Sometimes the temperature of the patient is taken by inserting the thermometer into the rectum; and, if this is done, the bulb should be passed well inside the external sphincter. Rarely the temperature is taken in the vagina. In the rectum and vagina the normal tempera-

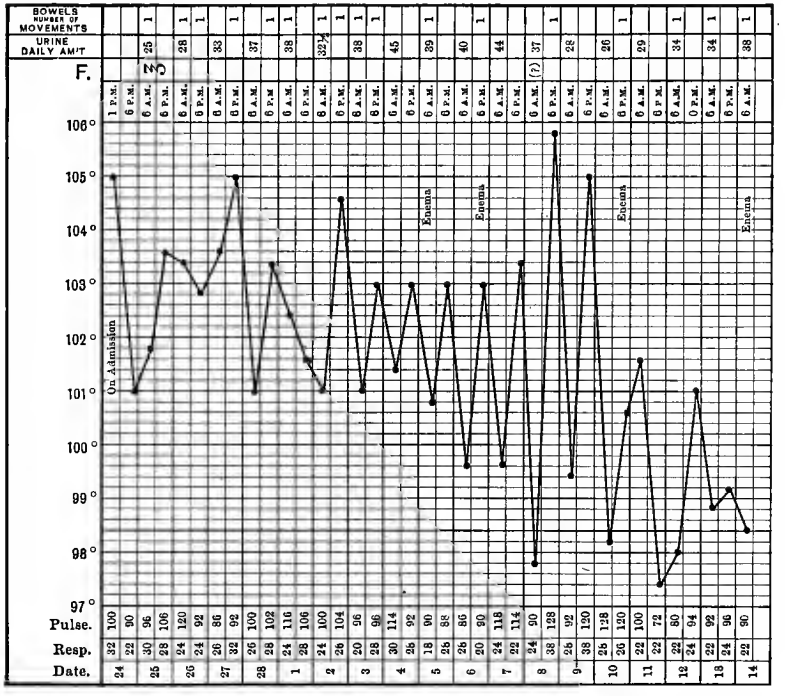


FIG. 181.—"Chills and fever" in course of typhoid fever.

ture is about one to one and a half degrees higher than in the axilla. In fat children the fold in the groin gives equally reliable results with those obtained by inserting the thermometer in the rectum.

The precautions to be taken in all cases in which a thermometer is used, in addition to those named, is to have a thermometer which is accurate, and to be sure that there is no acute or chronic inflammatory process present which will produce local heat, and so give an erroneous impression as to the actual temperature of the entire body. This is particularly apt to be the case in diseases of the mouth in children: thus, stomatitis may raise the local temperature

from 1° to 2°. Hot liquids, if taken into the mouth just previous to or during the time at which the thermometer is inserted, will so raise the temperature of the local tissues as to make the thermometer register several degrees above normal, and low records may be produced by cold liquids or ice held in the mouth. This subject has been studied by Lazarus-Barlow, who asserts that the effects of hot objects taken into the mouth last much longer than do those produced by cold, and that a mouth temperature should never be taken within one hour of the time that any hot food is ingested. He even shows that holding the mouth open for some time renders a true estimate of the body heat impossible, and advises that the temperature shall never be taken in the mouth if it is possible to take it elsewhere.

Febrile movements are generally associated with a dry, hot skin, but sometimes with a cold, wet skin. The latter condition is of evil significance, as a rule, and should be remedied if possible.

The Significance of Fever.—The significance of fever is great. It always shows the presence of an ailment sufficiently severe to make it wise for the physician to order the patient to bed until the fever abates or until he can surely determine its cause. The significance of a raised bodily temperature from a physiological point of view is that the nervous centres governing heat production and heat dissipation are disturbed by some substance circulating in the blood or by reflex irritation, or perhaps by both. The danger of high fever is that it may cause morbid changes in the protoplasm of the heart or in the vital centres at the base of the brain, but the danger of ordinary febrile temperatures has been greatly exaggerated. Indeed, in some cases moderate fever probably aids the body in combating or, rather, conquering the disease which has attacked it. This may occur in three ways, namely, by producing a temperature less favorable to the growth of certain disease germs than is the bodily temperature in health; by increasing cellular activity it may increase phagocytosis and the development of antitoxic materials; and, finally, by virtue of the increased temperature, the effects of poisons may be rendered *nil*. This is the case, for example, in regard to the drug digitalis, which will rarely produce its ordinary effects on the heart when well-marked fever is present. Another point of importance in this connection is, that the duration of fever has more to do with its importance as a symptom than has its degree, for a temperature of 105° for a few hours may be borne with impunity, whereas one of 103° for many days cannot fail to produce evil effects.

Fever in children does not possess nearly as grave significance as it does in adults, for children often develop high temperatures from slight causes and have speedy recoveries. The balance of their heat-

mechanism is easily upset. The older the patient the greater the significance of fever, and a rise of 2° or 3° in a man of sixty years is more alarming than one of 4° or 5° in a child of five or six years.

When fever is not due to a distinct pathological change in some part of the body, generally of an inflammatory kind, it may arise from a mild irritation of a mucous membrane, as when a catarrhal condition is set up. Such fevers are seen in cases of mild gastro-intestinal catarrh in children after the ingestion of bad food or exposure to cold. Sometimes fever apparently arises as the result of the reflex irritation produced by difficult teething (see chapter on the Mouth and Tongue), although in many instances the fever of dentition depends upon a more or less closely related, but overlooked, gastric catarrh. Sometimes after a urethral sound or catheter has been passed into the urethra of a man, in the course of a few minutes or hours he develops a severe chill, followed by a fever which may be quite high, but which rarely lasts long.

Fever in Infectious Diseases.[1]—Nearly all infectious diseases are ushered in by the development of fever of greater or less degree, and this is particularly true of the exanthemata. Inquiry should, therefore, be made by the physician as to the previous history of infectious disease. If one or more of the eruptive fevers have already been present, they can usually be excluded from the diagnosis of the illness present at the time of the visit. If, on the other hand, there is a history of pulmonary tuberculosis or acute articular rheumatism, this may indicate that another attack is coming on.

Typhoid Fever.—In typhoid fever the febrile movement is very characteristic in some cases, although in many instances it does not follow the description laid down in text-books. After several days of general wretchedness the patient develops a slight fever of from 100° in the morning to 101° at night, and this temperature progressively rises so that the next morning it may be 101° and that night 102°, the next morning 102°, that night 103°, and so on until the morning temperature may be 103° and the evening temperature 104° or rarely 105°. The fever usually reaches its acme by the end of the first week or ten days, and then for another week remains practically unchanged, there being a morning fall and evening rise of an almost equal extent. Toward the end of the third week, or sometimes earlier or later, according to the severity of the attack, the morning remissions become more marked, and then the evening rises fail to reach their former height. Often these marked morning remissions are the first indication of the tendency to recovery. Very high evening temperatures are indicative of a severe attack, but are not so indica-

[1] In this connection the student should also read that part of the chapter on the Skin devoted to the consideration of the eruptive infectious diseases.

tive of serious illness as are high temperatures in the morning. After the third week, in a moderately severe case, the temperature falls gradually until by the twenty-eighth day it usually reaches the normal. In very rare cases the temperature speedily reaches its acme at the very beginning of the disease, and then passes through the course already described. Such cases are generally prolonged, but may in some instances end by the fourteenth day. In other instances the development of high fever in the early stages of enteric fever, associated with severe general symptoms, is indicative of a short attack rather than a prolonged and severe one.

Sudden falls of temperature during the course of typhoid fever are nearly always of grave import. The most common cause of such a sudden fall is an intestinal hemorrhage, and the fall may occur sometimes before the blood appears in the stools. In other cases such a fall is an evidence of intestinal perforation. The other causes

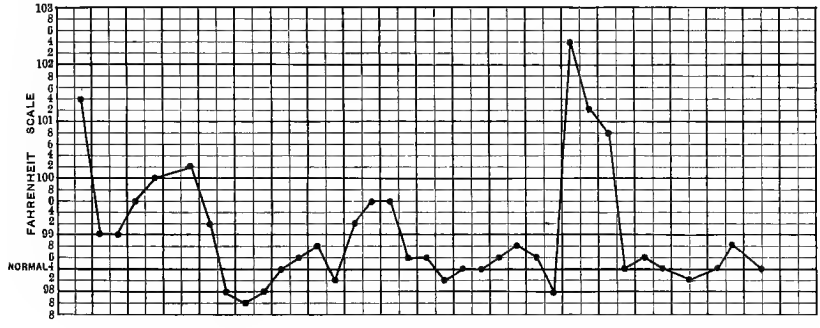


FIG. 182.—Showing recrudescence of fever in a case of typhoid fever

of a sudden fall are severe nose-bleed, or hemorrhage of any form; as, for example, that occurring in connection with abortion. Sometimes, too, without any of these causes, the temperature falls very rapidly, and the patient goes into collapse. Such cases are very grave and the prognosis is unfavorable.

A *recrudescence* or return of the fever, in which it rises quite rapidly to a point as high or higher than at any time during the attack, occurs in some persons who, during the stage of convalescence from typhoid fever, take solid food too soon, or are excited by the visit of a friend. Such rises are but temporary (Fig. 182). More rarely, as a result of getting out of bed too soon, or bad feeding, or other cause, a true relapse takes place, and the disease runs a second course, which is usually, but not always, of a shorter and milder character than the first attack (Fig. 183). Sometimes a mild, irritative fever, perhaps due to anemia, persists for some weeks, but the physician should not

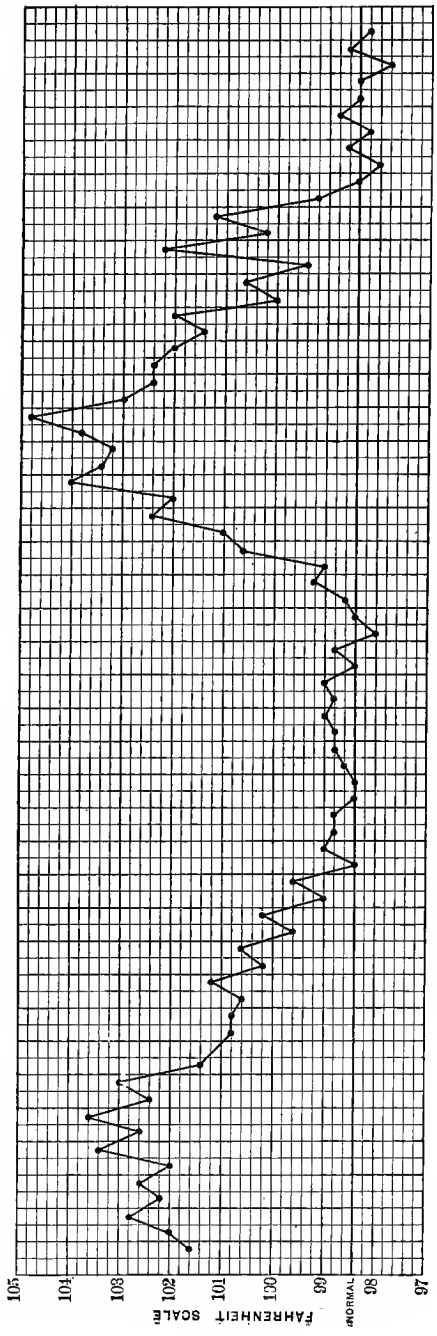


FIG. 133.—Showing a relapse in typhoid fever.

rest content with a belief that anemia is the cause until he has excluded all possibility of there being pulmonary, pleural, acute renal, or bone disease, as these conditions very commonly ensue as sequels of typhoid fever. In other instances, after the morning temperature has reached normal, the evening temperature remains pyretic for a number of days, and this may persist for some time. In a number of instances I have known the use of strychnine in full doses, at this period of the disease, to produce ranges in temperature, which ceased as soon as the use of the drug was stopped.

If the temperature in the course of a case of typhoid fever rises as high as 107° or 108°, the prognosis at once becomes very grave.

Very rarely enteric infection, so called, runs its entire course without any fever. Fisk, of Denver, and others have seen such cases, and the author had five of them at St. Agnes' Hospital in one term of service.

Strümpell asserts that as a rare occurrence the fever in this disease may become intermittent, being normal in the morning and as high as 104° at night during almost the entire illness.

The association of such a temperature curve as just described with the other characteristic signs of typhoid fever, as, for example, the development of the rose rash on the chest and abdomen, on or about the seventh day (chapter on the Skin), the ochre-colored, loose stools, the peculiar stupid, drowsy appearance of the face, the Widal test, and in some cases the peculiar typhoid odor about the patient, all make the diagnosis certain.

Irregular forms of malarial fever, particularly those forms due to infection by the estivo-autumnal parasite (see chapter on the Blood), may closely resemble typhoid fever. In many instances such cases are diagnosticated as typhoid fever, and probably some cases of true typhoid fever are thought to be malarial fever. The following differential table, drawn up by Thayer, is of interest in this connection. There is no such disease as typhomalarial fever, although there is no doubt, whatever, that pure typhoid infection may result in the production of a fever which closely follows the remittent and intermittent malarial types, and which is often associated with so much gastric disturbance and vomiting, and so lacking in the more prominent typhoid symptoms usually seen, that the picture of remittent malarial fever is clear, while the true picture of typhoid fever is clouded.

Remittent Fever.	*Typhoid Fever.*
Onset generally intermittent.	Onset gradual and progressive.
Irregular remissions.	Regular, though very slight morning remissions with evening exacerbations of temperature.
The temperature may arrive at 40° C. (104° F.) within twenty-four hours.	The temperature does not reach 40° C. (104° F.) before the third or fourth day.

Remittent Fever.	*Typhoid Fever.*
Headache rare in the beginning; of a neuralgic character, pulsating, variable in its position and intensity. Sclera subicteric from the onset.	Headache from the beginning, permanent, severe, frontal. Sclera white.
The apathetic expression of the face, the dryness of the tongue, and sordes upon the teeth are not very marked.	These symptoms are well marked and progressive.
Breath foul.	Breath has a peculiar mouse-like odor.
The delirium may come on in the early days; it is recurrent, but changes with the exacerbations of temperature and other symptoms, and may give way to grave symptoms related to other organs.	Delirium appears only when the disease is well pronounced; it is often persistent, and variable only in degree.
If there be pulmonary congestion, the cough and other symptoms come on suddenly; the areas affected change from one to the other lobe or lung, and may disappear and reappear again with varying intensity; dyspnœa is very pronounced; circulatory disturbances are marked, even syncope.	Pulmonary congestion is gradual and persistent; always hypostatic (the bases and dorsal surfaces of the lungs); the dyspnœa is less pronounced and later in appearing, depending more upon the abdominal conditions (tympanites, etc.).
There are usually restlessness and anxiety (jactitatio corporis).	There are usually relaxation, prostration, and stupor.
Peculiar grayish color of skin; sometimes a slight jaundice.	No jaundice.
Herpes common.	Herpes rare.
Anæmia more or less marked early in the course.	Anæmia absent, excepting in later stages.
No characteristic exanthem; urticaria not uncommon.	Characteristic roseola.
At times there may be transient tympanites or ileo-cæcal gurgling; they are but slightly pronounced and paroxysmal; diarrhœa is slight or absent, and has not the characters of that in typhoid fever.	Tympanites, gurgling, and diarrhœa appear slowly and may become well marked.
No distinct course.	Has a fairly characteristic course.
Urine high-colored; may show a trace of bile; Ehrlich's diazo-reaction rarely present.	Urine high-colored; bile absent; diazo-reaction present during the height of the process.
Blood shows no leucocytosis; eosinophiles not notably diminished; serum does not cause agglomeration of typhoid bacilli (Pfeiffer, Durham, and Widal); malarial parasites and pigmented leucocytes present.	Blood shows no leucocytes; eosinophiles diminished or absent; serum causes agglomeration of typhoid bacilli; malarial parasites and pigment absent.
Fever disappears under quinine.	Fever uninfluenced by quinine.
Is an endemic disease occurring particularly in rural districts; rarely epidemic.	Usually epidemic; prevailing commonly in cities.

Again, there can be no doubt that cases of true malarial infection occur in which the symptoms so closely resemble those of typhoid fever that a purely clinical diagnosis is almost impossible, particularly if an epidemic of typhoid fever is in full swing at the time. Finally, there can also be no doubt that it is possible for the patient to have a double infection with the bacillus of Eberth and the plasmodium of Laveran, in which case, however, the malarial mani-

festations are usually dwarfed by the typhoid infection, and are only marked at the onset of the enteric fever and at its termination. To this mixed infection the term typhomalarial fever may be correctly applied to indicate not a separate disease, but a double infection. Etymologically, this term might also be used to define a condition of malarial fever in which, because of profound debility, the patient was in a typhoid state—that is, in a condition of which typhoid fever is a type. Practically, however, it should be discarded or limited in its use to the double infection just described. For Malarial Fever, see page 455.

The febrile movement and other symptoms of enteric fever are often imitated very closely by those of *ulcerative endocarditis.* In addition to an irregular fever, there may be diarrhea, parotitis, stupor, and progressive feebleness in both diseases. An examination of the heart may reveal the presence of endocardial murmurs, which in association with signs of sepsis, or the existence of some focus of infection, such as a wound, a septic process, or the fact that the patient is in the puerperium, will render a diagnosis possible. (See also page 457.)

Acute Tuberculosis.—The differential diagnosis of acute tuberculosis from typhoid fever may be quite difficult in certain cases. When the symptoms of the two conditions are compared this is not difficult to believe, for we often have in both diseases headache, epistaxis, a very similar temperature chart, and a feeble pulse, while there may be in both conditions an eruption on the skin, which rather tends to confuse the physician than to aid him. Again, the delirium in each case is very similar, and the facial expression of the patient in both diseases is apathetic. Even the respiratory sounds in both diseases in their early stage may be apparently only those of a moderate bronchitis; and, finally, abdominal swelling, tympanites, and meteorism may occur in both maladies. Under these circumstances the hereditary and recent history of the patient may be of much value, as showing a tendency to tuberculosis on the one hand, or exposure to typhoid infection on the other. Again, if it be typhoid fever, the spleen on percussion is nearly always found to be enlarged. Then, too, the lesions in the lungs of a typhoid fever patient are generally at the bases, while in tuberculosis they are oftener at the apices. The stools may be loose in both diseases, but in typhoid fever they are apt to be ochre-colored; and, again, in tuberculosis the loss of flesh is often exceedingly rapid, and profuse sweats and high fever are frequently seen. The mental apathy in typhoid fever is more marked, as a rule, than it is in tuberculosis. The finding of Widal's reaction in the blood, or the discovery of the bacillus of Eberth in the feces and the presence of the diazo-reaction in the urine, would, of course, indicate typhoid fever. (See chapter on the

Urine.) An absence of leukocytosis would also indicate typhoid fever, for this condition is usually present in acute tuberculosis. Finally, careful and repeated examinations of the chest will usually, in the course of the disease, demonstrate the presence of tuberculosis of the lungs or bowels, if this be the cause of the illness. It seems hardly necessary to state that if any expectoration exists the sputum is to be carefully examined for tubercle bacilli in all doubtful cases, but while their presence proves tuberculous infection to be present, their absence does not prove the absence of this infection.

Trichinosis.—An irregular fever with muscular pains and a great deal of discomfort in the belly, the case simulating typhoid fever, may occur in cases of trichinosis.

Malta Fever.—A febrile movement closely resembling that of typhoid fever, a resemblance which is increased by the association with it of headache, insomnia, and anorexia, may be Malta fever, a disease which can be excluded in the vast majority of cases if there is no history of exposure to the exciting cause in the island of Malta, although it is not to be forgotten that Malta fever has been met with in persons returning from Porto Rico and elsewhere during the last few years. Sometimes it may be confused with relapsing fever, except for the longer febrile movement. Thus, after three or four weeks of illness convalescence seems to be established, and the temperature falls, but in a few days all the symptoms return with even greater vehemence than before. Such relapses may occur again and again. Violent pain in the joints on moving the body is often present. In doubtful cases the agglutination test with the blood may be performed, if it is possible to obtain cultures of the *Bacillus melitensis.*

Typhus Fever.—The temperature chart of typhus fever is so different from that of typhoid fever that it gives us a valuable differential point at the very beginning of the disease, for, after several days of languor, headache, and pain in the limbs, the fever suddenly springs on the patient, so that on the first night it may reach 105° F. Often it reaches 106° in a day or two, and while present is constant, the morning fall being very slight indeed. The development of the spots in a copious eruption on the third to the seventh day, which spots may develop into petechiæ before fading, or remain unchanged in appearance, the great exhaustion, the severity of the illness, and the sudden rise of temperature, followed by a constant fever, point to typhus fever. Finally, the conclusion of the febrile movement, in favorable cases by the end of the second week, by crisis or by a more rapid fall of temperature than we are accustomed to see in typhoid, all help to make the differential diagnosis, which is, however, in many cases very difficult or impossible in the early stages.

Relapsing Fever.—The temperature of relapsing fever nearly always rises suddenly at the beginning of the attack to from 103° to

105°, and remains high with slight morning remissions from three to seven days, when it suddenly falls as by crisis to the normal or below it, after being on the preceding afternoon or evening unusually high. Sometimes it falls as low as 92° or 93°. The patient now remains free from fever for from several days to two weeks, when with a sudden leap the fever and other symptoms of the first attack recur. A temperature of 105° to 106° in relapsing fever rarely indicates a grave outlook, as it does in typhoid. The only condition which resembles this temperature range of relapsing fever is intermittent malarial fever; but the rarity of relapsing fever in America, the frequency of malarial fever in certain parts, the presence of the spirillum of Obermeier in the blood in relapsing fever, and the malarial germ in the blood of intermittent fever, all make the diagnosis possible.

Scarlet Fever.—In scarlet fever the temperature suddenly rises on the first day to 104° to 105°, and still higher on the next day, and then remains constant as long as the eruption is on the skin in full

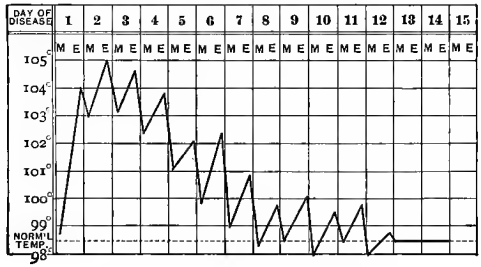


FIG. 184.—Chart of scarlet fever.

development. Just so soon as the eruption begins to fade the temperature also falls, not by crisis, but by lysis; not so slowly as in typhoid fever, but far more slowly than in croupous pneumonia (Fig. 184). This arrest of the fever usually takes place in simple cases by the end of seven days; and if it persists longer, is probably due to some complications, such as otitis, or the "collar of brawn," due to enlarged cervical glands. The characteristic strawberry punctated rash and scarlet hue appearing on the first or second day, the ultimate dermal desquamation, the violence of the onset of the symptoms, the sore throat, and the peculiar appearance of the skin, all complete the clinical picture, particularly if the symptoms be in a child. (See chapter on the Skin.)

In rare cases the fever in scarlatina is remarkably mild or almost absent, and these cases, as a rule, have a favorable prognosis. If the temperature be very high and persistent, on the other hand, the case is usually to be regarded as most grave.

Measles.—In measles the fever at first rises sharply to 103° or thereabout, then falls to a little above normal, is slight for several days, and then markedly increases with the development of the eruption on the fourth day, often ranging as high as 104° or 105°, at which point, with little variation, it remains for the two days during which the rash is well developed (Fig. 185). With the fading of the rash the temperature also falls by crisis. If fever persists to any extent, it is always due to some complicating cause other than the original disease; such a complication, for example, as a bronchial or gastro-duodenal catarrh.

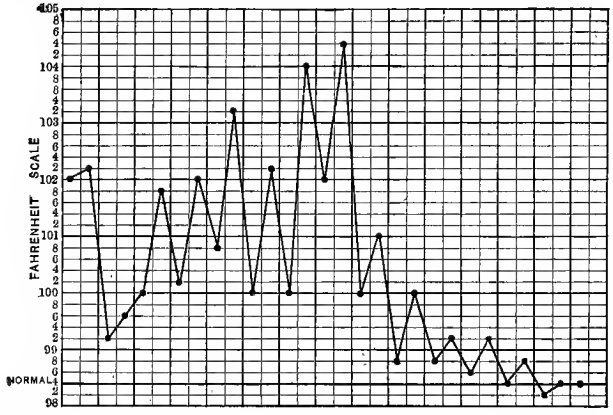


FIG. 185.—Showing initial fever with the subsequent fall and then a rise when the rash is well developed in a case of measles. Also shows an ending of the fever by crisis.

Rotheln.—The fever of rötheln, if any occurs, is very seldom more than 102°, and has no typical preliminary rise as has measles, so that the temperature chart of the disease may aid materially in a differential diagnosis. (See chapter on the Skin.)

Smallpox.—The febrile movement of smallpox is, with the exception of that of typhoid, the most characteristic of all the eruptive diseases. With a sudden onset of fever, pain in the back, severe headache, and malaise, the patient takes to his bed if possible, and his temperature if taken will be found speedily to rise even to 105° or more in some cases, and then falls back to almost normal for two or three days, during which time the eruption appears. In this way, therefore, the temperature chart of variola differs diametrically from that of the eruptive fevers so far discussed, for in these cases the fever rises with the appearance of the eruption, whereas in this instance the temperature falls with the appearance of the eruption. This lower temperature persists for several days, from $\frac{1}{2}$° to 1° above normal, until the ninth day of the disease or the sixth of

the eruption, when with the change of the pocks from vesicles to pustules the temperature rises again in what is called the fever of suppuration, which lasts with greater or less persistence for at least a week, when it ends by lysis or a gradual fall. Excessively high fever of 108° is a sign of approaching death or at least of very grave import.

Varicella.—The febrile movement of varicella, or chickenpox, is usually of very short duration and of little severity; but it may reach proportions entirely out of consonance with the general systemic disturbance, which is usually very slight in previously healthy children. Thus, it may rise in children who are prone to active febrile movements to as high a point as 105° for a very brief period, and yet may not seem to render the child ill.

Erysipelas.—The temperature range seen in cases of erysipelas is quite typical. At the beginning of the attack the rise is quite prompt and sharp to 105° or 106° or even above this, and, instead of remaining constantly high through the course of the inflammatory process in the skin, goes through marked intermissions or remissions, which frequently occur and are followed by rises in temperature as high as that which occurred with the first onset. The fever ends in some cases by crisis and in others by lysis, the latter mode of ending usually taking place in those cases which have had a very severe attack prolonged in character, or which have been in an asthenic state prior to the disease. The diagnosis of erysipelas is easily made by the brawny, swollen, and red skin, with the peculiar line of demarcation at the edge of the swelling. (See chapter on the Skin.)

Intermittent Malarial Fever.—A fever which rises sharply from normal to 103° or 104°, being preceded by a chill and followed in a very few hours by a sweat, the whole term of acute illness, if we exclude general physical discomfort, lasting but eight to twelve hours, is in the majority of cases that of intermittent malarial fever. The peculiarities of intermittent malarial fever, aside from those just named, are that the febrile movement begins to decline before the stage of sweating begins, and in some cases it begins to rise before the sensation of chilliness of the first stage leaves the patient. (See Fig. 186.)

The fall of temperature is usually less abrupt than the rise, and is sometimes delayed by slight temporary rises or arrests in its downward course. The febrile movement is repeated at intervals, ranging from one to seven days or even at longer intervals than this. If the attacks occur daily, they are called quotidian, and this is due to infection by two sets of tertian parasites which undergo segmentation on alternate days, or it may be due to infection with three sets of quartan parasites. If the attacks occur every other day, they are

called tertian (Fig. 187); if on the third day, quartan; if on the fourth, quintan. If two attacks come on the same day, it is called double quotidian.

Another point of importance in connection with malarial attacks is that they often occur earlier each day by an hour or more. Rarely, they are delayed.

Intermittent malarial fever is to be separated from other intermitting fevers by a number of facts. First, the presence of the malarial organism in the blood at the time of the attack, or evidences

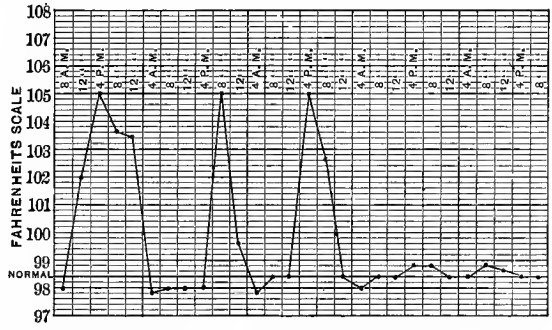


FIG. 186.—Showing daily paroxysm due to double tertian infection. One set of parasites segmented at 8 P.M. and the second set at 4 P.M. Paroxysm stopped by quinine on fourth day.

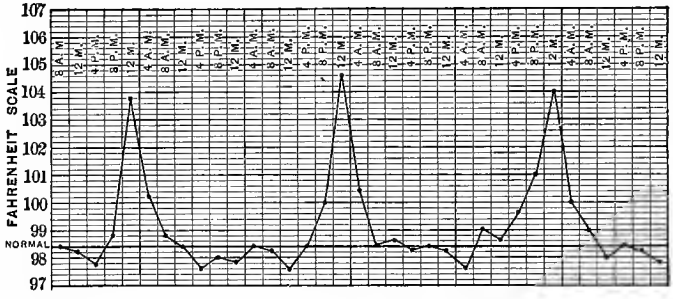


FIG. 187.—Showing paroxysms of tertian fever, the segmentation of the organism occurring at about twelve o'clock every other day.

of its presence at other times. (See chapter on the Blood.) Second, by the history of exposure to malarial influences. Third, by the marked effect for good on malarial fever produced by the administration of quinine.

As stated in the chapter on the Blood, an examination of this fluid will reveal in practically every form of infection, except malaria and typhoid fever, an increased leukocytosis; but in malarial infection the leukocytes are not increased in number.

Care must always be taken that the intermitting fever of the

various forms of sepsis is not diagnosticated as malarial intermittent fever. The most common error of this character is the making of a diagnosis of irregular malarial intermittent, because chills, fever, and sweat appear every evening, when, in reality, the real cause is an undiscovered pulmonary or abdominal tuberculosis. Again, acute ulcerative endocarditis and purulent phlebitis may cause similar symptoms, as may also hepatic abscess, impaction of gallstones, with suppurative cholangitis, causing the so-called Charcot's fever (see below). The absence of a history of malarial exposure, the possible presence of a cough, and the discovery of a tuberculous lesion in the chest or abdomen by careful physical examination will aid in deciding that the fever is tuberculous and not malarial in origin. (See chapters on the Thorax and on the Abdomen.)

Ulcerative Endocarditis.—The temperature curve may exactly resemble intermittent malarial fever; but in many instances the presence of an external wound, acute sepsis in some part of the body, or the presence of the puerperium will reveal the source of an infection. (See Fig. 188.) In the typhoid type of ulcerative endocarditis the profound asthenia and general prostration will separate the disease even if the temperature chart be useless. In this form the febrile movement is rarely typically intermittent. The crucial test of the differential diagnosis lies in an examination of the heart, in which a murmur may be heard in some but not in all cases, unless there has already been some grave valvular mischief. The cardiac feebleness and asthenia, on the one hand, and the result of the blood examination, on the other, aid the diagnosis. The duration of the case is not of much value in making a diagnosis, for cases of ulcerative endocarditis have lasted from two days to more than a year. Rarely, it lasts more than six weeks. Death usually occurs in ulcerative endocarditis, unless there has been previously present chronic endocarditis, in which case recovery may rarely occur.

The discovery of some spot showing a *phlebitis* may point to this cause for intermittent fever.

The fever of *catarrhal* or *suppurative cholangitis* often closely resembles intermittent fever, but the presence of hepatic symptoms, of marked jaundice, of a history of gallstone colic, and of exceedingly severe rigors, enables us to separate them. In obscure cases the malarial organism should be searched for, and if the condition be one of cholangitis an examination of the blood will probably show leukocytosis. (See chapter on Pain, Gallstone Colic.)

