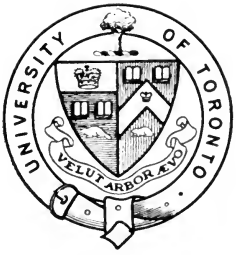

W. S. McClintock

1919

St. Mihiel Hospital.

A MANUAL OF

PHYSICAL DIAGNOSIS

O'REILLY

"The knowledge which a man can use is the only real knowledge, the only knowledge which has life and growth in it and converts itself into practical power. The rest hangs like dust about the brain or dries like rain drops off the stones."—*Froude.*

A MANUAL OF

PHYSICAL DIAGNOSIS

BY

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TO THE MEMORY OF

JOHN O'REILLY

Surgeon in Ordinary to the Households
of their
Majesties King George IV. and King William IV.
Died at Windsor, England, A. D. 1833

AND OF

THE HONORABLE JOHN ROLPH

M. A., L.L. D., M. R. C. S. ENG.

Founder of Medical Education in Upper Canada. Dean of "Rolph's School of Medicine," and of the "Toronto School of Medicine," from A. D. 1853–1870. Member of the Honorable Society of the Inner Temple, London, England. Member of the Legislative Executive Council of Upper Canada, A. D. 1835. Died, A. D. 1870.

PREFACE.

In the following pages an effort has been made to epitomize, for the use of students the various physical methods employed in the diagnosis of a medical case, and to as far as possible explain the origin and clinical significance of those described. The subject has been approached from a practical standpoint, the chronological order adopted being that employed in the actual examination of patients, special stress being laid on "Inspection", for which, however, the author feels no apology is needed. The "Appendix" was added merely to aid the student in following out the ordinary laboratory technic, only those methods being described which are in daily use in the routine examinations required by the clinicians of St. Michael's Hospital, Toronto.

Many authorities have been freely consulted by the author who declaims any title to originality, and who takes this opportunity to acknowledge his indebtedness to the works of others for the greater part of the material found in this volume. To both Dr. William Osler, "Regius Professor" of medicine in Oxford, England; and to Dr. Lewellys F. Barker, Professor of Medicine, Johns Hopkins Hospital, Baltimore, whose kindness in reading the manuscript of this manual and whose personal encouragement and pertinent suggestions have been of greatest value, the writer begs leave to convey his appreciation. To Dr. H. B. Anderson and to Dr. W. H. B. Aikins, of Toronto,

without whose unfailing kindness and assistance this volume would not have been completed; to Dr. James Mackenzie of London, England, for his courtesy in allowing the reproduction of certain of his original cardiograms, and to a number of his associates for their assistance in connection with the reading of proof, the author also desires to express his obligation.

BREFNEY R. O'REILLY.

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MANUAL OF
PHYSICAL DIAGNOSIS.

CHAPTER I.
INTRODUCTION.

No attempt has been made by the author more than to present in as concise a manner as possible the subject of **physical diagnosis.** In the following pages an effort is made to discuss certain fundamental principles and their practical application in the diagnosis of disease in order to supply to the student of medicine a working basis for further investigations.

Before proceeding let me remind the student who is beginning the study of **clinical medicine** that he has no longer solely to consider abstract facts and theories, but that he is about to apply his knowledge of anatomy, physiology, etc., to the living human subject.

It is at this stage that he encounters for the first time a "*personality.*" He has not now to deal with inanimate objects, but with intellectual beings, the intricate workings of whose minds demand consideration; and let me here emphasize the fact that the power to understand human nature is one most essential factor in the composition of a successful practitioner. It must also never be forgotten by the student that the patient before him has a mentality probably as capable of experiencing the "finer feelings" as his own,

and that he must in every case use toward him or her the same consideration that he himself would wish to receive from others.

Let me at this point once more emphasize the necessity for approaching the subject of clinical medicine not only from the narrower scientific, but also from the broader psychological aspect.

The student should lose no opportunity of studying the individual, visiting the "out-patients" in their homes, ascertaining their environment, talking with them, endeavoring to discover their peculiarities and fathom their personalities. Let him also not lose sight of the fact that the physician to be successful must not only be personally acceptable to the patient, but must endeavor to gain and hold the support and confidence of the friends and relations.

Since *tact and equanimity* occupy a position equal almost in value to that of scientific knowledge among the qualifications of the successful physician, the student is urged to neglect no opportunity of observing them in others and of endeavoring to acquire them for himself. Also, let him never forget that while the examination of the patient is proceeding he himself in his turn is being observed and analyzed by his subject. If this fact were constantly borne in mind many embarrassing situations would be avoided.

During the clinical examination of patients it is well to *avoid as far as possible discussions relating to unfavorable prognoses and the use of lay terms* such as cancer, consumption, etc., remembering that the psychical effect of an unguarded statement on the mind of a nervous subject may be at times far-reaching in its injurious results.

Before proceeding to discuss the methods used in the art of physical diagnosis it is necessary to define that term as well as certain others to which frequent reference will be made throughout this volume, and also to refer to our conception of the nature of disease.

We must first remember, however, that we not infrequently find in man considerable deviations from the normal type which are congenital in origin, or in other words are primarily due to some defect in the development of the embryo. For instance, the subject may have supernumerary digits, an undescended testicle or imperforate anus; these abnormal conditions we term malformations or anomalies.

It is next necessary to define the term disease, remembering at the outset that **disease** is not a stationary phenomenon, but a *series of progressive or active abnormal processes* occurring in the living body.

Slight deviations from the average normal physiological functions of the body must not lead us to conclude that a diseased process is in progress, since the healthy body has the capacity of accommodating itself to, compensating for, or overcoming the effects of some most abnormal external conditions in such a way as to prevent perceptible interference with the regularity of the vital processes. For instance, if a large quantity of water be imbibed, polyuria or excessive urinary secretion will result, in order to equalize and maintain the normal ratio of fluids and solids in the body.

Disease then is a condition in which the regulative mechanisms of the body, acting in opposition to one or more abnormal external influences, are no longer adequate to secure that the various physiological functions shall proceed undisturbed (Cohneim).

The severity, duration, and extent of such a process will therefore depend not only on the intensity and character of the abnormal external influences, but on the degree of the regulative capabilities or reaction of the affected organism.

It is well for the student to remember that frequently a diseased process may terminate and leave as a result certain pathological changes in the parts affected, such as we find, for instance, in the curtains of the mitral valve after the cessation of an attack of valvular endocarditis, and we should not, strictly speaking, refer to these changes as diseased conditions since no active abnormal processes are at present occurring in the valves themselves.

For a thorough understanding and conception of any disease, the etiological factors, clinical observations, and results of the postmortem examinations are of equally essential importance.

To sum up: Disease consists of a series of progressive pathological changes in the structure of any portion of the body or in the exercise of its function, or in both.

The term **diagnosis** refers to the differentiation of one disease from another. There are many different means which we may employ in order to facilitate our investigations, the most important of these, however, consist in certain methods which we group under the term physical diagnosis. Other methods are electro-diagnosis, serum-diagnosis, the use of the *x*-ray, etc.

Physical diagnosis refers to the application of certain physical methods used in the determination of the state of the organs of the human body.

In its practice we must endeavor to bring each of our five senses into play and to base our diagnosis not on any one fact we may elicit, but on the combined results obtained after having exhausted every means of investigation at our disposal.

Physical signs are those objective phenomena and conditions which the examiner is enabled to demonstrate by means of the methods described below (inspection, palpation, etc.).

A **symptom** is a subjective sensation recognized and usually complained of by the patient; symptoms differ materially therefore from the objective signs above referred to.

It is now necessary to describe in detail the methods employed in the elicitation of physical signs. Foremost among these is that of **inspection**, or the recognition of phenomena visible to the naked eye. Let me emphasize the importance of the results which one may obtain from its accurate application, and let me especially draw attention to its value in the examination of the comatose, of children, imbeciles and foreigners, or of those from whom for any reason we are unable to derive trustworthy information regarding the history of their illness or their present symptoms.

Having exhausted this means of investigation we next bring into play the sense of touch by employing the art of **palpation.**

By means of **percussion** we throw into a series of audible vibrations certain structures of the body, and it is from an appreciation of the notes elicited that we are enabled to make certain deductions.

Auscultation enables us to detect sounds produced in the body and its cavities, and **mensuration** to record

the body measurements as well as to confirm certain of our previous observations.

Finally, we may bring into play certain means of corroborating the observations already made, as for instance, the *x*-ray, bacteriology, chemistry, and the various scientific instruments of precision, such as the polygraph, sphygmomanometer, etc. It is, however, only after a careful application of every method at our disposal and consideration of the results obtained that we are justified in arriving at a definite diagnosis in a given case.

Before commencing, however, the physical examination of any patient who comes before us it is necessary to inquire into a number of facts regarding his past, as well as his present condition. The results of this interrogation constitute what is known as a **clinical history,** the details of which will be fully discussed in Chapter II. After having completed the patient's history we are in a position to proceed with an examination of his physical condition, and it is to the exemplification of the various methods employed by the clinician that the greater portion of this volume is devoted.

Since the human body is composed of certain *systems* (respiratory, circulatory, etc.) which are to a certain extent anatomically independent of each other, and since diseased processes are not infrequently confined to one or another, to exclusion of the rest, it seems natural to first examine each as a separate unit, and this will be proceeded with in as systematic a manner as possible.

Before investigating each individual system, in Chapters III and IV on general and special inspection

a number of phenomena visible on the surface of the body, not directly connected, however, with any of the great systems, will be described, as, for instance, the facies, skin, extremities, etc. A brief review of the topographical relations of the chief thoracic and abdominal organs and a chapter dealing with the practical analysis of the various pathological fluids, secretions, etc., are also added.

CHAPTER II.

THE CLINICAL HISTORY AND INTERROGATION.

INTRODUCTION.

From the time the patient enters the clinic room or is approached by the examiner he should be under the closest observation; as far as possible he should be put at his ease and, if necessary, reassured as to the methods of examination.

The student is again urged to neglect no opportunity offered to prepare himself for his future vocation; let him place himself mentally in relation to each case in a similar position to that of the clinician in charge, and not merely remain a passive spectator.

In the "*out-patient department*" as the patient crosses the room to the examiner's desk certain important observations must be made. The sex and apparent age are first noted; the latter, however, is to be compared later with the age according to the patient's own statement, since both frequently may be misleading. The gait (noting any peculiarities in the mode of locomotion), the general development, nutrition, and the presence or absence of gross deformities are next noted. The clothing also should receive close attention, and finally the station or the attitude assumed by the subject when at rest in the upright position must be determined.

The chair provided for the patient should have a

straight back and no arms, the light being arranged to fall over the examiner's shoulders on the subject's face. *Daylight* is essential for accurate observations, artificial light being frequently deceptive, especially in the examination of the color of the mucous membranes and the skin rashes.

Where the *patient is confined to bed* in place of determining the gait, station and condition of the apparel, note carefully the decubitus, or the position assumed when in the recumbent posture.

The "interrogation" may now be commenced. The following questions are suitable for casual examinations; a complete scheme for history-taking, however, will also be found below.

The patient's full name, age, sex, nativity, home address, occupation, marital relations, and name of previous attending physician must first be ascertained.

Inquiry is next made as to the complaint, its onset (if sudden or gradual), duration, if he has previously been subject to similar attacks, and if so, how often and when. It is also well to ask the question "How long is it since you were in perfect health?" as a check on the patient's statements regarding the onset of his illness. It is advisable also to ask if there has recently been a loss in weight.

It is usually better to allow the patient to describe his symptoms in his own language without interruption, and during this period to note carefully the mentality and also to as far as possible determine his temperament.

Inquiries should now be made with the object of determining the surroundings of his home and the health of its occupants, especially when an acute

infectious disease is suspected; also the details of an average day's routine and the presence of vicious habits, such as the abnormal use of tobacco or stimulants, must be determined.

Too much stress cannot be laid on a careful investigation into the habits and environment of the patient, as a means not only of determining predisposing and exciting etiological factors, but as a guide to treatment; for we must remember that a mere readjustment of the surroundings or mode of life is frequently of the utmost importance in the management of a given case.

The family history is now inquired into, noting the presence of an inherited tendency to tuberculosis, carcinoma, rheumatism, Bright's disease, gout, insanity, the neuroses, organic nervous diseases, etc., in father, mother, brothers and sisters, and also the causes of death in the cases of deceased members.

It is usual at this point to determine the temperature by means of a clinical thermometer, to count the pulse and respirations, and in the females to inquire as to the catamenia.

The examiner is now in a position to ask himself "Is this patient acutely ill or is he suffering from a chronic disease?"

On the completion of this superficial interrogation we may proceed to the physical examination of the patient, or when time permits to record a complete history, following the general principles outlined in the following table, as compiled by the author and in use in the "medical services" of St. Michael's Hospital, Toronto.

THE CLINICAL HISTORY.

Serial Number.....Medical Service Number.....Ward....
Date of Admission..... Date of Discharge.....
Diagnosis ..... Result .....
Previous Admissions.....

A. **General Interrogation.**

Name.	Nativity.
Address.	Marital relations.
Sex.	Previous occupations.
Age.	Present employment.

Complaint:

(1) Patient's spontaneous description of his illness.
(2) Rapidity of onset.
(3) Duration.

Personal History:

(1) Home surroundings.
(2) Habits.
  (a) Day's routine.
  (b) Exercise.
  (c) Sleep.
(3) Alcohol and tobacco.
(4) Tea, coffee, and drugs.
(5) Residence abroad.

Family History:

(1) Predisposition to tuberculosis, cancer, rheumatism, Bright's disease, gout, insanity, the neuroses, organic nervous diseases, etc.
(2) Health during life and present age, or cause and age at death, of parents, grandparents, brothers, sisters, children, and collateral relations.

Previous History:

(1) Former attacks of present illness and their duration.
(2) Previous treatment.
(3) History of the infectious fevers.
(4) Venereal disease (gonorrhea or "clap"; syphilis or "lues").
(5) Other illnesses or accidents, date of occurrence, duration, and results.

B. **General Inspection.**—A full description of this procedure will be found in Chapters III and IV.

C. **System Examination.**—It is next necessary to proceed with the physical examination of the various body systems, but it is usually advisable to first question the patient in a more detailed manner as to his symptoms, and as a guide for the student we will refer under this heading to several of those more commonly complained of in disease of each individual system.

After having determined which of the various systems is primarily or most markedly involved, still further investigations should then be instituted, having in mind the differentiation of the disease suspected from others presenting somewhat similar signs or symptoms.

I. **Respiratory System:**

(1) *History* of cough (character, pain), hemoptysis, dyspnea, "night sweats."
(2) *Objective* (see Chapter VI).
(3) *Analytical.*—Throat swabs, sputum, pleural exudates, tuberculin tests (see Appendix).

II. **Circulatory System:**

(1) *History* of precordial pain, palpitation, vertigo, syncope, dyspnea, cough, edema of the feet.

(2) *Objective* (Chapter VII).

(3) *Analytical.*—Examination of the blood, blood pressure, cultures and coagulation time, sphygmographic tracings, Widal's reaction (see Appendix).

III. **Digestive System:**

(1) *History* of excessive thirst or appetite, anorexia, dysphagia, eructations, nausea, emesis (time of occurrence, relation to meals and pain, quantity, hematemesis), flatulence, pain or "heart-burn" (location,character, relation to meals, radiation), defecation (diarrhea, constipation, character of stools, tenesmus, pain, hemorrhoids).

(2) *Objective* (Chapter VIII).

(3) *Analytical.*—Gastric contents, feces, etc. (see Appendix).

IV. **Genito-urinary System:**

Although a full description of the physical examination of the organs is not given in this work, it is always well, however, to inquire as to their functions, especially in women.

(1) *History:*

(a) Generative System.—In females note menstruation (frequency, pain, amount and character of flow. The patient is usually asked if she is "regular in her

monthlies," as to her "sickness," or if she is "regularly unwell"). Miscarriages, confinements, leukorrhea ("whites" or discharges). In men inquire as to nocturnal emissions, sexual habits, and discharges.

(b) Urinary System.—Lumbar or pelvic pain (character, location, onset, duration, termination, radiation, relation to micturition and bowel movements), micturition (frequency, nocturnal, control, precipitancy, tenesmus, pain). Remote symptoms due to renal insufficiency such as headache, drowsiness, dyspnea, defective sight, fits or convulsions and edema, especially of the eyelids. Note abnormal thirst or appetite and frequent micturition in diabetes, also inquire for a history of the passage of urinary calculi.

(2) *Objective:*

(a) Genital System (see special works).

(b) Kidneys (Chapter IX).

(c) Bladder and Urethra (see surgical works).

(3) *Analytical:*

Urine, semen, discharges, calculi (see Appendix).

V. **Neuro-muscular System:**

(1) *History* of headache, delirium, vomiting, defective sight, transient diplopia, vertigo, coma, convulsions (aura, onset, duration,

character, note also bitten tongue and involuntary micturition), peripheral pains, paresthesias (tingling, numbness, "pins and needles," formication, etc.), sexual impotence, mental changes, sphincter control, muscular stiffness or weakness.

(2) *Objective* (Chapter X).

(3) *Analytical.*—Cerebrospinal fluid (see Appendix).

VI. **Osseous and Articular Systems:**
For details of this examination the reader is referred to surgical works. The presence of heat, redness, swelling, pain, tenderness, crepitus, and mobility of the joints should, however, be noted; as also should tumors and deformities of the bones.

VII. **Report of Specialist's Examination.**—Eye, nose, throat, ear, etc.

VIII. **Report of progress, complications and treatment** of each case from day to day. Append also the temperature chart.

IX. **Result** of case.

X. **Report of postmortem** examination if any.

CHAPTER III.

GENERAL INSPECTION.

A. **THE GAIT,** or method of locomotion, is frequently of great significance in the investigation of a case. For proper study the legs should be fully exposed and the feet bared. Since certain *types* of progression are pathognomonic it is advisable to describe these in some detail, but it is first necessary to eliminate the possibility of any peculiarity noticed being due to congenital deformity or local surgical disease of the bones or joints.

I. **The Alcoholic.**—The staggering reel of the drunkard needs no description. It is to be differentiated from that seen in the severer forms of chorea, in certain cerebellar lesions, and occasionally in hysteria.

II. **The Spastic Gait.**—Bilateral spasticity is most frequently met with in lateral spinal sclerosis. Here the legs seem stiff, the knees refuse to bend, the feet are dragged along with the toes clinging closely to the floor. A compensatory tilting of the pelvis aids in the forward movement of the legs, but to a less extent than in the gait of hemiplegia.

III. **The Hemiplegic** gait is an example of one-sided spastic progression. The limb on the affected side swings from the pelvic girdle, flexion at the knee-joint being limited, and the foot passes through an arc of a circle whose convexity is directed away from the patient's side. The corresponding arm is usually also paretic.

This condition is sometimes simulated by that noticed in functional nervous disease, but here the leg is dragged along, no attempt being made by the patient to swing it forward; in contrast, also, the muscles of the limb are generally flaccid. Concomitant symptoms of hysteria will usually clinch the diagnosis.

IV. The Ataxic gait is seen best in tabes dorsalis. The gait is stamping, the feet are lifted suddenly and abnormally high, the limb being jerked forward, and the foot slapped down, frequently the heel touching the ground first. The patient walks on a broad base with the legs wide apart, the eyes fixed on the ground, and the body bent slightly forward.

In planting the feet uncertain swaying movements are exhibited, the leg not moving forward in one plane as is normally the case. The patient is unable also to advance in a direct line, to start suddenly or turn quickly on the word of command, all of which abnormalities become accentuated if the eyes are kept closed.

V. **The Festinating** gait is found in cases of paralysis agitans, and is also known as the *propulsive gait*. The patient advances with short, rapid, shuffling steps and increasing speed, with head and back bowed forward, as if he were attempting to reach his center of gravity, which lies apparently somewhere before him.

Retropulsion is occasionally seen. Here, if the subject be suddenly pulled backward, he commences to shuffle backward and is unable to arrest his steps, though throughout he is leaning forward, as in the former case. This gait in connection with bowing and stiffness of the spine ("poker-back"), the characteristic

facies (page 27), and certain rhythmical movements of the hands (page 47) is diagnostic of the disease.

VI. **The Steppage** gait is best seen in cases of multiple neuritis and is due to paralysis of the extensor muscles of the foot ("foot-drop"). In order to prevent the toes dragging the leg is lifted higher than normal, the foot jerked suddenly forward, thus throwing the toes in an upward direction. The patient presents the appearance of stepping over obstacles. It has also been termed "*prancing gait*," and is characteristic of the disease mentioned. It is not infrequently found also in cases of pseudo-hypertrophic paralysis in addition to the "waddling" movements.

VII. **The Waddling Gait.**—In pseudo-hypertrophic paralysis a broad base is effected, the spine being in a condition of lordosis. The body sways regularly from side to side, and the feet are often lifted high, as in the steppage gait, the heels and toes touching the floor simultaneously.

The difficulty found in maintaining the center of gravity, due to weakness of the legs and dorsal muscles of the trunk, is responsible for this peculiar gait.

The *method used in raising the body from a supine to an upright posture* is of interest. The patient first rolls over on his face, raises himself on his elbows and hands, then transfers his hands one at a time to the anterior surface of his knees, to his thighs, and finally lifts one leg at a time in order to gain the standing position—"climbing up himself" as it has been termed.

VIII. **The Vertiginous or Cerebellar** gait resembles that of an alcoholic. The steps are short, the feet are planted wide apart, and a tendency to reel is noted. In some cases support applied to the axillæ will over-

come this; in others the reeling is continually sideways or backward, especially in unilateral tumors of the cerebellum. This type may be closely simulated in certain cases of hysteria, but here, of course, the absence of an organic basis and presence of hysterical stigmata will prevent error.

B. **THE STATION**.—This term is applied to the attitude assumed by the patient when at rest in the *erect posture*. First note whether the patient can stand unsupported, and if a tendency to sway is present note if it is persistently in one direction, or if irregular.

The patient is now directed to stand with the feet close together. Ability to stand with eyes open with loss of equilibrium on closing the lids constitutes **Romberg's sign,** a phenomenon typically seen in tabes dorsalis. It is due either to interference with the central mechanism which controls the equilibrium, to plantar anesthesia, or to loss of "muscle sense."

Examine for the presence of *spinal curves* (lordosis or an abnormal bowing forward of the vertebræ, scoliosis or lateral curvature, and kyphosis or an excessive convexity backward of the column). Notice also the shoulders, marking whether both are of the same height.

The outline of the lower limbs, the manner in which they are set on the floor, and the condition of the upper extremities should also be determined, noting if they are held in any peculiar position or if they are used to aid in supporting the trunk, as we see in cases of disease of the vertebral column. In the latter instance the object in view is to attempt to transfer part of the weight of the body to the arms, and so relieve pressure on diseased and painful areas in the spine.

C. **THE DECUBITUS.**—This term refers to the position assumed by the patient when in the *recumbent posture*. First observe if the sitting position in bed is chosen by preference. This is known as **orthopnea** and is found most commonly in patients suffering from advanced cardiac, renal, or pulmonary lesions, dyspnea usually occurring on lying down.

The **dorsal** decubitus is common to many diseases, and is of little diagnostic value except when one or both legs are drawn up to relieve pelvic or abdominal pain or tension, as seen in infections of the peritoneum.

The **lateral** position is most frequently found in unilateral thoracic or abdominal disease. In acute pleurisy, for example, this position is assumed to aid in limiting the movements of the affected side and thus reduce pain. Also in cases of pleural effusion the patient may lie on the affected side in order to allow fuller play of the opposite lung and thus assist compensation. This posture is also frequently assumed when a cavity is present in the lung, that position being chosen which brings its opening uppermost, thus allowing for the accumulation of secretions and their discharge at intervals, instead of a constant trickling into the bronchial tubes with its consequent irritation and continuous cough.

Pleurosthotonos is a lateral bowing of the body, **opisthotonos** a condition in which there is backward arching of the spine, the body resting on the occiput and heels, the dorsal muscles being in a state of tonic contraction. The latter condition is found in its most typical forms in strychnia poisoning and cerebrospinal meningitis.

Emprosthotonos is the term applied to that condition

in which the patient assumes a bending forward attitude. It is uncommon, but may be noted in the same class of cases as is opisthotonos, of which it is the reverse. It is seen also in the acute abdominal inflammations and in renal and hepatic colic.

The **prone** posture is also rare, but may be assumed for relief of pressure on the anterior surfaces of the vertebræ; for example, in Pott's disease or aortic aneurysm; and less frequently in mediastinal disease or gastric ulcer.

The term **jactitation** is used to describe a restless jerking of the body, apparently purposeless. It is seen in hysterical attacks, also after large hemorrhages, in renal or hepatic colic, and in chorea.

D. **THE CLOTHING** should now receive careful attention. Is the patient neatly dressed or are there evidences of *carelessness* (buttons left undone, shoelaces untied) or is there a general appearance of untidiness such as we find in mental defectives?

One should notice the presence of *stains* over the upper garments, as when drooling has occurred, or over the trousers as an evidence of incontinence of urine. In this latter instance we may find the clothing wet, and an ammoniacal odor may also be noticeable. In diabetes whitish stains, due to deposition of crystals of glucose, may occasionally be seen.

The condition of the *shoes* will often point to some peculiarity in the gait, as, for instance, when the heels, toes, or one side shows evidence of excessive wear. Note if they are loosely laced, as we see where edema or tenderness of the feet is present.

One should also examine the seams of the underclothing for the adult pediculus corporis or other

parasites, especially if evidences of cutaneous irritation are present.

Lastly, is the *clothing too loose?* One may frequently from this fact suspect a rapid emaciation. The opposite may be found especially in the abdominal region, as an evidence of ascites, tumors, etc. This evidence is of special aid in dealing with foreigners or those who for some purpose attempt deception as to their condition.

E. **DEVELOPMENT AND NUTRITION.**—It is of interest first to note two *types of skeletal formation* which are congenital. These are, first, the tall, spare, small-boned subject with a long, narrow, shallow thorax and an acute subcostal angle; and second, the short, obese, thick-boned individual with a capacious thorax, horizontal ribs, and an obtuse subcostal angle. The former group seems to be especially prone to tubercular infections and the latter to emphysema and certain cardiovascular lesions.

Since every healthy person has his own normal *ratio of weight in proportion to stature*, we must consider each individual separately. Of far greater importance, however, than the average height, weight, and their relative relations as shown by statistics, are *variations from what is normal for a given subject.*

Rapid loss of weight is seen in fevers such as enteric, in chronic diseases as tuberculosis, diabetes, or carcinoma, but retention of weight in ascites and anemias is frequent and may mislead the patient as to the seriousness of his condition. In the last instance it is probably due to deficient oxidation.

It is necessary to endeavor to estimate and differentiate muscle from adipose tissue also to compare

the weight week by week or, in the case of infants, day by day, and to remember that the average *infant* at birth weighs from 6 1/2 to 7 1/2 pounds, and that at the end of the first three days a loss of 4 to 7 ounces is usually noticed, the original weight being regained by the tenth day.

TABLES OF AVERAGE WEIGHTS.

Infants.		Adults.	
Age.	Weight.	Height.	Weight.
1 month	8 lbs. 5½ oz.	5 ft. 1 in.	120 lbs.
2 months	10 lbs. 5 oz.	5 ft. 3 in.	133 lbs.
3 months	11 lbs. 15 oz.	5 ft. 5 in.	142 lbs.
4 months	13 lbs. 9 oz.	5 ft. 6 in.	145 lbs.
5 months	14 lbs. 14 oz.	5 ft. 7 in.	148 lbs.
6 months	16 lbs. 3½ oz.	5 ft. 8 in.	155 lbs.
7 months	17 lbs. 5 oz.	5 ft. 9 in.	162 lbs.
8 months	18 lbs. 10 oz.	5 ft. 10 in.	169 lbs.
9 months	20 lbs. 1 oz.	5 ft. 11 in.	174 lbs.
10 months	21 lbs. 5 oz.	6 ft. 0 in.	179 lbs.
11 months	22 lbs. 0 oz.		
12 months	22 lbs. 7 oz.		(*Hutchinson*)
	(*Ashby & Wright*)		

F. **THE FACIES.**—The physiognomy is one of the most fascinating studies with which we have to deal, the face bearing as it does the impress of character and mode of life, as well as evidences of disease and suffering.

The *general expression* should first attract our attention especially the placid, apathetic, or vacant stare; that of fear or anxiety; the haunted, suspicious look of certain mental diseases; or the shifty glances

of the malingerer or of those who have something to conceal. A careful study will frequently do much to aid us in understanding our subject's state of mind, and the information so elicited will often be an important guide to our method of approaching the patient.

The recognition of the *seat of disease* from facies showing evidence of suffering is possible in some cases from an analysis of the features. For instance, cerebral pain will often cause frowning or contraction of the muscles of the forehead; pain due to respiratory disturbances accentuates the nasal furrows; while abdominal inflammations show themselves by drawing down of the angles of the mouth. These signs are of peculiar value in foreigners and in infants who are unable to express their symptoms intelligently.

It is a difficult matter to describe *characteristic types* of facies, but for the guidance of the student we will endeavor to point out the more prominent features in a few diseases, commencing with those found in the acute conditions.

I. **Hippocratic.**—This type of facies is most commonly found in acute peritoneal infections. Its appearance is of ominous significance and is usually a sign of impending dissolution.

The patient is nearly always conscious, the eyes are open but dull and lusterless, the temples and cheeks appear sunken, the chin and alæ nasæ pinched, and the skin is pale and clammy. In addition, we notice a slight cyanosis (lividity), especially around the lips, which seem thin and drawn. Once seen, the impression received will never be forgotten.

II. **Pneumonic** is typically seen in lobar pneumonia.

Here the eyes are bright, expression anxious, the alæ of the nose actively dilating with each inspiration, and the respiratory rate is greatly increased in frequency. The lips are cyanotic, frequently exhibiting a herpetic eruption, and one or both cheeks are markedly flushed, usually, but not always, that on the side of the lesion being most affected.

III. **Typhoid.**—The expression is dull and listless, and the cheeks are usually flushed. Later in the disease the mouth is held slightly open and the lips are retracted, crusted and dry, exposing the upper teeth which often exhibit a fuliginous deposit (sordes) which consists principally of desquamated epithelium and inspissated mucus. Signs of progressive emaciation are also marked.

"*Coma vigil*," in which we find a loss of consciousness, low muttering delirium, with widely open staring eyes, may be seen in severe cases with intense toxemia, as also are carphologia, (clutching at the bed-clothes), and subsultus tendinum or twitching of the muscles of the forearms and hands.

IV. **Sardonic.**—The *risus sardonicus* is met with in strychnia poisoning. The angles of the mouth are drawn laterally, partially exposing the teeth, the jaws are tightly closed and the facial muscles in a state of spasm. Opisthotonos is usually also present, as are also evidences of muscular spasm in other parts of the body.

V. **Cardiac.**

(1) *Aortic Regurgitation.*—The face is pale; frequently capillary pulsation, throbbing of the temporal arteries, and in severe cases a nodding of the head synchronous with the cardiac systoles may be detected.

During attacks of *angina pectoris* a death-like pallor supervenes, the eyes are widely open and staring, pupils dilated, and the facies expressive of intense agony. As the attack passes off perspiration appears on the forehead and the normal color is regained.