When fever of an intermittent type has been observed, and intermittent malarial fever, tuberculosis, and cholangitis cannot be discovered as a cause, search should be made for tenderness and swelling of the liver due to *hepatic abscess*. Profuse sweats also may be found in such cases, as in most instances of septic fever. The diag-

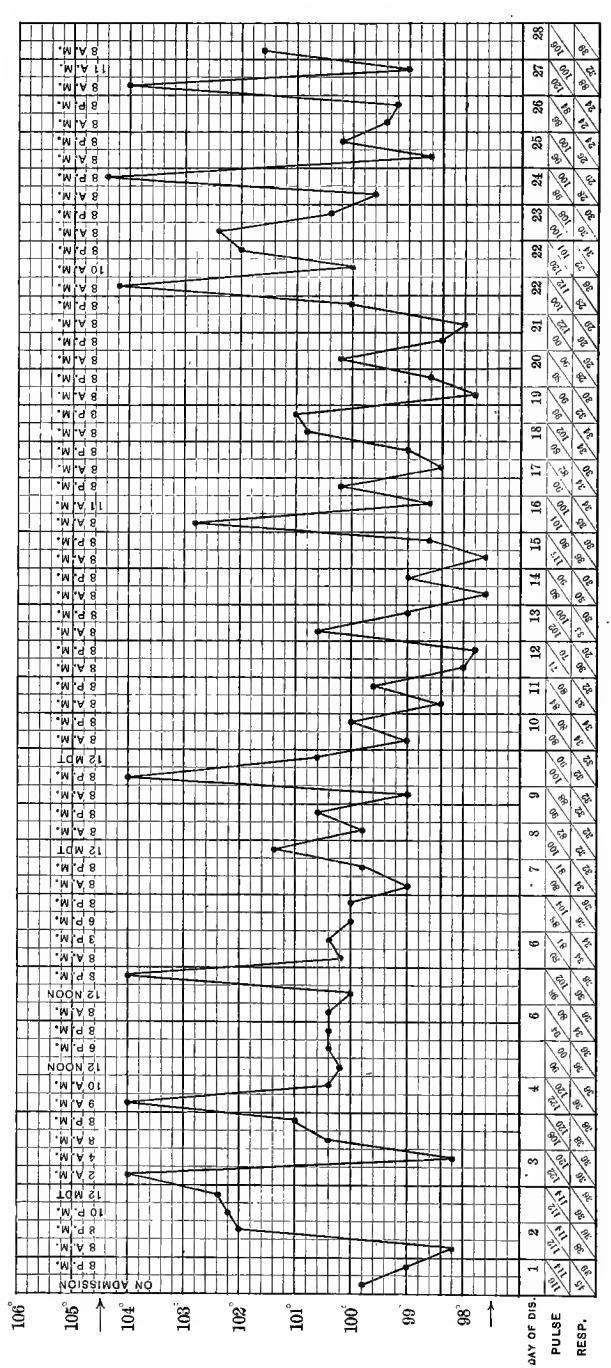
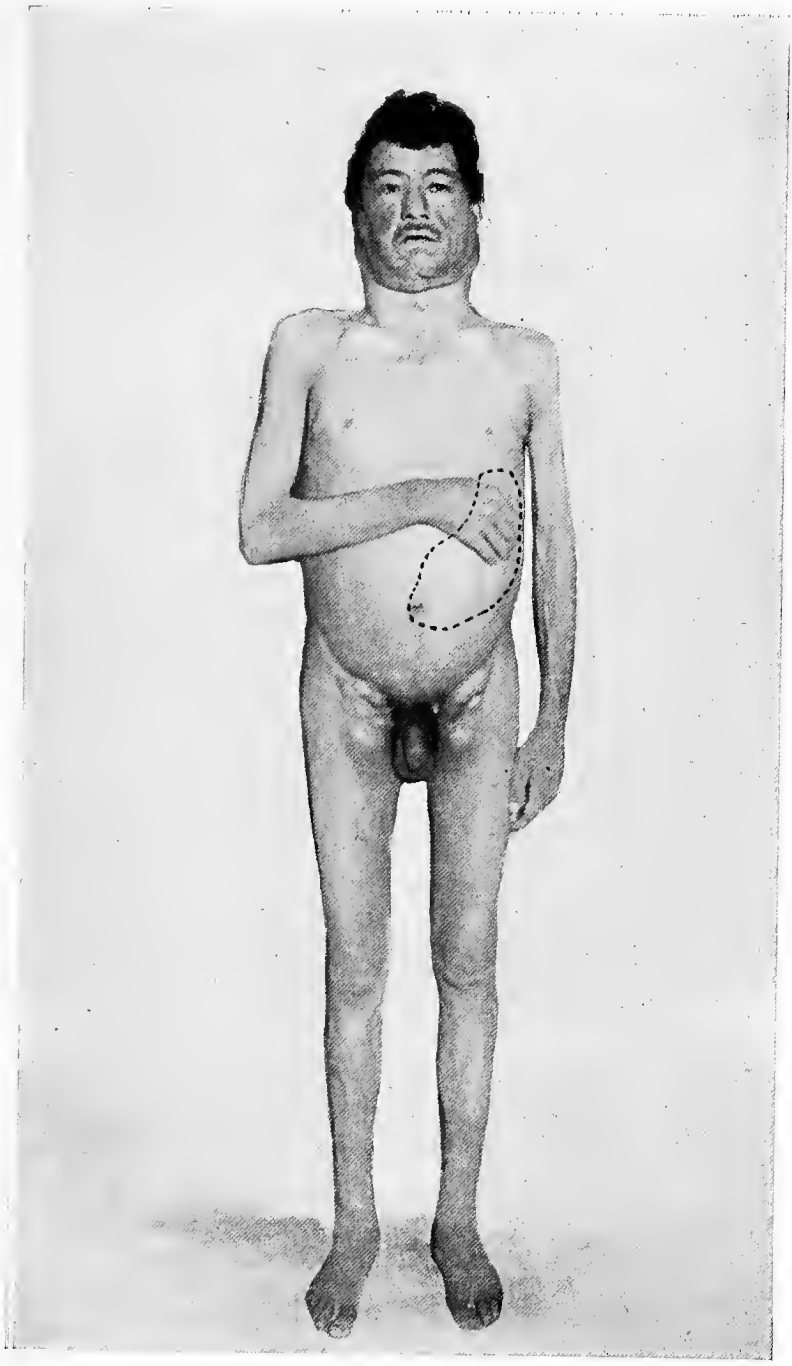


FIG. 188.—Showing temperature curves in a case of ulcerative endocarditis. (From a case in the author's wards.)

PLATE XVI.



Hodgkin's Disease. (De Forest's Case.)

nosis of hepatic abscess will be strengthened if there is a history of the patient having suffered from dysentery, as hepatic abscess is sometimes caused by amebic dysentery.

The presence of fever preceded by chills, the temperature rising to 104° or even 105°, followed by excessive sweats, in a person who is profoundly cachectic, may be due to *pernicious anemia* or to *septic poisoning*, as already pointed out; and it should be recollected that such a temperature chart is sometimes seen in cases of gastric cancer. Similar symptoms as to fever in association with enlargement of the lymphatic glands, particularly those of the neck, indicate Hodgkin's disease (see chapter on the Blood), or even more commonly tuberculous adenitis, which, however, is usually met with in the young and involves the glands near the jaw, while in Hodgkin's disease the glands near the clavicle are affected. Further, in Hodgkin's disease the swelling is usually bilateral, and to be found elsewhere than in the neck. (See Plate XVI.) Again, in tuberculous disease these glands often suppurate. The presence of the tubercle bacillus in an excised piece of the swelling will decide the diagnosis. In the opinion of some clinicians the two states are identical in cause. An intermittent fever may also be seen in *suppurative pyelitis*, in association with pyuria. This pyelitis may or may not be tuberculous.

Remittent Malarial Fever.—Remittent fever rising and falling every few days for two or three weeks, rarely rising above 103° to 104°, and even falling to the normal line, associated with enlargement of the spleen and liver, yellowing of the skin, or jaundice, bilious vomiting, and a history of exposure to malarial infection, is characteristic of remittent malarial fever, a form more chronic and very much more grave than the intermittent form just described, because it responds less readily to treatment; and, secondly, because it is accompanied by more marked changes in the viscera. It depends upon infection with the estivo-autumnal form of the malarial parasite. The conditions produced by this parasite are collectively grouped under the names remittent, continued, bilious remittent, and typho-malarial fever, or malarial fever of a typhoid type. In some cases the temperature and other symptoms will so closely resemble those of typhoid fever that nothing short of an examination of the blood can decide the diagnosis. (See chapter on the Blood.)

Care should be taken to recollect the fact that when typhoid fever develops in a young child the temperature may be so markedly remittent that an erroneous diagnosis of malarial infection may be made. In other words, "infantile remittent fever" is really typhoid fever in many instances.

A febrile process somewhat closely resembling remittent malarial fever, yet so rare, comparatively, as never to be confused with it, is *Weil's disease*. In this condition the fever runs a remitting course,

is associated with jaundice and swelling of the liver and spleen, and the stools may be clay-colored. There is one important point of difference between malarial remittent fever and Weil's disease, namely, that in the latter gastro-intestinal symptoms are nearly always wanting or are mild, whereas in the former they are apt to be marked. Usually the fever of Weil's disease ceases by the end of two weeks or earlier. It is probably an infectious jaundice.

Dengue.—In dengue, a disease seen most commonly in epidemics in certain parts of the southern United States, the patient, after suffering from violent aching pains in the body and limbs, swelling of the joints, and the development of a variable rash on the chest, develops an active fever, which lasts with the pain until the fifth day, when both the pain and fever decrease or cease, and then often return with equal force. These facts, combined with the fact that it is an epidemic disease, separate it from malarial fever. Dengue and influenza, of an epidemic type, closely resemble one another, but in dengue there is rarely marked involvement of the respiratory tract as there is in influenza; there is an eruption which is not seen in influenza, and it is not followed or accentuated by such grave complications as we see in the more severe cases of influenza. Dengue is a disease of the South and influenza one of the North.

Yellow Fever.—The fever of yellow fever is rarely over 103° or 104°, and is one of the milder symptoms of the disease; but it possesses this peculiarity, namely, that after the lapse of from twelve hours to several days there is a marked remission of the fever and all the other symptoms, and from this time on the patient may get well, or after a few hours this calm stage is followed by the true violent symptoms of the disease, such as black vomit, tarry stools, jaundice, and hemorrhages from the mucous membranes. Generally the full course of the disease to convalescence or death is run in about one week.

There are only two other diseases which can be readily confused with yellow fever, namely, dengue and bilious remittent fever. Dengue has been confused with yellow fever many times, and even the most experienced physicians have had great difficulty in separating them when the yellow fever has been mild. The most important points in their differentiation are the facts that in dengue there is usually a second onset of fever several days after the first onset, whereas this does not occur in yellow fever. Again, the eruption on the skin is not seen in yellow fever, and a rapid pulse is present in dengue, whereas in yellow fever the pulse is usually not very rapid. On the other hand, in yellow fever we usually meet with jaundice, albuminuria or suppressed urine, and a hemorrhagic tendency of a marked degree, all of which are absent in dengue. Further, death from dengue is very rare.

A case of bilious remittent fever occurring during an epidemic of yellow fever is almost certain to be incorrectly diagnosticated. In the absence of an epidemic, however, the probabilities of the case being bilious remittent fever are very great, and the presence of bilious vomiting rather than that of blood, the characteristic temperature chart, and, above all, the presence of a history of malarial exposure and of the signs of malarial infection in the blood, with the partial control of the symptoms by quinine in certain stages of remittent fever, point to the diagnosis of malarial disease rather than to yellow fever.

Stubbert gives the following differential tables of these fevers:

Yellow Fever.	*Pernicious Malaria.*
Headache bilateral-frontal, and post-orbital.	Headache generally unilateral-frontal, and temporal.
Temperature and pulse divergent; temperature rarely higher than 104° F.	Temperature and pulse correlative; temperature generally 105° to 107° F.
Albumin present in large quantities early in the disease.	Albumin rarely present.
Quinine has no effect on the progress of the disease.	Quinine has a specific effect if given hypodermically and early.
Stage of remission on third or fourth day.	Remission not present.
Attacks new arrivals.	Generally history of chronic malarial infection.
Always history of exposure to infection.	No history of exposure to infection.
Black vomit appears on third or fourth day.	Black vomit appears within thirty-six hours.
Hematuria very rarely present.	Hematuria a marked symptom.
Liver unchanged.	Liver enlarged and tender.
Yellow Fever.	*Dengue Fever.*
Cephalalgia and nephralgia are characteristic and constant.	Pain most severe in joints and muscles, and is *paroxysmal.*
Pulse and temperature divergent.	Pulse and temperature correlative.
The slowing of the pulse begins early in the disease.	The slowing of the pulse occurs late in the disease.
Congestion of face early in the disease. No edema.	Rash on face, followed quickly by edema.
Albuminuria.	Albuminuria absent.
Icterus.	Icterus absent.
Black vomit.	Black vomit absent.
No eruption.	Polymorphous eruption, followed by desquamation.

Cerebrospinal Meningitis.—Just as in yellow fever, so in spotted fever or cerebrospinal meningitis of an epidemic form, the fever itself is one of the least important symptoms, for, aside from the fact that it is apt to be irregular and intermitting, it is rarely very high, as compared with the violent cerebrospinal symptoms, the rigidity of the back of the neck, the headache, convulsions, and vomiting. The presence of these symptoms in an epidemic does more to confirm a diagnosis than the febrile movement. In some cases of spotted fever, however, of a very grave type, the fever becomes a hyperpyrexia, but in cases tending toward recovery the temperature usually begins to fall by lysis before any moderation in the other symptoms is manifested.

When the physician feels it necessary to determine the true character of the affection without delay, he may resort to lumbar puncture. An ordinary aspirating needle is introduced between the second and third, or third and fourth lumbar vertebræ (Fig. 27), and by this means some of the cerebrospinal fluid is obtained, which should be received in a sterile test tube, and examined microscopically for the characteristic diplococci of spotted fever, and for other microorganisms. The needle should be inserted 4 cm. (1¼ inches) in children, and 6 to 8 cm. (2 to 4 inches) in adults, to reach the fluid around the cord. It is not a dangerous operation. Pfaundler, on the other hand, recommends that the puncture be made in the lumbosacral space, and that it should be performed while the patient is in the sitting position. (For area for puncture see the figures on the spinal column in the chapter on the Feet and Legs.)

He believes that very high pressure of the fluid in the spinal membranes is present in tuberculous meningitis, and that this high pressure is so rare in other conditions that its presence is of diagnostic value. It is found, however, when the patient is suffering from purulent meningitis, from spinal tumor, and in some cases of functional neurosis. On the other hand, normal pressure excludes the presence of meningeal or cerebral affections. If the fluid which is obtained contains disintegrated blood, the patient is probably suffering from pachymeningitis or injury. If, on the other hand, the blood is fresh in its appearance, its presence is probably due to the puncture.

A very important point in this connection, insisted upon by Pfaundler, is that if the fluid is clear, every inflammatory affection of the meninges, except tuberculosis, may be excluded from the diagnosis. In tuberculosis, it is often cloudy, and in the later stages of the disease nearly always so. If the fluid is purulent, it indicates the presence of purulent or epidemic meningitis. A clear fluid is found in health, in serous meningitis, hydrocephalus, and functional neuroses, and it is generally clear in cases of tumor, uremia, and sepsis. The albumin present varies from 0.02 to 0.04 per cent., and if it is in excess of 0.05, it is probable that the disease is an inflammation or tumor; if it is present in still larger amounts, it is probably tuberculous meningitis. If, again, on analysis of the fluid the normal sugar which it contains should disappear, there is probably an inflammatory process present, and therefore if sugar is present an inflammation can be to a certain extent excluded.

Even in the presence of an epidemic of spotted fever it should never be forgotten that middle-ear disease often causes marked meningeal symptoms, and that croupous pneumonia often produces a similar train of manifestations, probably by infection with its particular microörganism. The possibility of tuberculous infection

producing such symptoms should cause the physician to examine the patient carefully for signs of tuberculous disease in other parts of the body from which infection might arise, as, for example, the lungs.

Septicemia.—The fever due to septicemia may produce a temperature chart which closely resembles that of enteric fever, but septic fever generally possesses one characteristic which, in the face of other symptoms suggesting sepsis, is of great importance, namely, the extraordinary rises from normal to 105° or 106°, and from that point even to a subnormal degree within a very few hours, so that the lines on the chart pass up and down in long sweeps. These sweeps are even more sharp and sudden than in an intermittent malarial fever, and their cause is determined by the discovery of some septic process in some part of the body. The presence of such a chart, in association with dull or violent headache, delirium, vertigo, and vomiting independent of taking food, would point to cerebral abscess, particularly if a history of injury could be obtained.

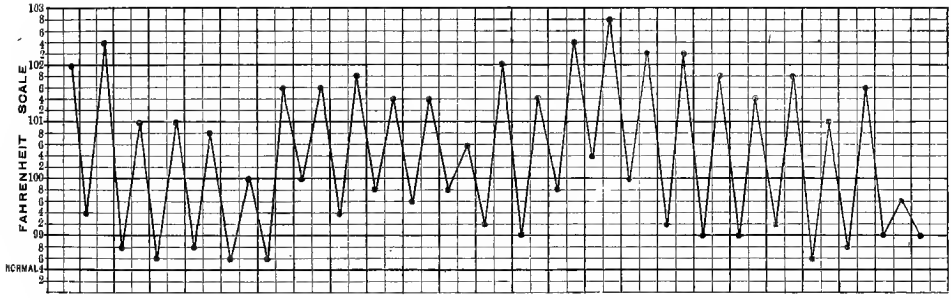


FIG. 189.—Chart of a case of pulmonary tuberculosis, showing rising and falling of the temperature morning and night.

A somewhat similar chart to this may occur in connection with cases of *active pulmonary tuberculosis*, when the lesions are well developed and septic absorption is active; but usually in the hectic fever of phthisis we have an approximately normal morning temperature, with a rise from 2° to 3°, or even more, toward night (Fig. 189).

This symptom of fever in any form occurring in a person with suspiciously "weak lungs" should cause the physician to be confident that he has overlooked some focus which another careful examination may discover, and it possesses another important diagnostic significance, namely, that the more active the febrile movement in phthisis pulmonalis the more active the disease process, and the less active the fever the less active the process. Fever may, however, be almost entirely absent in some tuberculous cases with extensive disease of the lungs.

Acute Miliary Tuberculosis.—The febrile movement of acute miliary tuberculosis has nothing characteristic about it, except that in some cases it may closely resemble that of typhoid fever, and if the physician does not carefully examine the case an erroneous diagnosis may be reached. If, however, the disease involves the meninges of the brain, a hyperpyrexia may be developed, and death speedily occurs in such cases, either in the fever or in a sudden collapse. The peculiar dyspnea, the cyanosis, the profuse sweats, and the diffuse pulmonary signs render a diagnosis of acute miliary tuberculosis possible in some cases.

Influenza.—When fever is associated with marked catarrhal symptoms, chiefly of the bronchial tubes and upper respiratory tract, with sneezing, lassitude, pains in the back and limbs, and excessive cough, the fever rising as high as 104° or 105° in severe cases, and then falling almost to normal, we may have before us influenza or catarrhal fever either of the sporadic or epidemic form. In this condition there may be in severe cases great prostration and cardiac failure or vomiting and diarrhea. The febrile movement is of the most irregular type, even when some grave complication, such as severe bronchitis or pneumonia, comes on, although croupous pneumonia rarely occurs as a complication of "la grippe."

Hay Fever.—The respiratory symptoms just described are also seen frequently in association with moderate fever, in "*hay fever*," that condition seen in susceptible persons during the haying season or late in the summer.

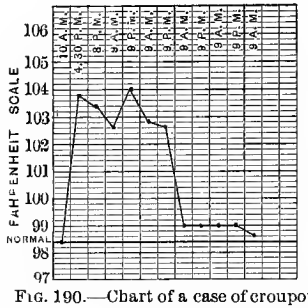


FIG. 190.—Chart of a case of croupous pneumonia, showing primary rise of temperature to 103 4° and crisis occurring as early as the third day.

Croupous Pneumonia.—The fever of acute pneumonia of the croupous type runs a very typical course in uncomplicated cases. Following a more or less severe chill, the fever quickly mounts to the high point of 103° or 104°, or even more than this (Fig. 190). For the next few days, if not modified by the antipyretics and the use of cold, the fever remains high; but there may be temporary remissions which look as if crisis was about to be established, when in reality they are followed at once by a return of the fever (pseudocrisis). Finally, in the majority of cases of croupous pneumonia the temperature suddenly falls by crisis on the seventh to ninth day (Fig. 191), and convalescence is established, although the sudden fall of fever may be associated with dangerous collapse. Sometimes convalescence is broken by brief and slight febrile movements. If the case

has been prolonged, or if it is of the typhoid type, the fever may end by lysis.

Catarrhal Pneumonia.—It is to be remembered that the fever of catarrhal pneumonia is rarely as high as in the croupous form, usually 101° to 103°, and ends by lysis, not crisis. (See chapter on the Thorax.)

The fever of *acute bronchitis* possesses no peculiarities over that of other acute inflammations.

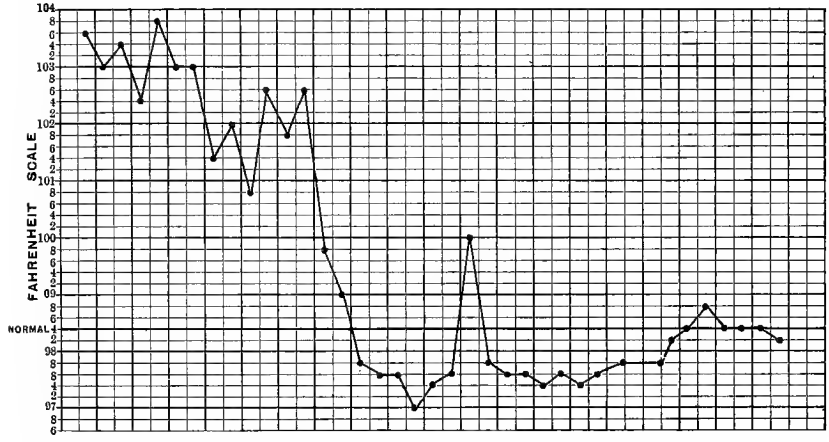


FIG. 191.—Chart of a case of croupous pneumonia, with crisis on the seventh day; admitted to the author's ward on second day of illness.

Syphilis.—It is not proper to leave the subject of fever due to the various infectious diseases without calling attention to that due to syphilis in the secondary period of its course. With the onset of the roseola or other skin lesion a fever, more or less marked, is nearly always present and is often preceded by chilly sensations and general malaise. This febrile movement may then follow one of three courses: it may never rise above 101°, and proceed as does a simple fever, with slight morning remissions and evening exacerbations; or it may be as remittent as is a malarial remittent fever; or, again, it may resemble a malarial intermittent, rising to a high point and then falling almost to the normal. Phillips, of London, has reported a case of syphilitic fever in which this febrile movement lasted for weeks, and, after being treated by quinine as a supposed tertian ague, ended at once under antisyphilitic medication. (See chapter on the Skin, Eruptions.)

Acute Articular Rheumatism.—The fever of ordinary cases of acute articular rheumatism is usually moderate, rarely exceeds 103°, and possesses no typical characteristics; but in very severe forms

of the disease with cerebral manifestations, a rheumatic hyperpyrexia may be developed, when, with delirium, convulsions, and cyanosis, the fever rises to 105° and even to 108°, after which death often ensues. The history of previous attacks of articular rheumatism, the hot, swollen joint or joints (usually the large ones), and the successive invasion of other joints as the ones first affected get well, point to the correct diagnosis. It must not be forgotten, however, that gonorrheal and other forms of septic arthritis occur with febrile movement. Pyemia, osteomyelitis, and purpura also may produce a fever with swelling of the joints. (See chapter on the Legs and Feet.)

Thermic Fever.—When a person, previously afebrile, during hot weather or when exposed to artificial heat in excess, is attacked by unconsciousness, convulsions, and very high fever, he is probably suffering from thermic fever or heat-stroke. Theoretically similar symptoms might be caused by a lesion due to embolism or hemorrhage in the neighborhood of the pons Varolii, but this is very rare. (See chapters on Hemiplegia and the Face and Head.) The fever in sunstroke may rise as high as 110° or 112° or even more; the skin is hot and dry, or more rarely cold and moist with sweat; but, even if this is the case, the rectal temperature will be found hyperpyretic.

A great rise of temperature (110° to 112°) often occurs after injuries to the cervical region of the spinal cord.

Acute Multiple Neuritis.—The rapid development of fever, pain in the back and limbs, and particularly in the nerve trunks, the temperature soon reaching 103° or 104°, may be due to an attack of acute multiple neuritis, and the history that the illness has followed exposure to cold and wet may, on the one hand, make the physician believe that his case is suffering from rheumatism or influenza, or on the other, in the absence of such a history, from the early stages of one of the infectious diseases. The early appearance of tingling, numbness, loss of power, and wasting of the muscles soon decides the diagnosis in favor of neuritis. The nervous disease which most closely resembles acute febrile neuritis is Landry's paralysis, and a differential diagnosis may be difficult; but in neuritis there are loss of sensation, muscular wasting, signs of degeneration, and fever, whereas in Landry's paralysis all these are wanting, excepting the sensory symptoms, which in both diseases may be similar. The predominant symptoms of Landry's paralysis are paralysis and loss of reflexes. (See chapter on the Legs and Feet.)

The prognosis of the severe form of febrile neuritis is grave, as death may ensue from respiratory paralysis.

SUBNORMAL TEMPERATURE.

Subnormal temperature of the body is seen as the result of any profound nervous shock, as after an accident or surgical operation, or prolonged anesthetization. It occurs, too, at the ending of the fever of croupous pneumonia and other febrile movements ending by crisis. It is also seen in severe cholera morbus and cholera Asiatica and sometimes in cholera infantum, and often is present either in the early part of the cold stage of intermittent malarial attacks or more commonly after the fever of the attack has fallen. A subnormal temperature of a dangerous degree is met with in the algid type of pernicious malarial infection, and can only be satisfactorily differentiated from other conditions by a blood examination. Subnormal temperatures are also seen in some cases of confusional insanity and of tuberculous meningitis and hysteria.

Heat Exhaustion.—An important variety of subnormal temperature is that seen in the form of heat-stroke called heat exhaustion, when, in place of fever, a condition of collapse is induced.

Severe injury to the dorsal region of the spinal cord often produces a great fall of temperature.

A temperature below 92.3° is nearly always fatal in its prognosis, but subnormal temperatures above this degree are not necessarily followed by death. A temperature of 95° is spoken of as one of moderate collapse.

CHAPTER XV.

HEADACHE AND VERTIGO.

The causes of headache—Digestive headache—Headaches due to the eyes—Headaches due to cerebral growths and abscess—Headaches due to syphilis—Headaches complicating acute diseases.

HEADACHE.

HEADACHE is, of course, always a symptom and never a disease, and it arises from such widely different causes that it is impossible in this book to discuss all of them. Only the more common conditions resulting in its development can be considered, more particularly in relation to its diagnostic significance in serious pathological states. The most common cause of headache is probably disorder in the function of the digestive apparatus, the next most common cause is eye-strain in its various forms, and the third is nervous exhaustion or neurasthenia with or without associated anemia. These may all be considered as perversions of function causing headache—that is, the pain in the head may be termed a functional headache. Less frequent, but far more important from a diagnostic standpoint, is headache seen in persons suffering from renal disease, brain tumor, and meningitis in its various forms. The remaining causes of headache are numerous, and some of them will be considered later; but the most important of the first class are the headaches of the gouty or the rheumatic, and of the second class those of cranial periostitis, middle-ear disease, and acute inflammation of the eye or in the jaw.

Headaches depending upon disturbance of the *digestive system* are nearly always accompanied by evidences of such disorder, consisting in gastric or intestinal distress, belching, hiccoughing or vomiting, or even by diarrhea. Often there is a distinct history of the ingestion of indigestible food or digestion-disturbing drink, but in other cases exposure to cold so congests the abdominal viscera that catarrh of the stomach and bowels is induced, and with it congestion of the liver followed by jaundice. The headache of disturbed digestion is nearly always frontal, and in many cases congestive to such an extent that the face may be flushed, or at least the intracranial circulation is so disturbed that the patient is unable to lower the head, because such a posture increases the pain. Such cases are relieved by hot

foot baths which relieve the congestion of the head; nearly always by the act of vomiting, which should be induced, if need be, by an emetic or by putting the finger into the back of the throat. Vomiting makes such headaches very much worse for a time, owing to the congestion of the head following the efforts at vomiting, and this is an important point in diagnosis, for in uremia due to renal disease and in some other states the vomiting is often so easily performed that no straining accompanies it.

That disturbances of the digestive tube are capable of altering the intracranial circulation is proved by numerous facts. Thus Brunton quotes the experiments of Ludwig and Dogiel, who showed that moving the intestines by the finger introduced through an abdominal incision caused a great increase in the flow of blood through the carotid arteries.

Headache due to disorder of the digestion rarely ensues immediately after food is taken, since some time must elapse before the ingested material becomes changed into an irritating or toxic mass by fermentation or putrefactive processes. As a consequence, several hours or even a day may pass without any discomfort in the head, after which time the full force of the headache develops. The headaches of indigestion are, however, characterized by two important facts, viz., that they are not constant, and, second, that they are often relieved or prevented by the use of a purgative, even if constipation has not been present. Such headaches are very apt to be pulsating and accompanied by great nausea. Sometimes such a headache takes a form called migraine or hemicrania, a condition in which the pain is chiefly, if not entirely, unilateral, and there is associated with the pain early and more or less persistent hemianopsia. It is to be remembered, however, that in some cases of hemicrania of nervous origin the sickness at the stomach seems to be secondary to the severe pain in the head.

Headaches resulting from digestive disturbance do not always depend entirely upon irritation of the stomach and bowel with reflex disturbance of the circulation and sensory nerves of the head, but upon the absorption of poisonous substances formed in the digestive tube. These poisons are usually formed only to be destroyed by the liver, or are developed in too small quantities to have any effect; but no sooner do congestion of the liver and deficient biliary secretion ensue than they are formed in large amounts, and enter the general blood stream, owing to the absence of antiseptic bile and the coincident or consequent constipation. As a result, we see very violent headache in jaundice due to catarrhal changes, particularly if the kidneys are not active in the elimination of toxic substances. Similar symptoms to those just described may occur in cases suffering from paroxysmal hemoglobinuria, for in this state severe head-

ache, nausea, vomiting, and persistent yawning are often present, with an icteroid discoloration of the skin. The reddish urine, pain in the liver, and sometimes an urticarial eruption will aid the diagnosis of this primary hemoglobinuria.

In other cases in which no jaundice is present violent headaches, which utterly incapacitate the patient, come on from auto-intoxication. Thus, a man apparently perfectly well goes to bed on a certain night and wakes in the morning feeling a little more drowsy than usual. On rising he may feel a little stupid, and perhaps be slightly vertiginous, but is able to eat his breakfast as heartily as usual. In the course of a few hours the mental heaviness becomes more marked and a pain in the brow develops, which gradually gets worse and worse until it is unbearable. The ordinary remedies for neuralgic headache are futile, and he finds no relief until by the use of a purgative he removes the source of his intoxication, and his kidneys have time to eliminate the toxins already absorbed. Sometimes vomiting comes to his relief, and the emptying of the stomach so stimulates his liver and intestines by the efforts of vomiting that the process of auto-intoxication ceases. Some of the intestinal poisons have been isolated by Brieger, Harnack, and others, and have a physiological action like many well-known drugs. Thus, one produces effects like those of digitalis, another like those of belladonna, and a third like those of aconite. Pulsating pain and a slow, full pulse may indicate the absorption of the digitalis-like toxin; a flushed face and hot, dry skin, the belladonna-like toxin; and pallor, faintness, and a feeble pulse, if no nausea is present, the presence of the aconite-like toxin. Persons suffering from headache of this type are nearly always much freer from discomfort in the head after such an attack than they have been for some time before.

Brunton has also pointed out that digestive headaches are often associated with an objective and subjective sensation of increased intra-ocular tension and tenderness on the upper surface of the eyeball, and the author has frequently confirmed this observation.

The headache of *eye-strain* is usually due to abnormalities in the ocular muscles. Most commonly, according to Noyes, the externi (abductors) are the muscles which are the seat of the difficulty, but this opinion is not generally shared by other ophthalmologists, who assert that the interni are most commonly at fault. Such headaches may be felt in any part of the head, but are most commonly said to be in the occipital region. If, in association with such headache, immediately after or long after reading there is blurred vision, pain in the muscles of the eye on suddenly moving the eyeball, a tendency to congestion of the lids, or hyperemia in the conjunctiva over the insertion of the muscle, the diagnosis of headache from eye-strain is practically certain. (See chapter on the Eye.) Violent pain in the

head may also be due to irritable retina and to astigmatism and spasm of the ciliary muscle. Acute inflammatory processes in any part of the eye may produce severe headache, particularly iritis, the pain of which is very apt to be worse at night.

Violent headache is often produced by *acute* or *chronic glaucoma*, and is usually felt about the eyes or orbit. Often it is of a unilateral character, and the sharp, shooting pain causes a false diagnosis of neuralgia to be made, or in some cases the patient is thought to be suffering from migraine, because in addition to unilateral pain there are often nausea, vomiting, and pallor of the face. The examination of the eye will show glaucoma to be present. Quite similar symptoms may appear as the result of a foreign body lodged in the cornea.

The headache associated with *nervous exhaustion* or neurasthenia may be superficial or deep; that is to say, neuralgic or apparently within the skull. It is often associated with some dizziness and vertigo, and is nearly always occipital in character, more rarely appearing over the brows. In addition to the pain, which is generally not very severe, there is often a sense of constriction about the head. Such a headache persists as long as a person who is overworked persists in fatiguing himself, and rapidly disappears when rest is taken. More rarely the pain in the head in neurasthenia is that of migraine, and is complicated by hemianopsia and hemicrania, often by a dilated pupil on the affected side, and flushing and pallor of one side of the face.

Headaches due to *rheumatism* are often quite severe, and are associated with much tenderness of the scalp or muscles covering the skull. Similar headaches, but more dull in character, are also seen in persons suffering from phosphaturia, and are relieved by benzoate of ammonium.

A headache is a symptom very commonly seen in persons who are subject to the *chloral habit*, and it may be general or limited to the forehead. It is commonly associated with vertigo, flushing of the face, and intense heaviness and drowsiness.

Headache of a violent, bursting character may be produced by full doses of nitroglycerin, the salicylates, and quinine, and by the use of large quantities of tobacco.

Leaving the headaches due to functional disturbances not associated with organic change, we pass to those due to organic disease. Those due to *renal disease* are of two classes, in that they are an evidence of uremia, or they are congestive and due to the high arterial tension so often seen as the result of chronic contracted kidney with its associated conditions of cardiac hypertrophy and arteriosclerosis. Uremic headache, as pointed out in the chapter on Vomiting, is often associated with nausea or vomiting of a persistent type, and

sometimes with diarrhea, for purging is an effort at elimination. The pain is not of the shooting, darting, or neuralgic type, but dull, even if severe, and is often associated with a sensation of fulness in the head. Sometimes the tendency to drowsiness is very marked, and, even if the patient does not sleep, he may seem on the verge of sleep all the time. Not rarely these cases instead of becoming comatose become wildly delirious.

These uremic headaches may occur in any form of renal disease, acute or chronic, which results in uremia; but, if the cause be chronic contracted kidney, there will be a high arterial pressure, and often a strongly beating heart with an accentuated second sound. This form with high arterial pressure will often be relieved by full doses of nitroglycerin, which not only relieves the tension, but also produces an increased renal activity. The urinary examination is of the utmost importance, and no surely correct diagnosis can be made in any case of suspected kidney trouble until this secretion has been examined and found abnormal. (See chapter on the Urine.)

While headache is far less common as a symptom of *diabetes* than of nephritis, it occurs in the former disease either as a dull pain with lassitude and depression of spirits or as violent neuralgia.

Headache which is constant, although it usually varies in degree, may be due to *brain tumor*, and is one of the most important symptoms to be noted in the diagnosis of a case in which such a lesion is suspected. The pain is often worse at night, and is usually more severe in persons suffering from tumor of the cerebellum than in cases in which the growth is in the cerebrum, probably because cerebellar growths often cause effusion which produces pressure inside the skull. A tumor of the cerebral cortex, as a rule, produces more pain than one in the white matter beneath. Meningeal growths are also apt to produce severe headache, but bony tumors of the skull often press upon the brain to an extraordinary degree without causing any symptoms.

Headaches due to brain tumor often have exacerbations with a regularity suggesting malarial disease, and, conversely, care should be taken not to mistake malarial headache for brain tumor.

After constant headache, the most valuable confirmatory evidence of brain tumor is papillitis of the optic nerve, which is present in about 80 per cent. of the cases. There may also be vomiting, and convulsions if the growth be in the motor cortex. Local paralysis, indicating the position of the growth, may be entirely absent, or it may exist and yet utterly mislead the physician as to the focal area which is diseased, since cases are on record in which, for example, a hemiplegia has existed, and at the postmortem examination the growth has been found in the frontal lobes. Tumors of the base of the brain cause focal symptoms most commonly, and in addition to

unilateral choked disk we find in many such cases ptosis from paralysis of the oculomotor nerve, disturbances in the functions of the trifacial nerve in its sensory filaments, so that painful tic (see chapter on the Face and Head) or anesthesia of the face may be present, and complete facial (Bell's) palsy may occur. If the hypoglossal nerve is affected by the pressure, the tongue is protruded to one side, it develops hemiatrophy, and disorders of speech result. Hirt points out that a tumor in the anterior fossa is apt to produce paralysis of the olfactory and oculomotor nerves and the upper branch of the trifacial. A tumor in the pituitary body causes pressure on the chiasm with resulting amaurosis, ptosis from oculomotor palsy, internal squint from paralysis of the abducens (sixth), and anesthesia of the skin and muscles of the eyebrow, forehead, nose, and eye, from involvement of the first division of the trifacial. A tumor of the middle fossa above the dura causes oculomotor palsy (ptosis), patheticus paralysis (downward deviation of eyeball from paralysis of the superior oblique), and amaurosis from pressure on the chiasm. On the other hand, if it is below the dura, the oculomotor, the pathetic, the abducens, and the fifth nerve are paralyzed. When tumors occur in the posterior fossa they cause paralysis of the trifacial, facial, auditory, glossopharyngeal, vagus, spinal accessory, and abducens, or, in other words, cause anesthesia of the upper part of the face, facial paralysis, deafness, loss of taste, irregular cardiac action, loss of power in the sternomastoid and trapezius muscles, and internal squint. Tumors of the lenticular and caudate nucleus, the interior portion of the thalamus, the corpus callosum, the fornix, choroid plexus, and of any part of the cerebellum except the vermiform process, may be present without any localizing signs.

Still more localizing symptoms are early paralysis of the oculomotor nerve from a lesion in the crus, hemianopsia in tumor of the occipital lobe, and tonic convulsions with preservation of consciousness and a staggering gait in tumor of the vermis of the cerebellum.

Should amaurosis be present, very valuable data as to the position of the growth are to be had from a study of the functions of the eye. If the pupils react properly to light, this shows that the optic nerves and tracts are intact, or, in other words, that the ocular reflex arc is perfect, and that the lesion must be in the ocular centres farther back. On the other hand, if the reflex is absent the growth probably presses on the nerve or tract. (See chapter on the Eye.)

The failure of a pupillary reaction may, however, depend upon amaurosis from lateral hemianopsia, in which case we examine the patient for what is known as "Wernicke's sign of hemiopic pupillary inaction." This is done by throwing the light by the ophthalmoscope so that it falls upon the blind half of the retina. If the pupil

does not react, we have in all probability a lesion of the optic tract of that side; whereas, if the pupil does react, we have evidence that the tract is intact, and there must be a bilateral lesion of the optic radiations of the occipital lobes, or in the centre of vision in the cortex. (See chapter on the Eye.)

Other general symptoms of brain tumor are slow breathing, particularly when the patient sleeps, a slow pulse, and, as the growth increases, symptoms of cerebral compression. It ought to be remembered that brain tumor may be closely masked by the results of chronic nephritis, for in the latter disease we find headache, local palsies or spasms, and, more important than all, an optic papillitis, which used to be thought pathognomonic of brain tumor. Albuminuria may be present in both diseases, but tube casts can usually be found in renal disease and not in tumor. Both diseases may exist side by side.

Care should be taken in a case of constant and severe headache that it be not thought due to brain tumor until the possibility of its being caused by a syphilitic arteritis, syphilitic meningitis, or syphilitic gumma, is excluded, for mental depression and crashing head pains occur in all of these states. This is the more important because arteritis is the most common result of syphilis, meningitis frequent, and gumma least frequent of all the cerebral complications of lues. The differentiation of gummatous tumor from cerebral tumor due to other causes may be impossible unless there be a history of specific infection or manifestations of syphilis in scars or other external signs of syphilis. Improvement in the symptoms under the use of iodides and mercury would indicate syphilis rather than a growth due to other causes. The presence of optic neuritis would indicate tumor or meningitis, and would exclude arteritis; and in tumor the pain is apt to be localized, while in arteritis and meningitis it may be diffuse. The chief symptoms of arteritis are those indicating failure of a proper blood supply to the brain, as evidenced by giddiness, weakness of groups of muscles, difficulty in speech, so that words are dropped out, and, it may be, the presence of symptoms like general paresis. Paralysis, when it develops elsewhere than in the ocular muscles, in such cases is usually the result of arteritis, since the arteritis results in a thrombus; but when the ocular muscles are affected the lesion is probably due to meningitis or to nuclear lesions. This development of ocular palsy is of great diagnostic significance. (See chapter on Eye and Face and Head.) In meningitis, on the other hand, the symptoms are irritative, such as spasmodic paralysis and irritative fever.

The following differential diagnostic table aids in making a diagnosis; but it is to be remembered that all these conditions may be very obscure:

Syphilitic Arteritis.	*Syphilitic Meningitis.*	*Syphilitic Gumma.*
Headache diffuse, often absent; not severe. Not started by pressure on cranium.	Headache diffuse and rarely wanting; sometimes localized. Started by pressure or by percussion on head. Very severe.	Headache usually localized.
Hemiplegia or monoplegia frequent. Muscles affected are flaccid, and reflexes are absent. Paralysis often fleeting and limited to a few groups of muscles.	Paralysis, if present, associated with rigidity and contracture, involuntary spasms, exaggerated reflexes. Paralysis more wideapread.	Distinct focal paralysis common. Paralysis associated with rigidity and spasm.
Optic papilla usually normal, sometimes syphilitic retinitis is present.	Optic retinitis with marked neuro-retinitis and abundant exudation along the vessels.	Choked disk often present.
Partial epilepsy rare.	Partial epilepsy common.	
Aphasia is transitory and intermittent.	Aphasia less complete but more permanent.	
Hallucinations rare.	Hallucinations common.	Hallucinations rare.
Pain in limbs rare and fleeting.	Severe pain in limbs of central origin.	
Intellectual functions feeble.	Intellectual functions not feeble, but may be drowsy.	
No active delirium.	Active delirium often present.	
Ocular symptoms rare.	Bitemporal hemianopsia due to compression of the chiasm. Homonymous hemianopsia. Amaurosis from pressure on optic tracts.	Ocular symptoms of gumma involve ocular cranial nerves (see text).
Disorders of sensation are fleeting.	Permanent zones of hyperæsthesia, anæsthesia, and paræsthesia.	
Paralysis of cranial nerves not common.	Paralysis of any cranial nerve.	Paralysis of cranial nerves if gumma is so placed as to injure them.
Temperature may be raised.	Temperature quite frequently raised.	Temperature very rarely raised.

In connection with this table, it must be remembered that should the arteritis result in degenerative changes descending the pyramidal tracts, or in thrombosis with degeneration, the flaccid paralysis characteristic of arteritis may become spastic. Again, should aneurysm arise from the arteritis the pressure upon a cranial nerve may produce paralysis, as does meningitis. Then, too, the meningeal symptoms may be varied. If the lesion is acute and at the base, there will be vertigo, compression of the cranial nerves, polyuria, and bulbar phenomena, and finally fatal coma. If it be at the convexity, then noisy delirium, convulsions, hallucinations, and paralysis in the form of hemiplegia or monoplegia appear. Death comes in coma. If it is chronic meningitis of the base, then we may have slowly developing alternate hemiplegia, crossed paralysis of the face and body, anesthesia of one side of the face, and paralysis of motion on the opposite side of the body. If the convexity be affected then

great irritability of the intellect, sensation, and motion may be present. Paralytic strokes are common, but coma is rare.

Violent headache is the most marked symptom of *brain abscess;* but focal symptoms—that is, localized palsy pointing to the area of the abscess—are very often absent, although the localizing symptoms which have just been described as due to tumor may, of course, be due to abscess if it is so placed as to press on nerve tracts or centres.

The rises of temperature which frequently occur in cerebral abscess are also indicative of the presence of pus, while the more rapid course of the disease, often only one or two weeks, points to abscess rather than tumor. Further than this, choked disk is rare in abscess and common in cases of tumor.

The difficulty of separating the headache of brain tumor from that due to brain abscess is very great, for the symptoms with the headache are almost if not quite identical in both cases. One of the most important of the differential points is the history of an injury to the head or of the presence of an infecting focus which could have caused cerebral abscess. Another means of aiding diagnosis is to examine the blood for leukocytosis. If the white cells are excessive, abscess is probably the cause of the illness.

In some cases of acute cerebral abscess, particularly in children, there is a curious tendency to bore the head into the pillow, or, if the child is still about the room, the head is rubbed or butted into the wall or against the body of the nurse. These symptoms are, however, absent in the slow, insidious forms.

When the physician has made a diagnosis of cerebral abscess because of the headache and associated symptoms, he must not be misled into a reversal of his diagnosis by marked improvement in the patient, who may so far recover as to go back to his occupation, for it sometimes happens that a remission or latent period develops in the subacute forms of abscess. During this apparent remission, however, the temperature is rarely constantly normal, the patient is anything but well, and chills may recur.

Severe headache well diffused over the skull, coming on rather rapidly and associated with fever, stiffness of the back of the neck, vomiting, photophobia, delirium, and, finally, stupor and paralysis, is probably due to meningitis or to *tuberculous meningitis*, effusion at the base of the brain, or, more rarely, to the onset of a severe attack of one of the acute infectious diseases. The differentiation of the former from the latter is sometimes difficult, but the finding of some local tuberculous focus, the insidious nature of the onset in some cases, the family history, and a set of symptoms pointing strongly to involvement of the base of the brain indicate that the bacillus tuberculosis is the cause of the disease. The fact that these symp-

toms are due in some cases to the onset of one of the acute exanthemata is established promptly by the appearance of a rash.

If the disease be tuberculous meningitis, the head pains will often be paroxysmal in character, so that the patient will at intervals of varying length give vent to sharp cries, evidently due to a sudden dart of pain. Vomiting may also be present and ocular symptoms develop, such as ptosis, strabismus, and unequal pupils, which have a sluggish reaction. The febrile movement will be irregular, now high, then very low; the temper peevish, if consciousness is present; and the skin pale and transparent. In the severe and rapid cases of tuberculous meningitis marked delirium comes on, the patient picks the bedclothes, and there are tenderness and stiffness of the nape of the neck. Pulmonary signs of tuberculous disease are often present, and even if absent a focus of tuberculous disease can often be found elsewhere. Care must be taken that the case is not mistaken for and thought to be typhoid fever, which it may closely resemble in its early stages, when headache, malaise, languor, and remitting delirium are present.

In children these symptoms of tuberculous meningitis may be so marked as to lead the physician to the diagnosis of this disease almost at once. Usually for some two or three weeks before the onset of the severe symptoms the child will have been feverish and cross. Vomiting of a more or less obstinate form now comes on, and constipation is present. The pulse becomes slow and irregular, a mild fever is present, and emaciation may be rapid. The general nervous state is one of apathy, but finally may be disturbed by the sharp hydrocephalic cry. Often the child makes chewing or sucking movements. The fact, however, that several other conditions produce identical signs in this class of patients renders caution necessary. It has just been pointed out that the onset of an infectious disease may so result, and it is to be remembered that inflammation of the middle ear of an acute type may cause every one of the symptoms just described. Such cases are often incorrectly diagnosticated until a discharge from the ear with great relief to the patient clears up all doubt as to the malady. Then, again, in some cases of croupous pneumonia all pulmonary symptoms may be masked in the violence of the meningeal manifestations, and, finally, it is not to be forgotten that in some cases of severe gastro-intestinal disorder there may be signs of meningeal inflammation, such as coma, squint, convulsions, myosis, Cheyne-Stokes breathing, and a depressed fontanelle.

A valuable symptom of tuberculous meningitis is change in the optic disk which may be distinctly swollen. Another ocular symptom of importance is the presence of tubercle of the choroid. The first of these is rarely present in cerebrospinal meningitis, and is common in tuberculous meningitis. The second is characteristic

of tuberculous meningitis. Another means of diagnosis of tuberculous meningitis is by lumbar puncture. A large, hollow needle is inserted at the side of the third or fourth lumbar vertebra in a line drawn between the iliac crests across the back. If the needle is inserted properly the subarachnoid fluid speedily escapes from it. This fluid, if centrifuged, will, in a large percentage of cases of tuberculous meningitis, reveal the presence of tubercle bacilli. Another indication that the case is tuberculous is the finding in the subarachnoid fluid of an excess of lymphocytes. While this excess of lymphocytes is not diagnostic of tuberculous meningitis, it is, when taken in connection with the other signs of the disease, of great diagnostic importance, and excludes the acute purulent forms of meningitis in which an excess of polymorphonuclear cells are

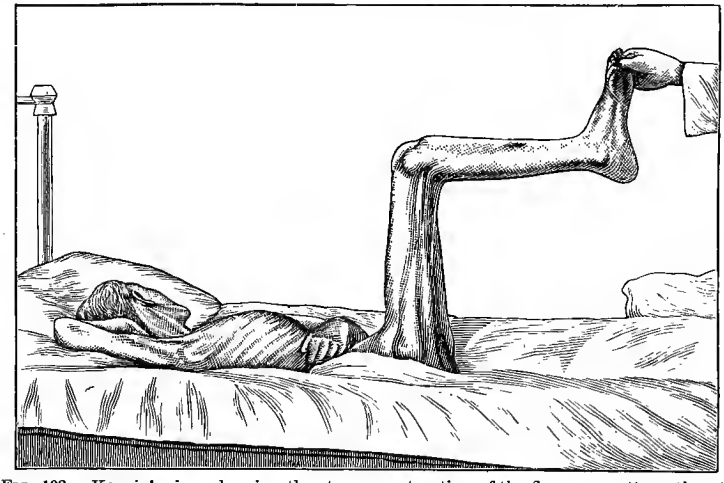


FIG. 192.—Kernig's sign, showing the strong contraction of the flexors on attempting to extend the leg. (After Osler's case.)

present. Koplik has emphasized the value of percussion of the skull in this disease in young children. The child should be upright, with the head slightly inclined to one side, and the percussion made at the junction of the frontal, parietal, temporal, and great wing of the sphenoid bones; in other words, about one and one-quarter inches behind the external angular process of the orbit. If, in percussing this area, a tympanitic note is developed, fluid is present in the latter ventricles of the brain, and tuberculous meningitis is probably present. Koplik believes that the percussion of the skull in the earlier stages of tuberculous meningitis is one of the most valuable aids in diagnosis.

In some cases of typhoid fever meningeal symptoms develop. In some of these the autopsy fails to show any signs of meningeal trouble. In others a true meningitis is found.

A valuable sign indicative of meningitis is that of Kernig. The patient is placed in a sitting posture at the edge of the bed, with the feet on the floor. If meningitis be present, it will be found that the leg cannot be extended on the thigh, because of contractures in the muscles. If the patient is too ill to sit up, then he should be placed on his back, the thigh flexed on the trunk and the leg on the thigh, and an attempt made to straighten the leg, which attempt will fail in a large number of cases if meningitis is really present.

The symptoms of meningitis closely resemble those due to *thrombosis of the cerebral sinuses*, so closely, indeed, that only the presence

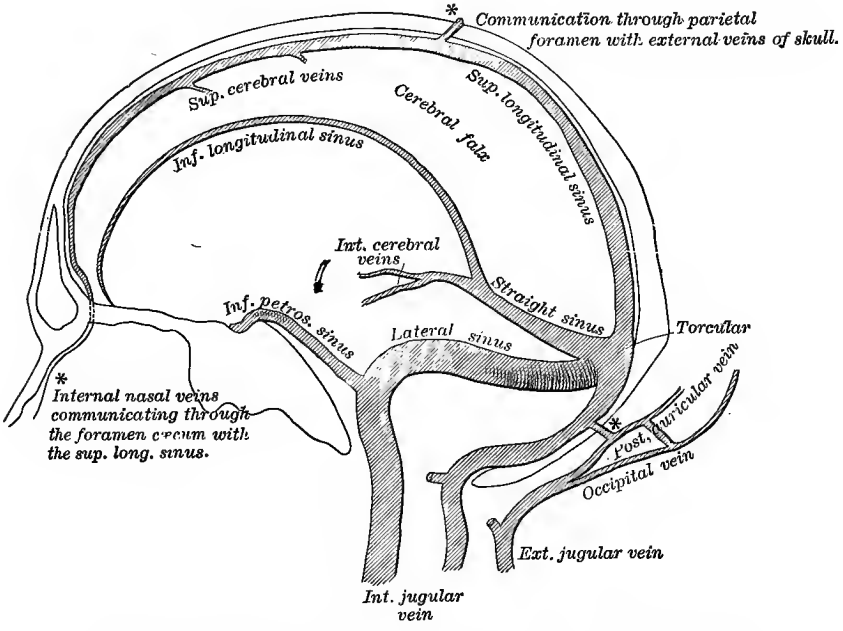


FIG. 193.—Showing the communications existing between the superior longitudinal and lateral sinuses and the external veins, indicated in the figure by *. (Leube.)

of the typical signs of such occlusions can determine the diagnosis. Thus, if the superior longitudinal sinus is affected by thrombosis, there may be epistaxis from distention of the nasal veins, and the temporal veins will be swollen, and the nearby tissues edematous through their close connections with the sinus through the emissary veins of Santorini, which escape from the skull by way of the parietal foramina (Fig. 193). In children there is usually in such cases bulging of the fontanelles and heaviness. Somnolence or delirium may be present with many of the characteristic symptoms of meningitis. This condition usually arises in connection with chronic

exhausting diseases, such as long-continued diarrhea and the continued fevers.

Thrombosis of the cavernous sinus is usually accompanied by quite typical symptoms. There is edema of the eyelids and finally of the entire side of the face on the side of the affected sinus, but this facial symptom may be absent or very fleeting in its duration. Sometimes there is exophthalmos, and if the thrombus is septic a phlegmonous inflammation of the orbital connective tissue may occur. These symptoms are due to the communication between the sinus and the ophthalmic veins. Finally, as pointed out in the chapters on the Face and Head and on the Eye, paralysis of the oculomotor nerve, the ophthalmic branch of the fifth nerve, and of the abducens and patheticus may occur, as these nerves pass through the cavernous sinus or in its walls. Nearly always thrombosis of the cavernous sinus results from some disease processes near by, as in disease of the middle ear and mastoid. Sometimes the affection is bilateral.

If the lateral sinus is affected by thrombosis, there is usually marked edema back of the ear, owing to the clot extending to the small veins of the scalp, which pass through the mastoid and posterior condyloid foramina. The external jugular vein on the affected side is partly collapsed, particularly on full inspiration (Gerhardt's symptom). Rarely, this vein may be unduly distended (Fig. 194). Thrombosis of the lateral sinus occurs far more frequently than that of the other sinuses. Suppurative otitis is its most common cause, and agonizing earache is, therefore, a symptom often associated with it.

Not only may cerebral thrombosis present symptoms resembling those of meningitis, but in addition those of cerebral abscess.

Violent headache, with vertigo, staggering, and confusion of thought, followed by unconsciousness, may follow *meningeal hemorrhage* due to disease of the bloodvessels, which are ruptured by some strain or by increased blood pressure under the influence of stimulants. Hemiplegia or localized spasms may be present. The patient may survive several days in severe cases, or may recover if the hemorrhage is small; but usually a hemorrhage large enough to cause marked symptoms is large enough to cause death.

The individual affected by meningeal hemorrhage will usually be plethoric, and, with the symptoms just described, will suffer from photophobia, extreme sensitiveness to the slightest noise, and pain radiating down the neck and trunk, which occurs in paroxysms. Localized paralysis is rarely present.

The presence of severe vertical headache in a middle-aged person who is insane and who is a male may indicate *pachymeningitis interna hemorrhagica* (hematoma of the dura); but usually the insane patient does not complain, and an antemortem diagnosis of this state is not made.

Headache resulting from *heat-stroke* or *thermic fever* is usually the result of meningeal congestion or inflammation, and is one of the most annoying symptoms of convalescence. It is apt to be greatly increased by moving the head, and is often relieved by venesection.

The earlier stages of *smallpox* and *pneumonia* of the croupous type are often periods of violent headache, which symptom in the

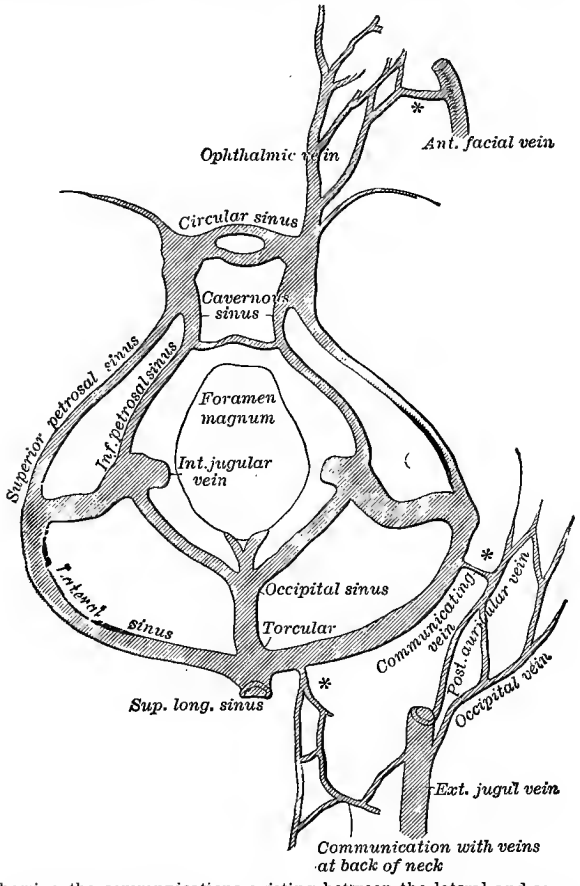


FIG. 191.—Showing the communications existing between the lateral and cavernous sinuses and the external veins, indicated in the figure by *. (Leube.)

former instance decreases with the appearance of the rash, and in the case of croupous pneumonia so closely resembles the headache and associated symptoms of meningitis that a correct diagnosis, if the pulmonary signs are not sought for, may be impossible. In every case in which such symptoms occur the lungs should be examined.

When headache is present in the course of *croupous pneumonia* it often lasts until crisis, but in some cases ceases by the third day.

The chest should always be carefully examined in all cases of severe headache with fever for signs of pulmonary disease.

Headache is a constant symptom in many cases of *typhoid fever* in the early stages, but the peculiar tongue (see chapter on Tongue), the tendency to diarrhea, the general systemic symptoms, and the facies of the patient will usually make its cause clear. More or less violent headache is often seen in measles, and depends probably to a great extent upon the engorgement of the nasal mucous membrane, or, in other words, has the same causative factor as has an acute "cold in the head" in producing cephalgalia.

Gruening is quoted by de Schweinitz as asserting that early morning headache is often due to *nasal catarrh.* This is, of course, only true if digestive troubles, which are often due to alcohol, and renal disorders are excluded. Severe morning headache, or dull headache on first waking up, may be due to *nocturnal attacks of epilepsy,* of which the patient is ignorant. If the tongue is bitten or the bed wet with urine, this diagnosis receives strong support.

Violent headache is often present during the febrile stage of *intermittent fever* and is often a complicating symptom of fever of the remittent type. In this connection the physician should remember that violent neuralgia of the supra-orbital nerve is sometimes due to malarial poisoning, and is called "brow ague."

Headache is often due to *anemia,* whether it be the result of hemorrhage or of the deficient formation of blood. The pain is usually frontal; there are often giddiness on movement, palpitation of the heart, a peculiar sensation in the head, and pallor of the skin. An examination of the blood will usually reveal the cause to be in this tissue.

Headache sometimes results from valvular heart disease. This in mitral regurgitation is often associated with vertigo, stupor, sleepiness, and, as night approaches, a mild delirium may come on. Its probable cause is congestion of the brain.

Rarely *intracranial aneurysms* produce headache, and when they are of the diffuse miliary variety this symptom may be a prodromal one before an attack of apoplexy. Large aneurysms may, however, exist without severe headache, and the position of the pain in no way indicates the seat of the aneurysm, save that aneurysm of the basilar artery may cause occipital pain. Auscultation might possibly reveal a murmur.

Headache may also arise from *disease of the skull bones,* either caries, osteitis, or periostitis, which result from injury, infection by syphilis or other infecting cause, such as typhoid fever or tuberculosis; but there is nothing diagnostic about the headache in these cases save that it is generally most severe in the area involved, and pressure over that part may elicit more or less pain or tenderness.

Violent neuralgia or shooting headache may be produced by exposure to cold, with resulting inflammation of the nerve sheath; by dental caries, and by middle-ear disease or disease in the external auditory canal.

VERTIGO.

Vertigo is a condition in which the patient feels as if he were losing his equilibrium. Sometimes he feels as if he were whirling around from right to left or left to right, sometimes as if falling forward or backward, and sometimes he seems stationary, while all his surroundings whirl round or rise up to or fall away from him. Although vertigo is a symptom which in itself lacks danger, it always produces great discomfort, if not fear. Functional vertigo arises from the patient being subjected to a whirling motion, from rough sea voyages, and from indigestion, deficient circulation, or excessive cerebral congestion. Often it is due to cerebral anemia arising from excessive hemorrhage. When it arises from indigestion it is probably due to reflex irritation, and perhaps to the absorption of toxic materials.

Vertigo as a symptom has a far more serious significance when it arises from organic disease. The most common lesions which cause it are middle-ear disease, Ménière's disease, tumors of the cerebellum, of the pons, of the crura cerebri, and the corpora quadrigemina. Vertigo also is not only a premonitory sign of an epileptic attack, but in the epileptic state called *petit mal* or minor epilepsy it is often the only symptom. In persons with atheromatous arteries it is very common, and sometimes it is a persistent symptom for some days before an apoplectic seizure. It is also present in disseminated sclerosis. Finally, many drugs, such as quinine and the salicylates, may produce it.

As the diagnostic points connected with most of the lesions here named are discussed elsewhere in this book, only Ménière's disease will be mentioned at this place. In addition to vertigo the characteristic symptoms of Ménière's disease are vomiting, noises in the ears, and, finally, deafness. The vertigo may be so severe that the patient falls to the ground. Aural examinations are usually futile in discovering any cause. Some authorities believe the disease to be due to a neurosis of the vasomotor nerves supplying the semicircular canals.

A form of vertigo unknown in America, the paralyzing vertigo of Switzerland, described by Gerlic, is a paroxysmal vertigo with great loss of power in the limbs, partial ptosis, and preserved consciousness.

CHAPTER XVI.

COMA OR UNCONSCIOUSNESS.

Coma is a condition of unconsciousness or insensibility from which the patient can be aroused but partially or not at all, and it may arise from injuries to the head, while the patient is in otherwise perfect health, which injuries produce laceration of the brain substance, cerebral or meningeal hemorrhage, or concussion. Again, it may be due to the influence of certain poisons, as alcohol, opium, chloral, cannabis indica, very large amounts of the bromides, or poisonous doses of other narcotics. Thirdly, it may arise from auto-intoxication, as in uremia, resulting from renal disease, in cases of diabetes; in cases of profound exhausting disease, like typhoid fever or ulcerative endocarditis; or from acute yellow atrophy of the liver and pernicious malarial fever. Fourth, as a coincident symptom or sequel of hemorrhage into the brain (apoplexy), as the result of an epileptic attack, of a cerebral embolism or thrombosis, of thrombosis of the cerebral sinuses, of cerebral abscess, of pachymeningitis, leptomeningitis, or cerebrospinal meningitis, of cerebral syphilis, of general paralysis, multiple sclerosis, and heat-stroke. The various points in connection with the diagnosis of coma from head injuries are to be found in surgical treatises, and the history of a head injury or the very presence of any injuries to the head is an important point to be sought after in the diagnosis. Care should be taken, however, to ascertain that any head injuries found to be present are not the result of a fall due to the onset of sudden unconsciousness, rather than the cause of the coma.

The coma of *acute alcoholic poisoning* is characterized by profound insensibility, great muscular relaxation, loss of the ocular reflexes, and great fulness of the bloodvessels of the neck and face in the early stages, and, finally, by ghastly pallor of the face as the coma deepens on the approach of death. The skin is moist and warm at first, but afterward becomes cold. The pupils are usually moderately dilated; the pulse is rapid, at first strong, then more and more feeble, and the respiration stertorous and heavy. The sphincters, as a rule, are not relaxed, although they may be so in rare cases. The bodily temperature in severe alcoholic poisoning progressively falls from 1° to 3° F. below normal.

Alcohol coma is to be separated from that due to opium poisoning by the absence of the contracted pupils and slow breathing of the latter condition, in addition to the other symptoms named below

in discussing that condition; from coma due to cranial fracture by the absence of any history or sign of head injury;[1] from chloral poisoning by the history, the greater fall of body temperature, and the great feebleness of the heart and respiration produced by chloral. It may be impossible to separate alcoholic poisoning from that of cannabis indica poisoning except for the fairly strong pulse generally found in the latter condition, and the history of the patient having taken the hemp or complained of the peculiar sense of prolongation of time before the coma came on.

The symptoms accompanying the coma of *opium poisoning* are heavy sleep, preceding the deep unconsciousness, during which the patient can usually be aroused by shouting in his ear or by violent shaking, but sinks back into slumber at once on being undisturbed. The face is suffused and reddened and may be finally distinctly cyanotic, and the breathing puffing and stertorous. When the patient is awakened he breathes more rapidly, and for this reason the duskiness of the face disappears and the normal hue returns. Death never occurs in the second stage of opium poisoning from the poison alone; but, if disease is present, death may take place at this time. The pupils are contracted to pin-points. The third or fatal stage emerges from the second by a gradual process, so that no abrupt line of separation can be noted. The face becomes at first more cyanotic, then pale and livid; the respirations, which have been 8 to 10 in theminute, are now only 4 or 5; and, finally, such prolonged pauses occur that all hope of another respiration is lost by the attendant. While the slow breathing is at first deep, it now rapidly becomes shallow, and relaxation is present to the greatest degree. The skin, previously dry, is wet with the sweat of death; the patient is so deeply narcotized that nothing can arouse him, and he dies from respiratory failure, although the heart ceases almost simultaneously from the asphyxia. The pupils do not dilate in the third stage, except in the relaxation of death.

In view of the frequency with which alcoholic and opium poisoning are confused, the following table is appended, which will be found of value in making a differential diagnosis as to the condition of the patient:

OPIUM-POISONING AND ALCOHOLISM.