(2) *Mitral Disease.*—The color is more or less dusky from cyanosis, and the venules are prominent. In cases of mitral stenosis the cheeks are often apparently flushed, but a careful investigation usually reveals the presence of slight cyanosis. In attacks of *cardiac asthma* pallor may accompany the lividity and signs of dyspnea appear.

(3) *Morbus cæruleus* refers to that deep blue cyanotic hue characteristic of certain congenital defects in the formation of the heart. The venules are unduly prominent and the cyanosis present is out of all proportion to the dyspnea.

VI. **Nephritic.**—Here we find, especially in parenchymatous nephritis, a marked degree of pallor, conveying a peculiar impression of "pastiness," associated with an edema or "puffiness" of the lower eyelids, most marked in the morning.

Frequently when *uremia* is present we may also detect evidences of dyspnea, characterized by cyanosis, rapid respiration, an anxious expression, and actively working alæ nasæ, which may closely simulate the pneumonic facies.

VII. **Phthisical.**—The bright wide-open eyes, the hectic malar flush, slight cyanosis and emaciation will usually direct one's attention to the underlying condition.

VIII. **Cachectic.**—Here the skin is pale and muddy, frequently also slightly yellow in color. The subcuta-

neous fat has been absorbed, and at times a slight brownish, patchy pigmentation is also demonstrable.

This condition is due to the combined results of anemia, emaciation, and the retention in the system of certain toxins. It is most commonly found in cancerous, syphilitic, and malarial subjects, also in that condition known as *cachexia strumapriva*, a combination of anemia with myxedema which follows extirpation of the thyroid gland.

IX. **Exophthalmic Goitre.**—The staring expression produced by the proptosis or protrusion of the eyeballs, infrequent winking, immobility of the occipitofrontalis muscle on rotating the globes upward, some enlargement of the thyroid gland associated with tremor of the fingers (page 47), and tachycardia are characteristic of this disease (page 139).

X. **Bell's Palsy.**—In paralysis of the seventh cranial nerve the forehead on the affected side is smooth, but the eye is open and the upper lid droops. The wrinkles have disappeared, the cheek is puffed in and out with each respiratory act, the paralyzed corner of the mouth droops and the opposite angle is drawn toward the healthy side.

XI. **Parkinson's Mask.**—The lack of expression and immobility, the "stony-stare" with the eyes directed to the ground, combined with disappearance of the facial furrows, constitute the physiognomy characteristic of paralysis agitans. The gait (page 17) and the peculiar tremor of the hands (page 47) clinch the diagnosis.

XII. **Marasmus.**—In the malnutrition of infants the expression is serious, the child's face resembling that of a "wizened" old man. The temples and cheeks

are hollow, the skin thin, harsh, and wrinkled. Pallor is also present. The nasolabial furrows are frequently accentuated and the subcutaneous fat absorbed.

XIII. **Adenoid.**—The facial expression of the *mouth-breather* is very characteristic, stupidity being the outstanding feature. The mouth is held open, the orifice being triangular with the apex uppermost, exposing the teeth. The lips are thick and dry, the nares pinched, and the nose itself small in size. Finally the eyelids show slight drooping. The expression has been compared to that of a fish, frog, or rabbit.

XIV. **Idiocy.**—Beyond the size and contour of the skull being in most cases abnormal, no single feature except the facial expression is pathognomonic. The variations are so numerous that no description suffices to convey the impression of mental deficiency so apparent to the observer.

Certain *stigmata of degeneracy* may be detected, notably malformation of the auricular lobes, a palatal arch which may be abnormally high and pointed or otherwise deformed, epicanthic folds, irregularities of the teeth, facial asymmetry and various anomalies of the hands and feet; but on no one of these signs can a diagnosis be arrived at, their value being merely confirmatory.

XV. **The Mongol** is a type of congenital idiot, obliquity of the palpebral fissures being the prominent feature, their inner angles slope downward as well as inward which, with the slight drooping of the upper lids (ptosis) gives an "almond-like" appearance to the orbits. The mouth is held open, the tongue is large and protruding, the head is usually small and globular, and the hands are broad and squat.

XVI. **Cretin.**—The face in a cretin is broad, the nose wide and flat, the forehead low, the eyes set far apart, the mouth open, the tongue slightly protruding, and the skin is thick and dry. In these cases the skull is flat-topped and of large dimensions, the stature small, the anterior fontanelle usually open, even as late as the eighth or tenth year, the abdomen prominent, the legs short, and the gait waddling and awkward.

Absence of the thyroid gland is responsible for the condition; it is therefore closely allied to myxedema of which it is the congenital analogue.

XVII. **Myxedema.**—Myxedema is an *acquired athyrea* due to either disease or removal of the thyroid gland, and therefore it closely resembles cretinism. To the post-operative form the term *cachexia struma-priva* is applied.

The features are coarse, the face full and round, the nostrils and lips thick, the mouth large, and the skin pale waxy, dry, and hypertrophic. Other evidences of this disease, such as bradycardia, a subnormal temperature, and a sluggish mentality corroborate the diagnosis.

G. **THE SPEECH.**—In the investigation of the above points the examiner cannot but have noted the speech of the subject and detected any abnormalities present (page 241), and also he will probably have formed some idea as to his mental caliber.

H. **MENTALITY.**—For details of this investigation the reader is referred to page 278, where this subject is discussed under the section on the nervous system.

CHAPTER IV.

SPECIAL INSPECTION.

Having thus completed a general survey or inspection of the patient we are in a position to proceed with a more detailed examination of the surface of the body and of certain of its members. Usually we first examine the skin, seeking by the presence of certain positive signs or the absence of others to arrive at a diagnosis.

It is necessary to emphasize the point that a negative fact or absence of a given phenomenon is frequently of equal importance to the finding of a lesion. It is therefore imperative to include negative observations in the history for future reference.

A. **THE SKIN.**—The following description may be applied to the cutaneous surface of any part of the body; in this way repetition can to some extent be avoided. No attempt is made to differentiate the various skin lesions; those changes only will be described which are associated with general diseases.

First notice the color of the surface with special reference to pallor, cyanosis, and pigmentation. In the latter instance note if localized or general. The degree of elasticity and the presence or absence of atrophic changes must also be determined.

Next examine for rashes and determine their characteristics; also note abnormal action of the sweat glands as shown by dryness or excessive perspiration. The condition of the peripheral circulation should likewise receive attention. Estimate the amount of the sub-

cutaneous fat and note the presence or absence of edema.

Scars either from trauma or resulting from certain skin lesions may be detected; if so, their position and character should be noted, as also should the presence of enlarged veins or parasites.

Endeavor to estimate the body temperature by palpating the skin of the trunk and compare the result with that shown by the clinical thermometer, as in emergencies this instrument may not be available.

I. **Pallor.**—May be general and permanent, as in the anemias; or transient, as in attacks of syncope, nausea, during chills, mental disturbances, shock, etc.

Pallor may even be localized, as seen in Raynaud's disease.

In **chlorosis** (except in cases of *chlorosis rubra* in which the apparent high color of the cheeks is deceptive), we find a greenish hue superimposed on the general pallor.

In **pernicious anemia** the color is a pale "lemon-yellow," the skin appearing at the same time waxy, the conjunctivæ being pearly white, in contrast to the condition seen in jaundice where an icteric tint is a frequent manifestation. The subcutaneous fat in both chlorosis and pernicious anemia is usually retained owing to defective oxidation, that however which lies beneath the ocular conjunctiva exhibits a yellowish tinge which unless care be taken may give rise to a diagnosis of jaundice.

Cachexia has already been described (page 26).

It is of great importance to remember that in persons with thick skins a *deceptive* appearance of anemia may be found, while in those with a delicate epidermis

marked anemia may be present in spite of the apparent good coloring; for this reason it is well to make hemoglobin estimations a part of every routine examination, but it is imperative that a complete analysis of the blood be undertaken in every suspicious case. The mucous membranes and conjunctivæ as well as the nails should always be examined for corroborative evidence as to the extent of the anemia.

II. **Cyanosis** may be acute or chronic, the color varying from a faint lavender, dusky or purplish tint, best seen in the lips or under the finger-nails, to the deep blue color of morbus cæruleus (page 26).

In asphyxia due to obstruction of air passages, either mechanical, inflammatory, or spasmodic, we see cyanosis at its height. In the subacute or chronic forms, however, the closest observation is at times necessary for its detection. Notice in these cases particularly the color of the nails and lips.

General cyanosis is generally dependent on cardio-respiratory lesions, and is then usually indicative of defective aeration; it may also result from qualitative changes in the blood due to toxins, such as those of pneumonia, the poisonous gases, certain drugs, or may result from vasomotor paresis.

Cyanosis may be also *local*, as seen in Raynaud's disease, chilblains and venous obstruction.

III. **Rashes.**—We must first differentiate those of the acute infective fevers from local lesions due to other causes, describing the character, situation, onset, color, presence of an exudate, desquamation, evolution, etc., and at the same time search for parasites, such as those of pityriasis versicolor and ringworm, which may be detected by means of the microscope.

Clinically we divide cutaneous rashes into (1) the *erythemas*, or areas of redness of the skin which can be made to disappear on pressure, as seen, for example, in scarlet fever, certain toxemias and drug rashes. (2) *Macules*, or discrete red patches, varying in size from that of a pin-head upward, nonpalpable, and disappearing on pressure. (3) *Papules*, or raised palpable lesions, usually of a reddish color. (4) *Vesicular eruptions* (bullæ or blebs), which are merely subcuticular collections of clear serum. (5) *Pustules*, or localized cutaneous abscesses. (6) *Dermatitis* or acute inflammation of the cutaneous surface.

Next examine the surface for discharges ("weeping"), crusting or scab formation, desquamation, infiltration, and also inquire for subjective sensations, such as burning, heat, tingling, etc. (paresthesia), and visible evidence of irritation as shown by "scratch-marks."

IV. **Edema** refers to the collection of serum in the subcutaneous lymph spaces. The swelling is painless and the skin tense and white, except in inflammatory edema where the color is red.

The phenomenon of "*pitting*" may be elicited; that is, by pressure of the finger over the suspected area for several moments one may produce a depression of the surface lasting some seconds. The eyelids, ankles, dorsum of the trunk, and sternal regions should be particularly examined where edema is suspected.

It may be *transient* as in urticaria (hives), in which it appears as wheals, or in angioneurotic edema. Edema is distinguished from *myxedema* by the absence of pitting, its distribution, and in the latter disease other evidences of thyroid insufficiency (page 29).

An accumulation of gases in the subcutaneous tissues

may be demonstrated by the presence of swelling and a sense of fine crepitation or crackling felt by the palpating finger. This will indicate the presence of *subcutaneous emphysema*, whose origin may be bacterial, due to loss of continuity of the cutaneous surface, or to rupture of an air-containing organ.

V. **Pigmentation** may be localized or general, and in both forms may be due to numerous causes.

In the *local* forms endeavor to discover an etiological factor, such as an irritant (chemical, thermal, mechanical or inflammatory), and note also if the pigmentation is associated with certain growths, as nevi, moles, melanotic sarcomata; or with general conditions, such as pregnancy (chloasma), hepatic disturbances, etc. It may be due to certain skin lesions as, for instance, tinea versicolor (due to the microsporon furfur), and is also seen in the copper-colored rashes of syphilis.

Generalized pigmentation may be due to the presence of bile-pigment (icterus or jaundice), in which the tint varies from a light yellow to a dark green and is seen most typically in the conjunctiva; to Addison's disease, in which bronzing of the skin and various mucous membranes is detected; to "argyria," or that deposit of pigment due to the administration and retention of silver salts in the body. In the last case the tint varies from a light-brown to a bluish color, depending on the severity of the case.

Leukoderma is an acquired condition recognized by areas of "milky white" skin surrounded by a narrow margin which show excessive pigmentation, the intervening skin remaining normal.

VI. **Ulcers** may be simple resulting from mechanical irritation, trophic or due to defective nutrition, malig-

nant (as in cutaneous cancer), or luetic (specific or syphilitic).

Note the situation and outline, the condition of the base as regards discharges, presence of exuberant granulation tissue, and its connection with the underlying structures (adhesions). The edges also should be examined and their condition described.

VII. **Scars.**—Note the situation, as, for instance, where present only over exposed surfaces of the body,

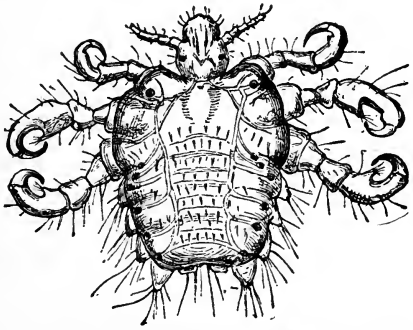


FIG. 1.—Pediculus Pubis. (*Tyson after Braun.*)

symmetry, age, outline, presence or absence of retraction, the consistence (for example the "tissue-paper" phenomenon characteristic of syphilis), the presence of pigmentation (as seen in the "copper-colored" tint of those of specific origin), or the exuberant scar tissue of keloids.

VIII. **Parasites.**—The various pediculi are easy to detect. The *pediculus pubis* (Fig. 1), and *capitis* may be found in the regions from which they derive their names; and the *acarus scabei* ("itch-mite," Fig. 2), at the bottom of tiny linear burrows in the epidermis, especially between the toes, fingers, and flexures of the

body, from which it may be removed by means of a needle.

The *pediculus corporis* ("body louse"), however, must be searched for in the seams of the clothing, since it is only present on the skin for the purpose of obtaining nourishment (Fig. 3).

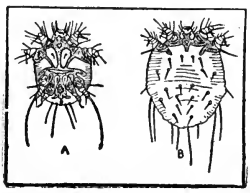


FIG. 2.—Acarus Scabei. A. Male. B. Female. (*Greene after Braun.*)

IX. **Nevi, Etc.**—Search for moles, new-growths, ecchymoses ("bruises"), petechial hemorrhages, spider angiomata, dilated venules, scratch-marks, and the "hemorrhagic specs" of pediculosis; endeavoring always to discover the etiological factor.

X. **Odor.**—The "*stale*" odor characteristic of typhoid, the "*mousey*" of favus, the "*pungent*" of hyperidrosis, the "*ammoniacal*" or "*urinous*" of vesical incontinence, and the "*sweet*" of the diabetic may serve as an aid to diagnosis, especially when examination is difficult, the history incomplete, where the patient is found unconscious, and in foreigners.

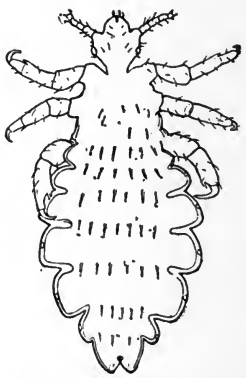


FIG. 3.—Pediculus Corporis. (*Tyson after Braun.*)

B. **CRANIAL VAULT.**—Notice first the size and contour of the calvarium. Search for local inequalities in the bones or superficial parts, and confirm their situation, extent, and consistence by palpation; noticing at the same time the condition of the sutures and fontanelles, the presence of craniotabes (unossified areas in the bones of the calvarium, usually

found in rickets and syphilis), and also the presence of tender spots or tumors.

Certain *types of skull* are fairly characteristic, the more common of these are found:

I. In **rickets** in which disease the skull is more square than normal, especially when viewed from above. The vertex and occiput are flattened, the forehead prominent, the fontanelles and sutures usually open at a late date, the latter occasionally until the twelfth month. The frontal and parietal bones may show craniotabes, "bossing" or thickening, and the scalp frequently evidences of excessive perspiration.

II. In **hydrocephalus** the skull is large, tending to become globular or pyramidal in outline, the face being relatively small and the forehead overhanging. The upper portions of the sclera are exposed on account of depression and protrusion of the eyeballs, the skull projects laterally, overhanging the auricles, and the fontanelles are widely open.

III. In **acromegaly** the head is somewhat enlarged, the face both elongated and broader than usual, of somewhat triangular outline, with the base directed downward (this is in part due to the enlargement of the maxillæ). The teeth are set wide apart, the features in general are coarse and thick, especially the nares, eyelids, tongue and ears which are markedly hypertrophic. Both this and the following are types rarely encountered.

IV. In **osteitis deformans** the outline of the face is triangular with the base upward. The head is carried well forward with the chin resting on the episternal notch. Search should be made for other characteristic skeletal deformities, especially in the hands.

V. In **leontiasis ossea** the head is large and globular, the malar bones and supraorbital ridges prominent, and the whole appearance conveys an impression of hypertrophy ("*lion-head*") to the observer.

C. **THE HAIR AND BEARD.**

I. **Quantity.**—Alopecia or loss of hair may be either general or patchy. In localized patches note carefully if the bald patches are completely devoid of hair, as in *alopecia areata*, or if broken hair stubs or signs of inflammatory reaction are present. If the latter conditions be found, subject the hairs to microscopic examination to determine the presence of the trichophyton or microsporon audouini (tinea tonsurans or ringworm, page 358).

II. **Color.**—Premature grayness should be noticed, especially if localized, as is sometimes seen in certain nervous lesions. The use of dyes must, however, first be excluded.

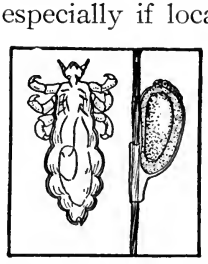


FIG. 4.—Pediculus Capitis and Nit. (*Greene.*)

The color of the hair in copper workers may acquire a greenish hue, while in cobalt and indigo handlers a bluish tint may be detected.

III. **Moisture.**—In atrophy of the skin and in myxedema diminished action of the glands is usually present, the hairs also are dry, thin and brittle.

In seborrhea oleosa, on the other hand, the scalp is greasy and desquamation of the epithelium ("dandruff") is found.

IV. **Pediculi.**—The adult parasite (pediculus capitis) may be found or merely the white oval nits attached to the hairs (Fig. 4).

V. **Crown.**—It is well also in cases of mental de-

generacy to examine the crown of the head, as abnormalities of the hair whorls may not infrequently be found.

D. **THE FOREHEAD.**—A description of the general examination of the skin of this region need not be reiterated. Certain *rashes*, however, show a tendency to appear in this situation, especially those of psoriasis, seborrhea, small-pox, and syphilis (corona veneris).

Profuse *perspiration* over this region in infants from six months to three years of age is suggestive of rickets.

Note the condition of the *wrinkles*, if less marked on one side, as in case of facial paralysis, or if the whole forehead be abnormally smooth, as in paralysis agitans.

The *muscles* may then be put into action by directing the subject to wrinkle his brows. In supranuclear paralysis of the seventh cranial nerve the forehead muscles are intact while those of the lower face are paralyzed on the affected side; in peripheral lesions, however, the paralysis is complete (page 255). Note also if on rolling the eyeballs upward wrinkling occurs. In exophthalmic goitre and double facial paralysis no movement results.

E. **THE CHEEKS.**—Their general condition has already been noted. In addition, several points should be observed.

Is there abnormal *flushing or sweating?* If so, is it unilateral, as is seen occasionally in one-sided lung disease, paralysis of the seventh cranial, and in lesions of the cervical sympathetic nerves.

Evidences of *facial paralysis* are seen in a loss of the normal furrows, apparent flattening of the affected side, and a drawing of the features toward the healthy side. In the comatose paralysis is recognized by a

puffing in and out of the affected cheek with each respiratory act (page 27).

F. **THE NOSTRILS.**—In addition to noticing the condition of the skin, inspect the *bridge* for the loss of normal contour such as we find after fractures, and also for the "saddle-nose" deformity so characteristic of syphilis.

Observe if the *alæ nasæ* are actively dilating with each inspiration as in dyspnea, or if the orifices are pinched, as in hippocratic facies and in "mouth-breathers." If a *discharge* is present, note its character and obtain sufficient material for bacteriological examination.

Rhinoscopy (see special works on this subject).

G. **THE AURICLES.**—The general development and symmetry should be noticed, as *congenital deformities*, such as malformation of the lobes or abnormal attachments, are not uncommon, especially in the mentally defective. Also note presence of the *hematoma auris* seen occasionally in the insane, in which condition the auricle is swollen, discolored and ecchymotic.

Pallor, cyanosis, discharges, and the presence of tophi ("chalk-stones") should be sought for. Occasionally transient anemia, cyanosis or even gangrene may be found, as in cases of Raynaud's disease.

Otoscopy (see special works on this subject).

H. **THE ORBITS.**

I. **Supraorbital Ridges.**—It is well to first examine and compare these structures, searching particularly for evidences of disease in the frontal sinuses.

II. **Eyelids.**—Note here the presence of the *edema* of the lower lids, so suggestive of nephritis, and of *ecchymoses* (hemorrhage). Observe the condition of the

margins, especially the presence of cysts, styes, and blepharitis. Occasionally parasites may be detected among the hairs.

Examine the condition of the *muscles* for blepharospasm as seen in photophobia (hypersensitiveness to light), and irritation of the seventh cranial nerve; look also for ptosis (drooping of upper lids) or retraction, and note if these conditions are unilateral. Is the act of winking properly performed? Infrequency is found in exophthalmic goitre (*Stellwag's sign*), and imperfect action in facial paralysis.

Imperfect coaptation of the lids is seen in Bell's palsy; it is usually also accompanied by *lacrymation*, or running of tears over the cheek, and *ectropion*. In this latter condition the punctæ are not kept in contact with the globe, either on account of the loss of tone of the orbicularis palperbrarum or the contraction of scar tissue. Lacrymation may also be due to stenosis of the nasal duct, to confirm which a probe may be passed.

The lids are sometimes also everted (ectropion) by a contraction of scar tissue following injury, trachoma, etc., or the lashes may brush against the globe from *entropion*.

Von Graefe's sign is seen in exophthalmic goitre. Here, on looking down the upper lid appears to lag, not following the globe in its downward rotation, as is normally the case. Finally, note the *axes* of the palpebral fissures, if they are altered as in mongols (page 28), and the presence of *epicanthic or semilunar folds* at their inner angles.

III. **The conjunctival sac** is next explored by everting the eyelids, the presence of foreign bodies or

inflammation (*conjunctivitis*) is noted, also if the latter is acute with swelling and ecchymoses. In acute inflammation the injection is brick-red in color, the vessels tortuous, and may be momentarily emptied by pressure on the lids, the inflammatory action being most marked in the cul-de-sacs, diminishing around the corneal margin.

Ciliary injection is recognized by a pink rim of congestion around the margin of the cornea; the vessels are straight and cannot be moved, while pressure on the lids leaves a momentary anemia. This condition should be contrasted with that of acute conjunctivitis, since it is indicative of iritis or corneal irritation.

The tiny pale ("sago-grain") nodules of *trachoma* ("granular lids"), or the grayish "false membrane" of *diphtheria* are occasionally seen. If a *discharge* is present note its character, and subject it to bacteri ological examination.

IV. **The Cornea.**—Its contour is inspected (*conical cornea*) and the presence of opacities (*leukoma*), such as remain after old inflammations (*keratitis*) or ulceration is to be noted. One should at the same time determine the depth of the anterior chamber and note if pus is present (*hypopyon*).

The presence of *arcus or circus senilis* (semilunar or annular opacities), due to fatty degeneration at the circumference of the cornea, are of no clinical significance in the aged, but are pathological in those under fifty and suggestive of arterial degeneration.

V. **The Iris.**—Alterations in its *color* are first noted, especially if asymmetrical; they may be congenital or the result of old inflammation (iritis).

Are the pupils dilated (*mydriasis*) or contracted (*myosis*)? Are they of equal, size first seeing, however, that both pupils are equally illuminated.

Is the outline of each a perfect circle? Irregularities in the *outline* of the pupil are found to be especially noticeable during its movements; they are frequently the result of adhesions following inflammation of the iris. *Hippus*, or the rhythmical alternate dilatation and contraction of the pupils, is sometimes seen in cerebral lesions.

Note also if the pupil appears black, or if there are changes in the transparency of the aqueous humor or lens (*cataract*).

The iris may be adherent to the cornea or to the lens (*anterior and posterior synechia*). Finally, as a congenital defect (*coloboma*) or as a result of iridectomy a portion may be absent at one part of the circumference.

VI. **The globe** should be inspected for abnormal prominence (*proptosis or exophthalmos*), such as we find in tumors of the orbit and exophthalmic goitre. *Recession or enophthalmos*, if present, should be noted. It is found after penetrating wounds of the globe and in paralysis of the cervical sympathetic nerves. We should also determine if the normal position of the ball in the orbit is altered, as we see in tumors of the globe or surrounding structures.

The *tension* is estimated by alternate pressure of the finger-tips on the upper surface of the globe as in obtaining fluctuation, two fingers only being used, the patient meanwhile looking downward. We designate plus tension as T_1, T_2, T_3. This estimation of course is only relative, the latter sign being applied

when no indentation can be made on the globe on account of excessive pressure in the vitreous. It is most characteristically seen in glaucoma. Minus pressure is frequently due to penetrating wounds.

VII. **The Ocular Muscles** (see page 251).

VIII. **Vision** (see page 247).

I. **THE LIPS.**

Examine the lips as to their color, prominence, presence of frothing, drooling, and tremors.

Herpes ("cold-sore") of the upper lip is frequently associated with pneumonia, and excoriations with irritating nasal discharges.

Ulcers must be carefully examined and described; they may be simple, malignant (cancer), or specific (syphilitic or luetic, being especially common as fissures at the angles). Evidences of loss of surface epithelium, as from burns or acid poisons, and discolorations also demand attention.

In "*mouth-breathers*" (adenoids and nasal obstruction, page 28) the upper lip is retracted, exposing the teeth to view. In *Bell's palsy* (seventh cranial or facial nerve) one angle of the mouth droops, while that of the healthy side is drawn away from the paralyzed side by the action of the muscles of the cheek (page 27). In tetanus and strychnin poisoning the jaws are tightly clenched and the lips in a state of spasm (see *Risus Sardonicus*, page 25).

The examination of the *oral cavity* is discussed in Chapter VIII.

J. **THE NECK**.—First note the "carriage" of the head, and test its movements and those of the neck muscles by instructing the patient to put these into

action. The presence of paralyses and spastic conditions (*torticollis* "wry-neck,"), and other spasmodic movements, either nodding or rotatory, are thus emphasized, and the movements of the cervical vertebræ, if painful or restricted, are at the same time investigated. *Retraction* of the head, as seen in strychnia poisoning, should also be sought for.

If a *tumor* is detected, its position, form, origin, consistence, mobility, pulsation, movement on swallowing, sensibility, and relation to the surrounding structures should be carefully determined.

The presence of enlarged *lymph glands* as found in tuberculosis, syphilis, leukemia, lymphadenoma and cancer, must also be excluded; particularly the enlargement of those sometimes found above the left clavicle in cases of gastric carcinoma, and those of the posterior triangles in pediculosis of the scalp.

Note, lastly, the occasional presence of a cervical rib, and refer for differential diagnosis of the various *cervical tumors* to works on surgery, where full descriptions will be found.

Turning next to the *skin*, note the presence of abnormalities as previously described (page 30), also the condition of the *superficial vessels*, especially as regards the presence of abnormal pulsations.

The phenomenon of "*tracheal tugging*," found in aneurysm of the aortic arch, is detected by the examiner standing behind the patient who is placed in the sitting posture with the head thrown well back. Grasp the cricoid cartilage gently between the forefingers of the two hands. A distinct downward movement of the larynx, synchronous with the apex beat, is found where this sign is present.

K. **THE EXTREMITIES.**—It is always well before completing our general inspection to examine the limbs in a superficial manner. We shall not here, however, describe lesions of the bones, joints, or congenital deformities.

I. **Circulation.**—As regards the hands and feet note signs of defective circulation, as evidenced by coldness, edema, cyanosis; also capillary pulsation if present, and the action of the sweat-glands, especially in the palms and soles. It is well to remember that cold, clammy extremities with excessive palmar perspiration are very common in neurasthenia.

II. **Nails.**—Examine these structures for atrophy, brittleness, transverse striæ, and alterations in contour. Severe illness often leaves its mark as a transverse marking or groove on the nails.

III. **Bones and Joints.**—As these investigations are being carried out the condition of the *joints* will have been observed, especially as regards anchylosis, swellings, deformities or crepitus; and also of the *bones* as regards evidences of rheumatic thickenings (*Haygarth's nodosities*) and syphilis. Examine also the *webs* of the fingers and toes for the burrows of scabies or of linear ulcerations (*rhagades*).

"*Clubbing*" *of the fingers*, which is frequently found in old-standing cases of cardiopulmonary disease, is recognized by a longitudinal curving of the nails which tend to overhang the finger-tips. The terminal phalanges are flattened and broader than normal, and other evidences of defective circulation, as cyanosis and coldness of the hands, are usually also present.

IV. **The Muscles.**—It is advisable at this point to test roughly the power of the muscles of the forearms

and hands by putting the principal groups into action and noting the results. Note particularly the condition of the interosseous muscles and the thenar and hypothenar eminences, especially if atrophy be present; or if fibrillary twitchings of the atrophic muscles occur, as seen in progressive muscular atrophy. Test also the power of writing. See also the *gait*, page 16.

For further details reference must be made to the chapter on the neuro-muscular system.

V. **Deformities.**—Certain nerve lesions produce characteristic deformities of the hands, such as the *main en griffe* ("claw-hand") of ulnar paralysis and the *wrist-* or *foot-drop* of multiple neuritis. The presence of *Dupuytren's contraction* must also be noted if present.

VI. **Tremors.**—The various forms of tremors of the hands will now be described. The more important are:

(1) *Athetosis*, or slow, irregular, deliberate, but purposeless movements of the fingers and hands may be seen, especially as sequelæ of infantile cerebral hemorrhage ("birth-palsy").

(2) A *volitional*, or *intention* tremor is seen most characteristically in disseminate sclerosis (see speech, page 245, and nystagmus, page 252) where a fine, static tremor is present, which is greatly exaggerated by any voluntary muscular action, as exemplified in raising a glass of water to the lips.

(3) In *exophthalmic goitre* a fine tremor of the finger-tips is usually found. It is best seen when holding the hands at arm's length with the fingers extended and separated, the vibrations occurring at the rate of about eight per second.

(4) In *paralysis agitans* a curious "pill-rolling" or

rotatory movement of the thumb and fingers of rather slow rhythm is found. It is pathognomonic of this disease (see also gait, page 17, and facies, page 27).

(5) *Toxic.*—This is best seen in the alcoholic. It closely resembles that found in the aged (*senile tremor*) and that of general paresis. A similar tremor is commonly found in the debilitated and in nervous individuals it may, however, be considerably coarser in character.

(6) *Choreic.*—Abrupt, irregular, involuntary contractions (increased by mental disturbances and diminished by voluntary muscular action) occurring in certain muscles or groups of muscles, and which may appear at times purposeful, are seen most typically in chorea ("St. Vitus' dance"), but may be closely simulated by those of hysteria. They may be unilateral, as in hemichorea.

CHAPTER V.

TOPOGRAPHICAL ANATOMY.

A. **REGIONS AND LINES.***—For convenience of description and to facilitate the projection of organs on the surface of the body, we arbitrarily divide it into certain regions or areas by means of *artificial lines*, both vertical and horizontal.