Opium-poisoning.	*Alcoholism.*
1. Pupils contracted.	1. Pupils normal or dilated.
2. Respiration and pulse slow and full.	2. Respiration nearly normal; pulse rapid, and finally feeble.
3. Face suffused and cyanosed.	3. Face may be pallid.
4. Skin warmer than in alcoholic poisoning.	4. Skin cool, perhaps moist.
5. Pulse slow, strong, and full till late in poisoning.	5. Pulse rapid, at first strong, then weak.

[1] The physician must not forget that a fall from alcoholism may result in a cranial fracture.

There is scarcely any difference as to consciousness in these two conditions.

When a *poisonous dose of chloral* is taken by man the person soon falls asleep and then sinks into a deep coma. The respirations become at first slow and labored, then shallow and feeble. The pulse, at first perhaps a little slowed, soon becomes rapid, thready, and shuttle-like, and is finally lost at the wrist. The face is white and livid, the forehead and the hands covered with a cold sweat, and the pupils, which are at first contracted, soon become widely dilated. Absolute muscular relaxation is present, and it is impossible to arouse the patient.

The coma of *uremia* may come on gradually, but most commonly its onset is sudden and follows a uremic convulsion. It possesses no diagnostic sign or signs which clearly separate it from the unconsciousness or coma following epileptic attacks, and, as the uremic convulsion is often typically epileptic in character, the differential diagnosis is very difficult. An examination of the urine, if it can be obtained by the catheter or otherwise, will indicate, but not prove, the presence of uremia if albumin be found in either large or small amounts, and the presence of very little urine in the bladder, indicating anuria, may be of some diagnostic significance. On the other hand, if the uremia be due to chronic contracted kidney, the urine may be plentiful, the albumin scanty, but the low specific gravity be noteworthy. The pulse is usually very slow, often but 40 to 50, but the arterial tension is high, so that the artery feels hard and unyielding. The temperature of the body is usually very low in those severe cases which are free from convulsions and have a progressively downward course; so low a point as 91° to 95° being sometimes reached, at which time the patient is usually moribund from collapse. When convulsions are present the temperature may rise as high as 108°, and there may be in some cases a severe chill, followed by fever, and this again by collapse. The respiration is nearly always very deep, and sometimes very much quickened, and at times has a peculiar hissing sound. Sometimes the patient may purse his lips as if to whistle. Cheyne-Stokes breathing may be present. Rarely the breathing is difficult and asthmatic in type (uremic asthma). If a preceding history of prolonged nausea, attacks of colliquative diarrhea, and vertiginous symptoms can be discovered as having been present prior to unconsciousness, these will add to the array of uremic probabilities. The coma of uremia is not necessarily a fatal symptom. Even in very severe cases remarkable recoveries sometimes occur.

Coma resulting from *diabetes mellitus* is of far graver import, as it commonly terminates the patient's life. There may not be any prodromes, and there may not be any history of an exciting cause

for the coma in a case of diabetes. Sometimes it is provoked in patients by severe exercise or great mental strain or emotion. When unconsciousness does not come on at once, the patient, after suffering from nausea, headache, and respiratory oppression, suddenly becomes anxious, delirious, and violent, then drowsy and deeply comatose. The pulse is not particularly noteworthy, but is usually full and not very tense. The respirations are deep and often very noisy, but at about the normal rate, although sometimes they may be rapid in the condition called diabetic dyspnea. The body temperature falls very greatly, even below 90° F. The respiratory changes and those in temperature may, therefore, be very much like those of uremia; but in association with the coma of diabetes mellitus there are two pathognomonic symptoms: first, the sweet odor of the breath, which smells like the aroma of a pear or an apple, or a faint odor of chloroform; and, second, the presence of sugar in the urine, which secretion becomes dark red on the addition of chloride of iron. Testing for acetone usually reveals it in the urine in excess. (See chapter on the Urine.)

Ordinary coma is rare in *typhoid fever*, and when it occurs is due to some complication, such as abscess or effusion at the base of the brain or meningitis; it is usually replaced by what is called coma-vigil, in which the patient, in a semiconscious state, keeps muttering day or night. This is a grave sign.

The coma of *acute yellow atrophy of the liver* is generally preceded by headache, nausea, anorexia, and perhaps fever, followed by nervous excitement or restlessness, and then mental hebetude, which is often accompanied by a noisy delirium which may amount to mania. Finally, after several days, coma comes on and gradually becomes more and more profound until death takes place. Some of these symptoms resemble those of uremia or diabetic poisoning, but the coma of acute yellow atrophy has in addition these characteristic signs, namely, jaundice, bile-stained urine, marked shrinking of the liver dulness, enlargement of the spleen, and hemorrhages into the skin, or these effusions may take place into the bowels and stomach. The urine is singularly free from urea, but contains leucin and tyrosin in large amounts. (See chapter on the Urine for description of leucin and tyrosin crystals.) Acute yellow atrophy of the liver is so very rare that this disease may be excluded by the law of probabilities.

When coma comes on as the result of *pernicious malarial infection*, it is most apt to be ascribed to sunstroke, uremia, or apoplexy, for its onset is usually sudden. Only a history of exposure to malarial influences, the presence of slight jaundice and anemia, and of an enlarged spleen will serve to separate it from these conditions, and an examination of the blood for the malarial organism may be neces-

sary before a positive differentiation can be made, for the diagnosis is by no means easy.

The coma of *apoplexy* may be sudden or gradual in its onset; generally it rapidly appears after the first symptoms of cerebral hemorrhage develop. The loss of consciousness may be partial or absolute, generally the latter if the leakage from a ruptured vessel be great. The respirations become stertorous, generally more rapid than normal, and, if a fatal result is in prospect, are rhythmically irregular; that is, they are now very slow, then gain in speed gradually until they become very fast, then the speed and vigor gradually fall until they are as feeble and slow as before (Cheyne-Stokes respiration). The history of preceding paralysis on one side of the body, or the presence of this loss of power if it can be demonstrated, the unequal pupils, the drawing of the face away from the paralyzed side, a strong, bounding pulse, and generally raised temperature complete the clinical picture of the coma of cerebral hemorrhage. If death does not ensue, consciousness may return, and the patient progress to recovery; but sometimes after several days of apparent convalescence a secondary fatal irritative coma comes on, associated with high fever. This is usually of ominous portent and is readily recognized because of the history. (See chapters on the Arms and on the Legs and on Hemiplegia.)

The coma of cerebral hemorrhage is unfortunately often taken for acute alcoholism, particularly as the latter state often induces the hemorrhage. The following table is designed to separate them:

ACUTE ALCOHOLISM AND APOPLEXY.

Alcoholism.	*Cerebral Hemorrhage.*
1. Pulse rapid, compressible, and weak.	1. Pulse apt to be strong and slow.
2. Skin moist, or relaxed and cool.	2. Skin hot or dry.
3. Bodily temperature lowered.	3. Bodily temperature raised.
4. Pupils equally contracted or dilated; generally dilated.	4. Pupils unequal.
5. No hemiplegia.	5. Hemiplegia; one side tossed, the other remaining motionless.
6. Breathing not so stertorous nor so one-sided in lips.	6. Respiration stertorous, the lips being inflated on one side on expiration.
7. No facial palsy.	7. Facial palsy.
8. Unconsciousness may not be complete.	8. Unconsciousness complete.

The smell of alcohol in the breath is no guide, as the high arterial pressure of acute alcoholism may have caused the rupture of a cerebral bloodvessel.

Coma due to *cerebral softening*, following embolism or thrombosis, has no signs other than those discussed in the diagnosis of these lesions in connection with hemiplegia (which see). Coma due to thrombosis of the sinuses of the brain is accompanied by the following diagnostic symptoms, namely, irritation or paralysis of the cranial

nerves resulting in strabismus, nystagmus, and lockjaw, stiffness of the neck, and clonic spasms. If the cavernous sinus is thrombosed, there will generally be found stasis of the veins in the eye, which means retinal congestion. The eyeball may be protruded, the eyelids swollen, and perhaps loss of function in the oculomotor nerve may be present, causing ptosis, and, if the abducens is affected, causing internal strabismus from paralysis of the external rectus. If the transverse sinus is involved, there will probably be edema behind the ear, and, if the petrosal or internal jugular be obstructed, the proximal part of the vein collapses. Thrombosis of the superior longitudinal sinus causes epistaxis and engorgement of the temporal veins. Thrombosis of any of these sinuses, however, may be present without these signs.

Coma due to *subdural hemorrhage* (pachymeningitis interna hemorrhagica) is peculiar in the fact that its onset is usually very slow, and the signs of nervous irritation last a long time and are quite violent, often amounting to epileptic paroxysms. Commonly, too, there will be rigidity of one limb, but the cranial nerves usually escape. The coma usually follows these signs, and the condition is peculiarly common in the chronic insane and in paretic dements.

Sudden unconsciousness with hemiplegia and vomiting may also come on in *Raynaud's disease.*

Coma from *cerebral abscess* is accompanied by symptoms closely resembling those of acute meningitis. The patient is dull and delirious; has headache, fever, and often has a hyperpyrexia. The sensibility becomes less and less, and deepens into the coma which ends in death if relief is not given. The localizing symptoms of paralysis may indicate that a lesion is in a certain part of the brain; but generally these signs are absent, because cerebral abscess is usually in the frontal lobes. If there is a history of injury, purulent otitis, infectious disease involving other parts, such as septicemia from wounds or empyema, and if there are vertigo, vomiting, and headache, fever, and an absence of choked disk of the optic nerve, the diagnosis is probably cerebral abscess; but a long duration of months is no sign that it is not abscess, as these cases often run a very prolonged course.

The coma of *purulent leptomeningitis* resembles that of abscess in many of its associated symptoms; but the intense headache, the rapid development of delirium and unconsciousness, the stiffness of the neck, the optic neuritis and disturbed movements of the ocular muscles, combined with the absence of a history of septic absorption, may make a differential diagnosis possible. Purulent leptomeningitis is rare, but it sometimes occurs in association with croupous pneumonia, and the presence of this disease will point to the cause of the coma.

The coma due to *epidemic cerebrospinal meningitis* is diagnosticated by the characteristic rigidity of the neck, excessive headache preceding the unconsciousness, the disturbances of the cranial nerves producing strabismus, unilateral or bilateral ptosis, nystagmus, impaired pupillary reaction, mydriasis, and myosis. The face is often painfully distorted. The presence of an epidemic, of course, makes the diagnosis clear.

It is well to remember that coma may be present from other forms of meningitis and arise in several conditions presenting similar symptoms, such as pneumonia of the meningeal type, otitic abscess, and gastro-enteritis. (See chapter on Headache and Vertigo.)

Cerebral syphilis may result in the development of coma by producing hemorrhage, embolism, arteritis, tumor of the brain, or almost any other lesion, and its diagnosis as the cause of an attack of coma is not easy. Of course, a history of syphilitic infection and the presence of symptoms of this condition in a patient who is too young to have secondary arterial changes from age render the probability of syphilis as a cause very great. Scars on the skin (see chapter on the Skin) may show specific taint.

When coma results from *general paralysis* it usually succeeds the peculiar epileptic attacks which come on late in that disease, and the history of delusions, tremor of the hands, peculiar speech, loss of the reflexes, with earlier milder attacks, like the one before us, combined with the age of the patient, render a diagnosis possible.

Practically identical symptoms may attend the development of coma from *multiple sclerosis*, and without the history of the latter affection the diagnosis may be impossible. If this history shows a spastic gait and intention tremor, nystagmus, mental weakness, and heightened reflexes, the probability of the attack being due to multiple sclerosis is increased.

Coma is sometimes seen as a later manifestation of *Addison's disease*, and it often develops very suddenly.

Heat-stroke produces coma as one of its almost constant symptoms. The history of exposure to heat and the hyperpyrexia are the two diagnostic points of importance. (See Fever.)

Sudden unconsciousness may arise from *heart failure* due to disease or fright; we call this fainting. Frequent attacks of this character should cause the physician to listen to the heart to discover if there is valvular disease, particularly aortic stenosis and fatty heart, and he should be on the outlook for renal difficulty. Sometimes sudden repeated attacks of unconsciousness are due to *petit mal* or minor epilepsy. Coma more or less profound follows attacks of true *epilepsy*. (See next chapter.)

CHAPTER XVII.[1]

CONVULSIONS OR GENERAL SPASMS.

Definition of a convulsion—The convulsions of epilepsy in its various forms—Of infancy—Of hysteria—Tetanic convulsions—Tetany—Spasms—Chorea.

A CONVULSION is a condition in which by reason of sudden tonic or clonic contractions of groups of muscles the body in whole or in part is thrown into spasmodic movements. Convulsions can be divided into those which are clonic or epileptiform and those which are tonic or tetanic. Further, it is a general rule that convulsions which are epileptiform or clonic in character have their origin in the cerebral cortex, while those of the tetanic or rigid type arise from excitation of the motor tracts in the spinal cord. The clonic variety of convulsions are represented by idiopathic, traumatic, reflex, and syphilitic epilepsy, hysterical convulsions of an epileptic type, uremic convulsions, and those convulsions which arise from the presence of growths or other sources of irritation in the cerebral cortex. Certain poisons may also rarely produce such attacks, notably lead and alcohol, and sometimes malingerers imitate very successfully the epileptic paroxysm.

The convulsion in *epilepsy* is characterized in some cases by the primary appearance of an aura—that is, a sensation in some part of the body, which the patient discovers comes on before each convulsion. This aura may be of any character and appear in any part. Most commonly it is sensory, and is as if a cloud or wave were passing up the body to the head. As the sensation reaches the head the patient may utter the peculiar epileptic cry or sigh, and with this sound the body becomes rigid from tonic spasm of the muscles. This spasm now relaxes for an instant, and then the patient's muscles pass into a state of alternate relaxation and contraction which throws the patient's body from one place to another.

The primary tonic spasm of the face produces risus sardonicus in some cases: the head is often drawn to one side, the eyes commonly turned to the same side, and the lower jaw locked tightly against the upper jaw. The arms are strongly flexed at the elbows, the hands flexed at the wrists, and the fingers bent into the palms of the hands with great force. As a rule, the evidences of the powerful

[1] For local spasms or tremors, see chapters dealing with the Face and Head, Hands and Arms, and Feet and Legs.

flexors overcoming the extensor muscles predominate; but sometimes the reverse is the case, and forcible, rigid extension of the parts affected takes place. The duration of these tonic contractions rarely exceeds two minutes, and in most cases is limited to but a few seconds.

It is followed by clonic spasms, already described, which are ushered in by more or less violent tossings, but whose onset is forewarned by peculiar vibratory thrills which run through all the affected muscles. The eyelids tremble, the body changes its position never so slightly, and then, as if the vibrations gained greater and greater power with each moment, the fibrillary tremors give way to muscular contraction. The expression of the face, which in the preceding stage was set and firm, is now constantly changed by the movements of the facial muscles; the jaws, no longer locked together, are gnashed and crunched one upon the other; the tongue is alternately protruded and drawn back, and, as a consequence, is often caught between the teeth and lacerated. The excessive movements of the muscles of mastication force the increased quantities of liquid secreted by the salivary glands from the mouth in the form of froth, which is often stained with blood by reason of the injuries to the tongue. The constancy of the convulsive movements now becomes less and less marked; well-developed remissions occur between each toss of the body, until the movements cease entirely; but it should be constantly borne in mind that the prolongation of the remissions does not produce any decrease in the severity of the intervening spasm, the final spasm often being even more violent than the first.

The intense discoloration of the face begins to pass away as soon as the remissions, by their length, permit the blood to be oxygenated, its disappearance being temporarily arrested by each paroxysm. Finally, the spasms having ceased, the patient lies before us relaxed, unconscious, and exhausted, and usually passes into a deep sleep or coma, which lasts a variable length of time, and from which he cannot be aroused, except very rarely, and then with great difficulty.

When one part of the body is involved in an epileptic paroxysm, the rest of it escaping, the condition is called *Jacksonian epilepsy.* By far the most important of its peculiar signs is the character of the onset, which always begins, in the typical Jacksonian form, in some peripheral portion of the body, and most frequently in the muscles of the thumb or hand, so that for the moment the convulsive movements are localized. They may remain localized at the point of origin, or immediately diffuse themselves over muscle after muscle until all the arm, leg, or other groups of muscles are involved. It is of the greatest importance, however, that the reader should keep the aura of an attack separate in his mind from the onset, remembering that the term onset is here used by the writer to designate the

beginning of the period following the aura, if there be one. Jacksonian epilepsy may be of almost any severity. In rare cases only one muscle may suffer throughout an entire attack, but in others the entire body may be at last convulsed. There may or may not be loss of consciousness, its presence or absence being dependent upon the seat of the lesion in the brain and the severity of the attack. In those instances in which only a few localized muscles are involved consciousness is more commonly preserved than lost.

Typical Jacksonian epilepsy may develop in the course of general paresis.

An epileptiform convulsion may be associated with the onset of an *apoplexy*, and usually indicates that the hemorrhage is in the motor cortex. Such an attack is generally Jacksonian in character; that is to say, one muscle or a group of muscles is involved, or, if not this, the attack is, at most, only unilateral. The cause is made manifest by the presence of the symptoms of apoplexy as generally seen, for there are inequality of the pupils, drawing of the face to one side, and a consequent hemiplegia which lasts indefinitely. Of the attack itself, it may be said that, so far as the movements are concerned, they differ in no way from those of the true Jacksonian epileptic seizure; and it should be remembered that hemiplegia often follows ordinary idiopathic epilepsy. Such a postepileptic hemiplegia is, however, usually fleeting, while that due to hemorrhage is more or less permanent. It should be remembered, however, that apoplexy may complicate epilepsy, being produced by the convulsion. Then again the lesions caused by a hemorrhage may ultimately result in epileptiform attacks, although this is certainly rare in adults. In some persons the history of this attack is very indistinct, owing to its occurrence in early life; while in others the paralysis has been so slight or temporary as not to bear any relation in the mind of the patient with the convulsive seizures following, which in many cases do not occur for some time after. The palsy and convulsions are not always due to hemorrhage, but to any pathological cerebral change. Heart disease, by causing embolism, may bring them on, and syphilis and puerperal sepsis may all produce a softening of the cortex, with an epileptic state following the paralysis.

We can very readily divide posthemiplegic epilepsy into two classes, for we find that in about one-half of the cases the convulsion occurs along with the paralysis and then follows at intervals, while in the other half the paralysis is not followed by convulsive seizures for weeks, months, or years.

Posthemiplegic epilepsy may occur at any age, but there can be no doubt that it far more commonly occurs in children than in adults. In at least two-thirds of the cases the onset is before five years of age, and in nearly one-half it is during the first two years of life.

The frequency with which posthemiplegic epilepsy comes on in the hemiplegia of childhood has been very carefully studied, and the conclusion reached that its occurrence is quite common. Thus, in Osler's cases 20 children out of 97 suffered from it. In the 80 cases collected by Gaudard 11 children had hemiplegic epilepsy, and 66 children out of 100 cases collected by Wallenberg were epileptic after hemiplegia. In another series of cases collected by Osler 15 children out of 23 were thus affected. (See Cerebral Diplegia.)

Very interesting results are reached if the statistics of this condition are analyzed. Thus, it will be found that in the cases which date from infancy females are twice as numerous as males, but in cases after five years of age there is no difference between the frequency in the two sexes. One of the theories of these infantile cases has been that they were produced by the use of instruments during labor, and repeated postmortem examinations have confirmed the possibility of this occurrence. On the other hand, every obstetrician knows that the birth of a boy generally means a more difficult labor than that of a girl, owing to the greater size of the head in a male child. *A priori* reasoning would seem to show, therefore, that the heads of male children would, accordingly, have instruments applied most frequently, and consequently that infantile cerebral trouble would be the result more commonly in males than in females; but, as has been said, this conclusion is contra-indicated by the facts. Another fact of great interest is that the paralysis in the infantile cases is more frequently on the left side than the right, but after the fifth year it is equally common on both sides.

The writer has already spoken of the fact that the convulsions may occur along with the first attack of paralysis, and continue, or that an interval may occur between the attack and the subsequent paroxysm. The chronic recurrent fits date from the onset in about one-third of the cases, but it is not uncommon for the paralysis to occur in infancy and the epilepsy to begin at puberty. It would seem that cells injured in early life may lie undisturbed until the increased demands of maturity call them out into diseased action. This prolonged interval occurring so commonly in children separates them from adults in this disease, for in the latter class it is very rare for the epilepsy to be delayed for more than one year.

Syphilitic epilepsy is only one of the many nervous affections which afflict those who may be so unfortunate as to contract this disease. There can be no doubt that syphilis produces epilepsy in adults.

There is also one symptom which may occur early in syphilitic epilepsy, or sometimes only late in the disease, namely, repeated partial, passing palsies, which while they may be in some cases hysterical, are in the syphilitic almost pathognomonic of brain involvement

—a momentary weakness in one arm; a slight drawing of the face to one side, which appears in a few hours; a temporary dragging of the toe; a partial aphasia which appears and disappears; a squint which tomorrow leaves no trace behind it. (See Syphilitic Arteritis.)

It is important to determine whether *idiopathic epilepsy* can be separated from that due to syphilis simply by the symptoms. This is very difficult to decide. So far as the convulsion itself is concerned, it is not possible to separate them. If, however, we can obtain any history, the matter becomes much more simple.

Fournier, in his lectures on epilepsy, in the Louvain, in Paris, in 1875, gave a summary of his views as follows:

1. In the *syphilitic epilepsy* there is nearly always absence of the shrill cry at the onset, so characteristic of the idiopathic variety.
2. There is frequently paralysis immediately after the attacks.
3. The seizure is incomplete or unilateral in character.
4. Attacks constantly increase in severity.

A therapeutic point, which may be used with the greatest success, is the administration of iodide of potassium in large doses. If the epilepsy be syphilitic, it will usually become less severe, and enormous amounts of the drug may be borne with impunity. As much as 450 grains in twenty-four hours will often do good.

Epileptic convulsive disorders may arise owing to the action of a very large number of toxic substances, of which only a few will be considered here, as an enumeration of all of them is manifestly impossible.

Alcoholic epilepsy consists of two distinct varieties produced by over-indulgence in intoxicating drinks. In one of these the convulsions are symptomatic of acute poisoning, and come on during a drunken orgy or immediately after a single large draught of liquor.

In the second variety the convulsion does not originate while there is alcohol in the blood, but in the intervals between the attacks of delirium tremens resulting from chronic excessive alcoholic indulgence. Under these circumstances the paroxysms are generally accompanied by hallucinations or by dementia or imbecility. In the alcoholic convulsion the symptoms may closely resemble those of true epilepsy, and not rarely the attack is ushered in by headache, gastric embarrassment, disorders of vision, and excessive tremors or some similar prodrome which may be looked upon as partaking of the nature of an aura. As a general rule, these alcoholic convulsions occur in paroxysms—two, three, four, or more, one after the other, at intervals of a few minutes. Not only may *grand mal* be closely simulated by alcoholic epilepsy, but simple vertigo or true *petit mal* may exist, either alone or associated with major convulsions. Alcoholic epilepsy is often associated with hallucinations, especially of terror, and not rarely is followed for days by a certain degree of

mental disturbance. Rather curiously these cerebral disturbances result rather in suicidal than homicidal tendencies, which is just the reverse of the insanity following simple epilepsy.

The symptoms of a uremic convulsion will be spoken of further when studying its differential diagnosis in connection with epilepsy.

The diagnosis of *lead epilepsy* from the idiopathic varieties is somewhat difficult, if the patient is seen for the first time during an attack; but the ordinary methods of determining chronic lead poisoning are, of course, of equal value here. The blue line on the gums may be present, and, if so, the diagnosis is almost certainly lead poisoning; but its absence is no proof that lead is not present. The administration of iodide of potassium also will so increase the elimination of the poison as to benefit the case and render it more easy to recover lead from the urine.

The history of exposure to lead in any form is, of course, exceedingly valuable evidence, but it should not be forgotten that in many cases this history is wanting. Thus, the poison may be derived from a hair-dye, or cosmetic, or from water which contains lead from pipes, or from an endless line of similar hidden and obscure sources. Amaurosis may be present in some cases, or optic neuritis with atrophy may occur. Where double wrist-drop is present the diagnosis is much more easy.

The symptoms of epilepsy due to chronic poisoning by lead are chiefly as follows: the man, apparently in his usual health, or who has had for a few days a feeling of weight in the head, or headache, is suddenly seized with most violent convulsions, which are often fatal, and which during their presence resemble ordinary epilepsy so closely as not to be separated from it. They end in coma, and are separated from each other by intervals of nervousness and disquiet. In some cases one convulsion follows the other so rapidly that death ensues from exhaustion, but in much more rare instances the attacks may resemble Jacksonian epilepsy very closely, and there may be no loss of consciousness. If such a condition occur, it is almost sure to be followed by a more violent fit. The attacks are not preceded by any aura whatever, but previous to the headache, already mentioned, the patient may have had amaurosis, and ophthalmoscopic examination of the eyes may show choked disk and neuritis of the optic nerve. As a general rule, such cases are fatal, but they may recover under careful treatment.

It is exceedingly important to differentiate between those convulsions which arise from *uremia* brought on secondarily by an action of lead on the kidneys and those which are due to a direct action on the brain. This may be difficult from the mere symptoms presented, but there are some points of difference. In the first place, the convulsion of uremia is, as a general rule, not so violent in its movements

nor so sudden in its onset. It is generally preceded by a few days of somnolence, or weeks of gastric disorder and headache, while lead epilepsy is generally sudden or preceded by cephalalgia by only a few days or hours. Again, examination of the urine in uremic convulsions will show a decreased amount of urea in proportion to the quantity of urine passed, while in plumbic epilepsy just the reverse will be true, unless the kidneys are affected *pari passu* with the cerebrum. If albumin be present, uremia is pointed to; but if the urine has a slow specific gravity and is passed in large amounts the indications are that there is chronic contracted kidney, which may or may not be the cause of the nervous disturbance.

Malarial epilepsy is an uncommon disorder, even in countries and regions which are notoriously malarial, but it has probably occurred, particularly in the southern part of the United States and in Brazil. The only cases which the writer can find recorded are by American or English observers, namely, Jacobi, Payne, and Hamilton. The latter gives but a passing glance at the subject, and the articles of the others the author has not been able to obtain, so that he knows them solely by reputation. In Hamilton's case a young man, who had lived for many years in an exceedingly malarious region, had more or less periodic epileptic attacks, attended by great preliminary rise of temperature and intense congestion of the face and head. He was unusually somnolent, and in the intervals frequently suffered from facial neuralgia. Change of the place of habitation and the use of quinine removed the disease entirely.

Undoubtedly the most similar convulsive condition that we have is that known as hysteria, and the diagnosis of one from the other is as difficult in some cases as it is essential and necessary for treatment and cure. The other conditions with which it might be confused are uremia, alcoholic epilepsy, tetanus, and syncope. Below are arranged all these disorders in a table, which briefly and succinctly shows the different points between them, although, of necessity, it is somewhat arbitrary on account of the lack of space.

As already stated, in epilepsy the movements are typically at variance with those of daily life, while in hysteria they are almost equally typical of ordinary muscular contractions, or, in other words, are more purposive in character, and frequently there is prolonged tonic contraction of the muscles, giving rise to the assumption of positions which bear more or less resemblance to normal attitudes. In hysteria, also, consciousness is impaired sometimes, but never so completely as in true epilepsy. Indeed, most commonly the individual knows all that goes on around her, for, while she may give no sign of consciousness by words or looks during the attack, she may afterward be able to narrate all that has occurred. Less commonly in hysteria, a condition known as automatic consciousness exists, in which, during

the paroxysm, the patient understands all that is said, but forgets everything on the return to quietness.

TABLE OF DIFFERENTIAL DIAGNOSIS OF EPILEPSY FROM HYSTERIA,[1] ETC.

Signs.	Epilepsy.	Hysteria.	Uræmia.	Petit mal.	Alcoholic epilepsy.	Tetanus.	Syncope.
Apparent cause.	None.	Emotion.	None.	None.	None.	None.	Mental shock.
Aura or prodromata.	Generally present, but short.	Globus hystericus; palpitat'n; cboking.	Headache, vomiting, and dyspepsia.	Faintness and dimness of vision.	Tremors.	Nervousness.	Not so well defined as in epilepsy.
Onset.	Sudden.	Often gradual.	Often gradual.	Sudden.	Sudden or gradual.	Gradual; begins in jaw.	Sudden or gradual
Scream.	At onset and sudden.	During attack.	Frequently none.	Frequently none.	May or may not be present.	None.	None.
Convulsion.	First tonic, then clonic.	Rigidity more pronounced, with more aching.	Rigidity generally absent.	No rigidity.	Movements more clonic than tonic.	Always tonic.	None.
Biting.	Tongue.	People, tongue, lips, and hands.	Tongue.	None.	Rarely.	None.	None.
Micturition.	Frequent.	Never.	Never.	Rarely, except when bladder is affected	Rarely.	Sometimes.	Never.
Defecation.	Occasionally.	Never.	Never.	Never.	Rarely.	Rarely.	Never.
Talking.	Never.	Frequent.	Muttering.	Never.	Never.	Never.	None.
Duration.	A few minutes.	Generally many minutes.	From a minute to hours.	Momentary.	May be prolonged.	Hours.	Indefinite time.
Consciousness.	Lost.	Generally preserved.	Lost.	Not lost always, but clouded.	Lost.	Preserved.	Lost.
Termination.	Spontaneous.	May be induced by shock.	Spontaneous.	Spontaneous.	Spontaneous.	Spontaneous.	Gradual, with no somnolence.

The movements of the hysterical patient after the tonic condition has passed away are as clonic as those of the epileptic, but still possess some purposive characteristics, and are not so bizarre as are those of the true disease. Thus the head, arms, and legs are struck with evident endeavor against the floor or surrounding furniture. Another point, which, when it occurs, is very distinctive, is the onset, toward the close of an hysterical convulsion, of a second stage of tonic spasm, such as occurred at the beginning. It will be remembered that this does not occur in epilepsy, although it must be borne in mind that in cases of the "status epilepticus" the rapid onset of another attack may show a second tonic stage. This can be separated, however, by the fact that it is followed by clonic movements, whereas the secondary tonic stage of hysteria is usually followed by relaxation and temporary recovery.

[1] This table is taken from the author's essay on Epilepsy, the prize essay of the Royal Academy of Medicine in Belgium, January, 1889.

In the secondary hysterical tonic contractions emprosthotonos and opisthotonos may occur, and are even more rigid in their character than they are in the first attack in some cases. Finally, too, in hysteria some peculiar emotional position is often assumed, as of the crucifix or of intense grief, or, perhaps, immoderate laughter is indulged in, with corresponding movements of the trunk. If the patient is quiet at this time, a smile may float across the face, while the eyes, with a look of pleasure, pain, or entreaty, may seem to be gazing at some object very far off. In some very well-developed cases the expression of pleasure is followed by a look of pain, with painful movements, or an appearance of intense voluptuous entreaty, with sensual venereal desire evidenced by gestures. Great terror may be present, and, as the scene constantly changes, the woman is now joyous, now mournful, now scolding, now praising her attendants or herself. Such is the history of a fully developed attack of hysteria.

In France there can be no doubt that the tongue is commonly bitten in hysterical convulsions, and that frothing of the mouth is frequently present; but in other countries this symptom may be regarded as indicative of epilepsy rather than hysteria. Doubtless the inexperienced reader will say, upon comparing these symptoms with those which were given as occurring in epilepsy proper, that the two disorders are easily separated from one another; but the author would insist upon the fact that in both cases he has given only the most typical characteristics of the diseases, and he repeats that all cases are not by any means so well defined. He would also remind the reader that the chief difficulty in making a diagnosis lies in the fact that frequently it must be made without any previous history of the case, as when a patient is brought into a hospital, in a fit, for treatment. When the history is obtainable or when the diagnosis can be put off until the case can be studied, the question is more easily solved.

If a large number of patients suffering from these hysterical attacks be questioned between times, it will be found that the so-called *globus hystericus* becomes an almost constant precursory symptom of an attack; and if the relatives be questioned, it will often appear that they have noticed that the fall to the floor is more gentle than in true epilepsy; but this is not always so by any means. Again, the expression of the face in hysteria is, between the attacks, often very characteristic, and the surrounding atmosphere of the patient seems, even to the inexperienced, to breathe hysteria. Very commonly areas of anesthesia and hyperesthesia occur in these patients, and are of all degrees of intensity and limitation. Search for them generally shows their presence after attacks of convulsions, but they may exist from one attack to the other, or develop sponta-

neously. In nearly all cases these areas are unilateral, and may extend entirely over one-half of the body, the line of demarcation of the anesthesia or hyperesthesia from the sound area being clearly and abruptly defined, generally at the median line of the front and back of the trunk. (See chapter on the Skin, that part dealing with Anesthesia.) It will be called to mind that such conditions are very rare in true epilepsy. Hallucinations are far more common after the fit in hysteria than in epilepsy, and sometimes they even occur during the attacks. They are always associated with the mental state; if terror is present, rats or disgusting objects are seen, and, according to Charcot, are generally seen on the side which, during the intermissions, is anesthetic. The pupil is more mobile in hysteria than in epilepsy, but may be contracted, normal, or widely dilated.