The lines used in examinations of the **thorax** are the *mid-sternal*, which passes vertically through the center of the sternum and the symphysis pubis, cutting the anterior surface of the trunk into two equal parts; the two *lateral sternal*, corresponding to the borders of this bone; the *mid-clavicular* (mammary), obtained by dropping vertical lines from the centers of the clavicles which, when projected downward bisect Poupart's (inguinal) ligaments and usually also pass through the nipples; and the *parasternal*, parallel to the last two lines and midway between them.

Passing around to the sides of the chest two others, the *anterior and posterior axillary*, are obtained by dropping lines vertically downward from the junction of the axillary folds with the chest wall.

* It has been the practice of the author and of the clinicians with whom he has been associated to spend several days instructing the students to outline the normal organs on healthy individuals with a dermographic pencil by means of the following methods, before proceeding to demonstrate their presence by physical examination; he has inserted this chapter dealing with topographic relations chiefly however as a review.

On the back three more lines are used, these are the *spinal or mid-dorsal*, dividing the posterior surface of the trunk into two equal parts; the others are the two *scapular* lines, which pass vertically downward through the angles of the scapulæ, parallel to the vertebral spines.

The following horizontal lines are utilized: the uppermost, or the *clavicular* line, corresponding to the prominence of the clavicles; *and two others* which pass around the chest at the levels of the junction of the third and sixth costal cartilages with the sternum to meet the anterior axillary lines.

It will now be seen that the anterior surface of the thorax is thus mapped out into certain definite **areas.** In the center line in front we recognize the *suprasternal notch* (jugular fossa) and below this the *superior and inferior sternal* regions, lying above and below the level of the third rib, in relation to the sternum. That portion of the neck on each side lying above the clavicles is designated the *supraclavicular* region, while below the bone and above the level of the third rib lies the *infraclavicular* region or fossa. The area lying between the third and sixth ribs, the lateral sternal and anterior axillary lines, is termed *mammary*, while immediately below this lies the *inframammary* zone.

On the lateral aspect of the trunk the *axillary* regions lie between the two axillary lines.

On the dorsum of the trunk the regions lying between the vertebral borders of the scapulæ and the mid-spinal line are termed the *interscapular*. The *supra- and infraspinous* areas lie above and below the spines of the corresponding scapulæ; and, finally, below the level of the lower angle of that bone lies the *infrascapular region.*

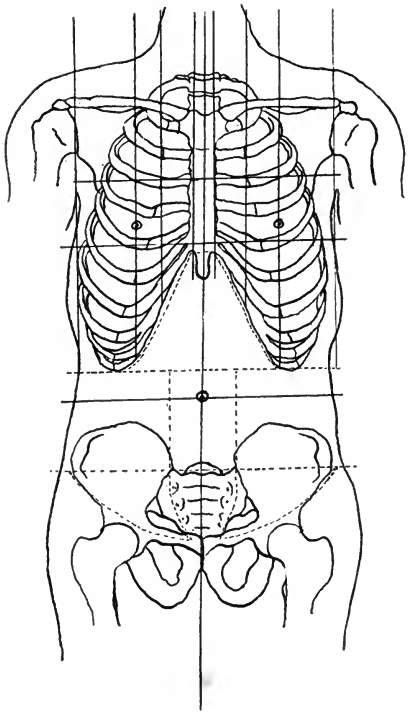


FIG. 5.—Lines and regions on the anterior surface of the trunk.

Passing to **the abdomen** two methods are employed; the simpler being to divide its surface into *quadrants* by means of two lines, vertical and horizontal, which cross each other at the umbilicus.

The other method is as follows: Two horizontal lines are taken, one cutting the lowest borders of the tenth ribs and the other joining the two anterior iliac spines; also two vertical lines which correspond to the outer margins of the recti until they meet the upper horizontal line, and then follow the borders of the rib cartilages to the sternum.

By this means the surface is divided into *seven regions*. In the center from above downward they are termed the epigastric, umbilical, and pubic (hypogastric). On either side are the lateral abdominal (lumbar) and inguinal (iliac), also from above downward. Above the two lateral abdominal lie the two hypogastric zones (Sobotta's atlas).

The **umbilicus** is situated opposite the disc between the third and fourth lumbar vertebræ, and is found to be 1 inch nearer the symphysis pubis than the base of the xiphoid in the healthy adult.

Opposite the junction of the second costal cartilage with the sternum, there may be felt on the latter bone a transverse ridge which is termed the **"angle of Louis."** This lies on the level of the disc between the fourth and fifth thoracic vertebræ and is of the utmost importance as a landmark, as from it we are enabled to obtain a reliable guide in counting the ribs.

The ribs may also be counted in front from above; as the first rib usually lies behind the clavicle its lower margin only may be palpated, so that the groove immediately below that bone corresponds to the first

intercostal space; or they may be counted from below and behind, commencing with the twelfth rib. It should be noted here that the ribs behind correspond in number to those of the vertebral spines lying next above, with the exception of the first rib which lies opposite the vertebra prominens; thus, for example, the seventh thoracic spine lies on the same level as the eighth rib.

In the male the *nipple* is usually opposite the fourth interspace, while on the posterior surface the *scapula* covers a region reaching from the second to the seventh ribs.

B. **ORGANS.**

I. **Respiratory System.**

(1) **The Trachea.**—This commences above at the lower margin of the cricoid cartilage, descends in the middle line of the neck, passes behind the sternum, and bifurcates opposite the disc between the fourth and fifth thoracic vertebræ; in other words, behind the "angle of Louis" where it is crossed by the arch of the aorta (see "tracheal tugging," page 45). At this point the two bronchi originate and pass downward and outward toward the roots of the lungs.

(2) **The Lungs.**—Commencing at the *apices*, which lie in the supraclavicular regions on the side of the neck and reach to a point 1 1/4 to 1 3/4 inches above the clavicles (that of the right lung normally rising to a slightly higher level than its fellow), the *outer margins* curve downward and outward, passing behind the junction of the inner and middle thirds of the clavicles.

The *anterior borders* of the lungs curve downward, forward, and inward, passing behind the sterno-

clavicular articulations toward the middle line, the two coming almost in contact behind the "angle of Louis." From this point the surface markings differ somewhat on the two sides. On the *right side* the anterior margin is continued downward, slightly to the right of the mid-sternal line, to the level of the junction of the sixth costal cartilage with the sternum, at which point it curves sharply outward, cutting the mid-clavicular line at the level of the sixth, the mid-axillary at the eighth, and the scapular line opposite the tenth rib, reaching the side of the spinal column about the level of the eleventh thoracic vertebra. The anterior margin of the *left lung* corresponds to that of its fellow from the apex to the level fourth costal cartilage, at which point it curves gently outward in an arched manner to cross the parasternal line at the level of the fifth and the mid-clavicular at that of the sixth rib. The remainder of the outlines are identical with those already given for the right lung, except that on the left side the lower edge is usually found to descend about 1/2 inch lower than that of its fellow.

These surface markings have been obtained during quiet breathing. The downward excursion of the lower borders of the lungs in ordinary respiration seldom amounts to more than 1/2 inch, but during a deep inspiratory act these lower margins may be found on percussion to have descended from 1 to 1 1/2 inches into the pleural sinuses.

The lobes of the lungs may be delineated by lines drawn around the thorax from the second thoracic spine to the level of the sixth ribs in the mid-clavicular lines. These correspond to the clefts between

the upper and lower lobes. A second line on the right side, commencing from the center of that above described, and passing forward to the level of the junction of the fourth costal cartilage with the sterum, marks the upper limit of the triangular middle lobe.

(3) **The Pleuræ.**—The reflections of the pleural sacs are as follows: They closely *cover the apices* of the lungs, but at the level of the "angle of Louis" their *anterior margins* slightly overlap behind the mid-sternal line, from which point their surface markings are somewhat dissimilar.

On the *right side* the anterior edge passes downward in relation to the mid-sternal line to the level of the eighth cartilage, curves outward cutting the mid-axillary at the tenth rib, the scapular line at the tip of the twelfth rib, and finally reaches the side of the body of the twelfth thoracic vertebra, overstepping the lower margin of the lung at these points by 2, 4, and 1 1/2 inches respectively.

On the *left side* the anterior margin passes downward behind the center of the sternum as far as the level of the fourth rib, where it curves outward, cutting the parasternal line at the fourth and midclavicular at the eighth rib, while the remainder of the lower margin corresponds to that of its fellow of the opposite side.

It will now be seen that to the left of the sternum, below the level of the fourth costal cartilage, a small triangular area of chest wall is uncovered by both lung and pleura, here we find later that a portion of the wall of the right ventricle of the heart lies in close apposition to the ribs and intercostal spaces, the pericardium alone intervening (area of "superficial"

or "absolute cardiac dullness," (page 157). This region corresponds to the *incisura cardiaca.*

II. **Circulatory System.**

(1) **Heart.**—An oblique line crossing the sternum from the lower border of the second left costal cartilage commencing 1/2 inch outside the lateral sternal line, to the upper border of the third right cartilage, terminating 3/4 inch beyond the lateral sternal line will correspond to the *base of the heart.* This part of the heart is composed of the right atrium (auricle), conus arteriosus, and the tip of the left atrium, from right to left.

The *right margin* is indicated by a curved line with its convexity to the right, passing from the right extremity of the "base-line" to the sixth right chondrosternal junction. This line corresponds to the outer border of the right atrium.

The *lower border* of the heart is shown by a line from the sixth right chondrosternal junction to the "apex beat" (a point 1/2 inch internal to the left mid-clavicular line in the fifth intercostal space, which in the average adult will be found to be situated 3 1/2 inches from the midsternal line). This border is formed by the outline of the right ventricle, the left ventricle entering into its composition for the last half inch of its length at its left extremity.

The *left margin* is shown by a line, with a strong convexity outward, drawn from the termination of the "base-line" on the left side to the apex beat.

That area bounded by the above lines is termed the *precordia.*

The position of the *chambers of the heart* may be projected on the chest wall as follows: The right atrium

occupies a triangular area bounded by the right border of the heart on the right, the "base-line" above, and a line from the third left to the sixth right chondrosternal junctions; of the left atrium the appendix only is in direct contact with the anterior chest wall, it lies behind the upper part of the third left costal cartilage. The right ventricle occupies the whole of the remaining area of the precordia, with the exception of a strip

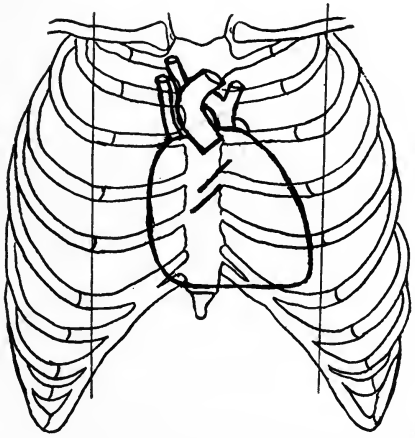


FIG. 6.—Topographic markings of the heart, valves and great vessels.

1/2 inch in width in immediate relation to the left margin of the heart, which corresponds to the left ventricle.

(2.) **Great Vessels and Valves.**—The situation of the *aortic valve* is shown on the chest by a line, 3/4 inch in length, passing obliquely downward and inward from the center of the third left chondrosternal junction to the midsternal line.

The *aorta* arises here and ascends immediately to

the right of the mid-sternal line, its right margin touching the tip of the second right costal cartilage. It then curves upward and inward toward the left, the highest point of the arch reaching the center of the manubrium. From here it curves backward and downward in the posterior mediastinum, where it lies between the pulmonary artery in the front and the bifurcation of the trachea behind. From its upper border spring the innominate artery opposite the second right chondrosternal junction, and from its highest point the carotid and subclavian arteries; while in immediate relation to its upper margin lies the left innominate vein.

The *pulmonary valve* corresponds to a horizontal line 3/4 inch in length, lying at the level of the upper edge of the third left costal cartilage, the left lateral sternal line passing through its center. This line is parallel to that given for the aortic valve.

From this point the *pulmonary artery* arises, the left lateral sternal line bisecting it longitudinally, so that one-half lies behind the sternum, leaving the remainder in relation to the innermost portion of the second left costal cartilage, the right branch passing beneath the arch of the aorta and the left backward toward the root of the corresponding lung.

The *mitral orifice* is shown by a line which passes downward and toward the right, from the center of the third left intercostal space, commencing 1/3 inch to the left of the sternal margin, to a point behind the sternum 1/3 inch from its left border and directly opposite the middle of the fourth left interspace.

The *tricuspid valve* lies altogether behind the sternum and is indicated by a line from the lowest point of the fourth left chondrosternal junction, passing obliquely

downward and toward the right, to a point opposite the upper edge of the fifth right chondrosternal junction, 1/3 inch to the left of the right lateral sternal line. It is situated to the right and in a plane anterior to that of the mitral valve.

Lastly it is well to note that the *abdominal aorta* bifurcates 1/2 inch below and to the left of the umbilicus, from which point the common iliac arteries extend to the centers of Poupart's iliac ligaments, and to remember that the celiac artery arises from the aorta half way between the umbilicus and the base of the xiphoid.

III. **Digestive.**

(1) **The Stomach.**—The *cardiac orifice* lies behind the seventh left costal cartilage, 1 inch from the sternum; the *pylorus* 1 inch to the right of the middle line, half-way between the end of the sternum and the umbilicus. The latter surface-marking is only approximate as the pylorus is not a fixed structure.

The *lesser curvature* lies deeply in the epigastric region; a slightly curved line, with its concavity directed to the right, joining the two orifices above described, roughly indicates its situation.

The *greater curvature and fundus* are shown by an arched line with its convexity upward drawn from the cardiac orifice to the level of the fifth rib in the midclavicular line; from here it curves downward and toward the left as far as the anterior axillary line, thence with its convexity downward to terminate at the pylorus. In health the lower border should not lie below the level of a horizontal line passing through the umbilicus.

(2) **The Colon and Appendix.**—The *ileocecal valve*

lies beneath the outer edge of the right rectus muscle, where it is cut by a line from the umbilicus to the anterior superior iliac spine. The *base of the appendix* lies 1 inch directly below this point.

The *ascending colon* is found chiefly in the right iliac and lumbar regions; the *hepatic flexure* beneath the ninth and tenth right costal arches; and the *transverse colon* usually in the upper half of the umbilical region, but the position of this colon may vary considerably. The *splenic flexure* lies behind the stomach in the left hypochondriac and the *iliac colon* in the left iliac and hypogastric regions.

(3) **The Liver.**—The outermost *angle of the left lobe* lies just below the "apex beat" in direct contact with the lower border of the heart, the diaphragm alone intervening.

The *upper margin* crosses the sixth right chondro-sternal junction, from which point it curves to the right, with its convexity upward, to cut the mid-clavicular line at the level of the fourth, the mid-axillary at the seventh, the scapular line at the ninth intercostal space, to reach finally the spinal column at the level of the ninth thoracic vertebra.

The *lower border* commences just below the "apex beat," which point marks the outermost angle of the left lobe, passes downward and inward to the tip of the eighth left costal cartilage, obliquely across the epigastric region to the ninth right costal cartilage (under cover of which lies the *gall-bladder*), thence along the costal margin to cut the mid-axillary line in the tenth interspace, and scapular line opposite the ninth rib, finally reaching the side of the body of the eleventh thoracic vertebra.

(4) **The Spleen** lies obliquely behind the ninth, tenth, and eleventh ribs, with its long axis corresponding to that of the tenth. Its anterior margin is found as far forward as the mid-axillary line, while its posterior edge reaches to within 1 1/2 inches of the ninth thoracic spine.

(5) **The Pancreas.**—Crosses the vertebral column opposite the first and second lumbar vertebræ, one-third lying to the right of the middle line of the body and the remainder to the left. The level of the gland on the anterior surface of the abdomen corresponds to a point mid-way between the base of the xiphoid and the umbilicus. Its head is in close relationship to the duodenum into which its duct opens.

IV. **The Kidneys.**—*On the anterior surface of the abdomen* the upper ends lie about 2 inches from the middle line, the lower about 3 inches. The lower edge of the right organ lies fully 1 inch above a horizontal line drawn through the umbilicus, and that of the left about 1 inch higher. The hilum of each is about 2 1/2 inches from the middle line on a level with the first lumbar spine, their upper edges reaching to about the level of the tip of the xiphoid.

On the posterior surface of the trunk, one-third of the left and one-sixth of the right kidney lie behind the twelfth ribs. The upper end of the left is on a level with the twelfth thoracic spine and the lower 2 inches above the iliac crest; the right organ lies about 1/2 inch lower than its fellow.

CHAPTER VI.

RESPIRATORY SYSTEM.

A. INSPECTION.

I. **THE UPPER AIR PASSAGES.**—Since we have already examined the external parts of the **nose** and referred the reader to special works on rhinoscopy for details of the examination of the nasal cavities, we may turn our attention to the **nasopharynx,** which in addition to being accessible to direct examination by means of mirrors and reflected light, may also be explored in children by the palpating finger introduced through the mouth. This method is frequently made use of in the detection of nasopharyngeal growths (adenoids).

The **oropharynx** may be fully exposed to view on widely opening the mouth, holding the tongue meanwhile close to its floor by means of a "tongue depressor." Evidences of inflammation (pharyngitis) or the presence of a tumor may be detected. In the latter event its origin, extent, and consistence must be carefully determined, especially since this is a common situation for the occurrence of retropharyngeal abscesses due to spinal caries (Pott's disease).

The **laryngopharynx** and the **larynx** require the use of a laryngoscope for their illumination, but the epiglottis may be felt through the mouth by the examining finger; this latter method is of the greatest practical value in detecting edema of that structure, a not uncommon cause of acute and frequently fatal dyspnea

in nephritis and laryngitis. For further descriptions of these organs special works on laryngology should be consulted.

The interior of the **trachea** and its bifurcation may also be seen by the aid of the laryngoscope, and in individuals with thin necks this tube may be subjected to external palpation, as also may the cartilages of the larynx and the hyoid bone. The phenomenon of "tracheal tugging" has already been described (page 45). Its interior may be rendered visible by means of Killian's *bronchoscope*, the commencement of the larger bronchi may also be seen; its application, however, is extremely difficult.

II. **THE THORAX.**

(1) **Introduction.**—It is impossible to overestimate the importance of the information to be derived from a careful and accurate inspection of the thorax, but for the results obtained to be of practical value, one must be thoroughly familiar with the various types of normal thoraces, and since in no two healthy individuals even are there to be found chests of precisely similar mould, the student is urged to neglect no opportunity offered him for their inspection.

Technic.—For satisfactory work the chest must be fully uncovered and the patient placed in a good light, which should fall over the examiner's shoulder. The upright, sitting or lying posture may be assumed.

The front of the thorax should first be inspected, with the examiner in a position directly in front of his subject, next it should be examined from both sides, and then the contour of the neck and the outlines of the chest as seen from below (with the patient in the supine posture), and also from above must receive

careful attention. In the latter instance the examiner stands behind the patient's chair or at the head of the bed, depending on whether the subject is sitting or lying on his back, and from this point observes the contour of the neck, prominences of the clavicles, and outlines of the chest wall in the vicinity of the level of the nipples, which are thus successively brought into view from above downward.

In this section it is our duty to lay stress on the results of our findings in regard to the general condition of the thoracic wall. The reader is referred to page 30 for a description of the inspection of the skin, and to page 125 for details of the special examination of the precordia, thus a certain amount of reiteration may be avoided.

In the inspection of the chest the "key-note" of success lies in the ability to **accurately and intelligently compare** not only the general outlines of the two sides, but in the comparison of **each part under examination with the corresponding area of the opposite side.**

Allowance must be made, however, for apparent skeletal asymmetry such as occurs for instance where the pectoral muscles of one side show excessive development, and for effect of the presence of the mammary glands on the thoracic outlines of the female; also the fact that it is *rare to find even a healthy thorax whose contours are perfectly symmetrical* must not be overlooked.

The general shape of the wall of the thoracic cavity having been determined, it is necessary to examine its constituent parts in detail in order that our observations may be of practical value. As regards *the ribs*, certain abnormalities may be detected, among these should be

noted enlargements of the sternal extremities ("beading" or "rachitic rosary," page 70), and excessive obliquity; also deviations from the normal of the substernal angle. The *intercostal spaces* may exhibit retraction or bulging, or the vertebral borders of the scapulæ may not be closely applied to the chest wall and hence show excessive prominence ("*winged scapulæ*").

Lastly, it is essential to carefully eliminate the possibility of any thoracic asymmetry being congenital, the result of trauma or due to the presence of spinal deformity.

(2) **The Normal Thorax.**—It is necessary before proceeding further to have a "mental picture" of an average normal chest before one, for purposes of comparison with those showing evidences of disease.

The following description applies to an **ideal healthy thorax.** It should be somewhat conical in form, elliptical in cross-section (except in infants whose chests are almost circular) with a transverse diameter in proportion to the antero-posterior as 7 is to 5 (Fig. 7), and a circumference at the level of the nipple of 34 to 36 inches in the adult male.

The shoulders, clavicles, and ribs should be symmetrical and the sternum and vertebral column should lie accurately in the mid-lines, after making allowance for the slight scoliosis which is normally present. The angles of the ribs with the vertebræ and with their costal cartilages should be equal on the two sides, and the subcostal angle be found to vary from 70 degrees in the male to 75 degrees in the female.

The contour of the chest should be rounded in its outline, and the ribs visible only below the level of the pectoral muscles. No deep hollows should destroy

its smooth outlines, with the exception of the slight recessions found normally just below the clavicles, and the faint central furrow, which corresponds to the position of the sternum.

Normally the precordia shows a very slight fullness due to the presence of the heart, similarly over the liver a "rounded moulding" of the chest wall is usually demonstrable.

The sternal furrow ends below in a depression termed the scorbiculus cordis; two slight secondary hollows may also be detected in the infraclavicular regions, one corresponding to the separation between the two divisions of the pectoralis major, and the other, which is the more distinct (*Morenheim's fossa*), to the interval between that muscle and the deltoid.

As regards the mammary gland, its development varies to such an extent in the female as to render descriptions useless, but in the adult male the nipple will usually be found on a level with the fourth interspace in the mid-clavicular (mammary) line.

Lastly, it is well also to remember that even in the normal adult the capacity of the right half of the thorax usually slightly exceeds that of its fellow, as may be demonstrated by means of the cyrtometer (page 118).

The **movements** of the thorax in health require description at this stage. The respiratory act is normally performed from sixteen to eighteen times per minute in the adult, a barely perceptible interval occurring between inspiration and expiration, of which acts the former occupies a slightly shorter period of time. In the new-born the number of respirations vary from 40 to 45 per minute, dropping to about

30 at the end of twelve months, and falling to 20 by the fifteenth.

In regard to the movements of the thorax, not only the rate, rhythm, and the general "type" of respiratory movement must be noticed, but a **close comparison of the amount of the excursion on the two sides of the chest in corresponding areas** must be carefully made.

The term **type of respiration** is used to describe the various manners in which the respiratory excursions of the thorax may take place. Thus, in certain individual cases little movement of the thoracic wall may occur, the movements of respiration being performed chiefly by the diaphragm (*abdominal type*), while in others the opposite is found. In the latter case there is very slight movement of the abdominal parietes, the intercostal muscles being chiefly responsible for the respiratory excursion (*thoracic*). In the adult male the diaphragm performs the greater share (*abdomino-thoracic*), while in females the reverse is true, resulting in the *thoracico-abdominal* type of respiration. (Page 74.)

Finally, in the normal adult the thorax is deepened, widened, and elevated by the contraction of the diaphragm, divergence of the lower ribs, raising of the clavicles and sternum, and convergence of the upper ribs. In health all parts of the chest dilate and contract symmetrically, with a regular rhythm, and convey to the observer the impression that the expansion is due to a "vital elasticity" and not to a mere mechanical increase in capacity as seen in emphysema.

(3) **Symmetrical Thoracic Abnormalities.**

(a) **Phthinoid Chests.**—This term is applied to certain types of thoraces which are found in individuals inheriting a proclivity to pulmonary tuberculosis. It

must be thoroughly understood that the anomalies described below are not due to past or present disease of the lungs, but are *merely indicative of a predisposition* on the part of the subject to tuberculosis. The results of lesions of the lungs on the external contour of the thoracic wall will be described in separate sections.

There are two types of chests which belong to this class; the first is the "alar," the second the "flat chest."

The *alar chest* is long and narrow with a shallow cavity, the ribs small and abnormally oblique, the vertebral borders of the scapulæ are not closely applied to the thoracic wall and hence show abnormal prominence ("winged scapulæ"), the shoulders narrow and sloping, the neck long and thin, the clavicles prominent, and the subcostal angle excessively acute.

The *flat chest* is usually associated with that just described; in it the costal cartilages have lost their normal convexity forward and became straightened, allowing the sternum to approach the spinal column and thus to materially diminish the antero-posterior diameter of the chest. No distinct line of demarcation can be drawn between these two types; the above descriptions are given separately simply for the sake of convenience, it being understood that intermediate types occur with features common to both.

These two varieties are found in individuals who have inherited a poor muscular system, or in those in whom, through illness or neglect, developement of the muscles has been interfered with. They have been termed "chests of full expiration."

(b) **Emphysematous chests** are found in subjects who suffer from pulmonary emphysema, the appearance produced being due to an effort on the part of nature

to increase the volume of the thoracic cavity in order to accommodate the large lungs, the thorax appearing in an exaggerated form as it does at the end of a deep inspiration. The shoulders are high and raised, the neck is short and thick, the sternum arched forward, the thoracic spine shows abnormal backward convexity, while the ribs are broad and set in a more horizontal plane than normal.

The total result of these changes is that the antero-posterior diameter of the chest is markedly increased, approximating that of the transverse, thus producing a "*barrel-shaped*" thorax whose capacity is greatly augmented, especially in its upper part, and whose cross-section approaches that of a square with rounded angles (Fig. 7).

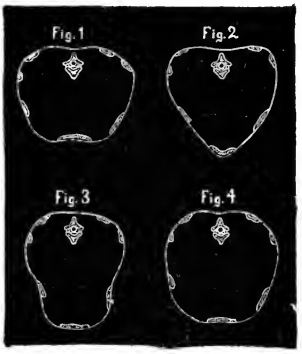


FIG. 7.—Cyrtometric tracings. (*Greene.*) 1. Normal chest. 2. Pigeon breast. 3. Rickets. 4. Emphysema.

In certain cases of emphysema, especially in those originating during the middle decades of life before ossification of the cartilages is complete, a slight, vertical localized bulging lying immediately to the right of, and parallel to the sternum, and having a width of about 1 1/2 inches, may often be detected. The costal cartilages here have been forced forward by the enlargement of the right lung, the presence of the heart on the left side being responsible for its unilateral occurrence.

It will be found also on inspection that the emphysematous chest appears rigid, very little real expansion

taking place during quiet respiration, although the thorax as a whole rises "*en masse*" at each inspiration, the type of breathing being abdominal.

It is well also to note that this form of chest may be closely simulated by alterations in the thorax resulting from kyphosis (Pott's disease).

(c) The **rachitic** type refers to that form of chest found in those who during infancy have suffered from rickets, a disease whose most prominent feature is an abnormal softness of the bones. These are in consequence more pliable than normal, and hence yield readily to pressure especially at their junctions with the cartilages, for it is here that the disease is most active. At these points there are frequently found rows of nodules, occasionally as large as small marbles, which are described by the terms "*beading*" *or rachitic rosary*, when, however, there is marked thoracic deformity they may be demonstrable only on the inner surface of the chest wall.

Vertical grooves along the line of the junction of the cartilages and ribs (Fig. 7), and the *rachitic girdle*, a transverse depression about 2 inches in width extending across the chest just above its lower margin corresponding roughly to that of the attachment of the diaphragm (*Harrison's sulcus*), may also be found.

These deformities are most marked in cases where obstruction to the entrance of air into the lungs has existed (adenoids, enlarged tonsils, pertussis, etc.), the result of which is to produce a chest whose antero-posterior diameter is increased in proportion to that of the transverse (Fig. 7).

In nearly half the cases also a posterior spinal curve (kyphosis) coexists; other evidences of rickets,

as shown in the calvarium and in curvatures of the long bones (knock-knees and bow-legs), will confirm the diagnosis.

A typical *pigeon-breast* may at times be seen. In it the ribs have become straightened in front of their angles, the sternum in consequence being projected forward as a ridge and the cross-section of the cavity assuming a triangular form. It is doubtful, however, if rickets is in this case the sole etiological factor (Fig. 7).

(2) **Unilateral abnormalities** consist in enlargement or diminution of the volume of the whole of one side of the chest. The more localized alterations of contour will be described under Section 3.

(a) **Unilateral enlargement** or bulging of one-half of the thorax may be due to the presence of a large amount of fluid or gas in the pleural cavity, to the existence of a tumor, a spinal curvature, or of compensatory or vicarious hypertrophy (emphysema) of one lung following diminution in the functional activity of the opposite organ.

In the case of fluid *effusions* this rule does not always hold good, in fact except in the more chronic forms the diseased side is usually the smaller of the two, the explanation of this, however, is not altogether clear. The increase in size of the side is regular, the general rounded contour of the affected half being retained and the heart is found to be displaced toward the opposite side. In the case of compensatory emphysema the boundaries of the lung extend beyond their normal limits.

(b) **Unilateral shrinking** or retraction depends on a diminution in the volume of the thoracic contents

and may result from shrinking of the lung from inflammatory action (fibroid phthsis), from atelectasis or collapse secondary to bronchial obstruction, or be due to non-expansion after pleural inflammation or absorption of a pleural exudate. Not infrequently the presence of fibrous adhesions obliterating the sac act as an additional factor in preventing expansion.

In this condition the intercostal spaces are abnormally hollowed and the whole side is shrunken. The heart will be found to be displaced to a greater or lesser degree toward the side of the lesion, and the opposite lung to be in a condition of compensatory hypertrophy (emphysema).

The presence of scoliosis must be carefully eliminated, since it produces an apparent flattening of one side in front, with a corresponding dorsal bulging.

(5) **Local changes in contour** are due to abnormal conditions of the thoracic wall, pleuræ, lungs or thoracic aorta; occasionally also an enlarged liver, kidney, or spleen may show itself by prominence of the overlying ribs.

Alterations of contour in the precordial region due to cardiac disturbances will be considered in the section on the circulatory system.

(a) **Localized bulgings** may be due to the presence of a neoplasm or abscess of the chest wall, tumor of the lung, or to an aneurysm of the aorta. Occasionally intercostal tumescence corresponding to a localized purulent pleural exudate may be present, especially if the abscess be about to "point" externally (empyema necessatatis). In rare cases the fullness may be due to the presence of air in the subcutaneous tissues, resulting usually from tearing of the underlying lung

and pleura by a fractured rib (surgical emphysema, page 34).

(b) **Localized Retraction.**—Flattening or shrinking of the chest wall occurs over any portion of lung which has been contracted by disease (pulmonary tuberculosis, etc.), this being specially noticeable where the process has been limited to the apices, as shown by an accentuation of the supra- and infra-clavicular fossæ. Retraction may also be a result of pleural adhesions, particularly such as occur after absorption of a localized empyema.

Over other regions of the chest the ribs in the affected area appear unduly prominent and the intercostal space sunken; not only as a result of the falling in of the chest wall, but also in consequence of muscular atrophy and loss of the fatty subcutaneous tissues which overlie the lesion.

Let me here emphasize the fact that frequently local abnormalities may be overlooked if the examiner only inspect the surface with the axis of his line of vision falling vertically on the area under consideration. In this case no *shadows* will be seen, whereas, if either the rays of light or the axis of the clinician's line of vision, be allowed to fall obliquely on the chest wall, shadows will become visible, and it is from their situation and movements that we derive our most valuable information.

(6) **Abnormalities of movement,** either in extent or rhythm, now require description.

(a) **Expansion.**—Departures from the normal may be seen affecting the chest *bilaterally* as, for instance, in *emphysema*, in which the thorax appears rigid and during inspiration to be lifted up as a whole;

although the movement is marked, little real expansion or increase in capacity takes place.

During attacks of *dyspnea* ("air-hunger") from any cause the respiratory excursions are excessive.

In bronchial *asthma*, however, the distress is most marked during expiration, the patient owing to difficulty in expelling the air from his lungs bringing his accessory muscles of expiration into violent action.

The "*thoracic type*" of respiration is marked in abdominal infections (peritonitis), tumors and ascites, and in paralysis of the diaphragm; and the "*abdominal*" where movements of the thorax are painful (pleurisy, pleurodynia, etc.) or restricted, as when the intercostal muscles are paralyzed.

Local deficiency in expansion occurs over areas showing evidences of retraction (page 73) and over the affected lobe or lobes in pneumonia. These areas, in addition to showing defective expansion, may also in time "lag" behind the corresponding regions of the healthy side.

Not only may the expansion be poor, but in marked cases local *inspiratory indrawing* of the thoracic wall may occur, especially when pleural adhesions are present. Generalized indrawing usually accompanies laryngeal obstruction, the intercostal spaces, lower end of the sternum and epigastrium all being involved in the process.

(b) **Frequency.**—Variations from the normal may occur, for instance in opium poisoning or with cerebral compression, where the rate per minute may be greatly lowered.

The opposite condition (*dyspnea*) occurs in various conditions, such as in defective aeration of respiratory

or circulatory origin (pneumonia, pleurisy, valvular lesions of the heart, anemias, etc.), in certain toxemias, (nephritis, diabetes, etc.), and after excessive exercise.

It is of great importance not only to time the respirations but to note the *ratio of their frequency to that of the pulse rate.* Normally, this should be as one to four. If, the respirations in febrile states become increased out of all proportion to the pulse rate, some complication of respiratory origin should be sought for.

(c) **Rhythm.**—First as regards the relation of inspiration to expiration, which in the healthy individual are practically of equal duration, we note that, in laryngeal obstruction especially, the *inspiration may show marked prolongation*, and is then usually accompanied by a loud harsh laryngeal murmur due to spasm of the glottis, and actively vibrating vocal cords ("stridor").

The *expiratory phase is prolonged* and more intense in diseases such as pulmonary consolidation and emphysema, where the prolongation is indirectly due to a sluggish recoil of the lung resulting from loss of its normal elasticity.

"*Jerky*" or "*cog-wheel*" *respiration* may be seen even in normal individuals if the act be influenced by consciousness (as when the patient realizes that he is under observation) and in healthy children. In pulmonary tuberculosis, a disease in which expansion of the affected area may occur in an arrhythmic or jerky manner, this phenomenon is frequent.

Cheyne-Stokes breathing consists in "the occurrence of a series of inspirations, increasing to a maximum and then declining in force and length until a state of apparent apnea is established. In this condition the patient may remain for such a length of time as to

make his attendants believe that he is dead, when a low inspiration followed by one more decided marks the commencement of a new ascending and then descending series of inspirations" (Stokes). It occurs in uremia, cardio-pulmonary and cerebral diseases in its most pronounced form, and is then often an indication of impending dissolution.

(d) *Litten's sign* refers to a narrow wavy thoracic *phrenic shadow*, seen to move down the axillary regions during inspiration, from the sixth rib or seventh space to as low as the level of the tenth rib. In normal chests its excursion covers about 2 1/2 inches, and in a deep inspiration about 1 inch more. An expiratory wave or rising shadow may be seen, but it is less distinct than the former.

At the commencement of inspiration the diaphragm lies closely applied to the thoracic walls, and as it descends becomes separated or "peeled off" from the internal surface of the chest in order to provide room for the expanding lung. The presence of this "wave-like" retraction of the intercostal spaces is probably due to the external or atmospheric pressure exceeding the intrathoracic at the beginning of inspiration, the pressures becoming equalized on complete expansion of the lung.

For its demonstration the subject should be supine with the feet pointing toward a light, and the examiner should stand several feet from the patient's side with his back to the light and should view the thorax diagonally.

Bilateral diminution in the shadow may point to a phthisical predisposition, moderate emphysema, the presence of a large abdominal tumor or ascites.

A *unilateral decrease* may indicate a small pleural effusion, an upward displacement of the liver, or subphrenic abscess. In effusions it is usually indistinct or absent, while in the latter instances its presence may not be interfered with.

B. **PALPATION.**

I. **INTRODUCTION.**—Palpation is the term we apply to the procedure used in bringing into play the *sense of touch* during the examination of any accessible portion of the body. For this purpose the hands are applied to the part under consideration and the presence or absence of certain signs may thus be determined, as for instance the shape of the chest, its movements, and the presence of adventitious phenomena such as tenderness or palpable vibrations.

It is found advisable to vary **the technic** used, depending on what signs we expect to elicit, and as the perception of touch is seldom equally developed in both hands it is always wise to use the same one when comparing the findings obtained in different areas.

In comparing the *contour* of the two halves of the thorax and when estimating the respiratory excursion it is usual to apply lightly but firmly the whole hand to the chest wall; during the examination of localized areas and over the apices of the lungs the palmar surfaces of the fingers are used. In the exploration of the intercostal spaces, however, the use of the tips of the fingers alone will be found to give the most accurate results.

In estimating the *expansion* of the thorax, especially that of its lower half, the two hands of the examiner should lightly grasp the chest with the thumbs touching

at the mid-sternal line, the fingers reaching as far around the sides of the chest as possible. It will be found normally that during inspiration the two thumbs will depart to an equal distance from the middle line; if defective movement, however, is present on one side, the corresponding thumb will exhibit less movement than its fellow.

Let me here emphasize the point that we must *never rely solely on the results obtained by any one method of examination.* Each one of the procedures used is of value usually only in the elicitation of certain facts, and it is not until we have estimated the reliability and correlated the various observations made that we are justified in arriving at a definite diagnosis.

Our first duty in the application of palpation is to *check the results obtained by inspection* of the thorax; thus its general contour, the movements, and the presence of abnormalities such as bulgings or retractions of the chest wall, must be confirmed. Since all of these points have been fully dealt with in the section on inspection of the thorax, no further description will be required; suffice it to say, however, that these two methods should always be used in conjunction, and that wherever possible mensuration should also be employed.

Having thus confirmed or modified our previous observations, we turn next to the consideration of certain signs which can be elicited only by means of palpation.

II. **VOCAL FREMITUS.**

(1) **Normal Fremitus.**—The term vocal fremitus is used to describe certain *vibrations which are felt over the lungs during the production of the voice.* To

elicit these the patient is directed usually to repeat the words "one, two, three" or "ninety-nine" slowly and in a deep tone, the examiner's hand meanwhile being applied to the surface of the chest.

These palpable vibrations are **primarily produced at the vocal cords** and after modification in the oral cavity are carried downward by the air column through the trachea and bronchi, thence by vibration of the lung tissue itself and of the chest wall to the hand. Any condition, therefore, which interferes with their passage will diminish or totally abolish vocal fremitus.

It is first necessary, however, to familiarize oneself with the *extent to which these vibrations should normally be felt in different areas of the healthy chest.* They are usually most marked over the lower part of the infraclavicular regions (where the bronchi are largest and nearest to the surface) and diminish in strength as the distance from the larynx increases. The effect of the muscles and subcutaneous fat in damping the fremitus (as, for instance, over the pectoral and scapular muscles) must be considered, as also must the fact that *normally fremitus is slightly greater on the right side* owing to the presence of the short wide right bronchus, the fact that it lies more nearly in the direct line of the trachea than its fellow, and that this lung is the larger of the two and in consequence allows of more numerous and freer vibrations.

We must always remember that *since the phenomenon of vocal fremitus is primarily due to the sound waves which occur during the production of the spoken voice, these vibrations will be accentuated in proportion as its depth of pitch and intensity increases.* So that in fe-

males, children, and those whose voices are high and thin we do not expect to find the phenomenon present in such a marked degree as in adult males whose tones are deep. The reason for this being that the "wave-lengths" of the vibrations produced by notes of low pitch are sufficiently far apart to be more easily perceptible to the hand than those of higher pitch; and we must remember at the same time that for obvious reasons whispered sounds are normally impalpable.

(2) **Increased Fremitus.**—There are certain abnormal conditions of the lungs, pleuræ and chest wall which modify to a greater or lesser extent the vibrations as felt by the palpating hand. In some diseases they are markedly accentuated, in others diminished or lost; we will first consider the former.

There are, however, no pathological conditions of the respiratory tract which will produce a complete and marked bilateral, *symmetrical* increase in vocal fremitus, although in the senile and in individuals with rigid chest walls, phthisis and emphysema, especially if emaciated, it may be slightly increased.

On the other hand, we frequently find in disease *localized areas* over which the fremitus is greatly exaggerated; such areas are usually in relation to underlying consolidation (pneumonia, tuberculosis, etc.) or excavation of lung tissue (tubercular and bronchiectatic cavities or vomicæ). In both these cases, however, for increase in fremitus to be present it is necessary that the air passages be unobstructed and the bronchi patent.

The **explanation** of the phenomenon found in cases of consolidation is that vibrations (sounds) are more easily transmitted by a medium whose consistence is

regular throughout (as found in a solid pneumonic lung) than by the normal organ whose continuity is broken in every direction by the septa, bronchial tubes, vessels, and air-containing alveoli. In the normal lung, therefore, the vibrations have to pass through structures of varying density, while in the case of consolidation a filling of the alveoli with exudate to a great extent converts the normal spongy lung into a tissue the structure and density of which are similar and equal throughout, and which hence becomes a better conductor of sound.

(3) **Diminished Fremitus.**—These vibrations may be diminished to a greater or lesser degree or may be completely absent as a result of disease of the lungs, pleuræ, thoracic parietes, or following plugging of the bronchial tubes.

In *vesicular emphysema* the conducting power of the lungs is interfered with and in consequence vocal fremitus is reduced or in severe cases may even be completely abolished. The same phenomenon is usually also present over tumors of the lung, but occasionally mediastinal growths, if in direct contact with the larger bronchi, may transmit the vibrations to the chest wall.

In cases where the *lung is removed from its normal relationship with the thoracic parietes,* either in consequence of a serous effusion, a greatly thickened pleura (the result of long-standing inflammatory action), the presence of air in the pleural sacs, or the collapse (atelectasis) which follows compression of the lungs and bronchial tubes by a pleural effusion, the vocal fremitus will be reduced in intensity or lost over the affected areas.

In the case of a *pleuritic effusion* it is not only the

presence of the fluid which interferes with the passage of the vibrations (fluid being a good conductor of sound or vibration), but the relaxation of the collapsed lung which underlies it that is responsible for its abolition.

The presence of an exudate in the bronchi (even if the surrounding tissue be consolidated) may prevent the vibrations from ever reaching the lung and in consequence the examining hand. *Temporary inequalities* in fremitus may therefore be due in cases with a profuse bronchial exudate to variations in the amount present; for instance, note the effect of an attack of coughing with expectoration. Massive tubercular consolidations and tumors also may abolish vocal fremitus.

III. **PLEURAL FRICTION RUBS.**—Friction rub is the term applied to a "to-and-fro" palpable vibration occasionally felt over areas of inflamed or roughened pleura (dry pleurisy), synchronous with inspiration and expiration, frequently accompanied by pain and hyperesthesia of the overlying skin, and usually intensified by compression of the chest wall.

It is rendered audible if the ear be applied to the naked chest and somewhat resembles the sound produced when two pieces of brown paper are rubbed together (page 114). It is to be differentiated from vibrations produced in the bronchial tubes, and from the "rub" of pericarditis. It is not affected by the act of coughing nor has it any relation to the cardiac cycle.

IV. **RALES.**—Palpable vibrations ("*wheezing*") may also be felt in cases where there are catarrhal changes in the bronchial mucosa, especially in the presence of a tenacious exudate or of localized con-

strictions. They may occur with either the low pitched and snoring or with the piping and sibilant sounds heard on auscultation (*sonorous or sibilant ronchi*, page 113). The presence of fluid secretions in the tubes may also produce a fremitus due to the bubbling of air through their contents during the respiratory acts (mucous râles, page 111). Both these forms are altered by coughing, unaccompanied by pain, and may be felt during inspiration, expiration, or may be present throughout both acts.

V. **OTHER PHENOMENA.**

(1) **Tenderness** may be demonstrable; if so, its origin should be sought. It may be due to a local inflammation or tumor of the parietes, to intercostal myalgia where it is increased by pinching the muscle affected, to neuralgia in which it is usually possible to find specially tender spots corresponding to the points of emergence of the nerve branches from the facia (*Valleix's points*), or finally to pleurisy. In this last condition it is usually aggravated by pressure or coughing, and on taking a deep breath.

(2) **Crepitus** refers to a crackling sensation produced on palpating the skin overlying subcutaneous connective tissue whose meshes contain gas (surgical emphysema). It may be bacterial or traumatic in origin, as, for instance where a fractured rib has punctured the lung.

(3) **Fluctuation** is occasionally found over collections of fluid lying in immediate relation to the intercostal spaces, as, for example, over large pleuritic effusions, or over simple abscesses of the chest wall.

(4) **Succussion** is a splashing sensation imparted to the examining hand in cases where both air and serum

are present in the pleural sacs (hydropneumothorax); if at the same time the chest be shaken from side to side. It is, however, rarely demonstrable.

(5) **Pulsations** are rarely due to disease of the respiratory apparatus (pulsating empyemata), but are sometimes found over certain tumors; they may, however, be communicated from aneurysmal dilatations of the aorta.

C. **PERCUSSION.**

I. **THEORY AND METHODS.**—Although percussion had probably been known to Hippocrates, Auenbrugger, in 1761, was the first to describe its application to physical diagnosis.

It consists in manipulations by means of which we are enabled to throw into *audible vibrations* certain structures of the body, and is applied by striking sharply, usually with the tip of the bent finger, a part under examination and interpreting by means of the sound produced the density, elasticity, and air-content of the underlying tissues.

For example, on tapping over air-containing organs and bones these structures will be found to resound to the blow, and the note elicited is termed a *resonant* one. On the other hand, solid tissues merely respond with a dull thud (as over the quadriceps extensor), and hence emit a *dull* or *flat* note, and it is in the production, appreciation, and interpretation of the various "notes" that lies the art of percussion.

Technic.—There are several methods we may use in order to throw into vibration the organs we wish to examine. That most commonly in vogue is applied as follows: The palmar surface of the terminal phalanx of the first or second finger of the left hand is

applied firmly to the body and the middle finger of the right hand, flexed at the first phalangeal joint, is used to tap the dorsal surface of the former in precisely the same manner as is used in striking the key of a piano. The forearm is flexed to a right angle and held motionless, while the act of percussion is performed by movements of the wrist and metacarpophalangeal joint. The intermediate finger (*pleximeter*) is at times replaced by plates, cubes or pillars of glass, metal, ivory, etc., and the percussing finger (*plessor*) by a small rubber-tipped hammer.

For several reasons the use of artificial aids is discountenanced; among these may be noted inconvenience in portability and application to the uneven body surfaces, and the fact that the "resistance" of the part struck to the blow of the plessor cannot be appreciated except by the finger itself.

The technic of percussion can only be acquired by demonstration and persistent practice. Several points, however, should be observed; for instance, the movements of the percussing arm should take place principally at the wrist and phalangeal joints, the elbow remaining stationary but not rigid; the blow must be given sharply and the plessor not allowed to remain in contact with the pleximeter, in order that it may not "damp" or interfere with free vibration of the part struck, it must also be made at right angles or perpendicularly to the surface under examination and with the tip only of the percussing finger.

The force of the blow employed will depend on whether the organ under consideration be superficial, in which case the lightest percussion must be used. On the other hand, in searching for deep-seated dullness

considerable force is required to elicit the required note; at times two or even three fingers may be used in conjunction as a single plessor.

The form of percussion above described has been termed **mediate** in contra-distinction to the method in which the pleximeter is dispensed with and the part under examination struck directly with the plessor (**immediate**). This latter was the original method but is now seldom employed except over the clavicles and sternum, or in a modified form in what will later be described as **palpatory percussion**, (page 116).

Having now defined percussion and described the technic of its application, we are in a position to analyze the results ("notes") obtained and briefly review the **theories** advanced as to their mode of production.

We must first remember that *resonant bodies* are the air-containing viscera and the bones; in other words, they are structures able to vibrate regularly and to emit a "clear" note when struck.

The osteal percussion sound is totally different in quality from that found over the hollow organs, although both are "resonant," the bones being enabled to vibrate in virtue to their elasticity.

The note obtained over the *hollow organs* (tympany), however, taking the stomach as an example, depends on the rhythmical vibration of the contained air and elastic walls. This note will be found to vary considerably, depending on the size of the viscus, the state of its walls, and the tension of the enclosed gas. Lastly, it must be remembered that simple vibration of air in a sac is not alone sufficient to produce a resonant note; a "unison vibration" of air and wall or, in other words, "consonance of the wall" is the essential factor.

The opposite conditions are found when *solid structures* are subjected to similar examination. They emit a "dull," "toneless" or "flat" note when thrown into vibration, and at the same time impart to the percussing finger a sense of **"resistance,"** whose degree is indicative of the consistence and elasticity of the underlying part, and the correct estimation of which will be found to give results of almost equal value to those obtained from the audible vibrations or sounds. It is for this reason that the examiner is able to appreciate differences more readily than a spectator who must rely solely on his auditory impressions.

Between the clear, resonant and dull or nonresonant notes above described are found innumerable others intermediate in tone; they are described by what we know as their **physical attributes**.

(1) **Quality or timbre** is that property which *distinguishes sounds from different sources*, which are otherwise similar, as, for example, the same notes produced on a violin and harp.

Let us note here that dull notes are "harder" in quality than clear or resonant ones, and that of the clear notes there are two subdivisions, namely, tympanitic and nontympanitic resonance, which will be further described.

(2) **Intensity** is simply loudness and consequently depends on the *amplitude of the vibrations*. Not only the amount of the productive force but also the density and quantity of the vibrating body will materially influence the note; for example, compare the intensity or loudness of the percussion note over, say, the second intercostal space in the mammary line, under which a large volume of lung tissue is situated,

with that over the thin margin of the lung overlying the liver in the fifth right interspace in the same vertical line.

In most cases the note over such hollow organs as the stomach (tympany or "drum note") is louder than that over the normal lung, and this clear, resonant lung note is in turn more intense than the dull note produced, say, over that part of the liver which lies in direct contact with the chest wall and still more so than that found over collections of fluid or, say, over the quadriceps extensor, which latter tones are described as "flat."

(3) **Pitch** depends on the *number of the vibrations per unit of time.* Thus the more frequent the vibrations and the shorter their "wave-length," the higher will be the "pitch" of the note produced; in other words, "*low-pitched*" notes are the result of few and long vibrations, while those of "*high-pitch*" result from more frequent and shorter waves.

The louder and clearer or more resonant the sound, the lower will be the pitch and *vice versa*, but we must note that in emphysema a loud, clear, but high-pitched tone is often obtained.

(4) **Duration** is of no great importance. It varies directly with the clearness and intensity; hence the louder, clearer (more resonant) lower-pitched a note, the longer will be its duration, and *vice versa.* In other words, a dull sound is of shorter duration than one more resonant.

(5) **Resistance** to the percussing finger *varies in inverse proportion to the elasticity of the underlying organ.* That is to say, the duller, the less intense, the higher the pitch, and the shorter the duration of the note

produced over any part, the greater will be the sense of resistance encountered.

(7) **Tympany** is the term applied to a peculiar clear or resonant note, produced when the *air in a cavity of appropriate size is set in vibration*, providing the vibrations be not modified by excessive tension of its walls.

If this cavity, however, is in direct communication with the open air (as we find in tubercular vomicæ in the lung), an "*open*" quality is appreciable; on the other hand, if the cavity be sealed or has no outlet (the stomach for example), the note produced is said to convey a "*closed*" or high-pitched impression to the ear.

Supposing a similar cavity be resolved into air-containing loculi by innumerable septa containing elastic tissue, as we find in the healthy lung, the note obtained on percussion will lose its tympanitic quality and a characteristic clear, low-pitched, "normal lung resonance" is produced

Remember that the pitch of tympany is higher than the normal lung resonance above described, and also that a tympanitic quality may be destroyed or become high-pitched over an organ whose walls are sharply distended and whose contents are under excessive pressure. A similar high-pitched, "boxy" note is usually obtained over pneumothoraces.

II. **THE NORMAL LUNG.**—Having now completed our review of the theories of percussion and described the methods employed, we may now put these into actual practice, commencing with the examination of the normal lung.

First regarding what are known as **"superficial"**

and "deep" percussion, we find that by gradually increasing the force of the blow dealt by the plessor we throw into vibration progressively deeper layers of the part percussed; in this manner we are able to differentiate lesions which lie in close relationship with the chest wall from others of a deeper or more central origin, also, for example, in the healthy chest we are enabled to examine by the use of a very light percussion the superficial layers of the lungs and their thin margins which overlie the heart and liver, in which latter areas, if deep percussion be employed, we should throw into vibration not only the lung but also the underlying organ.

Thus, by the use of superficial percussion we are also enabled to define accurately certain boundaries of the lung, and by the use of the deep method to determine the outline of solid and deep-lying organs, such as the heart and liver, and, in addition, to examine the central portions of the lungs themselves.

It is necessary now to determine the **boundaries of the lungs** or, in other words, to confirm or modify the normal topographical outlines described on page 53.

It is usual to determine first the height to which the *apices* rise on the side of the neck (remembering in all cases that we percuss from a dull toward a resonant area in determining the line of demarcation between them); we therefore percuss, using moderate force only, from above downward on the side of the neck, commencing at least 2 1/2 inches above the clavicles, and mark the point where dullness gives place to resonance on the skin with a soft, colored, grease crayon (dermographic pencil). In this manner we are enabled to define accurately the upper borders of the lung, and

also to compare accurately the physical qualities of the notes obtained on the two sides, always keeping in mind the fact that that of the *right side possesses a slightly higher pitch.*

It is impossible to outline with any degree of accuracy the *anterior margin* of either lung as far down as the fourth rib, or that of the right organ throughout its whole length, but on the left side the outline of the *incisura cardiaca* can be readily defined by light percussion.

The *lower margins* can be traced throughout their whole course, with the greatest ease, however, that of the right lung where it overlaps the liver. At times difficulty may be found on the opposite side on account of the tympanitic note superadded by the stomach.

It is necessary not only to define the margins during quiet respiration, but during a deep inspiration to note the expansion, or *respiratory excursion*, of the organs into the plueral sinuses, as shown by descent of the normal lung resonance. Respiratory excursion can usually also be demonstrated over the apices, but its detection requires expert percussion. By this maneuver we exclude also the presence of pleural adhesions which when present interfere with normal expansion.

On percussing over the sternum a clear note is elicited (the "direct" method may be used) which is due to the vibrations of the bone setting into acoustic activity the adjacent lung as well as the "osteal" note itself.

It should also be remembered that the note obtained over the *right lung* posssesses a slightly less clear quality, higher pitch, shorter duration, and

lessened intensity than does its fellow. This is especially marked over the upper lobe, and is due to the greater development of the muscles (in right-handed individuals), the arrangement of the bronchial tree, and to the fact that this lung rests on the solid, resistant liver.

We next proceed to examine the *clavicular regions;* here we may use the direct method, the clavicles taking the place of a pleximeter, always remembering the fact that the trachea may lend a tympanitic quality to the note over the sternal end.

Let me here lay stress on the vital point of the art of percussion; it is that our **deductions are based** not so much on the note elicited over any region of the chest as **on the results of comparison of the notes that are produced over exactly corresponding areas on the two sides, and under precisely similar conditions.**

For example, if the exact center of one clavicle be struck on its upper surface and that of the other on its anterior border, the notes obtained will be found to differ, and a false impression may in this way be obtained. Similarly great care must be taken to eliminate the possibility of fallacies occurring from ignorance of the influence of the part played by the ribs in altering percussion tones; thus it is necessary to compare rib with rib and space with space to secure reliable results, and to remember that the pressure exerted by the pleximeter must be the same on both sides, and that the finger must always be struck over exactly the same spot.

It is usual in routine examinations after having completed our comparison of the apices and determined the lung boundaries to *percuss from above downward in*

the mid-clavicular and axillary lines, comparing space with space and rib with rib on the two sides, after allowing for the influence of the presence of the mammary glands.

We next *examine the apices from the posterior aspect*, then the *supra-* and *infraspinous*, *infrascapular and finally the interscapular regions*. We must also take into consideration the fact that over the scapulæ a certain amount of "muffling" will be found, and that considerably heavier percussion will be required to throw the underlying lung into vibration than over areas such as the axillary, which are not clothed by masses of muscle or covered by bone.

It is only, however, by assiduous practice on a large number of healthy thoraces that we learn what note is normal for a definite area in a subject of a certain degree of muscular development and nutrition.

We have now by means of our manipulations determined not only the position of the borders and condition of the parenchyma of the lungs but have also defined the upper borders of the liver and the outlines of the heart and spleen, and where no abnormal alterations in the tones are present eliminated the presence of pleural effusions or fibrosis, and neoplasms.

Several other **important normal variations** must also be described. Thus we find that in the *aged*, whose chests are becoming rigid, the note is of a higher pitch and of a less clear quality than in adults of middle life, also that in *infants* the opposite condition is usually found, the tone being more intense, of lower pitch, clearer quality, and of a longer duration. We must also remember that if the patient be in the *lateral decubitus*, the lung on the dependent side will be subject to slight

compression and consequently will contain less air than its fellow, hence the note on the lower side will be slightly muffled or, in other words, will show defective resonance.

It is also necessary when searching for lesions to have the subject take several deep inspirations, and having thus fully distended every part of the lungs to again repeat the act of percussion, in this way many mistakes will be obviated, as occasionally, especially in the *bed-ridden*, all parts of the lungs are not fully distended at the same time, and the varying notes produced by this irregular inflation may lead to erroneous deductions.

Finally, what is known as the *lung reflex* must be described. We find that having percussed heavily a number of times over a single area, the note will alter, owing to a "reflex relaxation" of the lung tissue, and a tone of somewhat lower pitch and of a slightly tympanitic quality may be elicited. It is of no pathological significance, but must always be born in mind.

III. **INCREASED PULMONARY RESONANCE.**—What we have described as "normal lung resonance" may be altered by pathological conditions in one of two ways; either its clearness may be increased (hyperresonance) or the lung may emit an impaired, dull or flat note.

Hyperresonance may occur when the lung is overdistended with air, as, for example, in emphysema, either general or "compensatory." In the first instance the hyperresonance will be symmetrical, in the vicarious or compensatory forms unilateral or patchy, as found in the neighborhood of localized areas of nonfunctionating lung (*e.g.*, atelectasis or consolidation).

The note in these conditions will be clearer, more intense, of lower pitch, longer duration, and less resistance will be encountered by the percussing finger than over the normal lung.

Skoda's tympanitic resonance is found where relaxation of the lung imparts a hyperresonance of tympanitic quality to the percussion tone. Occasionally, however, the pitch may be raised and add a deceptive suggestion of dullness. Such a condition is found over the lung just above the level of a pleural effusion (if the latter be of sufficient size to cause a partial compression of the organ), over lobar pneumonia in the stage of congestion or during resolution, just above the level of a pneumonic consolidation, and occasionally also over early tubercular infiltrations, hypostatic congestion, and edema. Pneumothorax and cavities in the lung may also yield a high-pitched tympany on percussion.

IV. **DULLNESS.**—Certain diseases cause alterations in the normal note of such a character that resonance is diminished. If the interference muffle, damp, or render less clear to a slight degree the normal tones, we describe the condition found as **impaired resonance,** but if the vibrations are interfered with to a greater extent **dullness,** or even **flatness** may be produced.

The phenomenon of resonance being due, as we have seen, to the presence of air in the lungs, it follows that lesions which either lessen its amount in the lungs or interfere with the conduction of the vibrations we impart to the chest wall during the act of percussion will diminish its clearness.

Hence dullness conveys to us the information that either there exists consolidation or collapse of the lung tissue, an airless mass (tumor) in the thoracic

cavity, or that a pathological condition of the pleura is present (fibrosis or effusion).

It is partly by the recognition of the extent and position of areas of altered resonance over the chest wall that we are enabled to infer the state of the underlying tissues. For instance, in *lobar pneumonia* the dull region will be found to be limited by the boundaries of the lobe involved, while in *broncho-pneumonia* and disseminated *areas of tubercular infiltration*, they will be "patchy" or of irregular distribution and outline.

On the other hand, where a **pleuritic effusion** is present, its situation as defined by absolute flatness and marked resistance, will be found to follow the laws of gravity so that, providing there are no pleural adhesions, the phenomenon of "*movable dullness*" may be demonstrated. The upper boundary of the dull area will be found to vary on alteration of the decubitus in proportion to the volume of fluid present, to rise toward the clavicles as the quantity of exudate increases (400 c.c. of fluid being necessary to produce basal dullness), and its lower margins to conform to those of the pleural sacs. Its margins will also overstep those of the lungs to the same extent as do those of the pleural cavities. Dullness may be found over *Traube's space* (a triangular area in the left hypochondrium bounded by the lower costal arch, the anterior border of the spleen, and the upper limit of the fundus of the stomach) over which area normally we expect to obtain a tympanitic note on account of the underlying stomach. If, however, a previous inflammatory process has obliterated the pleural recess which underlies this area, tympany may be obtained over

Traubè's space even in the presence of a pleural effusion.

It is also well to note that the upper limit of dullness does not extend in a straight line around the chest, but rises highest in the axillary regions, assuming what is known as "*Ellis' curve*," and that just above this line will be found usually a band of tympany due to relaxation of lung tissue (*Skoda's resonance*, page 95).

It is impossible here to describe all the lesions which interfere with normal lung resonance; we have, therefore, merely outlined the general principles involved and drawn attention to several of the commoner pulmonary diseases.

V. **OTHER PHENOMENA.**

(1) The **"cracked-pot sound"** is a modified tympany caused by a sudden expulsion of air from a cavity which possesses but a small opening communicating with the atmosphere. It may be imitated by loosely clasping the hands, palms together and almost in contact, and then striking the back of one of them on the knee.

The phenomenon is occasionally present over a pneumothorax complicated by a fistula, but is more frequently obtained over a large vomica in the lung, providing it is in direct communication with a large or medium-sized bronchus. It can best be elicited if the percussion blow be given sharply, and the patient directed meanwhile to hold his mouth widely open and to breathe freely.