The following table gives, in as brief a manner as possible, the differential diagnosis between epilepsy and hystero-epilepsy, and is founded on a lecture by Professor Charcot, delivered at the Salpêtrière:

True Epilepsy.	*Hystero-epilepsy.*
Aura short.	Aura extremely prolonged.
Cry is violent.	Cry is more moderate and prolonged.
Spasms first tonic, then clonic, then followed by stertor.	Ataxic contractions, extension of limbs, turning of head, clonic movements, slight stertor.
Sometimes after fit of delirium or violent impulse or mania.	Bizarre contractions, no delirium, may be hallucinations.
Mental power is lost.	Mental power preserved.
No emotional attitudes.	Emotional attitudes.

A very useful differential point, strongly insisted upon by Charcot and Bourneville, is that in true epilepsy there is generally a very considerable rise of temperature during an attack, while in hystero-epilepsy the temperature remains normal or is only slightly raised.

In the diagnosis of true epilepsy from convulsions of a hysteroid character it is well for the physician to remember that the proportions of the two conditions in frequency of occurrence is, according to Gowers, 815 to 185 in every 1000 cases.

The differentiation of epilepsy from *uremia* is much more readily made, for there is usually a previous history of symptoms pointing to renal trouble, as, for example, some edema, or somnolence, or mental apathy, for some days or hours before the attack. Of course, in such cases recourse may be had to the ordinary tests for such conditions of the urine as are generally found when uremia exists; but it is to be remembered that epilepsy and kidney disease may exist hand in hand, and that for this reason the prognosis and diagnosis are to be carefully formed and given. If in a given case a prolonged history of dyspepsia, of frequent vomiting, occasional attacks of dyspnea, and failure of general health is found to be present, the correct diagnosis probably will be uremia. The preservation or loss

of consciousness in uremic convulsions is variable. Generally, if the convulsion is widespread and severe, the intellection is lost; but if it be only a slight attack, consciousness may be preserved. So long ago as 1840 Bright described cases of uremia, on the other hand, in which violent convulsions occurred without loss of consciousness, and Roberts has reported similar instances.

Fatal uremia may also occur in a patient whose urine is apparently normal; and, in cases of chronic contracted kidney, albumin may be absent from the urine for long periods of time. The specific gravity of the urine should be carefully noted, and in very doubtful cases careful estimations of the urea be made. If the specific gravity is constantly below 1.010, the kidney will nearly always be contracted unless diabetes insipidus exists. Tests of samples of the urine taken from the total daily quantity should always be made. Another means of testing the integrity of the kidney is to administer iodide of potassium and study its elimination. It is affirmed that, after a full dose, this drug can in an hour be readily recognized in the urine by adding nitric acid and then starch; but when contracted kidney exists the iodine fails to appear or is excreted only in very small quantities. A grain of methylene blue may also be used. (See chapter on the Urine.) The temperature of the body may also be used to differentiate between uremia and epilepsy. In 1865 Kien called attention to the fact that even when uremic convulsions are most violent they are accompanied by a fall of temperature of as marked a character as the rise noted in epilepsy. Since then this has been confirmed by Roberts, Hirtz, Hutchinson, Charcot, Bourneville, and Teinurier.

The diagnosis between *puerperal eclampsia* and epilepsy consists chiefly in the acuteness of the attack, and the fact that with no previous convulsive history a woman becomes suddenly convulsed during the pregnant or puerperal state. This is not the place for a discussion of the identity of uremia and puerperal eclampsia, although we believe that uremia is generally responsible for the nervous disturbance. If the convulsions are uremic, the temperature, according to the investigators just quoted, should fall; and according to Bourneville, puerperal convulsions are distinctly separated from those of uremia by reason of the fact that the temperature rises with great rapidity in the very beginning of the convulsions, and there remains with great steadiness. The condition of bodily temperature can, therefore, be used to differentiate puerperal eclampsia and uremia.

It is unnecessary to state once more that *petit mal* is but a variety or modification of *haut mal*. Nevertheless, it is useful to be able to separate it somewhat from the more severe form of the disease in the attempt to form a prognosis.

Some suppose that *petit mal* may be designated as consisting of one or two of the chief symptoms of epilepsy proper, and others have thought that the preservation of consciousness was the chief dividing line between it and fully developed epilepsy. The last idea is certainly incorrect, but it is impossible to give any outline which will absolutely separate the two conditions, so far as symptoms go. An important and useful point, is that, whereas the inhalation of amyl nitrite stops true epilepsy, the use of this drug, as a rule, increases the severity of an attack of *petit mal.*

The separation of *syncope* from epilepsy is one of the easier tasks imposed upon us. The color of the face, the weakened heart beat, sudden loss of consciousness, and the general appearance aid us here very much but it is to be recalled that in "Stokes-Adams disease" epileptiform seizures are often present.

Before closing this portion of this chapter the writer must bring forward the points to be used in differentiating epilepsy from those attacks simulated by malingerers. Often this is most difficult; and it is related by Fournier that, after his expressing an opinion that a man could always tell them apart, one of his assistants threw himself to the floor on his next visit in a pretended attack, whereupon Fournier, completely misled, exclaimed, "Poor M——; he is epileptic!" upon which the assistant, smiling, arose to his feet and refuted the statement.

Very serious injuries are sometimes submitted to by these persons to carry out their designs. The points to be looked into are: the condition of the pupils, which, in the simulated attack, always react normally; the corneal reflexes cannot be held back; the color of the face is rarely changed; and the thumbs are rarely flexed as they should be. Marc has pointed out that in malingerers the bystander can readily straighten out the thumbs and that they remain so; whereas in epilepsy they instantly become flexed again.

Suggestions as to movements are sometimes followed by malingerers, and the convulsant movements themselves generally lack the bizarre character so typical of epilepsy.

If tobacco or ammonia be held to the nose of the fraud, he generally is forced to disclose his true nature, but these drugs have no effect upon the unconscious epileptic.

The fact that in malingerers there is no rise of temperature may also serve as a differential point.

Convulsions Appearing in Infants or young children may result from injuries to the brain in birth, from the presence of growths, or from other distinct cerebral causes, and irritation of the alimentary canal. In these cases they may be reflexly produced.

There is one variety of infantile convulsive seizure due to meningitis, which is in itself often tuberculous and associated with retraction

of the head and squint; and another variety in which the symptoms very closely resemble those due to actual meningeal lesions, but in reality are quite independent of them. This condition has been called "pseudomeningitis", or "hydrocephaloid disease," and is seen in young infants generally after attacks of severe diarrhea. The fontanelle is depressed, the child is somnolent or comatose, and fever may or may not be present. The prognosis in the first class of cases is very bad. In the second class it is bad enough, but recovery quite often occurs if the treatment generally employed in the first class is set aside and a highly nutritious and supporting treatment is instituted.

If a child suddenly develops symptoms of acute meningitis, and has delirium, rigidity of the neck, and the major manifestations of the disease, the lungs should be carefully examined for croupous pneumonia, as this disease in children very often causes these cerebral or meningeal symptoms. Even in the adult maniacal delirium and rigidity of the neck may be present in croupous pneumonia, owing to meningeal involvement.

Convulsions, which are epileptiform, sometimes occur in the later stages of Addison's disease.

Epileptiform convulsions may come on in adults as the result of *multiple sclerosis*, and they are very commonly seen in sunstroke when the patient is first attacked.

Severe convulsions have been known to follow *irrigation of the pleural cavity* after aspiration, and they may also be seen in young children suffering from whooping-cough at the time of the paroxysm.

Tetanic Convulsions.—The convulsions which are of spinal origin, namely, those that are tetanic, are the result of *tetanus* or the *ingestion of strychnine* in poisonous dose, or its fellow ignatia, and sometimes are due to hysteria. The diagnosis is aided by what has been said in the last few pages in respect to the symptoms of hysterical convulsions, and finally in those patients that recover by the discovery of the hysterical stigmata.

Tetanus convulsions and strychnine poisoning are to be separated from one another by the fact that in tetanus the locking of the jaws comes first, while in strychnine poisoning it comes last. The convulsions of tetanus rarely, if ever, completely relax, while those of strychnine do have periods of complete relaxation. There is a different history in each case: in one, perhaps, of an injury, as of a nail run into the foot; in the other, of a dose of poison having been swallowed.

The differential diagnosis between strychnine poisoning and hysterical convulsions is more difficult. The convulsions are rarely so persistently tonic in hysteria as in strychnine poisoning, and the peculiar expression of the hysterical face is often seen in this

disease. The history of the patient, if obtainable, will throw much light on the case and aid very materially in the separation of the two conditions.

Tetany.—When a patient is seized with sudden and symmetrical tonic spasms of the hands, extending to the upper arms and shoulders, so that the fingers are flexed at the metacarpophalangeal joints and extended in the phalangeal joints, and the lower limb is flexed, while the legs are extended and the toes are flexed, the condition is one of *tetany*. (See chapter on the Hands and Arms, "accoucheur's hand.") It is most commonly seen in hysterical cases and has no relation to true tetanus. Pressure on a nerve trunk or bloodvessel will often produce an attack in such persons, and this is sometimes called "Trousseau's symptom." The pressure must be applied for several minutes in some cases, and the best place to apply it is the bicipital sulcus or the crural sulcus. Sometimes pressure on the brachial plexus or on the popliteal space will be provocative of an attack. It is not a constant symptom, but pathognomonic if found. Another equally useful diagnostic sign is called Chvostek's facial symptom. This results from the fact that the facial muscles are irritable, so that when they are tapped by the finger tip, or a rubber hammer, contraction results. The tapping is usually applied over the zygomatic arch in its anterior portion, and this will result in a spasm of the upper lid of the eye and the alæ nasi. In other cases stroking the area over the parotid will have the same effect. The muscles in tetany also have an increased electrical excitability (Erb's symptom).

It is worthy of note that both Trousseau's and Chvostek's symptoms are sometimes met with in rhachitic children, particularly if they have craniotabes. (See chapter on the Head.) Laryngismus stridulus will often be found associated with tetany and rickets.

Under the name Escherich's pseudotetanus a curious symptom complex characterized by persistent generalized tonic contractions of the muscles of the neck, back, legs, and jaw has been described. It may occur alone or in association with an acute infection as diphtheria. It is not a true tetanus as its name implies and is really a form of tetany.

Convulsions limited to a few muscles or more widespread in character may appear as symptoms in *acute yellow atrophy of the liver;* but the peculiar symptoms of this disease render easy the diagnosis of their cause.

Convulsions may also arise from *hematoma* of the dura mater (internal hemorrhagic pachymeningitis), but the diagnosis from those due to cerebral hemorrhage is practically impossible.

Spasms.—General spasms, in distinction from convulsions, are represented by chorea in its various forms, and by saltatoric and palmic spasm, paramyoclonus multiplex, and the occupation-

neuroses. There are other localized spasms from nervous diseases, such as facial spasm and wryneck, athetosis, and posthemiplegic chorea. Some of these conditions will be found discussed in the chapter on the Hands and Arms and that on the Face and Head.

When a patient is afflicted more or less constantly and more or less universally by disordered, irregular, jerking movements which throw the part or parts affected into unusual positions, which are not, however, maintained even for a moment, the condition is probably *chorea minor*. Often the speech is seriously disturbed by reason of the choreic movements of the lips and tongue or jaws, and some loss of power may be manifest in certain muscles. This true chorea or St. Vitus' dance may affect the whole body or only one arm or leg, but generally it is diffused. Commonly it ceases at night when the child sleeps, but it often persists day and night, and then becomes a serious malady, because of the exhaustion produced. It often follows fright, prolonged bad weather, and other causes which may upset the nervous balance of the child. Chorea in childhood is so characteristic in its manifestations that it can be readily recognized in most cases; but it sometimes has to be separated in adults from disseminated sclerosis, progressive muscular atrophy, hysteria, and Friedreich's ataxia. The movements in disseminated sclerosis are, however, fine muscular tremors, instead of minor jerking movements; and there are present nystagmus and scanning speech in sclerosis, but not in chorea. Again, in progressive muscular atrophy there is fibrillary muscular tremor, but not twitching of a marked form, and the muscles are wasted. In hysteria the muscular movements are rarely choreic, and the presence of changes in the color fields and the other stigmata of hysteria (see chapters on the Skin and on the Eye) renders a diagnosis of the latter condition easy. Friedreich's ataxia is to be separated from chorea by the scanning speech, scoliosis, slow incoördinate movements, and the family history of the disease.

Rarely when there is some paresis with chorea, the patient may present symptoms of acute poliomyelitis; but the paralysis in the latter affection is more marked, and there are no movements in the affected muscles, such as occur in chorea.

Chorea insaniens is a violent form of ordinary chorea associated with mania, which is not to be confused with choreic movements occurring in the choreic insane.

Choreic movements sometimes come on in the aged, and must be separated from paralysis agitans and senile trembling. This is possible by the fact that in paralysis agitans the movements are tremors, and there is loss of power with the peculiar facial expression ("Parkinsonian visage") and a hurrying gait (festination). Senile trembling is usually an affection limited to the head, and consists in a tremor

and not in marked twitching. (See chapter on the Hands and Arms, part on Tremors.)

A rare form of chorea has been called *Huntingdon's chorea.* It occurs in adults about the age of thirty to forty years. It is hereditary; that is, there is generally a history of the same trouble in the ancestors of the patient, and finally as it progresses psychical disturbances ensue. Irregular movements first appear in the hands, which movements become markedly incoördinated, the arms are thrown about in excessive and rapid jerkings, and when the infection involves the legs a characteristic gait is developed of a dancing or "hop, skip, and jump" character. Sometimes, early in the malady, the movements can be controlled by the will. The face passes through slowly formed grimaces, and the gait may be staggering. The speech becomes indistinct, and enunciation is not clear. Finally, dementia closes the scene. The movements of Huntingdon's chorea are not sudden as in true chorea; it is a disease of adult life, and mental disturbance is a prominent symptom. These facts separate it from ordinary chorea.

When the patient involuntarily bends over in a profound bow the cause of his movements may be rhythmical contraction of his abdominal muscles, producing the so-called *salaam convulsions* or chorea major.

A still more rare malady is *electric chorea* or "Dubini's disease," in which the muscles of the arm and then the leg on the same side are affected with a sudden muscular spasm or shock, such as is produced by the electrical current. Wasting of the affected muscles, loss of faradic irritability, occasional epileptic convulsions, and rarely elevation of temperature come on. The disease is a fatal one, and generally occurs in malarial regions in Italy. Under the same name of electric chorea Bergeron has described a state of rhythmical muscular spasm which usually ends in recovery.

When a condition of clonic muscular spasm affecting the trunk, limbs, and perhaps the neck is present, the hands and toes being uninvolved, as a rule, the possibility of the presence of *paramyoclonus multiplex* is to be considered. The spasms in this rare disease are bilateral and occur at intervals, often only on an attempted movement or speech. So violent are the muscular contractions in some cases that the patient may be thrown to the ground, or, if in bed, to the floor. These movements may vary from 3 or 4 to 120 per minute, but are generally about 50 per minute. The symmetrical bilateral involvement, the fact that the movements are not choreic in character, and that the patient is a male, are to be remembered in making the diagnosis. The ultimate prognosis is favorable unless the movements are so constant as to cause exhaustion. Care must be taken not to confuse hysterical movements with this condition.

The bilateral movements which affect only the larger muscles, and the fact that paramyoclonus multiplex is nearly always seen in the male, separate it in part from hysteria, while the hysterical stigmata when they are present will point to hysteria as the cause of the disorder.

Sometimes a patient will be met with in whom, when he attempts to stand, the leg muscles first become rigid and then are thrown into violent contractions, which cause him to jump up and down, or he may be thrown to the floor. This condition is called *saltatoric spasm* or "jumpers." It is to be separated from the condition of the legs seen in lateral sclerosis of the cord by the fact that in the latter disease the legs become spastically stiff on attempting to use them, from Huntingdon's chorea in that voluntary movements with the hands may be performed perfectly, and from chorea minor by the absence of small incoördinated twitchings.

Such a patient will often act on suggestions or in imitation of the acts of other persons or of animals.

Some writers confine the term "saltatoric spasm" to those cases which possess no imitative features. In such cases the disease is far more moderate in its manifestations.

Quite distinct from these clonic spasms of the muscles brought on by attempted movement is that in which the muscles become tonic on attempted movements. At first they are stiff and slow in their movements, but ultimately develop a tonic spasm, so that walking is at first almost impossible, but the limbs limber up on exercise. This is a rare affection, called *Thomsen's disease*, or one of the forms of *myotonia congenita*. (See chapter on the Feet and Legs.)

Forced gyratory movements of the body are sometimes seen as the result of a lesion of the middle peduncle of the cerebellum.

CHAPTER XVIII.

HICCOUGH, VOMITING, REGURGITATION, AND THE CHARACTER OF THE VOMIT.

Due to uremia—Cerebral lesions—Intestinal obstruction—Peritonitis—Cholera—Gastric disease—Hepatic disease—Poisons—The appearance of vomit.

HICCOUGH.

Hiccough or singultus may or may not possess considerable clinical significance. Often it arises from slight indigestion. In other cases it is produced by the drinking of sparkling wines or waters. When hiccough becomes persistent it is a symptom to be regarded with interest, for if it continues for a long period of time it is usually significant of hysteria or uremia, while if it develops in a patient exhausted by some prolonged or severe illness it shows deep depression of nervous tone, and is itself dangerous because of the exhaustion it speedily produces. Sometimes it is said to be an annoying symptom after passing catheters or bougies in cases of stricture in the urethra. Hiccough develops in peritonitis, and is a most distressing symptom. It is also seen in cases of intestinal obstruction and when abdominal growths are developing. Singultus also takes place in some cases of cerebral hemorrhage, in myelitis affecting the upper parts of the spinal cord, and in very rare instances because of severe mediastinopericarditis involving the phrenic nerve. It also occurs as a result of central nervous irritation in persons suffering from advanced anemia, and in cases of suppurative hepatitis.

VOMITING.

Vomiting is the act by which the contents of the stomach are forcibly expelled from this viscus through the cardiac orifice, the esophagus, the pharynx, and the mouth. The vomiting centre in the medulla oblongata gives rise to the necessary nervous impulses, and is provoked to this by direct stimulation or by reflex irritation. Thus in uremia the vomiting sometimes encountered is the result of irritation of the centre by some unknown poison. When apomorphine is given the centre is also stimulated. Centric vomiting is also caused by the administration of anesthetics, notably ether and chloroform.

On the other hand, gastric, intestinal, or other abdominal disorders may reflexly produce very persistent emesis, and for these reasons vomiting is of considerable diagnostic importance.

As vomiting is produced by many maladies, it is a symptom frequently met with. It occurs with a certain degree of constancy as a complication or symptom of uremia, diabetes, apoplexy, brain tumor, brain abscess, Ménière's disease, tuberculous meningitis, hysteria, intestinal obstruction from its various causes, gastric and intestinal indigestion, gastritis, gastric ulcer, gastric cancer, peritonitis, nephritic colic, hepatic jaundice, hepatic colic, in cholera, yellow fever, and a host of other ailments. Sometimes the onset of one of the acute infectious diseases of childhood is characterized by vomiting. Not infrequently this symptom associated with diarrhea masks the presence of the real cause of the illness, as in some cases of croupous pneumonia.

The vomiting of *uremia* may be one of the earliest manifestations of renal disease, and its presence, when persistent in the absence of local gastric or other causes, should always lead to an examination of the urine, since valuable time may be lost if the patient is considered to be suffering from some slight indiscretion in diet. Its association either as a preceding, concomitant, or consequent symptom of coma renders a diagnosis of uremia probable, while a history of uremic amaurosis, colliquative diarrhea, and failure of the general health will be very important points in reaching a decision. No pathognomonic symptoms of uremic vomiting exist unless we consider the urinary evidence a symptom, but in some cases the vomited matters smell strongly of carbonate of ammonium, resulting from the decomposition of the urea which has been eliminated from the blood into the stomach by the gastric mucous membrane. Uremic vomiting is, therefore, not only due to centric irritation by a poison in the blood, but to irritation of the stomach by the urea which is excreted into it. Diabetes comparatively rarely produces vomiting by the toxemia which it causes, but in any case the urinary examination and polyuria decide the diagnosis.

When vomiting results from *cerebral hemorrhage, embolism,* or *thrombosis,* the focal or hemiplegic symptoms characteristic of apoplexy are present. Possibly the vomiting is more indicative of hemorrhage than of plugging of the vessel. A sudden attack of vomiting in a previously healthy man of advanced years, or in one who is young but has a specific history, should raise the question as to the possible presence of one of these lesions; provided, of course, that ordinary gastric disorder is not present as a cause.

The vomiting due to *cerebral tumor* is generally accompanied by the characteristic severe and constant headache, vertigo, a slow pulse, impaired memory, and sometimes by epileptiform convulsions.

Further than this, the important diagnostic ocular symptom called "choked disk" of the optic nerve is to be sought for, and if found is of great positive value. Tumor of the brain, if near the base, often causes, too, involvement of the various cranial nerves. (See chapter on the Eye.) The vomiting of cerebral tumor is independent of taking food, and commonly comes on early in the morning, thereby differing from some of the forms of vomiting due to gastric disorder. The vomiting arising from *cerebral abscess* has symptoms precisely like those just named, so that a differential diagnosis is almost impossible. The history of injury or of an infectious process producing a secondary brain abscess may point to this cause of the vomiting: the real points of difference are that in abscess choked disk is rarely seen, fever is commonly present, and the cranial nerves generally escape. When *purulent meningitis* produces vomiting it may be impossible to tell whether this symptom is due to it or to an abscess, as the purulent collection may be localized. Vomiting sometimes results from *profound cerebral anemia* of an acute type due to hemorrhage, in fainting or in chronic anemia, as in chlorosis. Generally, however, the symptom is only a constant nausea. The presence of great pallor and other evidences of anemia aid in the diagnosis, but it must not be forgotten that some severe anemias are accompanied by febrile movement and by marked choked disk, which should not mislead the physician into a diagnosis of cerebral tumor.

When vomiting is due to *cerebellar tumor*, the diagnosis is aided by the presence of vertigo, the peculiar staggering gait, and finally by evidences of choked disk, on ophthalmoscopic examination, with disordered vision.

The vomiting of *meningitis* is quite frequently an early symptom, but it also often occurs later in the disease, and is caused by the meningeal irritation, and not by any condition of the stomach, unless that viscus has been disordered by the unwise use of drugs. This form of vomiting can nearly always be separated from that due to other causes by the excessively severe headache, chiefly of an occipital type; by the pain in the nape of the neck and in the spine; by the rigidity of the dorsal muscles, so that opisthotonos may be caused in severe cases; and, finally, by the disordered functions of the cranial nerves, as a result of which there are found trouble in the oculomotor nerve, strabismus, double or single ptosis, slowly reacting pupils, which may be unequal, nystagmus, and sometimes facial contractions due to involvement of the facial nerve.

Vomiting due to *acute miliary tuberculosis* often comes on at the very onset of the malady, and is associated with obstinate constipation, or, on the other hand, active diarrhea; but the fever, the very rapid pulse, the wasting of the patient, the possibly present physical signs of tuberculosis of the lungs, and, very important, the peculiarly

severe dyspnea, for which no adequate cause can be discovered on physical examination, all point to the general infection. If a skilful examination of the eye can be made with the ophthalmoscope, the choroid may be found to be studded with tubercles.

The reflex forms of vomiting are very numerous, and depend chiefly upon organic and functional disorders of the abdominal viscera. In some of these conditions vomiting is of little importance, except for its gravity if the patient is exhausted; in other words, it is simply a disagreeable symptom. In others the symptom vomiting is of considerable diagnostic value as indicating the grave mischief which produces it. One of the most important of the latter conditions is *intestinal obstruction*, whether it arises from intussusception, constrictions by bands, volvulus, or impactions.

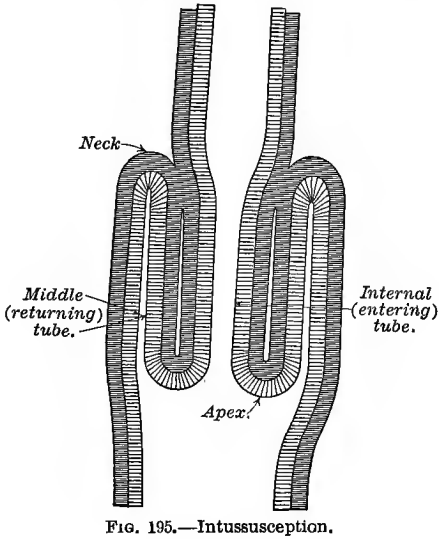


FIG. 195.—Intussusception.

In *intussusception* vomiting is practically a constant symptom, occurring with the sudden pain, or, at times, even preceding it. In children it continues until shortly before death, and is rarely feculent.

In the adult, and in the chronic form, there may be complete absence of vomiting, though this is certainly exceedingly rare. Leichtenstern takes exception to the statement that the seat of obstruction is indicated by the period at which vomiting is developed. The ileum invagination is most frequently accompanied by early vomiting, not because of its seat, which is usually but little removed from the ileocecal valve, but because it is commonly obstructive. The vomiting, both in time of development and in nature, will depend

not upon the seat of the trouble, but upon the presence or completeness of obstruction, and may be early if the obstruction is absolute in the sigmoid flexure, and feculent if the occlusion is in the upper part of the ileum.

The pain is usually sudden, violent, diffuse, or, if localized, usually centred in the ileocecal or umbilical region. After a few hours in children, a much longer interval in the adult, the pain ceases, often s suddenly as it commenced, and there is an interval in which there little to suggest that the pathological condition still continues. This is followed by a return of the pain, the paroxysms becoming more violent and prolonged, the intervals less marked as the disease progresses, or in the adult, if it passes into the chronic form, and intervals even of days may elapse between the paroxysms. The pain is frequently accompanied by tenderness, but this is an exceedingly variable symptom, and at times pressure seems to relieve the pain.

Blood-stained mucous evacuations are a symptom of intestinal obstruction which, in children, is rarely wanting. Of 108 cases of invagination in the first year of life this symptom was absent in but 4. It occurs within a few hours of the first attack. At the first the discharge is of blood-stained feces; later, if obstruction is developed, of blood and mucus, and is usually exceedingly offensive. In children diarrhea is common throughout the whole course of the case. At times, following complete constipation and feculent vomiting, there will suddenly appear copious evacuations from the bowel, mingled with blood, in which may be found evidences of the necrosed intussusceptum. Where this slough is extensive it may be lodged in a lower portion of the bowel and cause fatal obstruction.[1]

In connection with the mucosanguinolent evacuations the tenesmus or straining is a symptom so common that it is of some diagnostic import. That it is not dependent upon the character of the evacuation is shown by the fact that it is present in cases of complete obstruction. Brinton has shown that this symptom is seldom developed except in the ileocecal and colon invaginations.

A much rarer condition, and one which Leichtenstern ascribes to the secondary effect of intense tenesmus, is a patulous condition of the anus due to paralysis and dependent upon invagination of the descending colon and rectum. This is never produced by invagination of the ileum.

Leichtenstern's statistics show that a tumor can be felt either through the parietes or by rectal examination in 52 per cent. of all cases. In the first year of life this most important diagnostic sign was present in 63 per cent. The tumor is usually felt in the left

[1] For much information on the subject of intestional obstruction, see the Fiske Fund Prize Essay of the Rhode Island Medical Society for 1899, by Dr. Edward Martin and the author.

iliac region, or by the finger passed into the anus. The ileocecal invagination is most frequently accompanied by demonstrable tumor; the ileum invagination exhibits this symptom with less frequency.

Many authors have noted that the tumor varies in size and consistency from time to time, now, during an acute paroxysm of pain, being hard, knotty, and plainly perceptible, shortly afterward eluding the most careful search. Duchaussoy has described two distinct movements which can often be perceived in the tumor, namely, the erectile and the vermicular motion.

Distention of the abdomen is not of great significance, because it is often absent. In children especially it may not appear at all, or may appear only just before death. In adults, in whom obstruction is more common, it may become as well marked as in obstruction from any other cause.

In the chronic form of invagination the symptoms are less violent in onset; there may be nothing more characteristic of the attack than recurring paroxysms of pain, meteorism, and obstruction, with symptoms of intestinal stricture constantly manifesting themselves. These cases terminate either in cure by reduction or by extrusion of a slough, or perish from exhaustion, inanition, or in the course of an acute attack. In over one-half of the recorded cases a correct diagnosis was not made.

The additional symptoms upon which a diagnosis of vomiting from intussusception is to be based are the acute onset of colicky pain, and its intermittent character; passages from the bowels containing blood and mucus; the presence of tumor, commonly in the left iliac region, or felt through the anus, varying in size and consistency from time to time, with an erectile or vermiform motion; and the ordinary obstruction symptoms. The diagnosis is further confirmed if there are present violent peristalsis and tenesmus, and if these symptoms occur in an infant.

According to Leichtenstern, Bryant, and others, 40 per cent. of all cases of intestinal obstruction are due to intussusception, and this condition is most common in the first year of life, after which it becomes more and more rare until the fortieth or fiftieth year, when it increases in frequency. The prognosis is bad, the mortality varying from 73 to 90 per cent. unless early surgical relief is given.

Internal strangulation by bands occurs in from 25 to 30 per cent. of the cases of obstruction of the intestine, and affects males most commonly between twenty and forty years of age. In 133 out of 151 cases the small intestine was involved. The typical symptoms are as follows:

1. Sudden, agonizing pain, constant, and located about the umbilicus, with paroxysmal increments.
2. A rapid, weak pulse and subnormal temperature. This is

nearly constant in the early stages of the attack; later on, when local or general peritonitis develops, the temperature and pulse may assume the features characteristic of inflammation.

3. Vomiting. First of the contents of the stomach, then of bile, and, finally, in a large percentage of cases, of feculent matter. The feculent vomiting rarely appears before the third day, and in cases running a very acute course death may ensue before this symptom has time to develop. The vomiting is constant and gives no relief to the patient.

4. Constipation. Exceptionally there may be one or two passages representing the contents of the bowel below the seat of obstruction; after that the constipation is absolute, not even flatus passing by the anus. Treves has suggested that the evacuations sometimes observed toward the termination of the case, and not due to the relief of obstruction, may be dependent upon the beginning of peritonitis.

5. Tympanitic distention. Where there is a large segment of gut involved in the strangulation its early distention may give rise to a localized abdominal enlargement, which is exceedingly suggestive as to the cause of the attack. In general, the meteorism is not marked except when peritonitis supervenes.

Since in the large majority of cases the obstruction is localized in the lower portion of the small intestine, the primary distention will be observed in the middle abdominal region—*i. e.*, the epigastric, umbilical, and hypogastric areas. Laugier claims by this symptom to locate the obstruction with some certainty.

The violent peristalsis and repeated vomiting prevent the extreme meteorism characteristic of intestinal paralysis.

6. Localized tenderness and percussion dulness. When present these signs are of exceedingly great importance, since they denote the position of the strangulated bowel.

Exceptionally a tumor may be felt, formed by the congested gut or the matting together of the intestinal coils.

The urine is diminished in quantity and may be suppressed. In acute strangulation it commonly contains albumin, and it is stated that this is of diagnostic value.

In this connection the history is of great importance.

Other congenital deformities would suggest the possibility of Meckel's diverticulum as a cause.

A preceding typhlitis, pelvic peritonitis, or severe abdominal traumatism would respectively assign an adherent vermiform appendix, peritoneal bands, or rents in the omentum or mesentery as the causative agents in the production of the symptoms.

The age of the patient should also be considered, since this form of obstruction usually occurs between the twentieth and fortieth years.

The sudden onset of violent, persistent pain, subnormal temperature, and frequent pulse, the obstinate, absolute constipation, the persistent, repeated vomiting, becoming fecal, and the rapid course of the disease, all point to internal strangulation.

Auscultation of the abdomen is at times of value, a sound compared to the click of the water-hammer being heard most distinctly at the point of obstruction.

Palpation and percussion should not be omitted, as thereby the seat of obstruction has been distinctly located.

Volvulus is the most frequent form of intestinal obstruction after intussusception and that due to strangulation. Vomiting occurs, but is not so constant a symptom as in those forms first named. Thus it occurred in from 8 per cent. in Brinton's statistics to 2.5 per cent. in those of Treves, and 4 per cent. in Martin's and the author's. It is nearly always seen in men in middle life. The vomiting is rarely fecal, is very slight in many cases, and sometimes does not appear at all.

Vomiting, on the other hand, is quite commonly seen in the cases of *obstruction from impaction* or obstruction from foreign bodies. The distention is slight, the amount of systemic shock far less than in other forms of obstruction, and the duration of the attack somewhat longer than usually obtains in this class of affections. The symptoms of obstruction are frequently only partial, the vomiting being moderate in amount and not stercoraceous, the constipation not being absolute.

Except in the case of enteroliths and very large foreign bodies a tumor can rarely be felt.

It is often impossible to diagnosticate this form of obstruction from that depending upon a narrowing of the lumen of the bowel, such as is produced by cancer or stricture. The previous history is always of great importance.

In this connection it is to be remembered that *hemorrhagic infarction* of the intestine may produce symptoms similar to those just described, namely, pain in the region of the navel or more generally throughout the belly, fecal vomiting, diarrhea, and bloody stools. Actual obstruction may be present. Search should be made for a source from which an embolus may arise, as, for example, valvular cardiac disease, and if this is found the likelihood of infarction being present is increased.