It should also be remembered that this sound may be obtained during percussion over the chests of healthy, screaming infants.

(2) **Wintrich's sign** requires for its production condi-

tions similar to those mentioned in the above paragraph. It will be found that if the subject is directed to close his mouth, the tympanitic note elicited is lowered in pitch in comparison with the one obtained when that orifice is open.

(3) **"Friedreich's sign"** over a cavity depends on the fact that the pitch of the percussion note rises during a deep inspiration and falls as the air is expelled.

(4) **Gerhardt's phenomenon** may occur over a cavity in the lung containing a definite amount of fluid. On percussion it is found that as the patient assumes different positions alterations in the percussion note may be obtained on account of the movements of the contained exudate and its influence on the physical conditions of the parts under examination.

(5) **Amphoric Resonance.**—The "empty-bottle" note or "metallic ring" may be imitated by flipping the cheek sharply with the finger while the mouth is distended to a certain degree with air. It conveys an impression of "hollowness" to the ear. To obtain this sign a cavity with a diameter of not less than 2 inches and possessing smooth, tense walls are essential conditions. This peculiar note is due to the *prolongation and accentuation of certain overtones* which have been superadded to the tympany ordinarily present over pulmonary vomicæ. If the walls of the cavity are for any reason lax a "*cavernous*" quality is usually demonstrable. (Page 109.)

(6) **Williams' Sign** or **"Tracheal Note"** depends on the presence of consolidated or retracted lung in close proximity to the trachea, the dullness usually present over airless tissue being replaced here by a communicated resonance of tympanitic or tubular quality.

(7) **Biermer's phenomenon** may be present over a pneumothorax. It is found that the "metallic" note usually obtained is of a higher pitch with the patient erect than when the recumbent posture is assumed.

(8) **"Boxy" or "Wooden"** impressions are found when percussing over consolidated areas of lung surrounded by shells of relaxed tissue. The note elicited is less intense and emptier than normal lung resonance and possesses a subtympanitic quality. A somewhat similar tone may be elicited over pneumothoraces.

(9) **Grocco's triangle** is a dorsal area of dullness found on the side opposite to a large pleural effusion. It is a long narrow triangle with its apex directed upward, and reaching the same height as does the effusion, having a base of from 2 to 3 inches in length situated on the same level as the lowest margin of the pleural sac, and one side corresponding to the mid-spinal line. Several explanations for its presence have been advanced, but we do not consider it a sign of any value in diagnosis.

(10) The **"Hydatid Thrill"** is a peculiar quivering sensation imparted to the fingers during percussion over hydatid cysts. Their walls are elastic and contain a thin fluid under pressure, both of which conditions are essential factors in its production. It is rarely present over the thorax, but not infrequently met with in hydatid disease of the abdominal organs.

D. **AUSCULTATION.**

I. **INTRODUCTION.**—Auscultation is the term used to describe those methods by means of which we are enabled to appreciate through our auditory apparatus sounds produced in the body and its cavities and to interpret their significance. We are indebted for its

discovery to Laennec, who was the first to apply it to medicine in 1816, and who two years later published his first "memoir."

In this section we will confine our attention to the respiratory system, in which by the aid of auscultation we are enabled to detect sounds produced by the respired air in its passages, the phenomenon of vocal resonance, and the presence of adventitious sounds, such as friction rubs and râles.

Two methods are at our disposal; we may either apply the ear direct to the chest wall—**immediate, or direct auscultation**—or interpose one of several different instruments (stethoscopes) to concentrate or magnify the sounds. This latter method is termed **mediate auscultation** and has several advantages over the former. The chief of these being the lessened liability to contraction of contagious disease on the part of the examiner, the greater facility with which the less accessible parts of the chest may be reached, and the fact that inspection and palpation may be carried on at the same time; also for esthetic reasons. It is usual, however, for the sake of delicacy, to place a handkerchief between the examining ear and the chest wall.

In spite of the disadvantages of the direct method, the student is urged to practise it assiduously, for frequently in emergencies a stethoscope may not be available, and it is also well to remember that certain sounds, for example bronchial breathing, especially if high-pitched, are much more clearly heard by this method.

Stethoscopes are manufactured after numerous models. The form first used by Laennec was simply a roll of paper; a hollow tube of wood was next sub-

stituted (this form is most valuable in the investigation of heart sounds, since the cardiac impulses are simultaneously conveyed to the ear, thus facilitating the timing of murmurs and sounds); and later what is known as a binaural instrument came into use, possessing many advantages over the older type, especially that of adaptability.

The student is urged to use the greatest care in the selection of his instrument to see that the chest-piece has a wide bell, that its bore be not less than 1/8 inch in diameter, that the tubes are composed of heavy rubber, and finally that the ear-pieces fit both comfortably and accurately.

I strongly recommend the beginner to use none of the various forms of **stethophones** which magnify the sounds heard to a considerable extent, since they may become indispensable and if at any time be not procurable may seriously handicap the examiner. It is well also to remember that these instruments are but *poor conductors of high-pitched vibrations* and hence have a limited field of usefulness, also that it is clear and distinct impressions and *not mere loudness* that gives us the greatest amount of information.

The greatest difficulty probably encountered by the student in commencing the practice of auscultation will be found in the separation of cardiac and pulmonary sounds; it is possible, however, by concentration and repeated efforts that the mind can be trained to eliminate the one or the other as the auditor desires.

II. **NORMAL BREATH SOUNDS.**—It is found that on listening over a healthy chest certain sounds, distinct from those of cardiac origin, are clearly heard. These have been termed *breath sounds* or *respiratory*

murmurs, and are primarily **due to vibrations set up in the respired air as it passes through the glottis.**

Supposing therefore that we first apply the stethoscope to the side of the larynx or over the trachea, we should hear a double murmur as the air passes over the cords, one-half corresponding to inspiration and the remainder to expiration, with a short pause intervening. This sound has been termed **tracheal breathing;** it possesses great intensity, and is of a low pitch.

Bronchial breathing may be heard in the normal subject over the spine of the vertebra prominens, over the sternoclavicular articulations, and usually also in the interscapular regions. The sounds closely resemble those over the trachea, differing only in possessing lessened intensity and a slightly higher pitch.

Tubular breathing is merely bronchial breathing modified by the addition of a hollow or "tubular" element; it has, however, no definite pathognomonic significance.

Vesicular breathing is the term applied to the breath sounds heard over any region of the chest under which lies normal, functionally active lung. It is a double murmur, much softer than those already described, and conveys to the ear a "breezy" or rustling impression. Its first portion corresponds to the period of inspiration and its second, which is much less intense, of lower pitch, and of considerably shorter duration, follows the first after an almost imperceptible interval of time. The *expiratory murmur* has a duration equal only to the first quarter of the act of expiration, thus differing materially from those murmurs possessing a tubular or bronchial quality in which the expiratory and inspiratory sounds are of almost equal length.

Place the stethoscope next over the inner half of the infraclavicular region and a respiratory murmur, less harsh and tubular than those already described, may be heard. It will now be seen that this **broncho-vesicular** or "indeterminate" breathing is merely bronchial breathing modified by distance from its source, by its passage through the overlying lung tissue, and by the addition of the vesicular element.

It is usual to ascribe all the forms we have mentioned to a glottidean origin, *the sound produced at the glottis* being transmitted by the air column of the bronchial tree and the lung to the chest wall.

The *explanation of the abbreviation of the expiratory phase of the vesicular breath sound* lies in the fact that during expiration the direction of the air current is upward and antagonizes the propagation or transmission downward of the expiratory phase of the laryngeal murmur, and also that the force of the expiratory air current as it passes through the glottis, is considerably less than the inspiratory, since the act of expiration is due merely to an elastic recoil and hence lacks the forcible element due to muscular contractions.

We must now refer to certain **normal variations** in respiratory murmurs which may occur in health. It is found that the vesicular breath sounds are more intense over the anterior surface and upper portions of the lungs than behind and below, and that the murmur heard over the *left apex* is usually slightly more harsh and intense than that over its fellow on account of the arrangement of the bronchial tree.

In growing *children* on account of their thin chest walls and the greater elasticity of their lungs the murmur assumes a relatively loud, harsh or *puerile*

quality; on the other hand, in subjects with thick chest walls even the inspiratory stage of vesicular breathing may be almost inaudible, but can always be rendered more distinct by deep breathing, providing that no diseased condition is present.

III. **ABNORMAL BREATH SOUNDS.**—Having completed the description of the sounds which are normally heard over certain portions of the respiratory apparatus, we turn to the consideration of those due to disease.

The respiratory murmur heard over any suspected area should first be carefully described, noting the relative lengths of the inspiratory and expiratory phases, the quality (soft, harsh, tubular, etc.), pitch (high, medium, or low), and intensity before attempting to classify them under the following headings:

(1) **Diminished intensity or suppression** of the vesicular murmur may be found in a number of pathological conditions, among which are defective action of the respiratory muscles (from pain or paralysis), pleural fibrosis, pneumothorax, and pressure of tumors on the lungs or bronchi.

During the earliest stage of lobar pneumonia and over early tubercular lesions feeble or indistinct sounds are the rule.

(2) **Total absence** of the breath sounds may be secondary to large pleural effusions (especially if purulent), to a greatly thickened pleura, and also to complete obstruction of a large bronchus.

In the case of *pleural effusion* their disappearance is due not only to the presence of the fluid, which in itself is a good conductor of sound, but to the fact that the relaxed and compressed underlying lung is but a poor

conductor of sound. Not infrequently, however, *intense bronchial or tubular breathing may be heard over a massive hydrothorax.*

(3) **Prolongation of the expiratory murmur** occurs in certain conditions in which the act of expiration is unduly prolonged, either from obstruction of the bronchioles or from loss of elasticity of the lung parenchyma, as found in asthma and emphysema. It is also in some cases an early sign of incipient phthisis.

This condition is sometimes termed "*harsh*" breathing, but must not be confounded with the puerile type in which, although the sounds are harsh, the normal ratio of the two phases to each other are retained.

(4) **"Cogwheel" or "wavy"** respiration occurs with vesicular breathing and simply means that the act of inspiration is broken up into two or more parts.

If it is heard *symmetrically* in both lungs, and disappears on taking a long breath, it is usually due to nervousness. If *localized* and well marked on deep inspiration, it is indicative of irregular alveolar expansion and unequal elasticity of the septa, and hence it often suggests early pulmonary phthisis, especially if the respiratory murmur be at the same time lessened in intensity and the expiratory phase prolonged. Not infrequently the vesicular murmur may be resolved into a series of short phases synchronous with the cardiac systoles. This is most frequently met with over the anterior surface of the upper lobe of the left lung and is usually indicative of a slight loss of elasticity in the underlying parenchyma.

(5) **Bronchial breathing**, heard where normally the breath sounds should be of the vesicular type, is in-

dicative of disease. It does not differ in quality from that heard over the spine of the seventh cervical vertebra, but is of somewhat lessened intensity.

It is heard over consolidated (pneumonia and tuberculosis) or collapsed areas of lung (providing the bronchi be patent), and also over cavities (pulmonary and bronchiectatic).

The *expiratory phase* follows its predecessor after an almost imperceptible interval. It is somewhat more intense, possesses a higher pitch than its fellow, lasts throughout the greater part of the act of expiration, and accasionally occupies a period of time equal to that of the inspiratory murmur. For its production it is necessary that the bronchial tubes be patent.

The *explanation* advanced to account for this phenomenon probably lies in the fact that the solid lung and uninterrupted air column transmit the double laryngeal murmur more readily than does the normal lung, composed as the latter is of structures of various densities. (Page 80.)

Bronchial breathing is much more *frequently heard over large pleural effusions* than is generally indicated in text-books (an absence of breath sounds, however, being the more common sign), the tubular element produced in the underlying collapsed lung being carried by the fluid to the surface of the thorax. Since in these cases it is usually "high-pitched" and distant, it is best heard if immediate auscultation be employed.

Bronchial breathing may be either of *low pitch*, as is found over consolidated areas of lung tissue lying in direct relation to large bronchi, over dilated bronchi (bronchiectasis) and cavities in the lung substance. It may also be *high-pitched* providing the solidified

lung lies in immediate relation to bronchi of small caliber.

(6) **Amphoric breathing** is a sound somewhat resembling that produced by blowing across the mouth of a jar (amphora, a jar). It is found over vomicæ and in some cases of pneumothorax, providing the cavities have smooth walls and are in free communication with a bronchus. The "metallic" quality is due to an admixture of both high- and low-pitched tones.

(7) **Cavernous breathing** resembles amphoric, but lacks the ringing or metallic quality. It is heard over cavities and dilated bronchi; its significance is similar to the above.

IV. **VOCAL RESONANCE.**

(1) **Introduction.**—Much that has been said on the subject of *vocal fermitus* applies to the voice vibrations heard over the lungs during auscultation. The method of production, normal variations, and the alterations which occur in its intensity due to disease are precisely similar. However, instead of the vibrations being sought for by the palpating hand they are rendered audible by means of auscultation.

As we have already pointed out the **origin of vocal resonance lies in the larynx and air passages above it,** so we should expect on applying the stethoscope over the thyroid cartilage that the voice sounds should be most distinct in its region. We find this to be true, and depending on the point of application of our in-instrument, apply the terms **laryngophony and tracheophony** to the resulting phenomena.

Ordinarily during the production of the voice an indistinct "buzzing" is heard over the lungs, subject

to the same deviations from the normal as is the palpable vocal thrill, but in addition several other phenomena may be observed, which palpation fails to detect.

(2) The term **bronchophony** is used simply to describe a marked increase in the intensity of vocal resonance, such as is found to occur over an area of consolidated lung, especially if lying in direct relationship to a large and patent bronchus.

(3) **Pectoriloquy.**—Normally, over the lungs spoken words themselves are inaudible, but in certain conditions, notably where a large cavity and open bronchus lie in direct communication, just above the level of a large pleural effusion and occasionally over a consolidated lung, the words themselves become recognizable, and even whispered words may be rendered distinct, giving rise to the condition designated pectoriloquy or "*chest voice.*"

But let me, however, emphasize the fact that *pectoriloquy is merely an exaggerated bronchophony*, and that *bronchophony is merely a marked degree of increased vocal resonance*, and that these terms are therefore employed solely to convey an idea of the intensity of the resonance obtained in any given case.

(4) **Baccelli's sign,** the reliability of which is somewhat doubtful, depends on the fact that whispered sounds may be transmitted to the chest wall by serous but not by purulent pleural effusions.

(5) **Egophony** refers to a "nasal" or "bleating" quality superadded to ordinary bronchophony. It is found near the upper margin of pleural effusions and is usually best heard below and external to the angle of the scapula. Its origin has been ascribed to a

collapse of the bronchial tubes of the compressed lung which become flattened and act as do the reeds of a bassoon.

Not infrequently egophony is distinctly audible over *massive plural effusions*, in which cases the type of breath sounds heard is usually tubular.

(6) The term **Amphoric resonance** is sometimes applied to a hollow, echoing tone occasionally heard over large cavities and fistulous pneumothoraces during the production of the spoken voice. (Page 98.)

V. **RÂLES.**—The term "râle" is applied to describe certain adventitious sounds produced within the lumena of the trachea, bronchi, or lung alveoli by the passage of the respired air setting their contents into vibration.

Râles have been divided into two classes termed **"moist"** and **"dry,"** depending on whether they convey to the ear the impression that the respired air is bubbling through fluid, or that the sounds heard are otherwise produced.

It should be noted that the terms *crepitations* and *mucous râles* have been applied to the "moist," and that the dry forms have been designated *ronchi*. These terms are merely relative and so frequently misused that their application and interpretation is extremely liable to give rise to erroneous impressions.

It is not merely the ability to place certain sounds under their proper category, but a **clear understanding of the causative physical conditions present in the bronchial tree and alveoli** that is necessary for practical application so that, providing the auditor is able to appreciate the significance of what he hears, any nomenclature may be employed.

A word of caution is necessary at this point in regard

to the possibility of sounds produced by the friction of the stethoscope on dry, harsh skin (especially in the emaciated) or the crackling of the hairs of the chest under the bell-piece being mistaken for pleural rubs or râles. This may be obviated by moistening the skin with water or glycerin.

We can find no better **classification** than that originally introduced by Laennec, which commend itself not only on account of its simplicity, but also because it lends itself more clearly than any other to a practical application to diseased conditions.

(1) **Crepitant râles** have been likened to those sounds produced when the moistened tips of the thumb and first finger are suddenly separated, or when strands of hair are rubbed together between the fingers in close proximity to the ear.

They are found in the *early stage of pneumonia*, also in *edema* and *atelectasis*, and probably depend for their origin on the opening up of collapsed air-cells by inflation with the inspired air as it separates the sticky alveolar epithelium.

In both edema (hypostatic congestion) and ordinary atelectasis the physical conditions present are such as would be required to fulfill those requisite for the production of these fine crepitations. In both disseminated collapse of the air-cells is present to a greater or lesser extent, and in both it may be shown postmortem that these same alveoli may be readily inflated, and that therefore no alveolar exudate is present.

The same condition is present in the early stage of pneumonia (congestion or splenization), but as the disease progresses and exudation appears in the alveoli

(hepatization) air can no longer enter the cells and in consequence the physical conditions are altered and the râles disappear.

These fine crepitations are *almost indistinguishable, so far as the sound alone is concerned, from the finest form of mucous râles* which are produced in the terminal bronchioles, but the former are found characteristically only during inspiration, and their number over a given area greatly exceeds those of tubular origin. The latter are frequently present also during the expiratory phase of respiration.

Not infrequently during the earliest stage of a tubercular lesion these râles may be detected; if, however, sufficient air be not entering the lung to set the exudate into vibration (as shown by suppression of the breath sounds), a few coughs will often elicit fine audible râles.

Crepitations may occasionally be found over the base of one or both lungs in individuals, especially in the debilitated, who have been lying for some time on one side or on their backs, even when no lung lesions are present; a few long breaths will cause their disappearance and open up the temporarily inactive and partially collapsed alveoli. It is probable that a slight transitory hypostasis is also present in the most dependent parts of the lungs.

(2) **Mucous or moist râles** are due to the bubbling of the respired air through fluid bronchial exudates and vary greatly in their physical attributes. They may occur only during inspiration, expiration, or throughout both phases.

We find by experience that moist râles vary in **quality** from the finest crackling forms that are practi-

cally indistinguishable from crepitations to the large, coarse, gurgling bubbles of tracheal origin, and hence we see the impracticability of any set classification.

The value of auscultation therefore lies not so much in an artificial classification of the sounds heard as in an accurate knowledge of their origin, location, and significance.

In practice we divide them arbitrarily into **"fine," "medium"** and **"large"** or **"coarse"** moist râles, depending on their origin. If they are produced in the *finest bronchioles*, fine, high-pitched crackles, best audible at the end of inspiration, but also heard during the earlier part of the expiratory act, may be detected; if in the *medium-sized tubes*, their intensity is greater, their quality coarser, and conveys to the mind the impression of small bursting bubbles. Finally, if their origin be in the *large branches* of the bronchial tree they assume a coarse bubbling or gurgling character. The term "death rattle" is often applied to râles of *tracheal* or laryngeal origin.

It must also be remembered that râles may be produced in *pulmonary vomicæ*, in which event their intensity will usually be great and their quality coarse. Occasionally they may assume a *metallic, reverberating*, or *consonating* quality providing the cavities have smooth, tense walls, and that a unison vibration of wall and râle is possible. Hence they are usually found in association with amphoric breathing over large vomicæ, or over a pneumothorax.

From what we have said it is obvious that providing moist râles are detected over a lung, certain **diseased conditions** must be present. Foremost among these are *bronchitis*, in which the conditions necessary for their

production are found, *bronchiectasis* (dilatation of the bronchi), and during a breaking down of lung tissue with *cavity* formation. They may also be heard during the stage of resolution in lobar *pneumonia*, while liquefaction of the exudate is taking place (*redux râles*).

It should be noted at this point that occasionally râles which arise in deep-seated lesions cannot be heard on auscultation over the chest; they may, however, at times be rendered audible by placing the bell of the stethoscope well within the oral cavity and directing the patient to breathe deeply.

(3) **Ronchi** or **dry râles** are the third form of adventitious intrapulmonary sounds encountered. They also are produced in the bronchi, but are dependent for their origin on different physical conditions from those already described.

They depend on *bronchial stenosis* or narrowing, due either to pressure on the tubes from without (tumor, etc.) or to obstruction to the passage of air from within, as is found with spasm and swelling of the mucosa (asthma) or in the presence of a tenacious exudate adhering to the walls (bronchitis). In this latter instance not only may the passage be blocked, but the shreds of mucus themselves may be set into vibration by the passing air, providing they are at one point adherent and hanging free into the lumen. These sounds are therefore most characteristically heard in asthma and bronchitis and constitute what is known as "*wheezing*."

They also vary greatly in quality, those snoring sounds of low pitch which occur throughout a part or the whole of the respiratory cycle and are produced in

the larger tubes are termed **sonorous ronchi.** Hissing, squeaking, whistling, high-pitched ronchi heard principally during inspiration, and due to similar conditions in the bronchioles are described by the term **sibilant.** Between these two extremes innumerable variations in pitch and intensity may be encountered, depending on the caliber of the bronchus affected and the physical condition of its contents and walls.

Râles, especially the "dry" forms, must be *distinguished from the pleural friction* sounds described in Subsection VI. With the exception of the crepitant types, râles will usually be found to alter in character or disappear temporarily after the act of coughing; they are unaccompanied by pain, pressure of the stethoscope produces no tenderness nor does it increase their intensity. Râles also appear to have a deeper origin than the pleural rubs, which latter give the impression of a superficial origin and usually are of a to-and-from rhythm, whereas râles may occur during the whole or any stage of the respiratory cycle. At times, however, it may be almost impossible to distinguish between them.*

VI. **PLEURAL FRICTION RUBS.**—The palpable friction fremitus of plastic pleurisies described on page 82, may also be appreciated by means of auscultation, and hence needs little further notice.

Pleural friction rubs, are usually double and synchronous with the respiratory acts, abolished by their cessation, and may be heard during expiration or inspiration, or both.

* *The term "subcrepitant," as occasionally applied to the râles heard over the lung in the earliest stage of pneumonia, has after careful consideration and consultation been purposely omitted by the author.*

Their **quality** may vary greatly; in some instances they closely resemble showers of fine crepitant râles, in others may be squeaking and comparable to high-pitched ronchi, finally they may be rough, coarse, and creaking.

Pleural friction is usually *accompanied by pain and tenderness, is increased on deep inspiration, intensified by pressure of the stethoscope, and is unchanged by coughing.* It will also be found that during the inspection of the chest defective movement of the overlying thoracic parietes has been noted.

In cases of interlobar or diaphragmatic pleurisy it may be inaudible; in the latter instance the pain is often referred to the abdomen and the respiration assumes a thoracic type.

It is also necessary to remember that *pericardial sounds* may at times closely resemble those produced by a plastic pleurisy. The pericardial friction rub (pages 135, 184) has also a double rhythm, but its occurrence is synchronous with the cardiac movements. It is always precordial in location, and continues even if the patient holds his breath, and may thus be contrasted with simple pleural friction. It is also intensified by pressure and is apparently of very superficial origin.

It must be remembered that a *dry pleurisy* and its attendant rub *may be present over the cardiac area even during cessation of respiration,* in which case it is due to the communicated movements of the heart and takes on a rhythm synchronous with the cardiac cycle. This sound, however, will vary in intensity during the respiratory acts.

Finally a systolic *cardio-pulmonary murmur* may be

produced in the cardiac lappet by the contractions of the heart forcing the air from the overlying lung in a series of jets coincident with the ventricular systoles.

VII. **OTHER PHENOMENA.**

(1) **"Metallic tinkling"** refers to distant high-pitched sounds or râles found during respiratory movements over large cavities in the lung and pneumothoraces, due to disturbances of the fluid contents set up by the movements of the chest. It resembles that sound produced by striking a thin empty glass with a metal rod (Laennec).

(2) **"Bell"** or **"coin sound"** is a clear, ringing note heard during percussion over air-containing cavities (especially over pneumothoraces) when two coins are used as plessor and plexímeter (Trosseau), the stethoscope meanwhile being also applied over the space.

(3) **Succussion** or "splashing" sounds may be heard over cavities containing both air and fluid while the patient is shaken from side to side (hydropneumothorax).

(4) **Muscle sounds** are soft and rumbling; they are continuous and may thus be differentiated from those of pleural or pulmonary origin.

E. **PALPATORY PERCUSSION** is usually practised by percussing directly over the chest, with two or more fingers, using no plexímeter, in the same manner as in the immediate method, except that instead of the plessor being sharply lifted off the surface after the blow, the fingers are allowed to remain for several seconds in contact.

By this maneuver we are better enabled to detect the degree of **"resistance"** of the thoracic wall, which phenomenon is of the greatest value in the differen-

tiation of pleural effusions from pulmonary consolidations.

The phenomenon of *myotatic irritability* is seen not infrequently; in it the muscles covering the chest wall, especially the pectorals, a moment after being struck by the plessor, are seen to pass into a state of transitory contraction; this sign has, however, no diagnostic significance.

The "mediate" method may also be employed, the plessor again being left in contact with the pleximeter, the fingers, however, always being employed.

F. **AUSCULTATORY PERCUSSION** is used in mapping out certain organs (especially the heart, liver, spleen and stomach) and in outlining the boundaries of tumors, cysts, areas of consolidated lung and pleural effusions.

The stethoscope is applied over the part under examination, light percussion is carried out in radiating lines from the point of application of the instrument, and note is made of the points where an alteration in the tone is detected.

Light scratching movements of the fingers may be used in place of the percussion strokes.

G. **MENSURATION** is applied to the thorax in order to determine its *circumference*, and *semi-circumference*, especially at the level of the nipples, measuring from the vertebral spines to the mid-sternal line and comparing the two sides. It is also used to investigate the amount of the *respiratory excursion* or expansion.

It should be remembered that the *capacity* of the right half of the thorax usually exceeds that of its fellow, so consequently a slight difference in the measurements of the two sides may not be abnormal;

that during quiet respiration the amount of *expansion* as measured by a tape does not exceed 1/4 inch, and finally that during forced inspiration a difference of from 2 to 5 inches may be found in the adult male.

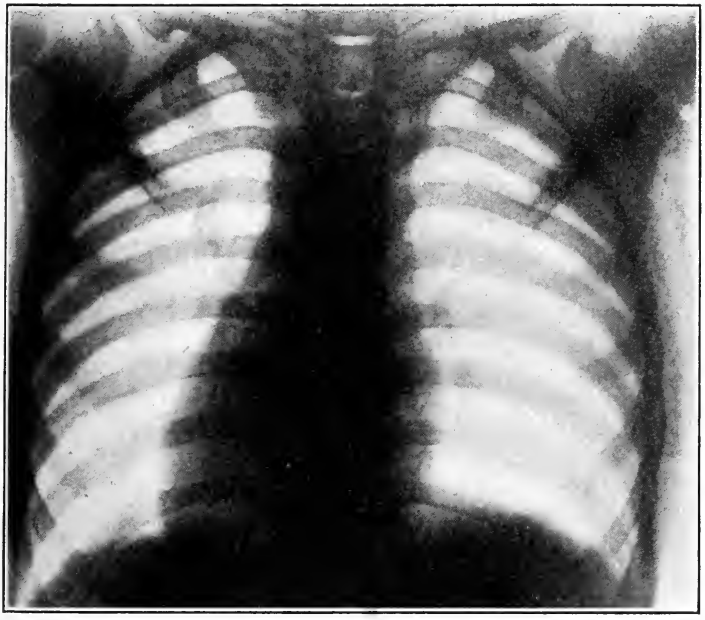
For permanent recording of not only the circumference of the chest but also of the shape of its cross-section a **cyrtometer** is used. It is an instrument consisting of two strips of flexible sheet lead about 1/2 inch in width, joined at two of their ends by means of a hinge. These strips are moulded accurately to the outline of the chest, the arms of the instrument being of sufficient length to meet in front of the sternum, the hinge meanwhile lying over the vertebral spines. The cyrtometer, which is thus seen to resemble a pair of calipers, is then carefully removed, laid on a sheet of paper, on which its outline is traced, and an accurate and permanent record of the condition of the two sides of the thorax thus obtained.

H. **RADIOSCOPY** has recently been used with considerable success, in the detection of pulmonary disease, especially in the recognition of apical lesions and mediastinal lymphadenitis.

To the courtesy of Dr. H. C. Parsons of Toronto the author is indebted for the reproduction of Plates I, II, III. Plate I shows a skiagraph of a normal child for comparison with Plate II, which was taken from a child, the subject of tuberculous disease of the apex of the right lung, and with Plate III in which both apices are seen to be extensively involved. In both these latter children tuberculous disease of the bronchial and mediastinal glands is present, as evidenced by the shadows lying to the right of the vertebral column.

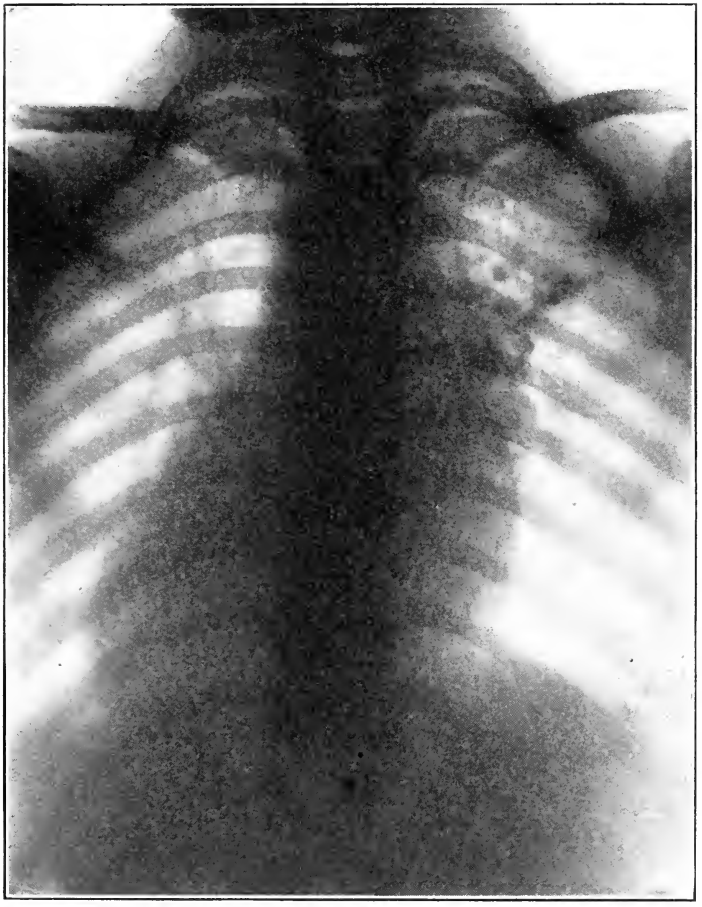
I. **THE SPUTUM** (see Appendix).

PLATE I.