When persistent vomiting develops in an infant during the first few weeks of life and no error in diet can account for the symptom, the physician should consider the possibility of the presence of *congenital hypertrophic stenosis of the pylorus*. At first the symptom may be present only after food is taken and the quantity ejected may be small or it may be greater in amount than the food recently

swallowed, indicating retention in the stomach of a former meal. Bile is never present in the vomit. In a few days the stomach becomes remarkably intolerant of food and the vomiting may be projectile in character. Rapid emaciation takes place and the physician may on palpation find a tumor at the pylorus. On inspection waves of contraction in the walls of the stomach may be seen passing from left to right.

When vomiting arises from *general peritonitis* it is often one of the earliest symptoms of the malady. It is almost always present, and is often a very severe symptom, and is associated with or replaced by a constant retching, which adds to the exhaustion of the patient. At first it may only follow the swallowing of food, but often it occurs without such a cause, and after the stomach is emptied of its ordinary contents glairy, watery mucus is expelled, which is often of a distinct, greenish tint. The great tenderness of the belly in acute peritonitis, the moderate fever, the rapid pulse, the anxious face, and the cold skin as collapse approaches, all render the diagnosis easy; but it is to be remembered that the distention of the belly by an overfilled bladder or pregnant uterus may mislead the physician into thinking that peritonitis is present because of the swelling, the pain, and the vomiting. Vomiting is not a severe symptom of appendicitis unless the peritoneum has become involved in the inflammatory process, although it may occur once or twice when the pain in the appendix is most severe. The localization of the symptoms in the neighborhood of the appendix makes the diagnosis possible. (See chapter on the Abdomen.)

When vomiting occurs in *typhoid fever* it is usually a symptom of bad feeding or imperfect digestion, and is rarely of grave importance except under two conditions. The first of these is when it occurs as a result and symptom of intestinal perforation, an accident commonly seen late in the disease; and, second, when it takes place as an obstinate and exhausting symptom after the fever has practically passed by, from unknown causes, probably toxic in character. The symptoms of perforation other than vomiting can be found in the chapter on the Abdomen and Abdominal Viscera.

Vomiting as a symptom of *cholera* is accompanied by serous diarrhea of profuse character, by the development of collapse, cramps in the muscles, anuria, and great circulatory failure. It should be separated from the vomiting due to cholera morbus or severe indigestion, antimonial poisoning, and arsenical poisoning. Cholera morbus is to be separated from cholera, first, by the absence of the comma bacillus in the stools; second, by the fact that there is a history of exposure to cold or damp, or bad food; third, by the absence of an epidemic; and, fourth, by the fact that its manifestations are milder.

No one can be skilful enough to separate symptoms of *poisoning*

by antimony from those due to cholera, for they are identical in every way. Nothing but the history of the ingestion of the poison and the discovery of antimony in the excretions can prove the case to be one of antimonial poisoning, particularly if an epidemic of cholera is present.

In *arsenical poisoning* the association of vomiting with bloody stools separates the symptoms from those of cholera.

Vomiting is often a very severe and early symptom of *cholera infantum* (see chapters on the Abdomen and on Bowels and Feces), and it occurs in attacks of *true dysentery* as a common symptom, when its underlying cause is readily discovered. (See Abdomen.)

The diseases of the stomach causing vomiting are cancer, ulcer, gastritis, catarrh (acute and chronic), true gastritis, and dilatation.

The vomiting of *gastric cancer* at first consists in the expulsion from the stomach of its contents—mixed particles of food, mucus, water, and sometimes bile. The vomit may be tasteless or sour from fermentation, and may have an offensive odor from similar causes. Often it contains blood, either in bright-red streaks or as a brownish-red fluid, or in similarly colored clots, which may be brown when they have been in the stomach for some time. Often the exuded blood, changed by mixture with the stomach contents, looks like coffee grounds, producing "coffee-ground vomit." This coffee-ground vomit is not pathognomonic of gastric cancer, but is very characteristic of this disease. Microscopically the vomited materials are seen to consist of particles of food, yeast cells, cocci, and broken-down blood corpuscles. (For the other symptoms of gastric cancer, see chapter on the Abdomen.)

Coffee-ground vomit is also sometimes seen in cases of *locomotor ataxia* following a gastric crisis. Roux asserts that overdoses of iodide of potassium may produce a gastric crisis in locomotor ataxia by irritating the stomach.

Vomiting due to *gastric ulcer* is preceded by pain, and is generally brought on by taking food, and so occurs soon after eating. The food is, therefore, only slightly digested, and evidences of fermentation are absent to a great extent. If blood is present, it is nearly always bright red and in considerable quantity, and indicates that a hemorrhage has recently taken place from the surface of an ulcer. Very profuse hemorrhages into the stomach may cause vomiting by irritating and distending this viscus. The history of vomiting after eating, the presence of blood in the vomit, the pain in the stomach, the age of the patient (generally twenty to thirty years), the sex (generally female), and the hyperchloric acidity, combined with the other symptoms (see chapter on the Abdomen), complete the diagnostic array of facts.

There are, however, other causes of vomiting of blood or hema-

temesis than gastric ulcer and cancer. Thus, it occurs from obstruction to the portal circulation from *hepatic cirrhosis*, and from growths and splenic affections which result in varicosity of the bloodvessels of the stomach. Hematemesis also follows severe blows, kicks, and other injuries to the epigastrium. Sometimes it takes place in cases of *heart disease* in which there has resulted hepatic engorgement with secondary gastric congestion, and it may be developed in small degree by any form of violent vomiting which strains the stomach, particularly if an irritant substance has already destroyed the mucous membrane. Again, hematemesis is seen in scurvy, typhus, yellow fever, and acute yellow atrophy of the liver, as a result of breaking down or destruction of the coats of the vessels. Sometimes it is seen in cases of dengue, in influenza of the epidemic type, and in relapsing fever. Hematemesis may also occur in purpura hemorrhagica, in hemophilia, and possibly as vicarious menstruation. In malarial fever of a severe character the dark-colored vomit is generally due to bile, but it may be due to exuded blood. Such a case is reported by Boon as occurring in a child.

Care should always be taken that the physician is not misled by the vomiting of swallowed blood into a diagnosis of gastric hemorrhage from any of the causes just named. It may enter into the stomach from the pharynx, as after epistaxis, or blood may be swallowed by a malingerer. Hematemesis is to be separated from hemoptysis by the fact that in the latter there are physical signs in the lungs, the sputum is frothy and bloody, there is absence of retching or vomiting movements, and the blood is brighter red in hemoptysis than in hematemesis, as a rule.

Under the name of *melena neonatorum* there is a condition of hematemesis occurring in children within the first few days or weeks of life. This condition has been thought by Leube to be due to gastric and duodenal ulcers, and his views are indorsed by Buhl and Huhn, Spiegelberg, Binz, and Landau. In one of the latter's cases thrombosis of the umbilical vein was present, and it has been thought that when expansion of the chest takes place in the newborn child small clots may escape from this vessel through the ductus arteriosus into the descending aorta and gastric arteries, and thus cause an ulcer of the stomach by embolism.

The vomiting of *acute gastric catarrh* is generally seen in children, and is often preceded by great nausea. The contents of the stomach are first gotten rid of, then mucus, water, and bile may be ejected, and finally exhausting retching ensues if the attack is severe. The tongue in such cases is coated and dotted with red spots from the enlarged fungiform papillæ, and the epigastrium is tender on pressure. There may or may not be fever and looseness of the bowels. The attack usually follows indiscretions in diet or exposure to cold.

Vomiting from *chronic gastric catarrh* is usually a condition met with in adults, and when seen in the male is most frequently the result of a frequent use of alcoholic beverages to excess. In women it often develops from excessive tea drinking associated with errors in diet. When due to alcoholism, the vomiting is often present only in the morning before or after taking food, and then is called the "morning vomiting of drunkards." (See chapter on the Tongue.)

Vomiting due to *true gastritis* or inflammation of the stomach in its deeper layers is very rare, except as a result of the ingestion of an irritant poison or hot liquid.

Perhaps the vomiting occurring in *dilatation of the stomach* is more typical in its character than any other. This act is often a prominent symptom of gastric ectasy, the matters vomited being often greenish and extremely fetid, and nearly always profuse in amount. Examination of the ejecta will generally show food swallowed days before, owing to the imperfect digestive action of the stomach, and this very inability of the stomach to act on the food generally gives, for a long period of time, a sense of weight and fulness often amounting to pain, and complained of bitterly. There is tenderness over the epigastrium on pressure, and the displacement produced by the palpation often brings on either acid or yeasty eructations or even the vomiting already named. Nausea preceding the vomiting is by no means common, there being simply a gush of foul liquids from the mouth. After such an occurrence the vomiting fails to recur for from twenty-four to forty-eight hours, or perhaps for a week—*i. e.*, until the viscus becomes overladen once more. The fluids which are given off on eructation are exceedingly acrid, nauseous, and bitter. Sometimes they are offensive, but more rarely odorless. The reaction of the vomit is almost always acid, lactic, and butyric being the acids most commonly found, but the normal hydrochloric acid is usually absent. Fibers of meat or masses of semi-digested and semi-decomposed food can be seen by the naked eye or under the microscope, and sarcinæ and many forms of bacteria swarm in the mass. Particular search should be made for the yeast fungus, *Torula cerevisiæ*, the presence of which is a certain evidence of active fermentation.

(For further information in regard to the symptoms of gastric dilatation, see the chapter on the Abdomen.)

Vomiting due to gastric dilatation should not at once lead the physician into a diagnosis of stenosis of the pylorus from growth or constrictions in this part of the stomach, or from innate feebleness of the gastric walls, for similar conditions may follow growths of the pancreas, which by pressure occlude the pyloric opening (see chapter on the Abdomen).

Sarcinæ ventriculi are found not only in the frothy, dirty looking

vomit of gastric dilatation, but in that of chronic gastric catarrh, cancer, and ulcer of the stomach. If iodine or iodide of potassium is added to the vomit, the sarcinæ become mahogany red or brown, and are easily recognized, occurring in squares which are separated by dividing lines (Fig. 196).

Vomiting also arises from *neurosis of the stomach* in several forms. Thus it is frequently seen in hysteria, in neurasthenia, pregnancy, and sometimes occurs in the form of what Gee has called "cyclical vomiting." It also comes on in association with gastric crises in locomotor ataxia. The vomiting of *hysteria* is generally characterized by its persistent character, often lasting for months, and yet the patient often maintains her normal weight to a surprising degree, either because the food which is taken is only vomited in small part or because she surreptitiously obtains food when her attendants do not know it, which she retains. It is generally associated with so many of the hysterical stigmata as to be readily diagnosticated. The vomiting of *neurasthenia* is seen in both sexes, and is particularly apt

FIG. 196.—Sarcinæ ventriculi, with starch granules and oil globules, from vomited matters. (Otto Funke.)

to follow any nervous muscular exertion. Thus in one case of the writer's, even a short railroad journey taken by an overworked man produced attacks of spinal tenderness with vomiting which lasted several days. In some neurotic cases the vomiting takes place as soon as the food is swallowed. The vomiting of *pregnancy* is usually a morning vomiting, though it may persist all through the day. It has no particular diagnostic features in itself, save that there are rarely any signs of gastric indigestion. The presence of pregnancy, of course, makes the diagnosis clear; and in such cases the physician should always examine the urine, since although the ordinary vomiting of pregnancy is a symptom of the first few months, that occurring later on may indicate grave renal complications. (See Uremia, in chapter on Coma and in chapter on Convulsions.)

The *cyclical vomiting* already mentioned is generally seen in children, and is of rare occurrence. It is characterized by attacks of vomiting recurring after intervals of uncertain length, during which the patient may seem entirely well. The attack may last from a few

hours to a few days. There are often pain in the epigastrium and constipation. Sometimes retching is the main symptom. It is generally seen in neurotic patients and is probably a form of acid intoxication.

In *acute pancreatitis* there is colicky pain in the epigastrium, deeply seated and extending to the right shoulder and back, and great restlessness, precordial distress, dyspnea, and faintness are present. The matters vomited are greenish, clear, and viscid, and the efforts at vomiting increase the pain. There is no jaundice, but death soon comes to the relief of the patient.

As an early diagnosis of acute pancreatitis may permit surgical interference, with possible recovery of the patient, the diagnosis is important. The mistake commonly made is to consider the case one of intestinal obstruction.

Vomiting of a peculiar character is always present in *phosphorus poisoning*. The symptoms associated with ingestion of the poison may not come on for some hours. At the end of that time the peculiar taste of phosphorus may be noticed in the mouth, the breath is heavily laden with its odor, and burning pain in the esophagus, stomach, and abdomen ensues. Vomiting and purging now assert themselves, and the matters vomited and those passed from the bowels may be luminous in the dark, owing to the presence of free phosphorus. The vomit is at first made up of food, then mucus, then bile, then perhaps blood. All the symptoms of a mild gastro-enteritis may develop, but it is to be noted that constipation of an obstinate type may replace the purging. Very soon the liver increases in size, and gives rise to general hypochondriac pain and tenderness, as well as local swelling. At the end of twenty-four hours, or perhaps after the second day, a cessation in the symptoms occurs, and, if the physician be not on his guard, this will lead him to a hopeful prognosis. In the course of a few hours jaundice begins in the conjunctiva and then extends over the entire body. With the onset of jaundice the vomiting and pain return with renewed vigor. The matters vomited are often the color of "coffee grounds," due to exuded and altered blood. The bowels are absolutely confined, or the few hard masses passed are white and clay-like, because of the absence of biliary coloring matter. There is no bile in the vomit in this stage, because the hepatic ducts have been closed by the inflammation set up in the liver. After this nervous symptoms ensue. Muscular twitchings, headache, vertigo, wild delirium, erotic convulsions, and, finally, unconsciousness and death occur. Sometimes the convulsions occur just before dissolution. Even if the patient survive the acute stage, he generally dies of the changes produced in his vital organs, which consist in widespread fatty degeneration, even in the acute stages. Atrophy of the liver, destruction of the gastric tubules,

pancreatic involvement, and kidney degenerations aid in producing the ultimately fatal result.

The symptoms may so closely resemble those of acute yellow atrophy of the liver as to make a differential diagnosis impossible, unless some evidence of the presence of phosphorus is obtainable.

Vomiting of a nervous type is a common complication of *exophthalmic goitre*, and when it occurs sometimes develops into a dangerously severe symptom, owing to its constancy, violence, and resistance to treatment. Oftentimes serous diarrhea is associated with it, and these two associated symptoms should not mislead the physician into a diagnosis of cholera morbus nor of jaundice, for icterus often comes on.

Ménière's disease is associated with vomiting, the contents of the stomach being expelled after the attack of vertigo and tinnitus aurium.

The *affections of the liver* which sometimes result in vomiting are chiefly catarrhal and obstructive jaundice, hepatitis, hepatic abscess, and pylephlebitis. The rapid development of jaundice, hepatic tenderness, and swelling, or a history of violent hepatic pain (colic), renders the diagnosis possible in the case of jaundice. (See chapter on the Skin.) Hepatitis—that is, hepatic abscess—is often accompanied by or produces vomiting which is apt to be very obstinate. The swelling of the liver, the tenderness in the hypochondrium on palpation, the pain in the hepatic region, often referred to the neighborhood of the right shoulder, and the febrile movement, which is intermittent, sweeping up to 104° or 105°, then down to normal, are the chief characteristic symptoms. (See chapter on the Abdomen.) Vomiting accompanied by a similar train of symptoms also occurs in cases suffering from pylephlebitis.

Violent vomiting associated with great pain in the loin, radiating down into the testicle, or inside of the thigh, indicates the presence of a *renal calculus*, either in the pelvis of the kidney or in the ureter.

Hemoglobinuria is sometimes accompanied by vomiting. The attacks are paroxysmal, and are often ushered in by persistent yawning, with pain in the limbs, headache, nausea, and vomiting, followed by moderate fever, which is preceded or accompanied by a chill. Pain may be felt in the liver, but, more pathognomonic than all, the urine is soon found to be dark, brownish red, or even black, owing to the presence in it of hemoglobin.

Vomiting is a frequent coincident symptom of headache, because in many cases the headache depends for its existence upon a disordered stomach or disordered bowels; but it also appears as a characteristic symptom of a condition in which the stomach is primarily not at fault, namely, in *migraine* or *hemicrania*, in which, in addition to violent pain in the head on one side, we may have

hemianopsia, scotomata, and sometimes great pallor or flushing of the face. (See Pain.) Usually the vomiting follows the pain.

When vomiting occurs in *yellow fever*, the presence of an epidemic, the suffusion of the eyes, the headache, the black character of the vomit, the slow pulse, scanty urine, and prostration, all point to the cause of the symptom.

Vomiting is a frequent symptom in some cases of *phthisis*, particularly if laryngeal tuberculosis is present. It also occurs as a result of swallowing the sputum instead of expectorating it, and very commonly excessive cough produces vomiting, especially if the cough follows closely after a meal.

Closely associated with the vomiting due to these causes is that occurring in cases of *pulmonary gangrene*.

In children suffering from *whooping-cough* vomiting often takes place at the close of the paroxysm, and is due to the spasmodic movements of the chest and diaphragm.

Finally, it is not to be forgotten that vomiting often ushers in any one of the *eruptive diseases*, such as the fevers, syphilis in its early secondary stages, and erysipelas.

Under the name of merycismus cases of *voluntary regurgitation* of food have been reported, chiefly outside of the United States. The patients have been nervous or hysterical persons.

The Vomit.—Aside from the diagnostic significance of the act of vomiting, the physician should remember that the vomit itself may give him information as to the condition of his patient.

Under the head of gastric dilatation we have spoken of the significance of vomiting large amounts of liquid and undigested food, so that it is not necessary to speak of this point here; but it is well to remember that small amounts of vomited material often possess considerable diagnostic importance. In the severe retching of cerebral disease or uremia very little material is gotten rid of, and this may be ammoniacal. In cases in which small quantities of exceedingly sour, clear liquid are vomited, we often find that the attack is due to migraine or nervous headache. If watery liquid and mucus are vomited, there is probably gastric catarrh. The vomiting of bile is usually only seen when repeated retching has drawn this secretion into the stomach. The liquid may be either golden yellow or greenish in hue. Somewhat like this vomit is that seen in peritonitis, in which disease grass-green material is often expelled. Similar material is also vomited in cases of intestinal obstruction before stercoraceous vomiting comes on.

The vomit of intestinal obstruction is sometimes fecal in odor for obvious reasons. If odorous poisons have been taken, the vomit smells of the poison; and if there be phosphorus present, the vomit not only smells of it, but in addition may be luminous in the dark.

The significance of bloody vomit has already been described. It only remains to name the test for hemin, which, if present, shows that the color of the ejecta is really due to blood. Some of the vomited material is filtered, and a little of the filtrate is evaporated on a watch-glass. This dried material is now scraped off the glass, mixed with a trace of finely powdered common salt, and placed on a glass slide. The powder is now covered by a cover-glass and one or two drops of glacial acetic acid allowed to flow under the cover-glass. This is then dried by exposing it to gentle heat, and after the drying is absolute a drop or two of pure water is touched to the edge of the cover-glass. Very minute crystals of hemin are now seen with the aid of the microscope.

CHAPTER XIX.

COUGH AND EXPECTORATION.

The varieties of and diagnostic significance of cough—The causes of cough—The sputum—Its pathological significance.

THE significance of cough as a symptom is very important, and, though it may arise from many causes, in the majority of instances it points to disease in the chest, in the trachea or the larynx, in the pharynx or in the nose. Rarely it is a purely nervous trick, and equally rarely it arises from irritation in the stomach ("stomach cough," so called). A cough is said to be dry and hacking when it fails to bring up into the throat or mouth any secretion, or when it is short and sharp. Often such a cough is paroxysmal; in other cases it consists in single but fairly frequently repeated, short, and forcible expiratory efforts, as if the patient was trying to clear his throat. A loose cough is nearly always paroxysmal; that is, it occurs "in spells," and at nearly every paroxysm results in the raising of some mucus. The first variety of cough is that seen in the early stages of phthisis pulmonalis, acute bronchitis, or pneumonia, before any exudation has taken place; in the early part of a paroxysm of asthma; in the early portion of an attack of whooping-cough and when the cough arises from irritation in the upper air-passages, whether this be due to the inhalation of dust or the presence of some growth, as a laryngeal papilloma. The loose variety of cough is seen in the later stages of acute bronchitis, pneumonia, asthma, whooping-cough, and in cases of emphysema with bronchiectasis, and in the stage of pulmonary tuberculosis associated with the breaking down of lung tissue, the formation of cavity, and the development of bronchitis with it, and in gangrene of the lung.

There are two peculiar forms of cough to be mentioned, namely, the so-called barking, brassy, laryngeal cough, which we hear most typically in false or spasmodic croup, and the cough of whooping-cough, which is, as its name implies, the most typical which we meet with. Suddenly the child begins to give a series of quick, sharp coughs, which become more and more rapid until the chest is emptied of air. In the early stages of the disease this is all that occurs, and unimpeded inspiration ensues; but later the cough no sooner ceases from exhaustion of the lungs of air than with the attempt of deep inspiration the glottis closes spasmodically, and the air is sucked

through the chink with a whooping sound. The flushed or cyanotic face of the child, associated with these paroxysmal attacks, renders the diagnosis easy.

There is nothing distinctive in the cough of early stages of pulmonary inflammation, whether it be bronchial or vesicular, although, if the bronchitis be very intense or if the pulmonary inflammation also affect the pleura, the cough may be partly smothered or suppressed by the patient, who endeavors to control or stop it in order to escape the pain it causes. To this end he sits or lies in bed, endeavors to fix the muscles of his chest so that they will not respond to the reflex cough impulse, and shuts his lips to hold his breath in, although very often the reflex irritation overcomes his will-power and the cough bursts through his compressed lips with an expression of pain. Such a cough is always indicative of pain.

In all forms of dry cough there is now and again a small plug of mucus expelled from some part of the respiratory mucous membrane. Such coughs possess no value to the patient, being merely a sign of reflex irritation; but a loose cough, unless it is very excessive, is of the greatest possible use to the patient, for it is an effort on the part of nature to rid the lungs of abnormal exudations or secretions. For this reason this symptom is not to be removed completely in cases of resolving pneumonia, pulmonary tuberculosis, or bronchiectasis with excessive secretion, since, if drugs are given which stop the cough, the lungs are speedily filled with the secretion; and in the case of tuberculosis or gangrene or mucopurulent bronchitis septic absorption with systemic infection results. Similar good results are reached by the cough heard in cases of pulmonary abscess, and when an empyema has broken into a bronchial tube. When the patient complains of chronic cough, which is worse in, or confined entirely to, the morning hours, and tells us that the cough finally causes the discharge of much secretion, and that this is followed by freedom from cough for many hours, the case may be one of tuberculosis with cavity, pulmonary abscess, empyema which has ruptured into a bronchus, or sacculated bronchiectasis. Such coughs come on in paroxysms whenever the lung must be relieved, and the length of paroxysm depends upon the looseness of the secretion and its situation in the lung. Thus, if the secretion be in the larger bronchial tubes, it is easily expelled; whereas if it be in smaller bronchi, or in the vesicles, or at the bottom of a cavity, great and frequently repeated effort will be required before the liquid can be raised into the mouth for expectoration.

The presence of an obstinate cough due to bronchitis, which resists all ordinary treatment, should lead the physician strongly to suspect that one of two ailments is present, namely, undiscovered tuberculosis, cardiac failure, or Bright's disease.

Cough brought on by changing the position of the patient often arises because of the alteration in position of a pleural effusion.

The cough of acute laryngitis may be quite severe, and occurs in short, sharp barks of a harsh or brassy character (like spasmodic croup) which is so typical as to be called a laryngeal cough. The association with this cough of partial or complete loss of voice and pain in the larynx, with a history of exposure to cold and dust, or of the excessive use of the larynx in speech or singing, renders the diagnosis clear, even if the laryngoscope is not used to discover congestion and inflammation of the laryngeal mucous membrane. In the false croup of children, which is always associated with laryngeal irritation, the barking, ringing cough is so characteristic as to render a diagnosis possible as soon as the sound is heard, and with it there is dyspnea due to obstruction to breathing.

The cough of the laryngeal phthisis is not so typically brassy and ringing as that of acute laryngitis, but the presence of pain in the larynx, hoarseness, and persistent laryngeal dryness and difficulty should lead to a search for tuberculosis by the laryngoscope, and an examination of the chest for physical signs of trouble in the lungs and of sputum for tubercle bacilli.

Sometimes cough of a laryngeal character is due to an aneurysm pressing upon the larynx. In other cases the cough depends not upon the pressure of an aneurysm, but upon the pressure produced by carcinoma of the esophagus, by a mediastinal tumor.

Cough due to the inhalation of irritant dusts or vapors is often present in girls who work in carpet factories, in the air of which there are immense quantities of fine particles of wool. Again, it is seen in knife grinders, needle workers, coal miners, and in workers in arsenical and lead pigments.

Sometimes in paralysis of the pharyngeal muscles (glossolabiopharyngeal paralysis) cough is produced by the slow passage of food, which may in fact enter the larynx.

A night or evening cough is very commonly seen in cases of respiratory catarrh or more grave disease. It is often absent all day, only to return in the evening in cases of laryngitis and in phthisis; and in those cases in which it follows getting into bed, it is due to chilling of the skin by the cold sheets, which results in congestion of the inflamed mucous membrane.

Quite frequently children suffering from chronically enlarged tonsils suffer from cough on going to sleep, especially if the uvula is relaxed or elongated. The cause of this cough is that in the relaxation of sleep the tonsils touch one another or tickle the uvula. As soon as the child wakes muscular contraction separates the approximating surfaces and the cough soon ceases.

If this cause of cough cannot be eliminated we must look farther

for its origin. Not infrequently hypertrophy of the mucous membrane over the turbinated bones, so that it presses on the nasal septum, may cause cough, and irritation of the inferior and middle turbinated bodies and the septum opposite the inferior turbinated body may cause reflex cough. So, too, enlargement of the pharyngeal tonsil may cause this symptom, as may also elongation of the uvula. When chronic enlargement of the tonsils, with follicular accumulations, is present, cough frequently results.

In regard to coughs in general, it can be said that in the absence of the early stages of acute inflammation of the respiratory apparatus a dry, hacking cough is either nervous or is due to reflex irritation in the ear or stomach, or to some hyperesthetic spot in the nasal, pharyngeal, or laryngeal mucous membrane; whereas a loose cough may arise both in adults and children from congestion and engorgement of the lingual tonsil. Such a cough is frequent, dry, and paroxysmal, and seems to arise from a sticking or pricking sensation in the throat; whereas a loose and prolonged cough depends upon causes of greater gravity farther down in the respiratory organs. Generally, if the stomach is at fault, a low grade of pharyngitis will be found.

The cessation of cough in advanced phthisis, suffocative bronchitis, or the bronchorrhea with bronchiectasis of old persons, or in severe pneumonia, indicates exhaustion, collapse, or approaching unconsciousness, and is a bad sign.

THE SPUTUM.

Macroscopic Examination—A careful examination of the materials expectorated by the patient, or, in other words, of the sputum, is of the utmost importance in all cases of disease of the respiratory tract, whether the abnormal process be primary or secondary. The methods which we resort to in examining sputum are macroscopic, or those of the naked eye, and microscopic. Of these, the microscopic are by far the most important. Sputum varies greatly in its general character on ordinary examination, sometimes being very fluid and even watery in consistency, and sometimes thick or tenacious. In some instances it is clear and glairy-looking, resembling somewhat partly beaten white of egg; in others it is yellow and opaque. Placed on a clean linen cloth, the sputum may evaporate to almost nothing, or leave a heavy mucopurulent deposit after all moisture is gone.

The naked-eye appearances of sputum are, however, quite characteristic in several conditions. Thus, in the later stages of acute bronchitis the sputum is apt to be thick and yellowish, and to contain lumps of half-inspissated mucus. In croupous pneumonia it is

rusty in color, is peculiarly free from watery ingredients, and is gelatinous to such an extent that it adheres to the spit-cup, so that when this vessel is well filled its contents do not readily fall out even when the cup is tipped upside down. The sputum may be less adhesive after resolution is well advanced. The brightness of the blood in the sputum in cases of pneumonia is also a guide to prognosis. Thus, Sir William Jenner said: "The less the weight for a given height, the more red blood in the sputum, the better the chance for the patient." In pulmonary hemorrhage or hemoptysis, after having, perhaps for a short time, a salty taste in the mouth, the patient suddenly brings up, with or without much preceding cough, a gush of nearly pure blood or blood freely mixed with ordinary sputum. The blood is bright red, not dark or prune juice in appearance, and the liquid is frothy, while the cough, which is always present after the hemorrhage has occurred, is suppressed and resisted by the patient, who fears further bleeding. This hemoptysis may be caused, first, by pulmonary tuberculosis; second, by valvular cardiac disease, generally involving the mitral valves, and resulting in pulmonary infarction; third, by aortic aneurysm; fourth, in persons suffering from severe purpura; fifth, from persons suffering from hemophilia; sixth, from vicarious menstruation; and, seventh, in rare cases of hemorrhagic smallpox.

Bloody sputum must be separated from bloody vomit due to gastric hemorrhage arising from ulcer or cancer. (See Vomiting.) This can be done by the cough, by the frothy character of the expectoration, by the presence of physical signs in the lungs, and by the history of pulmonary disease. It may, however, be confused with slight hemorrhage from a dilated and ruptured vessel on the posterior pharyngeal wall, in which case, after a little coughing, there may be expelled on a handkerchief a little blood-tinged saliva. Examination of the throat will usually reveal the ruptured vessel or other vessels dilated, but still intact. For a number of days after an attack of hemoptysis there may be expelled in the sputum dark clots of blood. So-called "currant-jelly" clots are expelled by coughing in many cases of malignant growths of the lungs.

Other causes of blood-streaked sputum are aortic aneurysm with leakage by oozing into a bronchus or the trachea; and particularly in children do we see streaks of blood in the sputum if there be present pulmonary gangrene.

Care should always be taken to discover whether the materials spit up are really tinged with blood, for they may be colored by some dyestuffs or the blood of some animal for the purposes of deception.

Finally, it is well to remember that a reddish-brown or brick-dust looking sputum is sometimes coughed up in cases of hepatic abscess communicating with the lung; and the sudden expectoration of a

brownish, purulent-looking sputum by a person who has been a sufferer from dysentery should cause the physician to examine the sputum for the ameba coli, in order to discover if the case is one of pulmonary abscess secondary to amebic dysentery. Symptoms of hepatic abscess may also be present. This has been called "anchovy-sauce" sputum.

In addition to the sputum already described we sometimes see a peculiar semiliquid sputum in cases of pulmonary phthisis, in which the sputum promptly separates into two layers on standing, the upper one being light and flocculent, unless there is a well-marked bronchial catarrh present, when it may be markedly mucopurulent, or, if a large cavity is present, its purulent character may be even more marked. To this list may be added several others, namely, the purulent sputum of pulmonary abscess or empyema, of mediastinal abscess opening into a bronchus, subphrenic abscess, hepatic abscess, pronounced bronchiectasis, and that from a large tuberculous cavity in the lung. Of these the more common are bronchiectasis, tuberculous cavity, and empyema breaking into a bronchus. In the first of these the cough is paroxysmal, and after it has been kept up for some time a gush of purulent sputum is suddenly brought up into the mouth, and the accumulation of pus is removed for a time. In the other the sputum is very fluid, and is so free that its expectoration rapidly fills the spit-cup, provided that the patient is strong enough to bring it up. A very liquid watery sputum is seen in pulmonary edema, particularly that seen in cases of Bright's disease or heart failure, and in some of the pulmonary forms of epidemic influenza.

Sputum which, on standing, separates into three layers, the top one frothy and dirty looking, the next clear and filled with shreds, and the lowest consisting of a sediment of pus and broken-down-looking materials, is seen in cases of pulmonary gangrene, and if the sputum when placed in a vessel containing water sinks to the bottom in disk-like masses or globules the disease may be tuberculosis.

The sputum expelled by an asthmatic at the time of the attack also has characteristics not so easily seen at a glance, but nevertheless demonstrable by the naked eye. Small pearls or plugs of mucus of the size of a sago-pearl are seen in the sputum, and if these are placed on a plate of glass under which is a black surface, and then teased out, they will be found to be rolled-up fibers, which when unrolled are found to be in the forms of curls or spirals. These are sometimes called Curschmann's spirals (Fig. 197), and they are rarely seen in several other conditions than asthma, namely, in chronic pulmonary tuberculosis and croupous pneumonia. Through the central core of the curl runs a bright and refractive filament, which is waxy, and is probably not an entity, but an optical effect.

Between the spiral fibers can be seen in many cases slight, bluish, octahedral crystals varying greatly in size, sometimes requiring a high-power lens to distinguish them. They are said by Salkowski to be composed of a mucous substance, but others believe them to be oxalate of lime, a phosphate of an unknown base, or ethylenimin. These are called Charcot-Leyden crystals, and are also seen in the sputum of chronic croupous pneumonia, chronic pulmonary tuberculosis, and in acute bronchitis.