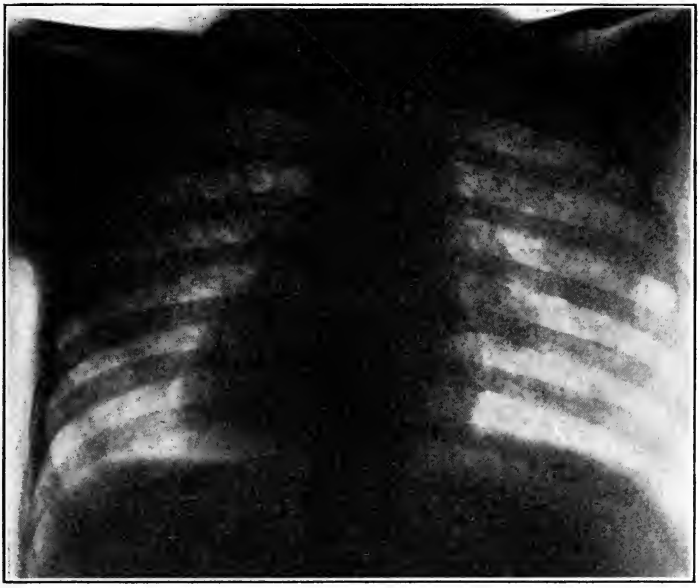
Radiograph of normal thorax of boy aged 10 years, taken from dorsal aspect of trunk. (*H. C. Parsons, M. R. C. P. (Lond.), Toronto.*)

PLATE II.



Radiograph of thorax of boy, age 8 years, showing shadows due to tubercular infiltration of apex of right lung, and also to the right of the spine shadows, due to enlarged tubercular bronchial and mediastinal glands. (*H. C. Parsons, M. R. C. P. (Lond.), Toronto.*)

PLATE III.



Radiograph showing extensive tubercular disease of upper lobes of both lungs, and also of the bronchial and mediastinal glands, from girl, age 12 years. (*H. C. Parsons, M. R. C. P.* (*Lond.*), *Toronto.*)

CHAPTER VII.

CIRCULATORY SYSTEM.

A. **INSPECTION.**

In this section we shall confine our attention to the inspection of the precordial area and of certain visible phenomena which although not present over this region, are nevertheless intimately connected with the circulatory system, noting particularly the condition of the superficial blood-vessels and the presence of thoracic, abdominal, and cervical pulsations, due to disease, displacement, or anomalies of the circulatory organs.

I. **THE PRECORDIA.**

(1) **Contour.**—The condition of the precordia, or that area of the anterior chest wall which lies in direct relation to the heart, must first be investigated. Normally its contour should be even and rounded, showing no excessive bulgings, retractions, or pulsations except those of the normal apex beat.

(a) **Abnormal prominence** may be due to a congenital anomaly, to distortion or disease of the chest wall itself (scoliosis, tumor, etc.), of the pericardium (effusion of fluid), of the heart itself (hypertrophy and dilatation), of the great vessels (aneurism), or, finally, to the presence of intrathoracic neoplasms.

It will now be seen that it is necessary not only to note the presence of abnormal fulness, but to determine whether its origin is superficial (parietal tumors, abscesses, etc.), osseous (due to alteration in the

position of the ribs, as from scoliosis or intrathoracic pressure), or due merely to bulging of the intercostal spaces (pleural or pericardial effusions).

(*b*) **Flattening or shrinking** of the precordia is also found to be either congenital in origin or due to scoliosis, or secondary to external pressure from instruments used in special trades, or to an indrawing of the chest wall following a shrinking of intrathoracic organs (chronic adhesive pericarditis or pleurisy, retraction of lung tissue).

Here also it is necessary to note whether the area as a whole shows retraction or if the shrinking is limited to the intercostal spaces, and also to determine the influence of cardiac and respiratory movements on both the bony and soft parts.

(2) **The apex beat** or point of maximum intensity of the cardiac impulse is found to lie in the adult male in the *fifth intercostal space, 1/2 inch internal to the mid-clavicular line* (3 1/2 inches from the centre of the sternum), and to be visible over a circular area having a diameter of about 1 inch. Clinically its position closely corresponds to the junction of the lower and left borders of the heart.

It is also found in the normal subject that slight alteration in its position takes place according to the posture assumed, the apex gravitating toward the dependent side of the body, *fixation* occurring where the pericardium is adherent to the chest wall.

In *infants* and young children it is usually found in the fourth space, while in the *aged* it sinks below the level of the sixth rib, in both cases, however, retaining a relationship to the mid-sternal and mid-clavicular lines which, allowing for differences in the size of the

thorax, corresponds relatively to that ascribed to the normal adult.

In disease the **extent of the impulse** may be diffuse and visible over a considerable area or, on the other hand, it may be invisible. It must not be forgotten that in healthy individuals with thick chest walls, and in those who possess a congenital skeletal formation which throws the apex behind a rib, it may also be almost imperceptible, while, on the other hand, in the emaciated it may appear to be abnormally extensive even in the absence of cardiac or pulmonary disease.

Disappearance of the apical impulse in disease may be due to weakened ventricular action or to the presence of pleural or pericardial effusions intervening between the heart and the thoracic wall. *Diffuse pulsation*, but normal in position, is found in healthy but thin-walled chests, and also during periods of forcible, excited heart action.

Inspection thus gives us information as to the position and extent of this pulsation, but for the confirmation of our findings and for the purpose of investigating its force we must bring into play the art of palpation, so that in order to avoid repetition we will describe the various abnormalities of the apex beat, such as alterations in position, force, extent, time, and rhythm under that section.

(3) **Abnormal Precordial Pulsations.**—After having located and investigated the apical impulse, search next for pulsations over the remainder of the precordial area.

In the precordial region abnormal *systolic** impulses

*The term "systole" is used clinically to define that period of the cardiac cycle which is ushered in by the first sound and which terminates with the commencement of the second.

may be found over any part. Their significance will be described under palpation.

We must also particularly examine the suprasternal notch (aneurysm of the aortic arch and pulsation of the thyroidea ima), the epigastrium (right ventricular hypertrophy, pulsating liver, or communicated pulsations from the abdominal aorta), and the second intercostal spaces of both sides where aneurysms and enlargements or displacements of the auricles may give rise to visible systolic impacts.

Occasionally impulses *diastolic* in time may be detected in the latter situations and, when present, are usually due to sudden and forcible closure of the aortic or pulmonary valves.

(4) **Systolic retraction** of the precordial intercostal spaces is a phenomenon most characteristically seen in cases when the *pericardium is adherent to the chest wall.* At times the adhesions are of such an extent as to bring about not only a recession of the spaces, but of the precordial thoracic wall itself (page 130).

In rare instances of old-standing fibrous pericarditis the epigastrium even may show systolic depression.

If, however, the apex beat be due to the right ventricle, systolic *retraction may occur, even in the absence of pericarditis.* This results in an "inverted cardiogram" and should always be kept in mind (page 12).

II. **THE BLOOD-VESSELS.**

(1) **The Arteries.**—Certain superficial vessels should next receive attention, noting first abnormal prominence and tortuosity, especially of the temporal arteries (arteriosclerosis), and second, excessive visible pulsations particularly in the common carotids, as seen

most characteristically in aortic regurgitation and in cases showing defective vasomotor tone.

(2) **The Veins.**—General venous *congestion with enlarged and tortuous* vessels is seen in its most marked form as a result of congenital cardiac anomalies.

In acquired valvular lesions the veins of the extremities frequently show evidences of enlargement. The lower half of the trunk and the legs are most affected where blockage of the inferior vena cava is present, while the upper extremities, head, and neck are alone involved if a mediastinal tumor compress, the superior vena cava.

The veins of the precordia may be markedly congested where enlargement of the heart exerts pressure on the mammary and intercostal vessels; edema over the sternum is also not infrequently present in the same class of cases.

Venous pulsation is best studied in the external jugulars. *Undulation* due to a slightly retarded emptying of the vein during auricle systole and *communicated movements* from the carotids must be carefully distinguished from *true venous pulsation.*

In order to differentiate one from the other *clinically* it is necessary to empty one of the veins from below upward by gently passing the fingers along it from the root of the neck toward the head, and then keeping its upper end closed by continuous pressure. During this maneuver it should be noted whether the vessel remains collapsed or refills gradually and slowly from collaterals, or if, on the other hand, it is distended from below by a series of pulsations such as occur when the valves guarding the tricuspid orifice are so altered as to allow of regurgitation of the ventricular contents.

A *centripetal venous pulse* is found where the arterial pulsation is propagated through dilated arterioles and capillaries directly to the peripheral veins themselves, and hence is accompanied by capillary pulsation. This condition is easily distinguished from those above described which chiefly involve the veins nearest the heart.

Freidreich's sign refers to a diastolic collapse of the cervical veins due to aspiration or rebound of the right auricle and *Kussmaul's sign* to an inspiratory swelling of the same vessels, both of which phenomena are found where mediastino-pericardial adhesions are present, (see subsection (4), page 128) as is also *Broadbent's sign* which consists in a systolic retraction in the tenth and eleventh intercostal spaces in the vicinity of the angle of the scapula. This latter phenomenon is produced through the cardiac contractions pulling on the posterior attachments of the diaphragm.

It is also possible by the use of a *polygraph* to obtain graphic tracings of venous arterial and cardiac pulsations simultaneously. The application and results of this method will be discussed later.

(3) **The Capillaries** are next inspected in order to determine if evidences of pulsation are present.

Capillary pulsation is best seen under the nails after producing a temporary anemia by gently pressing on the tip of the nail under observation and noting if the blood returns gradually as in the healthy individual, or in jets as we find in aortic regurgitation and diseases such as neurasthenia which involve a loss of vasomotor tone.

These pulsations may also be rendered more obvious by drawing the finger-nail of the examining finger

firmly across the skin of the subject's forehead and observing the manner of the return of blood to the ischemic area.

Evidences of sluggish peripheral circulation or *passive congestion*, whether from defective propulsive forces or obstruction to the return of the venous blood, are detected by the cold, clammy, edematous and cyanotic extremities present in these conditions. It must be remembered, however, that cyanosis may also be due to toxemias and certain congenital cardiac anomalies, as well as to defective aeration of the blood.

Lastly, it is not uncommon in certain hepatic and cardiopulmonary diseases to find a band of enlarged capillaries and venules around the chest at a level corresponding to the attachments of the diaphragm, due probably to pressure on the azygos veins or to portal obstruction.

B. **PALPATION.**

I. **THE PRECORDIA.**—After having completed the inspection of this area it is necessary to confirm our findings by means of palpation. Following the same order as in the section on inspection, first confirm or modify our previous observations regarding abnormal prominence or shrinking of the precordial region, paying strict attention to the relation of the ribs and interspaces to any pathological conditions present.

Next examine for the presence of certain signs which can be detected solely by palpation, especially thrills and the quality of visible pulsations, and then consider the examination of the pulse, the estimation of the blood pressure, and that of the body temperature.

Under the section on the pulse the use of the poly-

graph will be taken up; the following phenomena are, however, best appreciated by means of palpation.

(1) **The Apex Beat.**—The apex beat must first be located by means of palpation and the extent over which it is appreciable determined, thus confirming or modifying our previous observations.

Second, we estimate the **quality** of the impulse, differentiating the *heaving, forcible* quality found where the cardiac muscle is hypertrophied, from the *weak, slapping*, diffuse apical impulse of cardiac dilatation, and in cases of mitral stenosis noting the *sharp, thumping* or impact so characteristic of this special variety of valvular disease.

Diminution in the force of the impulse may be due to myocarditis, fatty degeneration, or, on the other hand, to extra-cardiac lesions such as pleural or pericardial effusions and emphysema.

It is necessary now to discuss in detail the conditions which cause **displacement of the apex beat.**

(a) The impulse may be found displaced *to the left* of its normal position as a result of the action of extrinsic influences such as a tumor of the right lung, a right pleural effusion or pneumothorax pushing the heart from its normal position; or of the presence of fibrosis and retraction of the left lung or pleuropericardial adhesions dragging the organ toward the diseased side.

(b) Displacement *to the right* may occur from the opposite conditions to those just described in subsection (a), in both of these cases presuming that little or no vertical displacement is present.

(c) The impulse may be displaced *downward and to the left* from hypertrophy of the heart, especially where

the left ventricle is most markedly involved, or from pressure of an aneurysm of the aortic arch.

(d) Displacement *upward* may be due to pericardial effusions, abdominal tumors or ascites exerting pressure on the diaphragm from below, or may be secondary to retraction of the upper lobe of the left lung.

(e) *Congenital anomalies* of the thoracic viscera may be responsible for an altered position, especially situs viscerum inversus in which the apex lies to the right of the sternum, as also may *acquired thoracic deformities*.

(2) **Abnormal pulsations** other than those due to apical impulses are frequently found. Look especially in the *second left interspace* for presystolic pulsations, due to enlargement of the left auricle, or systolic ones from dilatation of the pulmonary artery.

To the *right of the sternum* search for those secondary to displacement or enlargement of the right chambers of the heart or to aneurysms of the ascending part of the aortic arch, remembering in this latter instance that erosion of the ribs and sternum resulting in the appearance of a tumor, showing an expansile pulsation, may be found.

Abnormal pulsations may also be seen in the third and fourth left interspaces, where retraction of the left lung has taken place.

(3) **Other Phenomena.**

(a) **Endocardial thrills** may be found in cases of valvular disease and with dilatation of the aorta. They are *due to altered physical relations between the circulating blood, the orifices through which it passes and the cardiac chambers*, and are best exemplified in cases of mitral stenosis where the blood passes from a

dilated left auricle, through a stenotic or narrowed mitral orifice, to the relatively small ventricular cavity.

They bear the same relation to murmurs or "blood sounds" that vocal fremitus bears to vocal resonance, in being due to palpable vibrations.

As regards the *theories* advanced to explain their production, that most commonly accepted is that they are due to slow and forcible vibrations carried from the "fluid veins" (page 170) of the circulating blood by the cardiac walls to the precordia. It has also been suggested that in some cases an additional vibratory element may be superadded by the muscular contractions of the heart walls, which latter in the majority of cases show dilatation.

These thrills are in quality "purring," and may be in time *systolic*, and due then to aortic or pulmonary stenosis, mitral or tricuspid regurgitation, or to an aneurysm of the aorta.

In aortic stenosis two apparently separate thrills may be present, one at the base transmitted directly from the vessel itself, the other at the apex propagated by the ventricular walls. Both are in time strictly systolic.

Diastolic thrills may be found in cases of aortic or pulmonary insufficiency; those limited to late diastole, *presystolic* thrills as they are usually called, are found in cases of mitral or tricuspid stenosis; but of all the cases above enumerated the thrill of mitral stenosis is by far the most commonly encountered.

Systolic thrills may also be *muscular in origin* and are sometimes found over hearts showing uncomplicated hypertrophy. With true endocardial thrills the

fremitus is limited to the interspaces, in the other form the ribs themselves are found to vibrate.

Congenital heart anomalies are also at times responsible for thrills which may be present during any period of the cardiac cycle.

(b) **A Friction rub or fremitus,** as it is occasionally called, is found in acute plastic pericarditis. Friction is generally transient, for as soon as sufficient fluid accumulates in the sac the two layers of the pericardium are separated and the rub disappears.

In time they are usually double, of a "*to-and-fro*" *rhythm* synchronous with cardiac systole and diastole. They are always audible, limited strictly to the precordia, frequently accompanied by pain, and give the impression of a very superficial origin.

For details regarding their differentiation from other similar phenomena the reader is referred to page 114.

(c) **Fluctuation** in the interspaces of the precordia is an extremely rare physical sign, but when found it is usually indicative of the presence of fluid in the pericardial sac. It is of little diagnostic value on account of its late appearance.

(d) **Diastolic shocks** or impulses are most commonly met with in either of the second interspaces in close proximity to the sternal margin, and when found in these positions are due to sudden and forcible closure of the pulmonary or aortic semilunar valves, secondary to excessive pressure in, or aneurysmal dilatation of, the respective vessels. Occasionally also they are due to the sudden ventricular diastoles which occur in cardiac hypertrophy. In these latter cases the phenomenon is present most markedly over the central zone of the precordium.

II. **THE PULSE.**—From an examination of certain accessible peripheral vessels we are enabled to add to the information we have already elicited concerning the condition of the circulatory system. One may either employ the finger to palpate these vessels, or may make use of one of the various forms of sphygmograph, by means of which accurate and permanent records of the pulse-tracings are obtained.

Not only can we make graphic tracings of the various phenomena which occur in the peripheral arteries, but by means of certain instruments (polygraphs, cardiographs, etc.) we may simultaneously obtain tracings from the apex beat or other thoracic pulsations, the carotid arteries and jugular veins; from which tracings we are enabled to obtain information regarding the action of the heart itself, the relation of the events occurring during the cardiac cycle to each other, as well as to those observed in the vessels.

I shall first describe the ordinary clinical method of palpating the vessels, and then give a short résumé of James Mackenzie's observations and the deductions he has drawn from his polygraphic tracings. Since the apical and venous-pulse tracings are so intimately associated with those of the peripheral arteries, they will be taken up at the same time.

(1) **Digital Examination.**—Certain phenomena are best detected by means of this method and these will now be described. Others which cannot be fully appreciated without the use of a polygraph will be dealt with in the following section.

The **radial arteries** are those usually selected for examination. The subject should preferably be sitting or reclining, but it is always advisable to compare

the findings obtained with those found on assuming the erect posture, particularly in the investigation of cases of aortic insufficiency.

It is also necessary to examine both radials in order that anatomical anomalies or the asynchronism of aortic aneurysms may not be overlooked; and also to compare the relative volumes of the pulses of the upper and lower extremities after making due allowance for the effect of distance from the heart and the size of the vessels under examination. Thus, the pulse waves in the legs may be fuller than those in the arms where aortic stenosis coexists with a patent ductus arteriosus, some of the blood in this event being pumped directly from the pulmonary artery into the aorta below the point of obstruction.

To obtain the best results it is necessary that the forearm be pronated and the muscles relaxed, also that the three middle fingers of the examining hand be applied to the vessel under examination.

It is also frequently of service to examine and compare not only the radials but also the **carotids and temporals,** as asynchronism is not infrequent in aneurysmal dilatations of the aortic arch.

It must also not be forgotten that even in the normal individual the volume of the *pulse during inspiration* is greater than during the expiratory act, and that during deep and prolonged inspirations the cardiac contractions usually become less frequent.

As several points are to be noted during this examination, it is well always to follow the same sequence in routine investigations of the pulse in order to avoid possible omissions.

(a) **The rate** per unit of time is first noted. In the

healthy adult the rate per minute varies from 70 to 80, a normal increase of from 5 to 10 beats taking place on assuming the erect posture and during gentle exercise, while in sleep a decrease of from 10 to 15 occurs. The fact that certain individuals have normally a pulse rate varying widely from the limits already mentioned must also be taken into consideration.

At the end of fetal life the ventricular contractions occur at the rate of from 130 to 140 per minute, the beats being usually somewhat slower in males than in females. Immediately after birth they fall to between 125 and 130. By the end of the second year they reach 110, by the fifth 100, by the eighth 90, by the twelfth 80 per minute.

The pulse rate may be found to vary enormously in disease, occasionally in *bradycardia* falling to between 15 and 20 beats per minute.

On the other hand, in *tachycardia* the beats may follow in such quick succession as to be uncountable by palpation. An accurate estimate without the aid of a sphygmograph is impossible when the pulse rate rises above 150.

(i) **Bradycardia,** or abnormal slowing of the pulse, may be symptomatic of certain diseases. It is frequently found during convalescence from pneumonia, typhoid, diphtheria, influenza and the acute infectious fevers, in gastrointestinal disease, myocarditis and coronary sclerosis, asthma and emphysema. It may also occur as a result of certain toxic agencies, such as digitalis, alcohol, tea, coffee, etc.; cerebral disease, especially tumors, epilepsy, and hemorrhage; lesions of the cervical cord, vagus nerve; also in the neuroses and in jaundice.

It should also be noted at this point that in lesions of the "bundle of His" *Stokes-Adams' syndrome* occurs, in which the ventricular impulse and consequently the radial pulse, occurs only once, to two, three, or four auricular beats. This phenomenon will be more fully dealt with later (page 154), as also will those conditions in which, although the ventricle contracts regularly, an occasional impulse is of insufficient force to reach the radial artery (asystole).

(ii) **Tachycardia,** or excessive rapidity of the cardiac systoles, may be symptomatic of febrile states, cerebrospinal lesions such as tumors and degenerations, mental excitement and the neuroses (masturbation, hysteria, etc.), pneumogastric paralysis from pressure or neuritis, reflex irritation of gastrointestinal or peripheral origin, and of certain diseases of the generative organs. It is also a prominent sign in some toxemias, especially those of alcohol, tea, coffee, tobacco; in cases of hyperthyroidism (exophthalmic goitre), and it also may be found after violent muscular exertion.

Simple palpitation is a paroxysmal tachycardia in which the normal or sinus rhythm is present; no irregularity in the pulse is noticeable, except for the occasional occurrence of an extra-systole (Mackenzie).

True paroxysmal tachycardia refers to irregular attacks of increased rapidity in the heart's action in which the muscular contractions do not follow the normal sequence, and in which the ventricles contract simultaneously with the auricles, whose walls it has been suggested are in a state of fibrillary contraction.

(b) **The Quality.**—Simple inequality in the force of successive beats is recognizable on palpation, but for

accurate results it is necessary to take tracings with one of the various forms of sphygmographs.

In the normal pulse we note that the impulse as it reaches the palpating finger gradually fills the vessel, neither too abruptly nor in too tardy a manner. Distension of the artery being completed, the maximum pressure of the contents is sustained for a barely perceptible interval of time, after which a fall of pressure takes place, occurring somewhat more gradually than the systolic wave which filled the vessel.

Therefore to properly **analyze the pulse** it is necessary to note the manner in which the pressure rises, the duration of the fastigium or period of maximum pressure, and the time occupied by falling blood pressure.

First, as regards the *systolic wave* which in sphygmographic tracings is shown by the "up-stroke." Normally this should show a regular, continuous rise, but in certain diseases this wave occurs in too abrupt

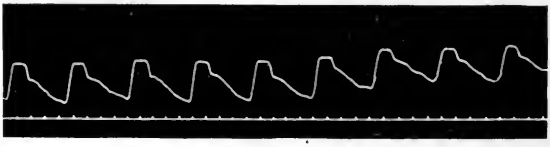


FIG. 8.—High tension pulse. (Blood pressure 240 mm. Hg.)

a manner, giving rise to sudden impulse under the finger and to an almost vertical sphygmographic "up-stroke," such as we find in cases where blood pressure is low (below 90 mm. of mercury).

The opposite condition with a slow, palpable rise, a slanting up-stroke, and a sustained summit is most commonly seen in individuals whose blood pressure is excessive (Fig. 8), where obstruction to the en-

trance of blood into the aorta (stenosis) exists (Fig. 9), or when an aneurysm is present.

Consider now the *period of maximum pressure* as represented by the top or "apex" of the sphygmographic pulse curve and note whether it shows a tendency to shortening, as evidenced by a sharp or pointed

FIG. 9.—Aortic stenosis and mitral regurgitation.

apex and precipitate down-stroke with a suddenly collapsing pulse, such as is typically felt in Corrigan's "water-hammer" pulse of aortic regurgitation. This phenomenon may be accentuated on raising the arm above the head (Fig. 10).

The opposite condition as shown by a well sustained apex and tardy down-stroke is found where arterial pressure is high and long maintained, as, for instance, in cases of arterial hypertension (Fig. 8).

FIG. 10.—Aortic regurgitation.

Next examine the period during which the *blood pressure is falling*, shown in the tracings by the "down-stroke." This may be sudden, with a vertical drop from the apex or highest point reached, or sloping and prolonged as when the pressure falls gradually.

At times, instead of showing a regular even descent, the down-stroke may be interrupted by a secondary wave, felt by the finger as an impulse following the primary systolic impact. That most commonly encountered is termed the "*dicrotic wave*" (Fig. 11) and occurs in pulses of low tension, particularly in typhoid fever. It is most readily recognized when the lightest pressure is exerted by the palpating finger,

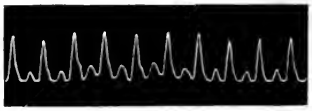


FIG. 11.—Dicrotic pulse.

and is due to reflection of a blood wave, produced by the impact of the back rush of blood on the closed aortic valve, along the arterial stream.

(c) **The Walls of the Arteries.**—The radial artery should be carefully palpated and "rolled" under fingers where it lies in contact with the radius. In every case it is advisable to determine also the physical state of the superficial temporal vessels.

The normal radial artery should be impalpable in individuals under about sixty years; thereafter slight thickening is not uncommon and is of slight clinical significance unless accompanied by evidences that the internal vessels of the body are also involved. Let me here emphasize the fact that there is **often no relationship between the degree of peripheral and that of central or visceral arteriosclerosis.**

Marked thickening or tortuosity of the superficial vessels, frequently visible as well as palpable, is found

in arteriosclerosis. In some cases a secondary deposit of lime salts produces what is known as a *pipe-stem artery*. Calcification may occur in placques or may be evenly distributed throughout the circumference of the vessel wall. It is well to remember that phlebosclerosis is not infrequently found.

(d) **Rhythm.**—Under this heading we must note deviations from the normal, rhythmical sequence of successive beats, as shown by irregular intervals of time intervening between the pulsations, the occasional complete dropping or loss of a beat, and also by inequality in force in successive impulses. This subject will be dealt with in a subsequent section.

Arrhythmia refers to simple irregularity in the sequence of the pulse beats, and *intermittence* to the complete and occasional loss of a radial pulsation. These conditions are frequently associated and may be found in nervous individuals, in cases of gastric disturbances, gout and neuro-muscular cardiac lesions, but occasionally are apparently due to the altered atmospheric pressure as found before thunderstorms.

Combined irregularity of both rhythm and force of successive beats is much more common than simple instances of arrhythmia. Here the differences in equality are due to the varying strength of the ventricular systoles, and are hence indicative of neuro-muscular cardiac defects. In its severest forms this combined irregularity has been termed clinically *delirium cordis*.

Pulsus paradoxus is most characteristically seen in cases of pericardial adhesions. In it the beats during inspiration are small in volume and rapid in rate, occasionally the radial pulsation completely disappearing at the end of a forced and maintained inspira-

tion, the pulse becoming fuller and less frequent during the act of expiration.

(e) **The Arterial Blood Pressure.**—Regarding its estimation by the finger, incessant practice is necessary to fix in the mind a standard of *normal pressure which varies from* 90 *to* 130 *mm. of mercury*, and to be able to detect variations.

For accurate results the use of a **sphygmomanometer** is essential. By its aid we find in low-tension pulses that the pressure may fall to 60 mm., while in the opposite condition, particularly in arteriosclerosis and renal disease, it may rise to 250 mm.

The minimum or diastolic, as well as the maximum or systolic, pressure must always be determined, in every case making due allowance for the condition of the vessel wall, as when this is markedly thickened by sclerotic changes observations regarding peripheral blood pressure are practically valueless.

(2) **Instrumental Methods.**—Certain instruments, usually termed polygraphs, have been designed which record graphically and simultaneously the movements which take place over the precordia (apex beat, etc.), hepatic, venous and arterial pulsations; since descriptions of the technic of their application accompanies each apparatus no reference need be made to this subject.

In order to appreciate the significance of the tracings we obtain it is necessary, on account of the recent advances which have been made, to review briefly certain fundamental principles connected with the mechanism of the heart's action, after which I shall attempt to epitomize James Mackenzie's studies on the heart's action, particularly those bearing on the subject of arrhythmia.

(a) The term **cardiogram** is applied to a graphic tracing of the heart movements obtained by applying one tambour of the polygraph over the precordia, usually over the apex beat.

The term apex beat is generally taken to mean that impulse produced by "the lowest and outermost point of the heart which strikes against the chest wall;" this part is usually formed by the left ventricle. It is well to remember that the term "point of maximum intensity" is frequently used to describe this precordial

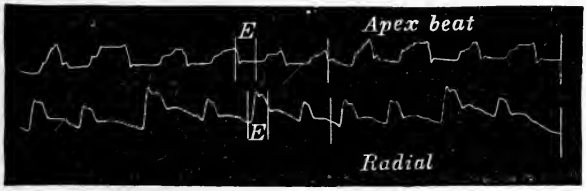


FIG. 12.—Simultaneous tracings of the apex beat and of the radial pulse. The rise in the apex tracing resembles the usual characters present in a tracing of the apex beat due to the left ventricle. On analysis it is found that the elevation is during the diastole, and the fall (E) during the systole, of the ventricle. (*Mackenzie.*)

pulsation, and it probably gives a more accurate conception.

Occasionally, however, in great dilatation or displacement of the heart the apical impulse may be due to the right ventricle, in which instance retraction or indrawing of the chest wall may occur in place of the normal impulse resulting in an "*inverted cardiogram*" (Fig. 12).

Clinically, therefore, every case showing systolic apical retraction, or an inverted cardiogram, is not necessarily due as was formerly believed to pericardial adhesions.

The **interpretation** of a normal apical cardiogram will now be studied (Fig. 13). It will be seen that the tracing may be divided into four distinct parts, each bearing a definite and constant relation to certain events in the cardiac cycle: they are (i) the presphygmic interval, or that period during which the ventricular muscle is commencing to contract, which forms the sphygmographic up-stroke (D) and (ii) the period of ventricular outflow or the systolic plateau (E). (iii) Ventricular

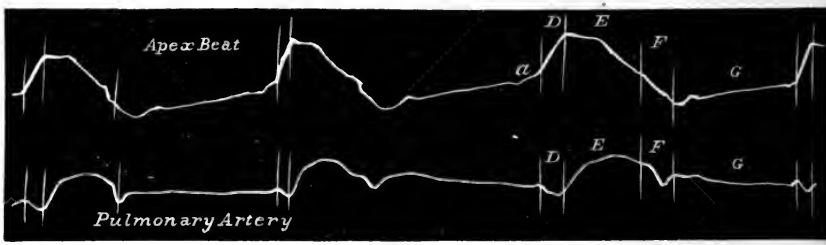


FIG. 13.—Simultaneous tracings of the apex beat and of the pulsation in the pulmonary artery. The letters represent the time during which the ventricle is passing into systole (D), emptying (E), relaxing (F), filling (G). *a* represents the small wave due to the auricular systole. While this tracing was being taken the cylinder was rapidly rotated. The letters D, E, F, have reference to the same periods in the cardiac revolution as in Fig. 14. (*Mackenzie.*)

relaxation corresponds to the down-stroke (F) and (iv). The period of filling of the ventricles to the gradual ascent which precedes the presphygmic interval (G).

In many, but not all, apical tracings a small secondary wave (a) may be found preceding the commencement of the ventricular systole by 1/10 of a second; it is due to the auricular contraction. In cases of "heart-block" this auricular wave may appear during the ventricular pauses; it may be absent however, in the tracings from a normal heart.

(b) An **arterial sphygmogram** may be defined as a diagrammatic representation of the variations of pressure within an artery, of the rate and rhythm

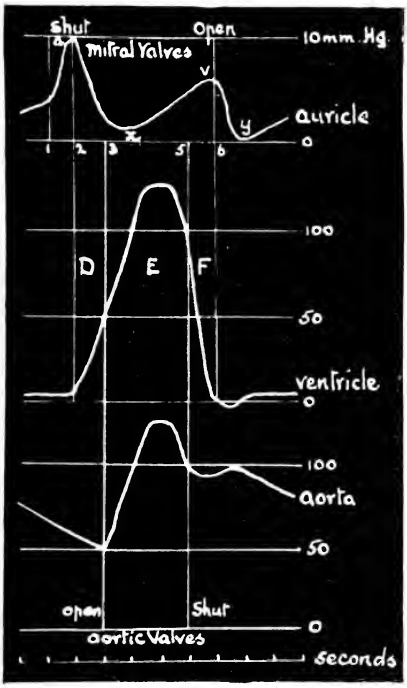


FIG. 14.—Semi-diagrammatic representation of the auricular, ventricular, and aortic pressures during one cardiac revolution. D, the presphygmic period of the ventricular systole; E, the sphygmic or pulse period; F, the postsphygmic period. The figures 1, 2 3, 5, and 6 have the same significance as those in Fig. 16. The divisions on the bottom line represent tenths of a second. (*Mackenzie after Frey.*)

of the cardiac systoles, and shows the sequence of certain events occurring in the cardiac cycle, as well as the character of the arterial blood pressure (Fig. 14).

The **interpretation** of a normal radial pulse tracing

(Fig. 15) shows that (i) during ventricular systole there occurs an abrupt rise or up-stroke, then a slight fall followed by a continuation of the primary wave at about the same level, forming the systolic plateau. This small extra wave is probably due to an instrumental defect; really the up-stroke and plateau together correspond to the period of ventricular systole (E). (The old term "predicrotic" or "tidal" waves should therefore be abandoned.) (ii) The diastolic period, or that during which arterial pressure is falling (G), is

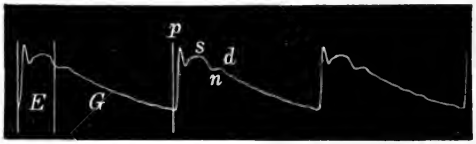


FIG. 15.—Sphygmogram of the radial pulse. The space E is the period of ventricular systole when the aortic valves are open; the space G the period of ventricular diastole; *s* is the pulse wave due to the ventricular systole; *n* the aortic notch; *d* the dicrotic wave; and *p* a wave due to instrumental defect. (*Mackenzie.*)

represented by the down-stroke of the tracing, which down-stroke is normally interrupted by a secondary or "dicrotic" wave (d). This impulse follows a rise in arterial pressure, secondary to a sudden tension of the aortic valves, set up by a negative pressure-wave due to ventricular relaxation, which wave is then reflected by the valve curtains as a positive one into the arterial system. (Fig. 11.) Other secondary waves may be seen, but they apparently have no clinical significance. (*Mackenzie.*)

(c) A **venous sphygmogram** is generally obtained from the jugular vein. By its aid we are enabled to gain information regarding the movements of the right auricle and ventricle, but in order that our observations be of value it is necessary to have simultaneous tracings

of the apex, radial, or carotid pulses in order to have a standard for timing any venous waves present.

Interpretation of the normal, negative, or auricular form of venous pulse presents considerable difficulty; for details the student is referred to James Mackenzie's work on diseases of the heart. The auricular wave a in figure 16 corresponds to the systole of the right auricle; a little below this c is a small secondary wave

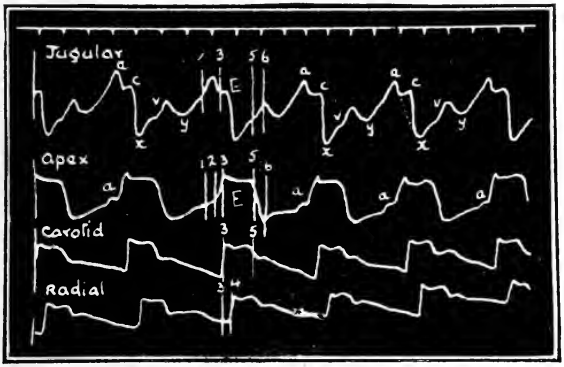


FIG. 16.—Tracings of the jugular pulse, apex beat, carotid and radia pulses. The perpendicular lines represent the time of the following events: 1, the beginning of the auricular systole; 2, the beginning of ventricular systole; 3, the appearance of the pulse in the carotid; 4, the appearance of the pulse in the radial; 5, the closing of the semilunar valves; 6, the opening of the tricuspid valves (compare with Fig. 14). (*Mackenzie.*)

due to a rise of auricular pressure interrupting the down-stroke which terminates at x. Next there occurs a rise or short up-stroke terminating at v due to the systole of the right ventricle; this termination corresponds to the opening of the tricuspid valves. The drop to y is due to the flow of auricular blood into the right ventricle after opening of the tricuspid valve; then as the ventricle becomes filled, stasis in the auricle and veins occurs causing the rise from y to

a, at which latter point contraction of the auricle occurs, thus completing the cycle. (*Mackenzie.*)

A secondary notch may occur on the ventricular wave; it probably corresponds to closure of the pulmonary valve. Another diastolic wave may be seen in slow-acting hearts shortly after the opening of the tricuspid valve; it is of little clinical significance.

The **ventricular, positive, or pathological venous pulse** (Fig. 17) is that form in which the auricular wave

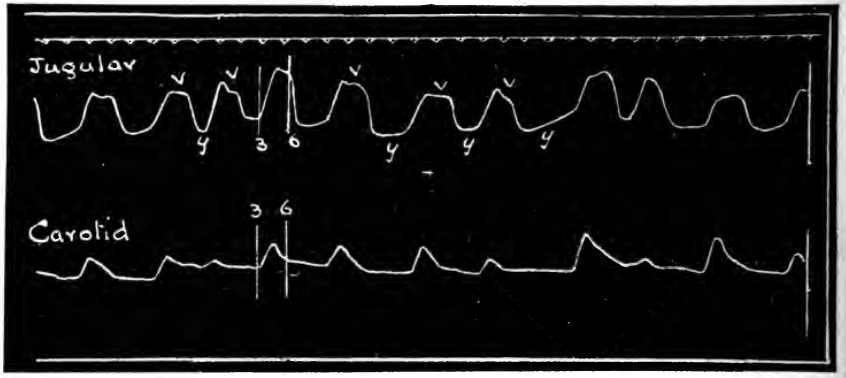


FIG. 17.—Simultaneous tracings of the jugular and carotid pulses, showing one large wave *v*, synchronous with the carotid pulse, and due to the ventricular systole, and one large fall, *y*, synchronous with and due to the ventricular systole. There is no sign of an auricular wave, and the jugular pulse is therefore of the ventricular type, and the rhythm of the heart is continuously irregular. (*Mackenzie.*)

disappears or coincides with the period of ventricular systole, there being *no sign of the auricular wave* at the normal period of the cycle. The single wave seen is due to the blood being forced back through the tricuspid orifice by the contraction of the right ventricle and obliterating the normal auricular wave. Again the rhythm is always irregular, the auricular muscle probably being in a state of fibrillation.

(d) **Arrhythmia.**—In order to appreciate the phenomenon of arrhythmia it is necessary to remember that in the **sinus venosus of the primitive cardiac tube** normally originated the stimulus necessary for a cardiac contraction, and that as development proceeded the sinus venosus became incorporated in the great veins just beyond the heart.

Normally it has been shown that *cardiac contractions originate* with a definite uniformity in a number of different sites, particularly, however, *near the mouth of the superior vena cava.* From this fact it is inferred that in these locations *remains of the original sinus venosus persist,* and that these remnants still possess the power of starting independent muscular contractions.

Recently these have been demonstrated as composing the **sinoauricular node** which is situated at the mouth of the superior vena cava, and as the **"bundle of His,"** a band of tissue which arises in the **auricular ventricular node** (which node lies near the mouth of the coronary sinus) and then passes across the auriculoventricular septum to divide into two branches which terminate as fine filaments in the ventricular muscle fibers. The fibers composing these structures are isolated by a delicate connective-tissue sheath, show a different histological structure from that of the cardiac muscle, and receive a separate blood supply.

Normally, cardiac contractions originate in the remains of the sinus venosus and are carried by the bundle of His from the auricle to the ventricle. This constitutes the ordinary *normal or sinus rhythm.*

(i) **Sinus irregularities** arise from some interference (usually nervous) with the remains of the sinus venosus, the result being a disturbance of the normal cardiac

rhythm. They are characterized by *varying lengths of the cardiac cycles*, chiefly affecting the diastolic intervals, the *quality of the successive beats, however, remaining constant.*

On auscultation the sounds are clear and the interval between the first and second is normal and regular; this type is not uncommon in adolescents, the irregularity, however, may appear only occasionally. Simultaneous tracings of the jugular pulse show that the rhythm of the auricle is similarly involved; when this is the case the *prognosis is usually excellent* and the condition demands no special treatment.

(ii) **Extra-systoles** are shown clinically by a sensation of "*fluttering*," *a* "*feeling of the heart having stopped*," or no subjective symptoms may appear.

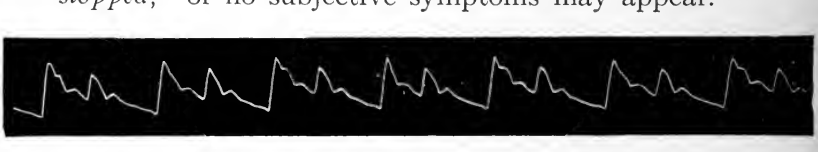


FIG. 18.—Pulsus bigeminus due to an extra-systole occurring after each normal beat. (*Mackenzie.*)

They are also shown by the occurrence of a *premature radial beat followed by an abnormally long pause* (intermittence).

Extra-systoles appear at long intervals, or regularly after one, two, three, four, or more normal beats; these latter types are termed **pulsus bigeminus, trigeminus,** etc. (Fig. 18).

If the extra-systole occur regularly after each normal beat and be of insufficient force to reach the wrist, the radial pulse will appear slow and a suspicion of the presence of bradycardia may arise, from which it may, however, be easily differentiated by auscultation.

Ventricular extra-systoles are supposed to arise in the auriculoventricular bundle beyond the auriculoventricular node. The simplest form is where a premature contraction of the ventricle is interpolated between two normal beats, the auricle preserving its normal rhythm (Fig. 19).

Auricular extra-systoles arise probably in some remnant of the original sinus venosus incorporated in the auricular wall. In these cases the heart sounds and

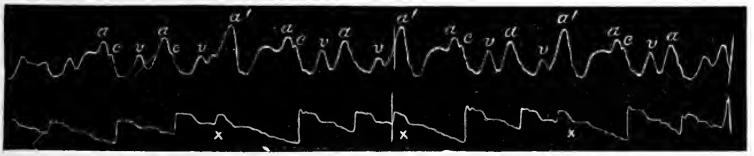


FIG. 19.—Simultaneous tracings of the jugular and radial pulses. The small beats x x x are extra-systoles. The auricle preserves its rhythm during the irregular periods in the radial pulse. The wave *a'* is the auricular wave during the premature contraction of the left ventricle. The absence of the ventricular wave *v*, after the wave *a'*, indicates that the right ventricle had contracted early, evidently synchronous with the premature contraction of the left ventricle, the large wave following *a'* being due to stasis. (*Mackenzie.*)

radial tracings are similar to those found where the extra-systole is ventricular in type; a simultaneous jugular tracing, however, shows the presence of the premature auricular systole (Fig. 20).

(iii) **Auricular fibrillation** is the term applied to a form of deviation from the normal or sinus rhythm.

In the majority of cases of continuous and disorderly irregularity, and also in true paroxysmal tachycardia, the heart beats more rapidly than normal, the auricular muscle being in a state of fibrillary contraction. This type of irregularity was formerly known as "*nodal rhythm.*"

It is found most frequently in cases of arterio-sclerosis and rheumatic myocarditis; and is of more *serious import* than simple sinus rhythm irregularities.

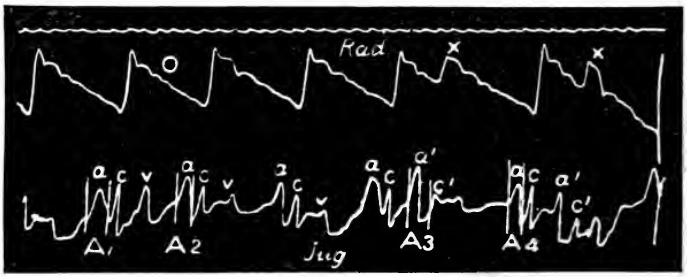


FIG. 20.—Shows two premature or extra-systoles of auricular origin (x). The waves *c'* in the jugular tracing occur at the same time as the small premature beats (x) in the radial tracing, and are therefore due to the carotid. These are preceded by premature waves *a'* due to the auricle. The interval *a'-c'* (space A_3) is greater than the average *a-c* interval (A_2), and is much greater than the following *a-c* interval (A_4). (*Mackenzie.*)

In tracings from this type *all sign of auricular contraction at the normal period has disappeared, as shown by the ventricular type of venous pulse* (Fig. 17).

(iv) The term **ventricular rhythm** (*Stokes-Adams'*

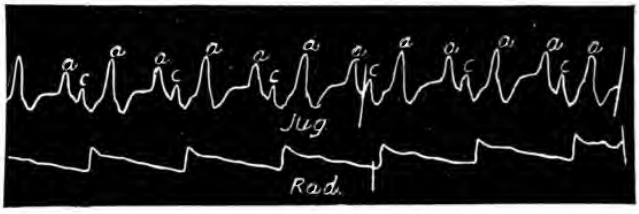


FIG. 21.—The ventricle only responds to every alternate auricular systole—ventricular rate 48, auricular rate 96. (*Mackenzie.*)

disease, heart-block) is used to describe that condition found where the *auricles and ventricles contract independently.*

It is always found to be associated with a destructive *lesion in the auriculo-ventricular or bundle of His.* The ventricular contractions originate in the remaining fibers of this bundle which lie on the ventricular side of the break in continuity; they show a much slower rate of contraction than do the auricles (Fig. 21), and may only respond to every one, two, three, or four of the auricular systoles or may beat as in "*complete heart-block*" in an altogether independent rhythm.

(v) **Pulsus alternans** is a condition in which, although the radial pulse *rhythm is regular*, there is a *variation in the force of successive beats;* it is found where there is

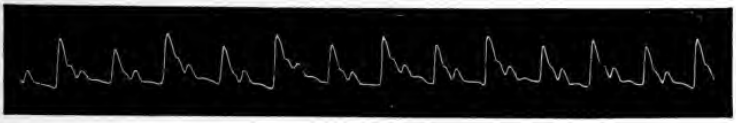


FIG. 22.—Pulsus alternans, male, 64, cardio-sclerosis and bronchitis. (*Mackenzie.*)

failure of the function of muscular contractility. This form of irregularity must be distinguished from that due to the presence of extra-systoles (Fig. 22).

(vi) **Pulsus bisferiens** refers to a double impulse felt at the summit of the systolic impulse; it is most often found in cases of aortic stenosis. Its origin is obscure.

C. **PERCUSSION.**

I. **THE NORMAL HEART.**—As we have already discussed the theory and practice of percussion in the section on the respiratory system, we are in a position to turn at once to describe its application in the investigation of the condition of the heart.

It is usual first to determine by heavy percussion

the limits of the **"deep," "relative or comparative"** cardiac dullness; in other words, the anatomical outlines of the heart itself. Before proceeding further, however, the student is referred to page 56 for the details of its topographical relations.

To determine the boundaries of the **left margin** of the heart it is necessary to commence deep percussion at the anterior axillary line, in the third, fourth, fifth, sixth, and seventh left intercostal spaces, where clear lung resonance is normally encountered, and to repeat the maneuver, gradually approaching the sternum, until the first evidences of defective resonance, indicative of the underlying solid heart, are detected. These points should be marked with a dermographic pencil. It must be remembered that the overlying lung is responsible for the superadded resonance and for the deep percussion which it is necessary to use in order to allow of penetration of the vibrations to the underlying heart.

It will be found in practice that *normally no dullness is present in the second interspaces*, as **the base** of the heart does not rise materially above the level of the upper border of the third cartilages and as the great vessels are too deeply placed to interfere with lung resonance.

After having delineated the upper border and left margin we turn to the **right boundary,** where it is found that although the topographic outlines show that the right auricle extends to the right of the lateral sternal line, in practice it is often impossible by this method in the normal individual to detect any alteration from the lung note to the right of the sternum. So that where a definite band of dullness is found in this loca-

tion enlargement of the heart is probable, providing displacement following left-sided pleural or pneumonic, pericardial and mediastinal disease can be eliminated.

It is impossible to detect by percussion any line of demarcation between the **lower margin** of the heart and the liver, since the two are directly related (the diaphragm alone intervening) and both are solid, airless organs, so that the determination of this margin is largely one of anatomic inference and is shown by a line drawn from the junction of the sixth right costal cartilage with the sternum to the apex beat. It is also necessary to determine the relation of the stomach to these organs, so as to be in a position to allow for the influence of its tympanitic note on the results of our percussion.

Next, by the use of very light percussion strokes, estimate the amount of heart uncovered by lung and sternum which lies in direct contact with the chest wall, thus marking out the triangular area of "*absolute*" *or* "*superficial*" *cardiac dullness* (page 56).

To determine this the lightest strokes are necessary, since the cardiac lappet of lung is at its margin extremely thin. It is well at this point also to note the slight diminution in the area of absolute dullness which takes place normally as the anterior margin of the lung expands into the pleural recess during forced inspiration.

Moritz's Method.*—The following method has been brought forward by *Moritz* as giving results more closely corresponding to those obtained by means of *orthodiagrams* than those reached by the above procedure, the underlying principle being to modify

* Methoden der Herz Untersuchung. Deutsche Klinik, 172 Lief, Berlin, 1906.

the force of the percussion strokes according to local conditions.

In determining the position of the *left margin* in the region of the apex, percuss from the left and below during shallow respiration, employing relatively feeble percussion strokes and holding the plessimeter finger firmly against the chest wall. It will be found that at the moment the heart is reached the stomach tympany gives place to a completely dull note.

As regards the *right margin*, percuss from right to left, the breath meanwhile being held at the end of deep expiration, with forcible "long drawn-out" strokes, the plessimeter finger being placed vertically and held firmly against the chest. The first dulling of normal lung resonance points to the presence of the right boundary (which on deep expiration has been shown to be displaced upward but not to the right) and should lie normally 3.5 to 4.5 cm. to the right of the mid-sternal line.

The position of the *upper margin* is determined during shallow breathing by using the same force of percussion strokes as for the right boundary, in this case, however, holding the plessimeter transverse to the long axis of the body.

II. **ABNORMAL CARDIAC OUTLINES.**—In disease the outlines of the heart may show marked alterations, as evidenced by increase or diminution of the normal **deep dullness.** These abnormalities may be due to changes in size of the organ itself, to its displacement, or to the results of pericardial, pulmonary, or mediastinal diseases.

Let me remind the reader that alterations from the normal percussion notes found over areas immediately

surrounding the normal precordial region, and apparently cardiac in origin, may be due also to certain pleuropulmonary lesions, for details of which reference must be made to the section dealing with the respiratory system.

In disease the **left border** of the heart may be found to extend considerably beyond its normal limits, owing to enlargement of the heart itself (hypertrophy or dilatation), particularly of the left ventricle when the area of cardiac dullness assumes a pyriform shape with the apex downward; to displacement as a result of pressure of a right pleural effusion, pneumothorax, tumor of the right lung, or aneurysm of the aortic arch; and, lastly, to the effect of retraction of the left lung or pleuropericardial adhesions dragging the heart toward the diseased side.

Dullness may be found in the **second left intercostal space** with obliteration of *Sibson's cardiomediastinal notch*, and when present is indicative either of an upward displacement of the heart itself which is usually secondary to abdominal pressure, to enlargement of the left auricle, or to an aneurysm of the pulmonary artery or aortic arch.

Dullness in the **second right interspace** may be due to the presence of an enlarged or displaced right auricle, or to an aneurysm of the ascending aorta. Occasionally a persistent thymus gland will give a dull note over the manubrium and second interspaces, as also may massive pericardial effusions.

The **right border** may extend at times as much as 1 1/2 inches beyond the sternum. When a marked band of dullness is found outside the right lateral sternal line displacement or enlargement of the right

chambers of the heart is probable, the cardiac outline in this latter event assuming a somewhat quadrilateral shape. Displacements result from the opposite conditions which produced dislocation to the left.

Any considerable displacement downward of the right half of the **lower margin** of the heart is rendered impossible on account of the liver, but the remainder is not infrequently found as far down as the sixth interspace. Lastly it must also be remembered that a distended stomach or abdominal pressure may cause a slight dislocation upward of the apex and left portion of this border.

Turning now to a consideration of the abnormal conditions which affect the area of **superficial dullness,** we find that this dull area may be almost obliterated and replaced by resonance in cases of emphysema, or it may be enlarged where there is retraction of the cardiac lappet of the lung. In some instances posterior mediastinal neoplasms may cause a forward dislocation of the heart, and in large pericardial effusions precordial resonance may also be practically obliterated.

The alteration in the cardiac outlines in *dextrocardia* must not be forgotten. This anomaly is usually a part of what has already been referred to as "situs viscerum inversus."

III. **EXTRACARDIAC DULLNESS.**—So far we have described the results of enlargement and displacements of the heart on the areas of cardiac dullness and the effects of aortic aneurysms and neoplasms.

It is necessary now to allude to the physical signs of **pericardial effusion.** Where this condition is present the areas of both deep and superficial dullness are apparently greatly extended. The dull area assumes

a pyriform outline with its apex uppermost, dullness being found external to and below the position of the normal outlines, and the apex beat either impalpable or displaced and floated upward and not corresponding as it should to the junction of the left and lower borders of dullness. The dullness to the right of the sternum extends gradually farther to the right as it approaches the upper margin of the liver, obliterating or rendering obtuse "*Rotch's cardiohepatic angle*," which in the normal condition should be one of about 80 degrees.

In massive pericardial effusions a slight displacement downward of the liver may be detected, and at times a small area of dullness posteriorly below the angle of the scapula is also demonstrable (*Pin's sign*), over which bronchial breathing is heard when the subject is in the erect position, altering to a vesicular murmur accompanied by fine râles and often by a replacement of the dullness by tympany on assuming the recumbent posture.

Ewart's sign refers to the presence of a dull area in the interscapular region opposite the level of the lower angle of the left scapula which is found in pericardial effusions, and over which both breath and voice sounds may be absent.

Hyperresonance over the precordium is found in pneumothorax or pneumopericardium. In both these conditions it may assume a high-pitched tympanitic quality.

D. **AUSCULTATION.**

I. **THE NORMAL HEART SOUNDS.**—It is hardly necessary to review the various events which take place during the cardiac cycle. Suffice it to say that in order to appreciate what follows it is essential to remember that the apical impulse is synchronous with the com-

mencement of the ventricular systole, that the carotid pulse is felt just after its commencement and the radial toward its completion, for, as we shall see, in order that our deductions from the various sounds heard over the heart be of any value, it is imperative that we know what relation they bear to the events of the cardiac cycle.

On listening over **the apex** of a normal organ two sounds are heard. The **first sound** is somewhat lower pitched and more booming than its fellow, occupies nearly three times as long a space of time, and corresponds to the period of ventricular systole, as may be confirmed by comparing the time of its occurrence with that of the apex beat.

This first sound is composed of two elements; one *valvular* in origin and sharp or high-pitched in quality, due to the closure of the mitral and tricuspid valves, the other, which is low in pitch and booming in character, results chiefly from the vibration of the *muscular* walls of the two ventricles. It is most necessary that this fact be kept in mind for reasons to be discussed below.

This first or systolic sound after a "short pause" is followed by a **second sound,** sharper in quality, higher pitched and of lessened intensity, which is *valvular* in origin, synchronous with the closure of the semilunar valves, and hence is early diastolic in time.

The second or "long pause" which follows, corresponds to the remainder of diastole and completes the cycle.

Thus at the apex we hear two sounds which have been likened to the syllables "*lubb dup*," the first of which is systolic in time and has been found to be

due to the combined audible vibrations of the muscular walls and those resulting from the sudden closure of the mitral valve.

Both of these sounds are transmitted by the wall of the left ventricle to the apex, and since it is in this region that the latter element is most distinctly audible it has been termed the "*mitral area.*" This term, of course, has no anatomic reference.

The second or diastolic sound is purely valvular in origin and has been shown to have been carried by the wall of the left ventricle from the aortic orifice, to the closure of which valve its origin is mainly due.

Next place the stethoscope over the **xiphosternal articulation,** and two sounds, in time and quality closely resembling those already described, but of somewhat lessened intensity, are audible.

The *first sound*, which is systolic in time and low in pitch, is due to the contraction of the right ventricle and its sharper element to the closure of the tricuspid valve. The *second sound* heard is diastolic in time and high in pitch, and is due to the closure of the semilunar valves.

The region of the xiphosternal articulation is termed the "*tricuspid area.*"

As we approach the **base of the heart** and listen over the two second interspaces, in close proximity to the sternum, a marked alteration from those sounds heard over the apex is detected. Here two sounds and two pauses, bearing the same relation to the cardiac cycle as those already described, are audible.

The *first sound* loses largely its booming element as well as its intensity, while the diastolic or *second sound* gains in volume, the result being illustrated

by the syllables "*lup-dupp*," with the accent on the second part.

These diastolic sounds are purely valvular, that to the right of the sternum corresponds to the closure of the aortic, and its fellow to that of the pulmonary valves. The inner portions of the second interspaces have been termed therefore the "*pulmonary and aortic areas*," respectively. Again remembering, however, that no reference to anatomic relations is intended.

The first sound heard over the base of the heart has thus lost a considerable degree of its intensity in transmission, especially as regards the muscular element, the valvular second sound becoming intensified as we approach the proximity of the semilunar valves.

This completes the description of the normal heart sounds. We will next analyze the various changes in their quality which take place in disease, and then discuss murmurs and certain other adventitious sounds heard over the heart and vessels.

II. **MODIFICATIONS OF THE HEART SOUNDS.**—Several important variations from the normal cardiac sounds are frequently heard in disease, and under certain conditions audible adventitious vibrations may also be present, such as murmurs and friction rubs. These last will be considered later.

(1) **Accentuation,** or increased intensity of the heart sounds, may be found to involve either those of systolic or diastolic origin, and further to be limited to, or heard best over, certain areas of the precordia.

The **first sound** alone may be accentuated or intensified in *hypertrophy* of the ventricular walls. Its duration becomes prolonged and quality more intensely booming, and conveys the impression of a forcibly

acting organ to the ear. Other signs of hypertrophic changes already described will clinch the diagnosis.

On the other hand, in simple *dilatation* the sound, although at times showing slight accentuation, differs from that heard in the former condition in being of a shorter duration and possessing a clearer, sharper, higher-pitched quality, resulting from absence of the preponderating "muscular" or booming element so characteristic of hypertrophy. Other physical signs already referred to in previous sections should be used as confirmatory evidences of the presence of an enlarged and thin-walled ventricle.

Also it is necessary to remember that in uncomplicated *mitral stenosis* the first sound at the apex acquires a short, "thumping" quality; this, in connection with an apical presystolic murmur (page 174), thrill (page 134), and accentuation of the pulmonary second sound, is diagnostic of this special lesion.

Accentuation may be limited to either the aortic or pulmonary areas, and effects there the second or diastolic sounds. It is then due to sudden and forcible closure of the semilunar valves.

In the case of the **aortic second sound,** an increased intensity of a "ringing" or metallic character is found wherever an increase in the intra-aortic blood pressure takes place during diastole. This condition occurs in arteriosclerosis and aortic aneurysms in its most marked form.

Where the accentuation is limited to the **pulmonary second sound** increased blood pressure in the pulmonary artery secondary to left-sided cardiac lesions or to obstruction to the pulmonary circulation is evidently present.

To recognize accentuation it is well to remember that where the first sound at the mitral or tricuspid areas approaches that of the second in intensity, either the first is weakened and less intense or the second is accentuated. When the first sound over the base of the heart (pulmonary and aortic areas) approaches its fellow in intensity, it is either the first which is the louder of the two and therefore that which shows departure from the normal, or the second which shows lessened intensity, as where lower pressure exists in the aorta or pulmonary artery.

Accentuation of all the sounds occurs in "functional" derangements such as accompany febrile conditions, toxemias (tea, coffee, tobacco, hyperthyroidism, etc.), mental excitement, certain forms of hysteria, and excessive physical exertion. By "*functional*" disturbances we mean those in which no organic valvular or muscular lesions can be found.

Apparent accentuation is present in individuals with thin chest walls, in cases where there is massive consolidation of, or a large vomica in the neighboring lung, also in pneumopericardium and occasionally in pneumothorax.

Lastly, it is well to remember regarding the aortic and pulmonary second sounds as heard over the base of the heart, that *in the young* the latter is usually somewhat louder, while in those of advanced years the aortic normally becomes the more intense.

(2) **Diminished intensity** of all the sounds occurs in the obese, where the heart is removed from its relationship to the chest wall by effusions (pleural or pericardial) and in emphysema.

Where **myocardial degeneration** is present and the

contractions diminish in force the sounds become feeble and less intense. Since it is the *muscular element of the first sound* that is specially affected, this sound as heard at the apex *loses its booming character* and approaches the second in quality; as now *only its sharp valvular element remains* it becomes, therefore, the preponderating feature.

That condition in which the first and second sounds approach each other in quality and become equidistant has been termed *embryocardia*. It is indicative of a weakened ventricular wall and hence from a prognostic standpoint is of grave significance.

In dilatation the same condition is frequently present, but it must not be forgotten that occasionally if the muscle shows no marked degeneration, the first sound may be relatively sharp and loud.

Since the prognosis in cardiac lesions depends far more on the muscular condition of the heart walls than on any valve defect, the necessity of being able to appreciate and distinguish between the muscular and valvular elements is obvious.

In the second and third stages of *mitral stenosis* the intensity of the second or aortic sound as heard at the apex may be markedly lessened, or the sound may disappear on account both of the low arterial pressure and the rotation of the heart to the left (which follows right ventricular hypertrophy) displacing the apex and wall of the left ventricle from contact with the chest, and hence interfering with the transmission of the aortic second sound (Broadbent; see also page 175).

II. **ARRHYTHMIA.**—Much that has been said on this subject in the section on pulse will require no further notice. The sequence of ventricular systoles,

their relation to the diastolic pauses and to each other, intermittence, etc., have been studied in the section dealing with the pulse for the sake of convenience and in order to simplify the examination, especially since the presence of the second and adventitious sounds heard during auscultation may tend to confuse the examiner.

There are, however, certain phenomena which may disturb the regular cardiac rhythm that can only be appreciated by means of auscultation. We will now discuss these in detail.

(1) **Embryocardia, or fetal rhythm** has already been noted as the result of weakening of the muscular element of the first sound and abbreviation of the long pause. In this condition the two sounds therefore approach each other in character and may become practically equidistant. This "pendulum-like" sequence must therefore be regarded as of *serious import*, indicating as it does pathological muscular change, and in cases in which digitalis is being exhibited as giving indication of toxic effects.