Sometimes, in cases of diphtheria, casts of the larynx and upper bronchial tubes are expelled by coughing. Small casts are also seen in the sputum of that rare affection, fibrinous bronchitis. Some-

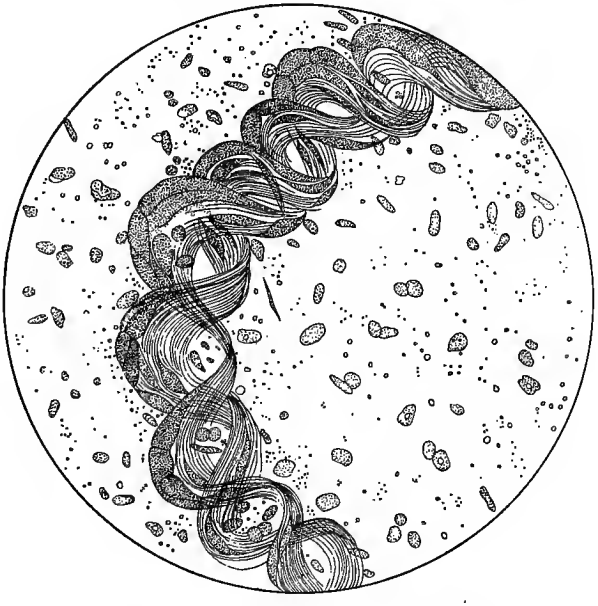


FIG. 197.—Curschmann's spirals. (Von Noorden.)

times these casts consist of a perfect mould of several branching bronchial tubes and bronchioles, and they may be white, yellowish, or even pinkish in color from bloody exudation. Sometimes they are only visible to the naked eye if placed in water and shaken, when what has appeared to be a roll of mucus spreads out into the characteristic shape of the tubes from which it comes. Casts of the finer tubes can sometimes be found in the sputum of cases of croupous pneumonia.

Microscopic Examination.—The examination of the sputum by the aid of the microscope should be made with care. A portion of the sedimentary part of any sample may be carefully separated from

the rest by means of a pipette; but to facilitate the examination of sputum for tubercle bacilli when but few exist, for the rapid and thorough examination of the sputum for elastic fibers, which has been heretofore a tedious and complicated process, and for the discovery of Charcot-Leyden crystals and fibrinous coagula, the centrifuge is a much better apparatus. (See chapters on Urine and Blood.) The tubes used for the precipitation of the sputum are 50 mm. long, with $2\frac{1}{2}$ mm. lumen, and are fitted into the hematocrit frame.

A small quantity of sputum is placed in a clean porcelain dish and actively stirred for a few minutes with a glass rod until it becomes thin and free from lumps and apparently homogeneous. Without any dilution whatever the sputum is drawn into the sputum tubes by means of a medicine dropper connected with a small rubber tube. The two precipitating tubes, filled with sputum in this way, are placed in the hematocrit frame and revolved for at least three minutes, making about 15,000 revolutions. The solid portions of the sputum collect in the distal extremity of the tubes. A small portion of the sediment is placed on a slide and examined microscopically for elastic fibers, Charcot-Leyden crystals, etc. The sediment from the second tube can be stained for tubercle bacilli or other microörganisms as desired.

When the centrifuge is not used and a small particle of sputum is placed upon a glass slide under a cover-glass and gently pressed, there will be seen, if the sputum be mucous or mucopurulent, threads of mucus and mucous corpuscles with white blood corpuscles, which are particularly numerous if the sputum be purulent. These latter are granular, fatty, and sometimes pigmented by soot or other substances which have been inhaled. Epithelial cells derived from the respiratory passages are also found in large numbers, often broken down and fissured, granular, and generally a nucleus can be distinguished in their centre. Of far more importance than these, however, are the particles of elastic fiber or elastic threads, which, if present, show positively that a breaking-down process is going on in the lung, or more rarely in the bronchial tubes. These are usually seen in the sputum of advanced or rapidly progressing tuberculosis of the lung and in that of abscess and gangrene of the lung. If there is doubt as to their presence because they are sparse, we obtain them by the following process: boil equal parts of the suspected sputum and a 10 per cent. solution of caustic potash, and dilute the jelly-like mass which results with water. After this has stood for twenty-four hours the elastic fibers may be found in the sediment as swollen threads, for which, however, small particles of food which may come from the mouth may be mistaken.

The appearance of fine, needle-like crystals of fatty (margaric) acid, which may be bent like a curved needle and often grouped in

bunches, may indicate pulmonary gangrene or purulent bronchitis. They are found chiefly in the plugs or lumps which the patient expels in his sputum, but they possess no indicativeness of the pulmonary changes just named if follicular tonsillitis, either acute or chronic, is present, since the plugs derived from the follicles of the tonsils also contain similar crystals. Again, they are of no diagnostic value if found in stale, mucopurulent sputum, as they may form in this after it has been expectorated. The peculiar crystals called Charcot-Leyden crystals have already been described.

There are four remaining objects to be seen in the sputum of diseased persons, all of which are of great diagnostic importance when found. The first of these is very rare, namely, the eggs of the *Distoma pulmonum,* which are found in the sputum. This parasite sometimes produces hemoptysis without any physical signs of pulmonary change, is rarely if ever seen in this country, but is common in

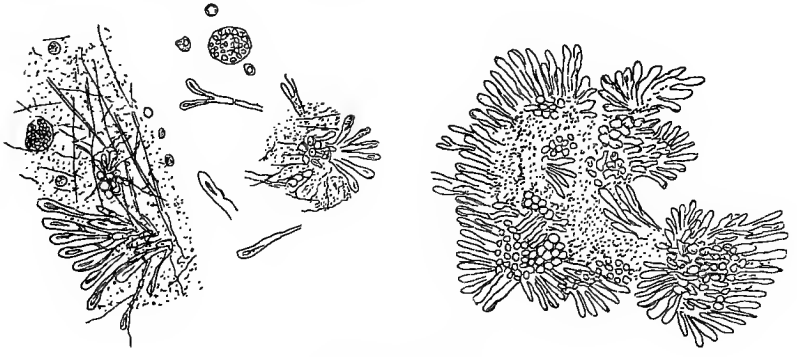


FIG. 198.—Actinomyces.

Japan, Corea, Formosa, and China. The second is the evidence of echinococcus infection by the appearance of portions of the cysts or of the hooks of the scolices when the cyst bursts into a bronchus from the lung or adjacent structures. Such cases are also rare in this country. The third condition, which is also very rare when involving the lung, is actinomycosis, in which infection we find radiated fungi or actinomyces in the sputum. This fungus appears as a number of club-shaped projections attached to a heterogeneous mass of irregular-looking material (Fig. 198).

The fourth and most important of all these finds in sputum is the *tubercle bacillus,* both from the point of diagnosis, prognosis, and treatment, and because the disease tuberculosis is so widely distributed in every class, in every part of the country, and is so constantly prevalent. The discovery of this bacillus in a sample of sputum which has not been exposed to the entrance of bacilli after it has been expectorated, is a positive sign of tuberculous infection of the lung

unless there be tuberculous disease of the upper air passages or mouth. While their presence gives positive evidence, their absence in a given sample of sputum is not negative evidence of an absolute character, for that particular specimen may be free from bacilli or they may have escaped the staining or the eye of the examiner.

The method for examining sputum for tubercle bacilli is as follows: The specimen which is brought to the physician is poured into a shallow glass vessel having a blackened bottom, or into a glass saucer placed on a piece of blackened paper. If this sputum is now closely examined, it will be found to contain small, yellowish masses, one of which should be picked up by means of a spoon or platinum needle, freeing it as much as possible from the mucus surrounding it. A very small part of this mass is now placed on a cover-glass and well distributed over its surface by means of a needle or teasers, or it is

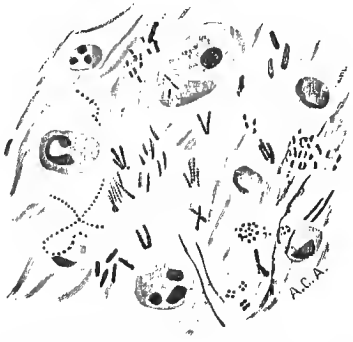


FIG. 199.—Tuberculous sputum stained by Gabbett's method. Tubercle bacilli seen as red rods; all else is stained blue. (Abbott.)

spread by placing another cover-glass over the first and pressing them together with a to-and-fro movement. The quantity of sputum used must not be large enough to extend over the edges of the glass. This having been done, the glasses are now separated by a gliding movement, and the thin film of sputum covering each one is allowed to dry by exposure to the air, after which, being held by forceps, it is passed through the flame of an alcohol lamp to fix the coating. Care must be taken that too much heat is not used. Some carbol-fuchsin stain[1] is now placed in a watch-glass and the cover-glasses are immersed in it. As soon as this is done the cover-glass is held over a flame until the steam begins to rise from it, when it is withdrawn, then heated again until this process has been repeated several times. The cover-glass is now washed in water, and the film covering it will be found to have an evenly distributed red color. The glass is next

[1] Carbol-fuchsin stain, or Ziehl's solution, is made by dissolving 1 gram of fuchsin in 10 c.c. of alcohol, and adding 100 c.c of a 5 per cent. solution of carbolic acid.

placed for a moment in a 25 per cent. solution of nitric or sulphuric acid in water and gently moved about, to decolorize the deposit on the surface of the glass. As a result the albumin and cells on the glass are decolorized, but the bacilli are not. The glass is next washed in dilute alcohol (60 per cent.) to remove any free fuchsin. Should the decolorization be imperfect, so that the film still has a red color, it must be still further decolorized by returning the glass to the acid solution and then washed again in the dilute alcohol and water. The cover-glass is now placed in a solution of methylene blue (saturated watery solution). The glass is then finally washed in water, and afterward examined under the microscope, or dried and mounted in Canada balsam for permanent preservation. The tubercle bacilli are distinguished by the fact that they retain the red color from the fuchsin solution, while other bacteria, having been decolorized by the acid solution, take the contrast stain and appear blue.

Another method, Gabbett's, is perhaps more useful than that just given. It consists in staining, as already directed, with the carbol-fuchsin solution, and then placing the cover-glass in a second solution, which contains the acid for decolorizing and the contrast stain. This latter solution is composed of 100 c.c. of a 25 per cent. mixture of sulphuric acid and methylene blue 1 to 2 gm. The cover-glasses are left in this solution for a couple of minutes, and then washed in water. When placed under the microscope the tubercle bacilli will appear as red rods in strong contrast to the blue background.

CHAPTER XX.

PAIN.[1]

The kinds of pain—The significance of its locality—Colic.

It is manifest that it will be impossible for the author in this chapter to enumerate all kinds of pain, both as to the situation, degree, and character. He can only mention those forms which possess considerable diagnostic importance. It should always be remembered that pain is the sign adopted by Nature to notify the individual of some abnormal condition in his body, and in many instances pain is only developed when the attempt is made to move a part which from its condition had much better be allowed to rest.

Pain is generally described as darting or stabbing in character, when it occurs in a single or repeated paroxysm; as throbbing or pulsating, when it rises and falls in severity with the pulse beat; as dull and aching, when it resembles the feeling associated with a bruise. Sometimes stabbing or darting pains are called lancinating, or the patient may state that the pain is tearing and rending in character. All pain is associated with direct or indirect irritation of nervous matter, but, if the nerve or nervous centres connected with a part be destroyed organically or arrested in function, we have a condition called anesthesia. (See chapter on the Skin.)

Not infrequently darting or stabbing pain is associated closely with actual disease of nervous tissue, which may be primary or caused by the pressure or irritation of a growth or some foreign body. Such pains are seen in neuralgias due to inflammation of the sheath of a nerve or its surroundings as it passes through a bony foramen; in neuralgia due to meningeal thickening; in the lightning or tearing pains of locomotor ataxia; from pressure upon the spinal nerves by spinal disease or in that caused by fractured bones. Again, we often meet with violent pain as the result of true neuritis, whether it be produced by infection, by injury, or by poisoning.

Throbbing pain is nearly always associated with the presence of congestion or local swelling in the part where the pain originates, and arises from the fact that the peripheral nerves are subjected to pressure, which is increased with each additional beat of the heart. Dull, aching pain is often produced by slow inflammatory or pathological processes in organs not well endowed with sensory nerves.

[1] See also chapter on Headache.

There are two forms of pain yet to be considered which are peculiar in their character, namely, that nauseating pain due to a blow or injury to the testicle and ovary and that boring pain which occurs in cases of inflammation or morbid growth affecting bony tissues, particularly in the long bones.

There is another point in connection with the study of pain as a symptom of disease, namely, that pain is often referred to a point far away from the source of the symptom. Thus, the child with hip disease complains of pain in the knee or in the ankle; the one with dorsal caries, of pain in the intercostal nerves anteriorly; and a stone in the kidney may cause violent pain in the penis or testicle.

The physician should always remember that the degree of pain must be determined in part by the expression of the face and movements of the body, for often these features of a case will show that the pain described so vividly in words is much exaggerated. The general systemic signs of pain are a tense pulse, if the pain be recent in onset and acute; a somewhat accelerated respiration unless the pleuræ or lungs are involved, when it may be retarded; dilatation of the pupil; more or less sweating, particularly on the forehead; faintness; and sometimes the passage of clear, limpid urine if the pain be abdominal.

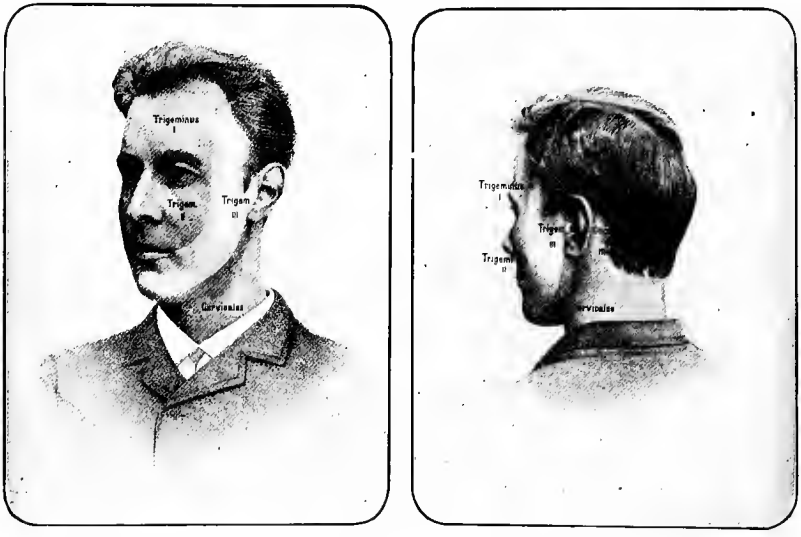
The first kind of pain which will be discussed is the darting or paroxysmal pain in what may be called the *neuralgias*. These depend upon one of three causes, and, though they may occur in any nerve of the body, are most commonly seen in the nerves of the head; or in nervous women in the nerves of the pelvic organs and external genitals. The three causes are generally debility with anemia, reflex irritation, and irritation of the nerve by poisons or by the presence of growths.

Violent neuralgia of the head is commonly seen in overworked or overdanced young women, who lack sufficient sleep and fresh air and who are anemic. It also arises from the reflex irritation of a decayed tooth, or from inflamed or overstrained eyes, or from a diseased ear, so that an examination of any one of these parts may reveal the cause of an obstinate neuralgic pain. Similarly we see cases of neuralgia, particularly of the supra-orbital nerve, which are due to chronic poisoning by one of the metallic poisons, such as lead and arsenic, and also as a result of malarial infection (brow ague). If the neuralgic pain be due to neuritis, it will not only be typical of neuralgia, but along the track of the nerve marked tenderness will be developed on pressure, and rarely an eruption will appear on the skin, as a herpes zoster. Pure neuralgia, on the other hand, is often relieved by pressure upon the nerve involved.

When the fifth cranial nerve is affected by neuralgia, we find that if the upper branch is involved the pain is felt in the forehead, the

eyebrow, and the eyeball, the conjunctiva often becoming injected If the pain be in the upper lip, the posterior nares, and the cheek, the infra-orbital or second branch is affected; while if the pain is in the lower jaw and chin, the third division of the fifth nerve is involved.

A peculiar form of neuralgic pain coming on in attacks or paroxysms of great severity is *migraine* or *megrim*, in which the pain is usually confined to one side of the head, associated with great tenderness of the scalp, and may be preceded in many cases by disorders of vision, such as hemianopsia or dimness of visual perception. Associated with this pain at its zenith we frequently see vomiting and retching, faintness, with sweating localized in the pain area, or diffused, and great facial pallor. Pressure by the fingers upon the



FIGS. 200 and 201.—Showing the distribution of the three branches of the fifth nerve.

painful area often produces no more pain or even gives partial relief, but a light touch may cause increase in the pain. Rarely a somewhat similar condition to migraine, which is not unilateral but bilateral, is found in connection with rheumatism of the scalp. As migraine may be due to a rheumatic state, care in making a differential diagnosis is necessary. The pain of migraine is, however, unilateral, more severe, more transitory, and associated with the symptoms named, whereas in the rheumatic head pain the history of rheumatic tendencies of a marked character, the diffuse pain, the increased soreness on exposure to cold or changes in the weather, aid in separating it from migraine.

When syphilis or injury causes a *periostitis* of the skull, violent

pain of a neuralgic character may be present, particularly at night; but the local symptoms are manifest, and when compared with the history make the diagnosis possible.

It is also necessary to separate the headache of *cerebral tumor* or *cerebral abscess* from neuralgia of the head. The pain of such a cerebral condition is constant; the headache is sometimes worse at night, sometimes in the daytime, and greatly increased by physical or mental effort. The danger of confusing the pain of neuralgia with that due to tumor is great unless the physician remembers that the constant pain of tumor may vary from slight headache to sharp paroxysms of pain. The occurrence of convulsions points strongly to tumor if associated with headache of this character, and, finally, the presence of tumor as a cause of headache and not ordinary neuralgia is decided by evidences of optic neuritis, vomiting, vertigo, and the development of focal symptoms of localized paralysis. (See chapters on Headache, Vomiting, Convulsions, and Spasms.)

The most common seat for neuralgic pain in the head, other than in the brow, is the occipital region, the posterior branch of the second cervical nerve or great occipital being the one most affected (Fig. 201). As this nerve supplies the occipital region and the posterior part of the parietal regions, these areas may be involved in the painful manifestations, and all these parts may be tender to the touch. Brushing the hair may be impossible, because of the pain produced by the brush touching the scalp. Occipital neuralgia is oftentimes bilateral. It may simply arise from cold or injury; but, if persistent and severe, caries of the cervical vertebra should be sought for as a possible cause.

Pain of a neuralgic or darting character in the neighborhood of the heart is found as the result of several causes, as a rule in the following order of frequency: (1) Pain with palpitation of the heart from the accumulation of flatus in the transverse colon just as it turns to descend. Many patients who come to the physician complaining of heart disease suffer only from this condition, due to fermentation in the large bowel. Again, the pain due to gastralgia, or, as it has been called, cardialgia, may be referred to the heart by the patient. (2) To intercostal neuralgia due to debility. In these cases a tender spot will often be found, one in the precordium, another in the outer edge of the scapula, and a third on the vertebral column. These are sometimes called the "spots of Valleix." In other cases the pain will be due to spinal disease, anemia, or the tight lacing of corsets. (3) To pseudo-angina. (4) To true angina pectoris. (5) To locomotor ataxia.

Pain of a character somewhat resembling true angina pectoris is also sometimes met with in patients who have that rare disease, *acute aortitis*. The pain is constant under the sternum, but it has

terrible exacerbations, and a sensation of rending of the retrosternal tissues. Death may occur in an attack. It is seen chiefly in gouty patients and in syphilitics. Very rarely it is seen in patients who have suffered from malarial poisoning.

Pain is felt much more commonly in disease of the aortic orifice than in lesions of the mitral orifice of the heart.

Pseudo-angina occurs most commonly in anemic, nervous girls, or young women whose vessels are normal but who have hysterical tendencies. *True angina* occurs in those of middle age or advanced life or in young persons whose vessels are affected by syphilis. In the false form the sensation is as if the heart would burst. In the real form it feels as if the heart were squeezed tightly in a vise. In this form, too, the bloodvessels will usually be found hard and corded, atheromatous, and the blood pressure high. The additional diagnostic points in favor of true angina pectoris are that the principal seat of pain is somewhat to the left of the lower and middle sternum, from which spot it may extend to the axilla and back and turn off to the occiput or extend down the arms to the hands, where a sensation of coldness may be felt. Sometimes even the abdominal organs and testicles seem to be affected. The patient is motionless, the face anxious and covered with a cold sweat, and respiration is shallow. The disease is usually seen in persons over forty years of age. The thoracic pain of locomotor ataxia is rarely felt in the precordium, but commonly in the axilla, and it rarely radiates down the arm. The other symptoms of tabes dorsalis should be sought for in all doubtful cases. (See chapter on the Legs and Feet.) True angina pectoris is far more rare in women than in men. Violent cardiac pain, exactly resembling that of true angina pectoris, is met with, according to Vergely, in diabetes mellitus.

Very severe pain, paroxysmal or constant, felt in the chest may also be due to *aortic aneurysm*, and, if so, will be found associated with pain shooting down the arm on the left side, dilatation of the pupil, unilateral sweating of the face and neck, and the physical signs described in the chapter on the Thorax.

Severe pain of a darting character felt in the chest, not due to angina or the causes just named, is nearly always an indication of one of four things: (1) Intercostal neuralgia, already named; (2) pleuritis, with or without pneumonia; (3) pericarditis, if it is felt in the precordium; (4) a morbid growth in the chest, particularly a mediastinal tumor or enlarged bronchial glands.

Both *intercostal neuralgia* and *pleurisy* are associated with severe pain, increased by taking a deep breath, the pain occurring sometimes with inspiration and sometimes with expiration. They are to be separated from one another by the presence of cough, fever, and of a friction sound in pleuritis, and by the fact that the entire side

is more or less tender to the touch in this state. When the pain is constant and lasts for a long time, it may be due to a low-grade pleuritis, resulting from pulmonary tuberculosis, particularly of the apex of the lung, the morbid process affecting the pleura. *Pericarditis* is frequently caused by pneumonia, and is painful.

Pain felt at the right of the left scapula or between the shoulders is often due to gastric ulcer or dyspepsia.

The pain of mediastinal growth is due to pressure on nerve trunks, and the diagnosis may be very difficult unless bulging and dulness on percussion are present. The condition is rare.

Neuralgia of the *pelvic viscera* in women is frequently seen as the result of functional or organic disease. It may be ovarian, when it is very apt to occur with greatest severity half-way between the menstrual epochs or just before them. Sometimes the neuralgia may be present in the labia majora or in the perineum. It usually occurs simply as a sudden, darting pain, which does not last, and, indeed, rarely continues more than a moment, although there is usually associated with it more or less constant uterine or ovarian tenderness. Care should be taken that these pains are not thought to be due to cancer or other severe organic lesion. Pain in the sacral region is often an indication of uterine or rectal disease. If higher up the back, it is often due to myalgia or lumbago; and lumbago, if not due to rheumatic tendencies, is often due to the colon being loaded with feces.

If the patient is a child, pain in the back should cause us to suspect *spinal caries*, *rickets*, or *scurvy*. If the former, any jar will greatly increase the pain; but if the child be placed over the knees, face downward, and the knees separated so that intervertebral pressure is removed, the pain disappears. Such a child if told to jump down from a stool will not obey, but will take care to slide off gradually and gently on to the floor, in order to avoid jarring the spine.

In scurvy the tenderness of the spine is usually diffuse, but it may mislead the physicians into a diagnosis of spinal disease, but investigation of the gums will reveal scorbutic blisters and the diet will be found imperfect. (See chapter on the Lower Extremities.)

When a patient suffers from violent pain, increased by motion, extending from the sciatic notch in the buttock down the posterior part of the thigh, even to the ankle or heel, the pain signifies an attack of *sciatic neuralgia* in an adult, or if it occurs in a child gives grave reason for suspecting *hip disease*. If it is not sciatic neuralgia, it is due to *sciatic neuritis*, or, rarely, to a *growth in the pelvis* pressing upon the nerve before it emerges from the pelvis. The pain is fairly constant, generally worse at night, and becomes agonizing at times, even if the patient remains absolutely quiet and does not move the limb. The following points will, when pressed on,

increase the pain if it be neuritis: the point of exit of the nerve from the pelvis, on the lower part of the sacrum, the head of the fibula, and behind the malleolus on the outside of the ankle. If these points are found, combined with a history of exposure to cold, injury to the nerve, rheumatic tendencies, and a persistency and tendency to return, the diagnosis of sciatica is clear. If the pain be due to sciatic neuritis there may be found wasting in the muscles supplied by the nerve, and some anesthesia of the skin, and herpetic eruptions may appear on the skin along the course of the nerves. There will be also a history of long duration, and the leg will be apt to feel numb and tense from effusion into the sheath of the nerve. (See chapters on the Skin and on the Feet and Legs.) The heel and toes will be tender, but the inner aspect of the plantar surface will usually escape. Again, in sciatic neuritis, if the leg be extended, and then while in extension flexed at the hip until it is at a right angle with the trunk, pain will be felt at the sciatic notch. When the pain is a pure neuralgia, which is rare, it is not increased by moving the limb, there is little or no tenderness on pressure on the nerve trunk, and the patient often suffers from neuralgia of other nerves.

Sciatica is much more common in men than in women, which is the reverse of all other nerve pains of like character, and far more usual in middle or advanced age than in the young.

Double sciatic pain should arouse suspicion of *locomotor ataxia*, of malignant growth pressing on the spinal cord or on both nerves in the pelvis, the presence of lumbar abscess or of diabetes mellitus causing neuritis.

When there is a hysterical, painful joint at the knee or hip in a woman, care is necessary to discover that the pain is over the entire leg rather than in the course of the nerve. Care must also be taken that rheumatism of the muscles of the thigh be not taken for sciatica. This can be separated from sciatica by the diffuse character of the pain and tenderness and by the fact that in the rheumatic condition the slightest muscular movement causes pain all over the thigh. Sometimes a *malignant growth of the femur* may produce symptoms of sciatica, and the writer not long since had under his care a case of osteosarcoma of the femur which had been treated for sciatica for several months.

Finally, *renal calculus* may cause violent pain to be felt down the leg. (See below.)

It should also be remembered that malingerers, particularly soldiers desiring to shirk duty, often pretend to have sciatica.

Abdominal Pain.—Abdominal pain of sufficient severity to cause a patient to seek medical aid may be due to a large number of causes. Its locality is of some value in helping to determine its cause, but too much reliance should not be placed upon the statements of the

patient as to its site, because even the most intelligent may be unable to correctly indicate its actual point of origin. In a general way it may be stated that a pain which is most severe in the upper right quadrant of the abdominal area is probably due to gallstone colic, to cholecystitis, or to disease in the pyloric portion of the stomach. So, too, pain in the right lower quadrant is probably due to appendicitis or it may be due to carcinoma of the caput coli, and in women, to disease of the ovary or Fallopian tube. Pain in the upper left quadrant is most commonly due to wind at the angle of the transverse colon and rarely to gastric ulcer, while pain in the left lower quadrant is due to gas, to a growth in the bowel or to a sigmoiditis or to ovarian or tubal disease. By far the most frequent form of abdominal pain is that due to flatus arising from indigestion. A peculiarity of this form of pain is the fact that it is rarely limited to one spot for any length of time, and is usually relieved by the passing of gas from the rectum. It may be associated with diarrhea and a history of the ingestion of indigestible food. Further than this, percussion will elicit a tympanitic note in the area of greatest pain and distention, whereas, in intestinal obstruction due to tumor or intussusception, percussion may reveal dulness. (For the various forms of intestinal obstruction, see the chapter on Vomiting.)

In *hepatic colic* the patient often, after some days of wretchedness and "biliousness," is seized by sudden and violent pain in the right hypochondrium, which is paroxysmal in character. Jaundice ensues in such cases with more or less rapidity, and fever of an irregular type may occur in the renal form of colic. It is worthy of note that a gallstone when in the gall-bladder, or cystic duct, rarely causes much pain, and its impaction in this duct does not cause jaundice. If it be in the hepatic duct jaundice is present, and pain is often marked, but is not so severe as if it be in the common duct.

If the stone be impacted in the common duct, then the most violent pain is present, and jaundice is marked, as a rule, the stools being clay colored. If the symptoms develop suddenly, and pass away equally rapidly, the stone has probably escaped into the bowel, whereas if the stone becomes impacted in the papillæ of the duct, the jaundice and other symptoms become progressively worse. It is entirely possible for the same stone to slip back, and cause temporary relief, then to slip into place and precipitate another attack, or for another calculus to follow its predecessor, causing a return of the symptoms. Pain is more indicative of stone than any other cause of obstruction of the common or cystic duct. Thus, in 80 cases of common-duct obstruction pain was marked in 51, absent in 10, and unrecorded in 19, while in 79 cases due to other causes than stone, pain was present in only 9.

If the stone is movable, there may be attacks of acute radiating

pain with chills, and sweats due to absorption of toxic matter through the abraded mucous membrane. (See Charcot's Fever.)

Boas has pointed out that in many cases of gallstone a spot of tenderness may be found at the level of the twelfth dorsal vertebra three inches to the right of the vertebral line.

Severe pain in the hepatic region may also be due to *acute cholecystitis*. There is usually local muscular rigidity, tenderness, and vomiting, but the liver is not enlarged unless the cholecystitis is associated with cholangitis. This condition follows the infectious fevers, notably typhoid fever, and may be confused with appendicitis, which often causes cholangitis or abscess behind the liver.

The pain of *diffuse hepatitis* varies with the severity of the inflammation, and it may not be present unless the hypochondrium is palpated. In cholangitis it is rare unless suppuration is localized, but in *cholecystitis* of a chronic type the pain often comes on in paroxysms, and is associated with fever, enlargement of the gall-bladder, and great tenderness in its area on palpation. The liver itself is not enlarged. If suppuration is present, the symptoms of general sepsis may develop. The paroxysms of pain may vary like those of gallstone in the endeavor of the gall-bladder to extrude its thick mucopurulent contents. Care should be taken not to mistake a subphrenic abscess for cholecystitis.

When there is a history of violent colic situated near the hypochondrium, or in the epigastrium, not accompanied by jaundice and enlargement of the liver, the possibility of the cause being a *pancreatic calculus* is not to be forgotten. Such a condition is rarely met with it is true, and still more rarely do we find pancreatic stones in the feces, because they are friable and broken up in the feces before they are passed. Boas states that they are a frequent cause of so-called "neuralgia of the liver." It should not be forgotten that blocking of the common duct by gallstone also produces blocking of the pancreatic duct, and this in time may cause a complicating attack of acute pancreatic necrosis.

The pains just described are so severe and characteristic in their distribution that they cannot well be confused with those of *intestinal indigestion*, in which condition we have a history of the ingestion of bad food, a state of more or less flatulent distention of the entire belly, and, it may be, diarrhea.

The pain arising from the presence of a *gastric ulcer* may, with its associated symptoms, so closely resemble that due to cholelithiasis as to make a differential diagnosis almost if not quite impossible. The same statement holds true of duodenal ulcer, but the comparative rarity of this lesion excludes it on the ground of improbability. The pain of all these affections may be so near the area of the biliary passages that its localization by the patient gives the physician no

aid whatever. Jaundice is so often absent in cases of hepatic colic that its absence in no way excludes this state. While the presence of an excess of hydrochloric acid in the gastric contents points to ulcer it sometimes occurs that hyperacidity arises from reflex irritation from the gall-bladder. Even the development of pain a definite time after the ingestion of food, due to the irritating effect of the acid upon the gastric ulcer, may also develop in cases of gallstone if there has developed around or about the gall-bladder inflammatory exudations and adhesions which interfere with the movements of the pylorus and duodenum as the food passes into the small bowel. The presence of hematemesis is strongly indicative but not pathognomonic of ulcer.

Finally, the physician must bear in mind that occasionally severe abdominal pain with vomiting, diarrhea, and even the passage of blood may be the result of *angioneurotic edema* with an abdominal crisis. This pain is usually diffused throughout the belly and not confined to one area although at onset it may be in the epigastrium. These symptoms may seriously mislead the surgeon unless he first makes inquiries, designed to discover the fact that attacks of angioneurotic edema have affected other parts of the body. This is the more important in view of the fact that these attacks are often precipitated by indiscretions in diet as is appendicitis and gallstone colic. The skin should always be carefully examined to discover traces of urticaria.

The sudden development of pain of great intensity in the right lower quadrant of the abdomen (see McBurney's point, Fig. 202), associated with muscular rigidity of the abdominal wall, tenderness on palpation, a quick pulse, and in some instances vomiting indicates the development of *appendicitis*, a diagnosis which is aided, but not confirmed, if on examining the blood a distinct leukocytosis is present. It is worthy of note that the pain of appendicitis is often referred in its greatest intensity, and by intelligent patients, not to the neighborhood of the appendix, but to the epigastrium, or even to the region of the sigmoid flexure, and it is by careful palpation only that the area of greatest tenderness is found in the appendicular region.

Before reaching a diagnosis of acute appendicitis the physician must exclude the following causes of pain referred by the patient to the appendicular area. One of these is diaphragmatic pleurisy; still another is subphrenic abscess and a fourth is ovarian or tubal disease if the patient be a woman. Rarely a floating kidney may be provocative of similar symptoms as may also phlebitis of the iliac vein. If the pain is very severe, so severe that the patient is incapacitated from giving a clear description of its chief seat, it may be due to gallstones, renal stone, or intestinal perforation. Severe pain may also be due to inflammation or twisting of Meckel's diverticulum.

Generally diffused pain of a constant severe character felt all over the abdomen or localized at first in some particular spot, and greatly increased by pressure, should lead the physician to examine the case for a possible *peritonitis*. Nothing else causes such violent, diffuse pain. The well-flexed legs, the anxious face, the drawn upper lip, quick pulse, the exquisitely tender abdominal surface, the thirst, the

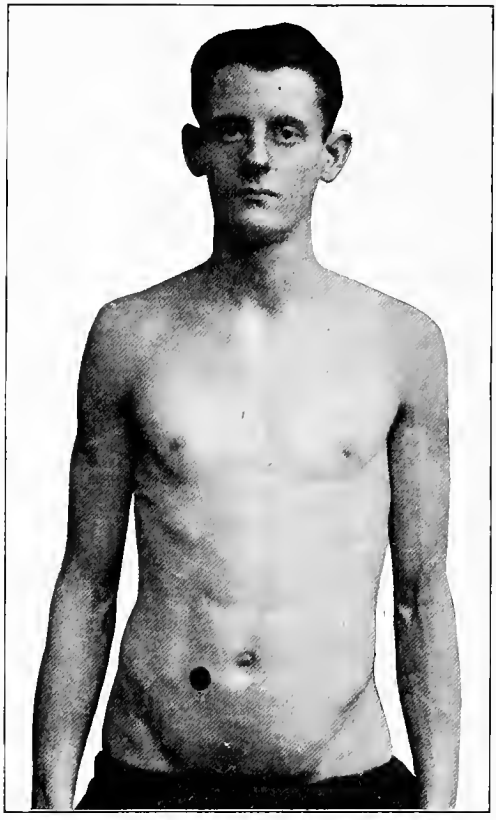


FIG. 202.—The black spot is at "McBurney's point."

moderate fever, and the rapid onset of collapse in fatal cases render the diagnosis easy.

In *pancreatitis* the pain is sudden in onset, violent, and usually felt chiefly in the left upper zone of the abdomen. The belly is distended, nausea and vomiting are present, and fever may be present also; delirium may come on, and death generally speedily ensues unless operative measures are resorted to. (See Abdomen.)

The onset of severe pain in the abdomen in the course of typhoid fever, while not necessarily an indication of *perforation of the bowel*,

is nevertheless deserving of careful study. If it is due to perforation, as a rule it is violent enough to make the patient cry out if he be not stuporous, and the following symptoms will probably be present in whole or in part: The epigastrium and general abdominal wall will be fixed; the abdominal wall may be scaphoid or distended, but is usually hard. Percussion of the area of the liver may show obliteration of hepatic dulness, as a result of gas being between the wall and this organ, but while this is an important sign if present, its absence does not exclude perforation. Hiccough may be present, and perhaps vomiting. The facial expression is that of grave abdominal disorder. (See chapter on the Face.) An examination of the blood may show leukocytosis, and if such an increase in white cells is present, it is an important positive factor in confirming the diagnosis of perforation. An absence of leukocytosis not only does not negative the diagnosis of perforation but it may confirm it, because if the shock be great leukocytosis may be prevented by the very intensity of the insult to the peritoneum. As in suspected appendicitis so in suspected typhoid perforation, the chest must be carefully examined lest the pain of an acute pleurisy be mistaken for abdominal disorder. So, too, it is essential that the pain of acute cholecystitis, appendicitis, and renal calculus be excluded before a diagnosis of perforation is determined.

Severe abdominal pain in typhoid fever may, however, be the result of wind colic, acute pleurisy, a distended bladder, cholecystitis, and impaction of feces.

Sometimes a patient who has *floating kidney* will suffer from severe renal pain, nausea, vomiting, and collapse, the symptoms simulating renal calculus. These attacks are known as Dietl's crises.

If the pain be due to *chronic lead poisoning*, it centres about the umbilicus, and is of a twisting, knotty character, "as if the bowels, were being twisted around a stick." There is a history of exposure to lead in many cases, and a blue line on the gums can often be found.

If due to *fecal impaction*, there is a history of a continued tendency to constipation, with dry, hard stools, and a lump of hardened feces may perhaps be felt through the belly wall.

If due to *intestinal obstruction*, the pain has no characteristic seat in any part of the abdomen, as a rule; but the general symptoms of this condition will be found present in the case. (See chapters on Vomiting and on the Abdomen.)

Abdominal aneurysm may cause severe pain by pressure on nerve trunks; and uterine and peri-uterine disease also cause, reflexly, epigastric pains.

Reference has already been made to the pain of *renal colic*. The characteristic symptoms of this condition are as follows: In renal

colic the patient is suddenly seized with violent pain in the region of the kidney on one side, which passes down to the groin and even to the end of the penis. It is paroxysmal in character, and so excessively severe that it often produces sweating, vomiting, and even fainting. The condition is seen much more frequently in men than in women. The pain often suddenly subsides, leaving only a sense of soreness and tenderness in its track. The urine may be partly suppressed and bloody if the stone injures the ureter to any great extent. Pain simulating renal colic may be due to neuralgia or arise from several organic causes not connected with calculus. Thus, Habershon has stated that in valvular disease of the heart, particularly of the aortic valve, severe and colicky pains frequently radiate down into the right hypochondriac region, and Ralfe says into the renal region. Again, pain in this part may be due to aneurysm of the aorta or of the mesenteric artery. Further, the accumulation of hard fecal matter in the colon may cause nephralgia. Finally, Ralfe calls attention to the renal pain felt generally in the right kidney by women who have exercised violently while wearing a tight corset, which has pressed upon the liver and kidney with great force on making a jump or a sudden bend of the trunk. Sometimes a sudden "storm" of uric acid or an accumulation of oxalic acid in the kidney causes pain and tenderness.

The presence of constant pain and soreness in the abdomen on one side, frequent micturition, and occasionally the presence of blood in the urine, are symptoms of several renal states, such as *pyelitis, renal calculus, a tumor in the renal pelvis, and tuberculosis of the kidney.* The pain is chiefly in the region of the kidney and along the course of the ureter where it crosses the pelvic brim; and as it is often made more severe by standing and is of a bearing-down character, it may be thought in women to be uterine. Jolts or jarring of the body also aggravate it, as does the act of urination. Sometimes a good deal of pain is felt on the sound side.

If an examination of the urine reveals pus, this will indicate pyelitis; and if the pus contains tubercle bacilli, tuberculous pyelitis. The latter diagnosis is confirmed if tuberculous disease is present in other organs, for renal tuberculosis is usually secondary. Small cheesy masses may be present in the urine in the latter state. Renal calculus will be attended by attacks of renal colic, which, as a rule, are more severe than those produced by the passage of masses of broken-down matter along the ureter. A history of having passed a stone at some previous time, and the discovery of blood corpuscles in the urine are of importance in the diagnosis.

Pain in the abdomen of the darting neuralgic type, other than that due to gallstones, renal calculi, ordinary gastralgia, lead poisoning, enteralgia, or malignant growth, may be due to *locomotor ataxia.*

This possibility should never be forgotten, and the fact that the patient is an adult, complains of the most violent pain in the belly, and has no other abdominal signs, should make us search for the other signs of tabes dorsalis. Generally these attacks will be of a tearing, rending character; they may be beyond description in severity, and after they pass off the patient is left in a condition of nervous wreck. Sometimes the pain is in the stomach, sometimes in the bladder.

SOME ABDOMINAL CONDITIONS IN WHICH SUDDEN AND ACUTE PAIN FORMS A PROMINENT SYMPTOM.[1]

Disease.	Mode of onset.	Character of pain.		Seat of pain.	Tenderness and pressure.
		In kind.	In intensity.		
Acute intestinal obstruction:					
a. Strangulation due to bands.	Very sudden.	More or less continuous.	Most intense, agonizing.	Epigastric or umbilical region.	Pressure at first relieves, afterward aggravates.
b. Acute intussusception.	Sudden to very sudden.	Paroxysmal.	Severe.	Epigastric or umbilical region.	Pressure at first relieves, afterward aggravates.
c. Acute volvulus.	Sudden.	Paroxysmal, but less than *b*.	Moderate.	Umbilical or over heart.	Pressure never causes pain.
d. Due to gallstone or stricture.	Less sudden, gradual.	Paroxysmal later.	Moderate.	Often near seat of obstruction.	Tenderness over obstruction.
Appendicitis.	Very sudden.	Quite continuous.	Agonizing.	At first periumbilical, later about appendix.	Greatest over appendix or in left groin.
Acute peritonitis.	Very sudden.	Continuous.	Very severe	All over belly, but chiefly epigastric or umbilical.	Very tender everywhere except at very first.
Hepatic colic.	Sudden.	Aching, tearing, paroxysmal.	Agonizing.	Epigastric, radiating to between shoulders or to shoulder-blade.	Pressure at first relieves, then unbearable over gall-bladder.
Renal colic.	Sudden.	Aching, tearing, paroxysmal.	Agonizing.	Affected loin, passing down in front into testicle and bladder.	Tenderness over affected kidney.
Intestinal colic.	Sudden or gradual.	Paroxysmal.	Varies in severity.	Varies in position.	Relieved by pressure.

Grube has recently reported cases showing that diabetes may produce attacks of violent abdominal pain resembling the crises of an ataxia.

Neuralgic pain in the back and abdominal parietes very closely resembling, if severe, renal or hepatic colic is sometimes due to *gastralgia*, the paroxysms being very sudden in onset. In other

[1] Andrews' table, slightly modified.

instances the pain is in the epigastrium or hypogastrium, and is associated with so much tenderness on light pressure as to impress the careless with the belief that gastric ulcer or cancer is the cause. Neuralgic spots can generally be isolated in such persons if the skin is carefully tested for its degrees of sensation, and these spots will be found to exist near where the nerves are given off from the spine or over the spine of the ilium. In some of these cases the cause of the so-called gastric pain really exists in the presence of otherwise quiescent gallstones.

Circumscribed abdominal pain of a constant character and generally of less severity than that just described, may be due to an *abdominal tumor* (see Abdominal), or to ulcer of the stomach or bowel.

In *dysmenorrhea* the pain is sometimes so severe as to render the patient almost insane, but it differs from that of inflammation in that it is paroxysmal and that there is no real tenderness on pressure; and, again, the patient does not lie still, but tosses from side to side in the bed. The pain of *pelvic tumor* is usually produced by pressure on a nerve, and is increased by palpation in some cases, as is also that of ovaritis. In *cystitis* the pain is deep in the pelvis, radiating upward, and is associated with tenderness, vesical spasm, and tenesmus.

The pain of *fissure of the anus* is not at all proportionate to the lesion producing it. This pain may be atrocious and agonizing, and often is produced by a movement of the bowels, after which it lasts for some hours.

(For abdominal pain due to conditions associated with movements of the bowels, see chapter on the Bowels and Feces.)

Pain in the Feet and Limbs.—Neuralgia of the toe and foot is not a very rare condition, and is sometimes called "Morton's painful toe," or *metatarsal neuralgia.* Severe pain at the base of the fourth toe comes on suddenly, and may radiate up the anterior aspect of the leg. Sometimes it is only dull, at other times it is so sharp and excruciating as to cause the patient to scream. It is separated from gout by the absence of any signs of inflammation in the part, by the fact that the big toe is not affected, and by the age and history of the patient. At times the base of the second toe is affected. Such a case will usually indicate that the patient has worn an ill-fitting boot.

Finally, in connection with this class of cases there should not be forgotten two others, namely, those in which idiopathically or otherwise, growths form on nerves and cause pain; and, secondly, cases in which the arm or leg having been amputated, a *neuroma,* or catching of the end of the nerve in the scar, causes violent pain in the lost part, according to the patient's sensation, because the perceptive centres have been trained to regard pain impulses coming along this

nerve as from its peripheral end. Thus a man whose leg may have been amputated years before will complain of severe pain in the amputated foot, although he knows it is off.

Pain in the back is often very severe in the early stages of smallpox, of epidemic influenza, and in lumbago.

One of the most misleading forms of pain of a severe character, involving the entire body, with fever, delirium, and a variable skin eruption and swelling of the joints, may in the early stage be thought to be smallpox or rheumatic fever, when in reality it is due to *dengue* or breakbone fever.

CHAPTER XXI.

TENDON REFLEXES AND MUSCLE TONE.

The knee-jerk and ankle-clonus—The arm-jerk—The significance of decreased and increased reflexes.

We have already had occasion, particularly in those chapters devoted to the Legs and Feet and the Arms and Hands, to speak of what are called the reflexes or "muscle-jerks." There is much discussion as to whether the muscular contractions produced by tapping the tendon attached to a muscle are the result of a reflex action, in which the spinal cord is directly involved, or whether it depends upon muscle irritability or tone. It is not necessary for purposes of diagnosis to enter into a discussion of this character, because the facts in our possession prove conclusively that variations in these muscle-jerks are of great diagnostic importance in diseases of the nervous system, whether they be true reflexes or not. The knee-jerk, or, as it has been called, the patellar reflex, is the diagnostic sign most frequently sought in studying nervous diseases associated with lesions in the spinal cord, because it is most easily developed.

The methods of developing the knee-jerk, elbow-jerk, and ankle clonus have already been described in the chapters on the Hands and Arms and the Feet and Legs, to which the reader is referred.

The chin-jerk is elicited by having the patient open his mouth slightly and then placing a flat object, like a paper cutter or tongue depressor, on the edges of the lower teeth. A sharp tap is now given to the flat object, when in health there will be a slight upward jerk of the chin.

The scapulohumeral reflex of von Bechterew is elicited by tapping the skin on the spinal border of the scapula near its inferior angle. This usually causes a slight adduction and external rotation of the arm. It is exaggerated in neurasthenia, but its chief diagnostic value is in connection with lesions in the pyramidal columns above the cervical enlargement, in which state it is greatly altered in that we now find contraction of the posterior fibers of the trapezius, the deltoid, biceps, and forearm mucsles, so that the shoulder is raised, the arm thrown from the side, the forearm flexed, and the fingers extended that the muscles on the opposite side also respond. If the

brachial plexus be diseased, the response on that side may be greater than on the side that is tapped.

Having learned how to test these muscle-jerks, we now turn to a consideration of what they mean when absent or abnormally increased.

A loss of knee-jerk is not characteristic of any disease unless this loss is associated with other symptoms which only need the discovery of this symptom to confirm the diagnosis. The nervous conditions in which we find the reflexes decreased or lost, taking the patella reflex as a type, are locomotor ataxia; peripheral neuritis; poliomyelitis, acute or chronic; transverse myelitis, if the disease involves the reflex arc; Friedreich's ataxia; diphtheritic paralysis; apoplexy, immediately after the shock; Landry's paralysis; spinal meningitis; spinal injuries, immediately after the accident; epilepsy, immediately after an attack; and chorea. We also find a total loss of reflexes in advanced diabetes mellitus and sometimes in diabetes insipidus.

By far the most common cause of the loss of the knee-jerk is locomotor ataxia, but any lesion involving the posterior columns of the cord or the posterior nerve roots in the second, third, or fourth lumbar segment will produce the same results. Therefore, loss of knee-jerk is symptomatic of transverse myelitis of this region as well as of ataxia. Again, if the motor tract of the cord at these levels is diseased the knee-jerk is lost, as, for example, in acute and chronic poliomyelitis or myelitis involving the motor part of the reflex arc; and, finally, peripheral neuritis, which blocks the pathway from the periphery to the cord, and from the cord to the muscles, also causes loss of knee-jerk.

If the cause of loss of knee-jerk be locomotor ataxia, we will probably find in addition to this symptom some difficulty in walking, particularly if the eyes are shut; a lack of steadiness if the feet are placed together when the patient stands with his eyes shut; Argyll-Robertson pupils, or a reaction to accommodation but not to light; attacks of severe pain in the body or limbs; and, it may be, laryngeal crises or spasms and atrophy of the optic nerve.

If the cause of loss of knee-jerk be neuritis, we will find tenderness on pressure along the nerve trunks, diminished muscular tone, and some wasting; an absence of any disturbance of the bladder and no Argyll-Robertson pupil, laryngeal or other crises, nor optic atrophy.

Again, if the cause be acute poliomyelitis, there will be a history of sudden onset with fever, the limbs will be relaxed and flabby, the muscles will rapidly waste and become very feeble or paralyzed, and there will be no sensory symptoms whatever. The patient will usually be a child if the disease is acute. If the loss be due to transverse myelitis of the second, third, and fourth lumbar segments,

the symptoms of paraplegia, parasthesia, and anesthesia, with atrophy of the muscles and loss of control of the bladder and rectum, will be present, and a girdle sensation may be marked.

In Friedreich's ataxia the history of heredity, the nystagmus, the early age of the patient, the absence of pupillary symptoms, the ataxic gait, and the loss of reflexes, are the facts which go to form our basis for a diagnosis. In the remaining diseases named the history of the case points to the cause of the loss of the knee-jerk very clearly.

The conditions in which we find the knee-jerk *increased* are apoplexy after the attack; disseminated sclerosis; cerebral palsy of childhood; paretic dementia (not constant); primary lateral sclerosis; amyotrophic lateral sclerosis; ataxic paraplegia; hysterical paraplegia; transverse myelitis if the lesion is above the reflex arc; epilepsy some minutes after the attack; unilateral lesions of the cord on the paralyzed side; injuries to the spinal cord, after the recovery from first shock; pressure on spinal cord above the reflex arc; hereditary cerebellar ataxia; sciatica; tetanus; rheumatoid arthritis; and neurasthenia.

The history of sudden paralysis and unconsciousness in a case of apoplexy with stertorous breathing, followed by loss of the knee-jerk, and then its return in an exaggerated manner, make the diagnosis clear unless the attack be one of the apoplectiform attacks of disseminated sclerosis, in which case there will be present a history of the intention tremor, nystagmus, and the syllabic speech, so that though the knee-jerk is exaggerated in both diseases the diagnosis can be readily made. In the cerebral palsy of childhood the age of the patient, the contractures and gait, with the history, decide the diagnosis. In lateral sclerosis the spastic rigidity, excessive exaggeration of the knee-jerks, absence of sensory disturbances, and ocular symptoms, all render the diagnosis possible. Similar exaggeration is also seen in amyotrophic lateral sclerosis, in which disease there is wasting of the muscles, particularly of the hand. In both, these ailments the exaggeration of the knee-jerk is due to disease of the lateral pyramidal tracts, which block the inhibitory fibers from the higher centres. For similar reasons we find exaggerated knee-jerk in ataxia paraplegia.

In hysterical paraplegia the age and sex of the patient, the peculiar facies, the areas of anesthesia and hyperesthesia, and the peculiar gait point to the diagnosis.

The increased knee-jerk in cases of transverse myelitis occurs when the lesion is situated at such a point in the cord that the lateral tracts are cut off and the reflex arc is preserved.

In neurasthenia the knee-jerks are exaggerated, but are easily exhausted.

Leaving the knee-jerks as a type of a reflex, we find that the skin

reflexes are often lost in cases of apoplexy when the deep reflexes are exaggerated. The table on page 78, from Taylor's *Index of Medicine*, shows the area of skin reflexes very well.

The plantar reflex obtained by tickling the sole of the foot has recently attained considerable diagnostic importance through the work of Babinski, who has found that in normal persons such irritation causes flexion of the toes on the metatarsus, whereas in disease of the pyramidal tracts extension of the great toe, with or without

FIG. 203.—Babinski reflex in health, somewhat exaggerated. The toes are drawn up toward the knee.

extension of the other toes, takes place, as, for example, in spastic paraplegia. The normal reflex, somewhat exaggerated, is shown in the figure. It is a noteworthy fact that in infants in health extension takes place instead of flexion, and that the administration of full doses of strychnine to adults will sometimes reverse the normal reaction. The Babinski reflex is not pathognomonic of disease of the pyramidal tract, although it is indicative.

In glossolabiopharyngeal paralysis the reflexes of the tongue and throat are lost and those of the face sometimes increased; in progressive muscular atrophy the reflexes of the arms are lost, while those of the legs are preserved; and in tuberculous meningitis the reflexes are apt to be more marked on one side than the other.

In athetosis the reflexes are increased in the affected part.

Ankle clonus is found most marked in lateral sclerosis, in disseminated sclerosis, and in amyotrophic lateral sclerosis. A false clonus is sometimes seen in hysteria.

A Table of the Reflexes.

Reflex.	Point of stimulation.	Situation of centre.	Significance.
1. Plantar,	Irritating skin of soles.	Extreme end of cord.	Usual in health.
2. Gluteal,	Irritating skin of buttocks.	Origin of 4th and 5th lumbar nerves.	Rare in health.
3. Cremasteric,	Irritating skin of inner side of thighs.	Origin of 1st and 2d lumbar nerves.	Usual in health; best marked in boys, on account of the newly formed cremaster.
4. Abdominal,	Irritating skin of abdomen in line of nipples.	Origin of 8th to 12th dorsal nerves.	Frequently absent.
5. Epigastric,	Irritating skin of chest in 5th and 6th spaces.	Origin of 4th to 6th dorsal nerves.	May be absent in health.
6. Erector spinæ,	Irritating skin from scapula to crest of ilium.	Origin of all the dorsal nerves.	Rare in health; frequent in wasting disease.
7. Interscapular,	Irritating skin between scapulæ.	Origin of 6th cervical to 3d dorsal.	Rare in health.
8. Palmar,	Palms of hands.	Cervical bulb.	Only in infants.
9. Cranial: Conjunctival,	Sclerotic, or inner surface of eyelid.	Medulla.	Absent in disease of 5th nerve only.
Iris (to light),	Pupil.	Anterior portion of oculomotor nucleus.	Absent in disease only.
Palate,	Soft palate and uvula.	Medulla.	Absent in disease only.
Nasal (sneezing),	Naso-respiratory passages.	Medulla.	Absent in disease only.

CHAPTER XXII.

SPEECH.

The changes in the speech and voice—Their significance—Aphasia—Apraxia—Alexia—Paraphasia.

THE character of the speech and the tones of the voice often convey a considerable amount of diagnostic information to the physician. While in many diseases no marked alterations from the normal manner of speech are present, in others marked changes take place. Thus, in acute laryngitis due to exposure to cold or irritant vapors the patient has a *whispering* voice. In persons suffering from pulmonary tuberculosis the development of hoarseness and whispering or labored speech tells us only too well of the fact that the grave and distressing complication called laryngeal tuberculosis has arisen, and that the progress of the case will be more rapid toward the fatal result. Again, the sudden onset of whispering voice or complete aphonia occurring in a young girl whose facies is hysterical, should always arouse a suspicion of hysteria, while if the signs of this condition are absent and the patient has none of the signs of tuberculosis, we should examine the larynx for a papillomatous growth. Again, if *hoarseness* or a whispering voice is manifested by a male of adult years, who is also suffering from dyspnea, unilateral flushing or sweating of the face and neck, and unequal, rapid radial pulses, we should suspect aortic aneurysm or a mediastinal tumor which is pressing on his recurrent laryngeal nerve. There are also other causes of hoarseness due to nervous lesions arising from the long and tortuous course of the nerves supplying it. These have been well grouped together by Felix Semon. (See Table on next page.) It is interesting to note that in suspected cases of disease of the parts herein named it is well to make a laryngeal examination, since this may reveal a paralyzed cord, although the voice has not indicated such a condition, because by great retraction of the opposite cord the laryngeal opening is kept patulous and phonation is possible.

Hoarseness may also be due to tuberculous infection of the larynx, to laryngeal papilloma, and hysteria.

Hysterical mutism may occur in both males and females. It usually follows a fright or violent emotion, or it may follow an hysterical seizure. Sometimes it develops without any such history. The condition lasts from a few hours to months or years, and recovery

is often as sudden and unsuspected as the onset. As a rule, the tongue, lips, and jaws are unimpaired in their functions. Sometimes, however, these parts are affected by hysterical spasm. Often there will be hysterical anesthesia with the mutism. Usually there is no evidence of cerebral lesion in such cases in the sense of impairment of intellect.

TABLE SHOWING THE POSSIBLE CAUSES OF LARYNGEAL PARALYSIS.

I. BULBAR AND BULBO-SPINAL AFFECTIONS.	II. PERIPHERAL AFFECTIONS.
1. Hemorrhage and softening.	1. Acute rheumatic influences.
2. Syphilitic processes.	2. Catarrhal neuritis.
3. Tumors.	3. Toxic influences (lead, arsenic, etc.).
4. Diphtheria.	4. Tumors in the posterior cavity of the skull or in the foramen lacerum or foramen jugulare.
5. Progressive bulbar paralysis.	5. Pachymeningitis.
6. That curious form of systemic central nervous disease first described by Hughlings Jackson and Morell Mackenzie, in which one-half of the tongue, the corresponding half of the palate, the corresponding vocal cord, and, in a number of cases, the corresponding trapezius and sterno mastoid muscles are affected.	6. Traumatism (unintentional ligature of nerves, injection of iodine into a goitre, cut throat, stabbing, injury during extirpation of goitre, etc.).
7. Amyotrophic lateral sclerosis.	7. Tumors of neck (goitre, peritracheal glands, etc.).
8 Disseminated cerebro-spinal sclerosis.	8. Aneurisms of the arch of the aorta, innominate, subclavian, carotid.
9. Syringomyelia.	9. Mediastinal tumors (malignant, tuberculous, calcification of bronchial glands, etc.).
10. Tabes dorsalis.	10. Pericarditis.
	11. Pleurisy.
	12. Tuberculosis and pleuritic thickening of apex of right lung.
	13. Chronic pulmonary affections (chronic pneumonia, anthracosis, etc.).
	14. Infectious fevers (typhoid, etc.).
	15. Œsophageal carcinoma.

When a child speaks with a *nasal twang* or indistinctly we suspect the presence of adenoid vegetations, and will probably find that he or she is addicted to mouth breathing. Stuttering or stammering may also be due to this cause.

A *feeble, hesitating speech* is often a sign of exhausting disease, and a short and quick but feebly spoken sentence generally indicates that the patient is suffering from some cardiac or pulmonary complaint, which renders him short of breath, so that he hurries through the sentence in order to be able to breathe freely again. In cases of pneumonia or of pulmonary edema this hurried speech is a very characteristic sign.

Again, in cases of typhoid fever, when the tongue is dry and immobile from accumulated sordes, a mumbling character of the speech is present, even if the brain is entirely clear, and in severe stomatitis the same quality of the voice may be present.

It is in connection with the disorders of the nervous system, however, that the most typical alterations of the voice occur. Let us suppose that a patient in middle life or in more advanced years develops a *slow, scanning speech,* with intention tremors (see chapter on the Hands), nystagmus, and more or less muscular weakness. In all probability he is a sufferer from insular or, as it is otherwise called, disseminated sclerosis. When he speaks each syllable is sharply accentuated and slowly pronounced. The only other condition in which a slow, scanning speech is of great diagnostic importance is in that rare disease Friedreich's ataxia; but the facts that this disease begins in childhood, that several members of the family are apt to be affected, that there are ataxic symptoms and early talipes equinus, render it easy to separate this affection from insular sclerosis. (See Paraplegia, in chapter on the Feet and Legs.)

A *hesitating, halting speech* associated with Argyll-Robertson pupils, unequal pupils, delusions of grandeur, and tremor of the tongue, which last symptom may be so marked as to cause the speech to be indistinct and blurred, is indicative of paretic dementia.

If an incoherent speech develops in a child who is not suffering from an acute illness causing delirium, there will usually be found in association with this symptom the nervous twitchings of chorea, for speech disturbances occur in about one-third of the patients suffering from this disease.

A very *indistinct speech of a mumbling character,* great difficulty being experienced in the pronouncing of dental and lingual sounds, and perhaps associated with feebleness of the voice, if the larynx is involved, is seen in cases of glossolabiopharyngeal paralysis. If the cause of the defective speech be this disease there will be found, as associated symptoms, wasting of the tongue, lingual tremors, some dribbling of saliva from the mouth, and immobility of the lips, the face about the mouth being expressionless.

Somewhat similar symptoms due to paralysis of the lips, with escape of the tongue and pharynx, at least for a long time after labial paralysis develops, are sometimes seen in advanced cases of amyotrophic lateral sclerosis; and a still more close resemblance may be produced by the so-called "pseudobulbar paralysis," the lesion of which is in the motor cortex of the brain on both sides, in the lower part of the ascending frontal convolution. Rarely the latter is only a unilateral disease.

A rather *shrill, piping voice,* the sentence being begun with hesitation and then hurried to an end in a rapid volley of words, is sometimes seen in paralysis agitans.

Aphasia.—By far the most interesting speech defect is that called *aphasia.* It is divided into motor aphasia and sensory aphasia.

Before studying these conditions we must discuss the nervous mechanism of speech. When a child learns to talk it performs a purely imitative act. Its auditory nerve conveys the sound to its perceptive centres, and from here an impulse is sent to its motor speech centres, and these again send impulses to the inferior speech nuclei in the medulla oblongata, which in turn move the muscles of speech. Simultaneously the child learns the words and stores them in memory centres for sounds, and also stores in memory centres "motor memories," which tell him how to repeat the muscular movements a second time. Again, when he learns to recognize objects and call them by name he must use "visual memory" centres. These centres are all best developed in the left hemisphere of the brain in right-handed persons and in the right half of the brain in left-handed persons.

If a person suffers from pure aphasia, he simply loses the memory of how to say certain words, and the lesion is in the third left frontal (Broca's) convolution. This state is sometimes called *aphemia.* The patient knows what a knife is when he sees it, but he cannot recall his motor memories so as to move the muscles to say "knife." He can read to himself, because he has not forgotten the meaning of the words, and for this reason he understands what is said to him, and may be able to repeat a word immediately after it is said, by a purely imitative process. Generally, we find with aphasia a condition called *agraphia,* in which the patient cannot write his thoughts, but can copy perfectly. In the great majority of cases of aphasia, however, the patient is paralyzed in his right hand, so that the symptoms of agraphia cannot be demonstrated. Under the name of *paraphasia* we sometimes meet with a condition in which the patient can speak quite freely, but transposes words or interpolates useless words to such an extent that what he says is unintelligible. When the patient slips words, the lesion is in the associating tracts between the speech centres, and this is called *conduction aphasia.*

In another condition closely connected with aphasia we have a state in which the patient can spell out words from a page set before him, but he cannot read, because the words convey no idea to him. This is called *alexia,* or "word blindness." Again, he may forget the use or significance of certain objects, such as a knife and fork; this is called *apraxia.* Still further, words when spoken to the patient in his native language may be heard perfectly, and yet understood no more than if in some unheard-of language. This is called "word deafness."

If the patient has simple aphasia he has a lesion in the third frontal convolution in its posterior part. If he has word blindness or alexia, the lesion is in the angular gyrus, extending back into the occipital convolution. If he has apraxia or the loss of memory

of objects, the lesion is in the same area as in alexia; and if "word deafness" is present, the lesion is in the posterior part of the first temporal and upper part of the second temporal convolution of the left hemisphere. As the various symptoms of aphasia in all its forms are closely associated with those of focal lesions of the brain, resulting, for example, from hemorrhage or embolism, the reader should read the chapter on Hemiplegia in this connection.

Aphasia is quite frequently met with as a symptom of hysteria, and may occur independently of any organic lesion, so far as we know, in children during convalescence from an attack of a severe infectious disease. In the latter cases speech may return many months after.

The following plan of testing a patient, devised by Eskridge from a shorter one by M. Allen Starr, may be followed with advantage:

1. The power to recognize objects seen, heard, felt, tasted, smelt, and their uses.
2. The power to recall the spoken names of objects seen, heard, felt, tasted, and smelt.
3. The power to understand sounds other than speech.
4. The power to understand speech and music.
5. The power to call to mind objects named and point them out at request.
6. If word-deaf, can he recognize his own name when it is spoken?
7. The power to recognize a word spelled aloud.
8. The power to call up mentally the sound of a note, figure, letter, or word.

The examination thus far will test the various sensory areas, but more especially the auditory and the association tracts between the different sensory areas connected with speech.

9. The power to recognize letters, figures, notes, and colors seen.
10. The power to understand printed and written words seen.
11. The power to read printing, writing, and music aloud and inaudibly, and to understand what he reads.
12. The power to recall objects, the names of which are seen.
13. The power to write voluntarily.
14. The power to write at dictation.
15. The power to copy, and the manner of copying, printing, and writing.
16. The power to write the names of objects seen, heard, felt, tasted, and smelt.
17. The power to read aloud and inaudibly, and to understand what has been written.
18. The power to write his name and the ability to read it when written by himself and by another person, or when it is printed.
19. The power to recognize a letter by tracing it with the index finger or with a pencil, the movements being guided by another.

20. The power to call up mentally the appearance of an object, a figure, a note, letter, or word, when word-blind.

These additional tests will aid in determining the condition of the visual word memories in the angular gyrus, and the connection between this area and the surrounding sensory and motor areas.

21. The power to speak voluntarily, and, if impaired or lost, the character of the defect.

22. The power to repeat words after another.

23. Does the patient recognize his mistakes in speaking and writing, and can he correct them?

24. Can the patient think in speech (propositionize)?

25. Is there any special difficulty in the use of nouns, verbs, or other parts of speech?

26. The power to understand pantomime or gesture expression.

27. The power to employ intelligently gesture in expression.

28. The power to read figures and to calculate.

29. The power to count both money and in numbers.

30. The power to play a game of cards or other games.

It is not to be forgotten that speechlessness is often present in melancholia and dementia. Further, temporary speechlessness or apparent aphasia may follow severe typhoid fever as a result of cortical exhaustion without the development of hemorrhage, embolus, or thrombus. The prognosis is favorable.

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