(2) **Reduplication, or doubling** of either sound may occur.

(a) Reduplication of the **first sound** is probably due to the asynchronous closure of the mitral and tricuspid valves, and hence is secondary to systolic ventricular asynchronism. It is rarely met with, but is of serious import.

It is found in renal, pulmonary or myocardial disease, also in valvular lesions after failure of compensation.

(b) Reduplication of the **second sound** is indicative of a pathological increase of the blood pressure in

either the pulmonary artery or aorta. The valve of the vessel on the affected side as a result closes at a slightly later period than its fellow and usually also with considerably more than normal force (accentuation).

Hence this phenomenon is present over the second left interspace when excessive pressure in the afferent pulmonary vessels exists, such as is found in obstruction to the passage of blood through the lungs (fibrosis, consolidation, etc.) or in passive congestion of these organs secondary to left-sided valvular lesions.

In mitral stenosis three sounds may be heard at the apex, the third being usually in time presystolic and due to a sudden increase in ventricular tension and "bellying" of the mitral flaps or to early papillary contraction. This condition has been termed "*gallop*" or "*canter rhythm.*"

Regarding those cases of basal reduplication found in mitral stenosis physiologists differ in their views as to the cause, as they also do where it is apical in location.

Lastly, it must not be forgotten that even in health, during a deep inspiration a *physiological reduplication* of the second sound at the base may be encountered.

(3) **Extra-systoles** are easily recognized by the regular sequence of the sounds being interrupted by two short, sharp sounds, followed by a long pause. For confirmation use the cardiogram (page 152).

III. **ENDOCARDIAL MURMURS** are adventitious sounds produced within the cavities of the heart itself, due to the results of altered physical relations between the cavities and orifices of the heart acting on the blood stream, and hence are frequently accompanied by organic valvular lesions.

On the other hand, murmurs or bruits may be heard

over hearts showing no gross anatomical alterations; these sounds are most frequent in the anemias, and are termed "hemic" in contradistinction to those already referred to which have been designated "organic" murmurs.

(1) **Organic Murmurs.**—A murmur is a sound produced by the flow of fluid (or gas, see "respiratory murmurs") along a tube whose caliber or lining membrane is uneven.

But it must be remembered that no matter how swiftly liquids or gases pass through a cylinder whose diameter is constant and whose walls are smooth, no murmurs will be produced; also that in health the relative sizes of the cardiac orifices and cavities are so adjusted that the movements of the flowing blood are inaudible.

When a jet of fluid passes rapidly from a narrow orifice into a cavity whose diameter is greater than that of the opening (for example, when a stream of blood flows from a narrow or stenosed mitral orifice into the left ventricle), for the first part of its course its constituents cohere and form what is known as a **fluid vein.**

In such a case the stream from the narrow orifice flows (as does the "Gulf Stream" in the Atlantic Ocean) through the contents of the larger cavity. Both the molecules of this stream and those of the surrounding fluid are thus set in vibration, and, providing these vibrations are of sufficient strength, they become audible over the precordia.

Again, fluid passing from a wide to a narrow space (as from the ventricles into the great vessels) ordinarily causes no sound, but if the stream impinge on the stiff,

bevelled or roughened edge of a valve, even if no true stenosis be present, audible vibrations are at once produced by the presence of abnormal eddies or "fluid veins."

Organic murmurs are due therefore either to gross pathological alterations in the valves of the heart, to congenital valvular anomalies, to the persistence of passages normally patent only during fetal life (such as the ductus arteriosus), or to structural defects in the walls, as seen in an imperfect interventricular septum or patent foramen ovale.

In *acquired valvular lesions*, however, two types of alterations occur. The valve affected may through structural change be unable to close completely; being therefore "*incompetent*" it will allow of an abnormal backward flow of blood and a **"regurgitant murmur"** will result. On the other hand, it may become thickened and distorted with adherent cusps, and as a result narrowing and consequent obstruction to the onward flow of blood through the orifice may occur. This condition is termed *stenosis* and gives rise to an **"obstructive murmur."**

Lastly, although the valve curtains may show no organic change, stretching of the fibrous ring to which their bases are attached is sometimes present; or as a result of dilatation of the ventricles the cordæ tendinæ may become too short to allow of proper approximation of the margins of the valves and closure of the orifices they guard. The defective closure of the valve curtains in these cases results in what is termed **"relative incompetence,"** which condition may be present at any of the four orifices.

"Relative incompetence" is not found in cases of

hypertrophy, unless accompanied by marked dilatation, since the papillary muscles also hypertrophy and elongate to compensate for the cordæ tendinæ which cannot stretch to allow valvular approximation.

Having now described the general conditions associated with the production of organic murmurs, we must next investigate these sounds more fully in order to determine their significance.

Note carefully the **point of maximum intensity,** the size of the area over which the murmur is audible, and its **time** in relation to the events of the cardiac cycle. The time is best ascertained by endeavoring to fit the murmur into the "frame" formed by the normal sounds or pauses; and only when this is impossible should simultaneous palpation of the apical or carotid impulse be resorted to. Next determine its **transmission** or the direction in which it is carried from the point of maximum intensity, its **quality** (low-pitched sounds being described as "soft," "blowing," or "rumbling," and those of high pitch as "harsh," "sawing," or "musical"), its **intensity** (which depends on the force of the blood stream and consequently varies directly with the strength on the ventricular contractions and also with the nature and degree of the causative lesion), and its **duration** (long or short). Finally determine the *results of posture, exercise, and the respiratory movements* upon its character.

Turning now to a detailed account of the murmurs resulting from valvular defects, we will first describe simple or uncomplicated cases, then those in which combined or multiple lesions are present, and finally take note of several of the more common congenital anomalies.

(*a*) **Mitral Regurgitation** is the most frequently met with of all the valvular lesions. In cases of insufficiency or incompetence of this valve, defective adaptation (from valvular disease or "relative" insufficiency) or the presence of a perforation in the curtain allows of a backward flow of blood from the left ventricle to the auricle during systole.

It is to the vibrations set up in the ventricular contents and the hemic "fluid veins" that the murmur is due.

Bruits produced at this orifice have their point of *maximum intensity* over the "mitral area" (a circular region having a diameter of about 1 inch and a center which closely corresponds to the position of the apex beat), but may be audible over the greater part of the precordia. In **time** they are strictly systolic and may almost completely replace the valvular element of the first sound as heard at the apex.

They are **transmitted** most commonly around the chest toward the axilla and are frequently heard near the angle of the scapula. The mechanism of this phenomenon is that the murmur is transmitted from the posterior surface of the heart to the dorsal chest wall by the bodies of the vertebræ.

In **quality** they are usually soft, blowing, and low-pitched; not infrequently, however, they are high-pitched and may exhibit a "musical" quality. Their *intensity* varies greatly according to the condition of the heart muscle, the amount of regurgitating blood and the size and shape of the orifice.

(b) **Mitral stenosis** or narrowing of the left auriculo-ventricular orifice, results in the production of an opening relatively too small for the size of the ven-

tricular cavity ("button-hole" or "funnel-shaped" orifice) and hence in the formation of "fluid veins" as a result of the altered relationship between orifice and chamber.

The murmurs heard in this lesion have their point of *maximum intensity* somewhat internal to the "mitral area," are limited usually to a very small area of the precordia, have no tendency to selective **transmission,** and in **quality** are abrupt, harsh, rough, and often rumbling. Their *intensity* is usually great, but occasionally in the latter stages of the disease they may completely disappear.

In *time* the typical murmur is presystolic and corresponds therefore to the period of auricular systole, during which the blood is passing through the stenotic orifice with greatest force.

This lesion is in the majority of cases accompanied by a sharp, thumping, apical impulse (page 132), a presystolic thrill (page 134), or accentuation of the pulmonary second and apical first sound (page 164), evidences of an enlarged left auricle (page 159), and reduplications, apical or basal (page 168).

The murmur in many cases will be *found to vary* from day to day in both quality and intensity, also to become louder on assuming the supine posture or to disappear or become faint if the opposite be assumed. It must not be forgotten also that cases with marked mitral narrowing may occasionally be encountered, in which no murmur can be detected; other evidences of stenosis, however, will usually point to the diagnosis.

The murmur may also disappear on the inception of auricular fibrillation; the auricular wave in these cases disappears from the jugular pulse and the ventricular

form of venous pulsation with irregularity in the heart's action takes its place.

In some instances a *protodiastolic murmur* followed by a mid-diastolic pause and then a presystolic bruit, resulting in two distinct though short murmurs during ventricular diastole may be present, or these two may become fused and result in a prolonged bruit occupying the greater part of the long pause.

Three "stages" are found clinically in cases of mitral stenosis. With *full compensation*, or that period during which the heart is enabled by hypertrophy to accommodate itself to the altered physical conditions and to adequately maintain the circulation of the blood, a presystolic murmur "running up into" and terminating abruptly in a sharp first sound and followed by the second after the normal interval is recognizable.

As compensation fails the right ventricle, which in this lesion bears the brunt of the work of carrying on the circulation, dilates rapidly, and as a result of its enlargement occupies still more of the anterior surface of the heart's outline than normally, and consequently rotates the whole organ and displaces backward the narrow margin of the left ventricle whose wall normally carries the aortic second sound to the region of the apex beat. The second sound fails, therefore, to reach this point, and as a result only the presystolic murmur and first sound are audible in the "mitral area." (Page 167.)

In the third stage a *complete loss of compensation*, or failure of the heart muscle to respond to the extra work imposed upon it takes place, and sufficient force is not generated to throw the blood stream into audible vibrations, in consequence of which nothing

remains at the apex but the "thumping" first sound of the ventricular systole (Broadbent).

A systolic murmur is not infrequently associated with cases of mitral stenosis which is apparently not secondary to a mitral leak; it commences immediately after the first sound and rises in intensity ("*crescendo murmur*") until it terminates in the second sound, thus differing from the ordinary mitral regurgitant murmur.

Lastly, this murmur must be distinguished from that which occurs in some cases of aortic regurgitation (page 177).

(c) **Aortic Regurgitation.**—In this lesion the semilunar valves at the aortic orifice are incompetent, either from a distortion of the segments interfering with proper closure and adaptation of the margins or from a rupture of one of the cusps, the result of this being that during diastole a stream of blood passes backward through the aortic orifice into the left ventricle, and audible vibrations are set up by and in the resulting "fluid veins."

This murmur has its point of *maximun intensity* in the "aortic area" and over the sternum at the level of the angle of Louis, and is frequently also audible over the inner part of the second left intercostal space. It is *transmitted* in the direction of the regurgitating blood current or, in other words, down the sternum and toward the xiphoid or apex. In *time* it usually completely occupies the diastolic interval and replaces more or less completely the second sound. Its *quality* is softer than the bruits of aortic stenosis and its *intensity* is very variable, depending largely on the amount of regurgitating blood, and it is

but little affected by posture or respiratory movements.

Austin Flint Murmur.—In addition to the typical "basal" diastolic murmur of aortic insufficiency a second bruit, presystolic or late diastolic in time, with its point of maximum intensity at the apex and showing no tendency to selective transmission, is sometimes found even in the absence of organic mitral stenosis. This murmur was first described by Austin Flint and bears his name.

The following theories have been suggested as explanatory of its production. The first of these supposes that the audible vibrations are due to the impinging of the two streams of blood which enter the left ventricle during diastole, the one from the aorta and the other from the left auricle during its systole; the maximum vibrations therefore being during the latter part of the diastole or presystole.

According to the second theory the murmur is due to the vibrations of the aortic segment of the mitral valve as it lies between these same two streams of blood during the auricular contraction.

The third theory holds that owing to the increase in the size of the cavity of the left ventricle, which always accompanies aortic insufficiency, the normal relationship between its diameter and that of the mitral orifice is disturbed, resulting in a "relative" mitral stenosis.

This murmur must be *differentiated from that produced by an organic mitral narrowing*, by the absence of other signs pathognomonic of mitral stenosis (page 174). Additional signs confirmatory of aortic insufficiency are to be found in hypertrophy, especially

of the left ventricle, in the capillary (page 130) and Corrigan's "water-hammer" pulse (page 141), as well as in certain other arterial phenomena to be described later (page 185).

An aortic diastolic murmur best heard at the apex has been held by Foster to be indicative of disease of the left posterior valve cusp.

(d) **Aortic Stenosis.**—A *true stenosis of the aortic ring is seldom found*, but narrowing just beyond the orifice as a result of thickening and stiffness of the aortic cusps may occur and offer obstruction to the entrance of blood into the aorta during ventricular systole.

The auscultatory phenomenon consists in a bruit, with its point of *maximum intensity* over the "aortic area" and often audible over a great part of the precordia. It is *transmitted* in the direction of the blood current or, in other words, into the great vessels of the neck. In *time* it strictly corresponds to the period of ventricular systole, and in *quality* is usually harsh, of moderate intensity, and is uninfluenced by posture or respiratory movements.

It may almost completely replace the first sound, be accompanied by a systolic thrill (page 134), hypertrophy of the left ventricle, and a characteristic pulse (page 141).

Although **true narrowing of the aortic ring is rare, systolic murmurs over the aortic area are frequent.** They are usually due to roughened or merely rigid valve segments, to atheroma, simple dilatation, or to an aneurysm of the aortic arch. In these instances absence of confirmatory evidence of the presence of stenosis and the presence of signs pointing to the other conditions will clinch the diagnosis.

Usually, however, these "basal" systolic murmurs are "hemic" in origin.

(e) **Tricuspid Regurgitation.**—Acquired organic disease during adult life of any of the valves of the right side of the heart is so rare as to be almost a curiosity, but during intrauterine life they are not infrequently attacked by endocarditis and these valves are also specially liable to present congenital anomalies.

On the other hand, a *relative incompetence* is frequently encountered when dilatation of the right ventricle exists. A similar explanation to that given for "relative" mitral regurgitation will apply (page 171).

The murmur is limited to, and heard best over, the "tricuspid area" at the base of the ensiform and is transmitted slightly upward and toward the right axilla. Its rhythm is systolic, quality soft, and intensity usually low. Evidences of enlargement of the right ventricle, venous pulsation, and congestion of the liver will corroborate the diagnosis.

(f) **Tricuspid Stenosis** is almost unknown as an acquired lesion. With it a presystolic murmur is found, which is best heard over the area of "superficial or absolute" cardiac dullness. It has no selective transmission, and is of a quality closely resembling that found in mitral narrowing. Evidences of dilatation of the right auricle, venous engorgement, and cyanosis are usually also present.

(g) **Pulmonary Regurgitation** is also a very rare condition. It may be due to relative dilatation or stretching of the pulmonary "ring" (aneurysm, etc.), to acquired valvular disease, or congential anomaly. The murmur, which is best heard over the "pulmonary

area," is in time diastolic and is usually transmitted for a short distance down the left sternal margin.

(h) **Pulmonary Stenosis** is practically always congenital in origin. The murmur is in time systolic and most distinctly heard over the "pulmonary area." It is usually transmitted upward and outward beneath the left clavicle.

Although disease of the pulmonary valve is seldom encountered, systolic murmurs over the base of the heart (especially over the "pulmonary area") **are frequently heard.** They belong to that class already alluded to as "hemic" or "functional," and will be further described on page 183.

(i) **Combined Valve Lesions.**—When one considers the etiological factors of endocarditis (the cause of all cardiac murmurs except those resulting from congenital anomalies or "functional" disturbances), it is obvious that frequently more than one valve will be diseased, and also that the same valve may not only be so altered as to produce stenosis of the orifice it guards but, on account of certain structural changes, may also be insufficient to close the orifice and so allow of regurgitation.

Again, from what we have said of murmurs and their transmission, it will be obvious that the same bruit may be audible over more than one valve area, and also that functional or hemic murmurs may at times be coincident with those of organic origin. For the clinical differentiation of these latter forms reference should be made to page 183.

The following are the more **important combinations of left-sided valvular lesions** given in their order of frequency:

(i) *Aortic stenosis and regurgitation, plus mitral regurgitation,* produce in the first place a double, systolic-diastolic or "to-and-fro" murmur at the aortic area, as well as one systolic in time at the apex. Each murmur is accompanied by certain other phenomena indicative of the primary lesion. In this instance it is also well to remember that the two aortic lesions tend to a certain extent mutually to neutralize one another, and thus each in a measure compensates for the vicious results of the other.

(ii) *Aortic stenosis* and *mitral stenosis* together produce two murmurs, the first of systolic and the second of presystolic rhythm. The latter murmur must in every instance be differentiated from that described by Austin Flint (page 177) by the absence of confirmatory signs of mitral obstruction.

(iii) *Aortic and mitral regurgitation.*

(iv) *Mitral stenosis and regurgitation.*

(v) *Aortic and mitral stenosis.*

(vi) *Mitral stenosis and aortic regurgitation.* In all the above combinations we find a combination of auscultatory and other signs characteristic of the lesions present.

In the presence of two or more murmurs the points of maximum intensity, direction of propagation, correct timing, character of the pulse, presence of reduplication, diminution in intensity or accentuation of the heart sounds must all be noted. The presence of signs indicative of secondary alterations in the heart walls and chambers will also aid in arriving at a correct deduction as to the lesions present.

(j) **Congenital Cardiac Anomalies.**—*Dextrocardia*, or that condition in which the position of the heart in the

chest is reversed, the apex pointing to the right, is practically always a part of that condition termed "situs viscerum inversus" and, unless accompanied by intracardiac changes, the auscultatory signs may show no deviation from the normal, except those due to altered anatomical relations of the valves to the chest wall.

The term dextrocardia is also applied to the displacement of the apex to the right of the sternum which is secondary to a left-sided pleural effusion or to retraction of the right lung.

A congenital *pulmonary stenosis* is that lesion most commonly encountered. It is frequently associated with a *patent foramen ovale*. In the latter instance two murmurs may be present; the first corresponding to that described as characteristic of the acquired pulmonary lesion; the second, which may not always be present, is basal in position, presystolic in time, and soft in character. It may also be heard over the back and is probably dependent for its existence on differences in pressure in the two auricles.

A persistent and *patent ductus arteriosus* is usually associated with a loud, harsh, systolic bruit over the pulmonary area, which is sometimes accompanied by a thrill.

A defective or *patent ventricular septum* frequently causes a systolic murmur in the region of the third interspace in the left parasternal line or at the base of the sternum. Occasionally a continuous hum, varying greatly in intensity over different areas, is audible over the whole of the precordia.

Also it must not be forgotten that the murmurs of many cases of congenital malformation are present

only on exertion, as when the child cries, or that they may give rise to no auscultatory phenomena whatever.

As regards *differentiation from those of acquired origin*, congenital murmurs are rarely diastolic, usually basal, show no pathognomic transmission, but are accompanied frequently by cyanosis, dilated venules (morbus cæruleus), clubbing of the fingers, an abnormal number of red cells per cubic millimeter of blood, and sometimes also by evidences of malformations in other organs.

Lastly, moderator bands or aberrant cords may account for the accidental muscle murmurs which are occasionally encountered.

(2) **Functional murmurs** or, as they are also called, **inorganic, hemic, dynamic, anemic or accidental,** are frequently found over hearts showing no evidences of structural or valvular changes.

They may be *heard over any part of the precordium, but most commonly over the base of the heart in the pulmonary or aortic areas, as systolic bruits, soft in quality, showing no characteristic transmission, are often transient, increasing in intensity at the end of deep inspiration and on assuming the erect posture.*

An absence of any of the secondary cardiac or peripheral changes pointing to organic valvular disease, and the presence of an anemia, fever, or evidences of a cardiac neurosis, suggests that the bruit heard is of the functional type.

It has been suggested that these murmurs all owe their *origin* to the "fluid veins" set up by a relative mitral insufficiency resulting from alterations in the myocardium (due to qualitative changes in the blood producing muscular weakness and loss of tone), and

the consequent dilatation of the left ventricle and auricle. The basal location of such a murmur has been ascribed to its conduction to the chest wall by the enlarged left auricle.

Numerous other *theories* have been advanced. Dilatation of the pulmonary artery and conus with fibrillary tremors of the muscular fibers involved, altered viscosity of the blood, compression of the pulmonary artery by the enlarged left auricle, and the possibility of venous murmurs in the great vessels being carried to the precordium have been brought forward by different authorities as accountable for their production.

In addition to the characteristics noted above, it will be found in cases showing these murmurs that slight *dilatation of the heart is almost invariably demonstrable*, also that extracardiac signs indicative of organic disease are wanting.

The transient apical "*bruit de consultation*" heard in nervous subjects comes under this category.

IV. **EXOCARDIAL SOUNDS.**

(1) **Pericardial friction** sounds exhibit a "to-and-fro" character and are dependent on the cardiac contractions for their rhythm.

They are very superficial, frequently scratching in quality, often transient, increased in intensity by pressure of the stethoscope, and are definitely limited to the precordia. The point of maximum intensity may vary from hour to hour.

These sounds are usually most intense over the base of the heart and during a deep inspiration. They are sometimes modified by changes in posture, often accompanied by pain and tenderness near the third

left costosternal junction, and continue even if the patient holds his breath.

They must be carefully differentiated from pleural and pleuropericardial friction sounds and also from cardiopulmonary murmurs (page 115).

(2) **Churning and splashing sounds** of pericardial origin are found where air and fluid are both present in the sac. They have also been termed "water-wheel" sounds.

(3) **Arterial murmurs** may be audible notably in cases of aortic insufficiency where one of double rhythm, often accompanied by the "*pistol-shot*" phenomenon, is heard especially if slight pressure on the vessel be exerted by the bell of the stethoscope.

In aortic stenosis the endocardial murmur is usually transmitted to the carotids.

Over the subclavian artery, in cases of phthisis with dense apical pleural adhesions, a murmur systolic in time is not uncommon.

Aneurysms, aneurysmal varices, and aortic atheroma are also frequently accompanied by bruits.

(4) **Venous murmurs** are best heard over the jugulars. They are soft or "buzzing," continuous, and are found in anemic states.

They owe their origin either to altered physical conditions in the circulating blood or to the unaltered caliber of the internal jugular veins as they pass through the unyielding cervical fasciæ ("*bruit de diable*").

They are heard best during a period of apnea with the patient standing, especially if slight pressure on the vein be exerted by means of the stethoscope.

E. **AUSCULTATORY PERCUSSION** is occasionally made use of in determining the outlines of the "relative cardiac dullness."

F. **MENSURATION** is of value in permanently and accurately recording the position of the apex beat and for the purpose of comparing from time to time the size of the heart during the observation of any given case.

Recently the *x*-rays have been extensively employed with great success in determining the outlines of the heart; the pictures obtained are termed **orthodiagrams.**

G. **THE BODY TEMPERATURE.**—The normal temperature of the average adult varies from 97.2° *F. in the morning to* 98.4° *F. at night.*

In children it is usually slightly higher on the average than in adults. It must be remembered also that the young are liable to show much greater variation and higher temperature ranges than adults suffering from the similar diseases, also that high fever in a child usually is not of such grave significance as a similar condition in the adult.

(I) **Palpation.**—An approximate estimate of the body temperature may be made by the palpating hand. This should be applied to some part of the trunk, especially the axillary regions, since the cutaneous surfaces of the head and the extremities do not furnish a reliable guide on account of the extensive surface available for radiation.

It is also obvious that when the patient is freely perspiring the amount of evaporation will also interfere materially with the estimation of the body heat.

(II) **The Clinical Thermometer.**—To insure accurate results these instruments must be standardized and accompanied by a certificate stating the individual error of each instrument. Both Fahrenheit and Centigrade scales are employed, the former, however, almost entirely in England and Canada.

Before using the thermometer it must be carefully washed in cold water and the column of mercury "shaken down"; after use it is placed in an antiseptic solution (1 to 20 carbolic acid or 1 to 2000 bichlorid of mercury solution) for some minutes and again washed in cold water.

It is imperative also that patients known to be subjects of tubercular disease, syphilis, or any of the contagious fevers possess their own individual instruments.

The thermometer may be placed in the *mouth* under the tongue, the lips meanwhile being kept tightly closed and the patient instructed to breathe through the nose. This is the usual method of procedure.

In children and in the comatose, the instrument may be placed in the fold of the *axilla* or *groin;* in these situations it should remain at least one minute longer than if placed in the mouth or rectum, and it must be remembered also that the temperatures recorded in these areas will usually be found to be at least half a degree lower than in the other situations.

Rectal temperatures are of the greatest value, and the results obtained are much more accurate than those of any other method.

(3) **The Temperature Chart.**—The temperature should be taken at certain fixed hours; in cases showing a febrile reaction (*pyrexia*), every two hours, in the ordinary run of cases, however, every four hours is generally considered sufficient. Morning (8 A. M.) and evening (8 P. M.) temperatures are often all that is necessary.

No description of the various forms of "charts" in use will be given; they must be seen to be appreciated.

Certain **types of fever,** however, deserve a short note.

They are the "*continued*," in which fluctuation does not occur to any marked extent (1 to 1 1/2° F.), the temperature curve as shown by the chart remaining at an almost constant level.

"*Remittent*" fevers are those showing daily variations of at least 2° F., usually the evening temperature reaching the highest point.

"*Intermittent*" fevers show febrile reactions only at certain hours in the day, but in certain diseases (malaria, etc.) these paroxysms may occur daily and constitute the "*quotidian*" type.

When the febrile period, however, is present only on every other day, the term "*tertian*" is applied; and when two days intervene between paroxysms the term "*quartan.*" "*Double tertian*" refers to those states in which febrile reactions occur daily, but in which those on the first, third, etc., differ materially from those observed on the alternate days.

The term "*fastigium*" is applied to the period of full development of any fever, "*crisis*" to describe the sudden termination of any febrile state, and "*lysis*" when the febrile movement gradually falls to normal.

H. **THE BLOOD** (see Appendix).

CHAPTER VIII.

DIGESTIVE SYSTEM.

A. **UPPER FOOD PASSAGES.**

I. **THE LIPS.**—The points to be noticed in the examination of the lips have already been dealt with on page 44, so we may pass at once to the investigation of the oral cavity and its contents.

II. **THE ORAL MUCOUS MEMBRANE** should be carefully inspected, noting especially evidences of *inflammation* (stomatitis), as shown by diffuse or

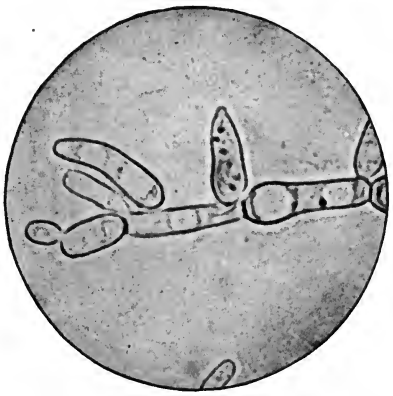


FIG. 23.—Oidium albicans. (*Kolle and Wassermann.*)

patchy hyperemia, vesicles, ulcers (aphthæ), and the presence of raised gray or white, adherent points or patches of exudation, found when a parasitic (thrush, (Fig. 23) or diphtheritic infection is present.

The *rashes* of scarlet fever, small-pox, measles and of varicella may appear on the mucosa, especially on that

of the palate. In measles the presence of *Koplik's spots* must not be overlooked. They are found early in the disease and often appear before the cutaneous eruption. They consist in tiny bluish-white points surrounded by inflammatory areolæ.

Burns or discolorations such as are caused by the caustic mineral poisons and the presence of *pigmentation* should never be overlooked. *Epitheliomata* and the raised, soft, dirty-gray "*mucous patches*" of syphilis must also receive attention.

A grayish-white thickening of the oral mucosa is not infrequently seen as a result of repeated acute attacks of ulcerative follicular stomatitis in adults, the submucous follicles being usually also visible as tiny nodules about the size of pin-heads scattered over the thickened mucous membrane.

Gangrenous stomatitis (*cancrum oris*) is occasionally seen in debilitated children. It commences as a small dark red, inflamed and rapidly spreading area, which ultimately undergoes necrosis and may result in perforation of the cheek.

III. **THE TONGUE** now demands consideration; and since we shall refer later to hypoglossal *paralysis* (page 261) we may at once return to the inspection of the mucosa.

Note the color of the *mucous membrane*, its moisture, the presence of furring, swelling, indentations of the teeth, ulceration (loss of surface epithelium and fissures, which may be simple, specific or malignant in origin), tumors, scars (suggestive of old syphilitic lesions or epileptic seizures, a bitten tongue being commonly the result of the latter), and the "strawberry tongue" of scarlet fever.

Tongue-tie, ranulæ, calculi in the salivary ducts, frenal ulcerations, etc., must be looked for in order to render this examination complete. A detailed description of superficial glossitis, the "geographical tongue," leukoplakia, and other local diseases, however, are beyond the scope of this work.

IV. **THE GUMS.**—The color should first be noted, and the occurrence of retraction, ulceration along the line of the teeth (mercurial or due to *pyorrhea alveolaris*, the latter a common cause of digestive disturbances), the spongy, bleeding gums of scurvy, and the "blue line" found in cases of chronic lead, or the "green" of copper poisoning must not be overlooked.

V. **THE TEETH.**—In children we must, however, in addition to ascertaining the number present and the dates of eruption (see "below") search for evidences of congenital syphilis.

TABLE OF ERUPTION

Temporary		Permanent
6 to 8 months,	2 lower central incisors.	6 years, first molars.
8 to 10 months,	4 upper incisors.	7 years, central incisors.
12 to 14 months,	lower lateral incisors.	8 years, lateral incisors.
	upper front molars	9 years, anterior bicuspids.
	lower front molars.	10 years, posterior bicuspids.
18 to 20 months,	upper canines.	11 to 12 years, canines.
	lower canines.	12 to 13 years, second molars.
2 to 2½ years,	posterior molars.	17 to 25 years, third molars ("wisdom teeth.")

Relations of Temporary to Permanent Teeth.

		M.	C.	I.	—	I.	C.	M.							
Temporary	Upper,	2	1	2		2	1	2 = 10	} = 20						
	Lower,	2	1	2		2	1	2 = 10							
		M.	B.	C.	I.	—	I.	C.	B.	M.					
Permanent	Upper,	3	2	1	2		2	1	2	3 = 16	} = 32				
	Lower,	3	2	1	2		2	1	2	3 = 16					

"Hutchinson's teeth," or the two upper permanent incisors of congenital luetics, are *peg-shaped*, the cutting-edge showing a crescentic outline or semilunar notch with its concavity directed downward; their cross-section approaches that of a circle and their free edges slope toward the middle line below. Not infrequently their surfaces show discoloration.

The term "*screw-driver*" is used to describe the earlier stages of Hutchinson's teeth, before the loss of enamel and the formation of the crescentic notch has taken place.

Since it has been shown that the *calcification* of the first permanent molars commences during intra-uterine life, the central incisors at the age of one month, the canines during the third or fourth, and that of the remaining teeth not until some time later, it follows that any disease (syphilis or stomatitis) which attacks the child the first year of life will effect only the calcification of the central incisors and canines. The "*test teeth*" for syphilis are therefore the upper central permanent incisors and those for infantile stomatitis the first molars. These teeth also frequently show transverse furrows or pitting.

The presence of caries, excessive tartar, loss of enamel, or irregularity of the teeth, loosening and ulceration or retraction of the surrounding gums should be noted, and in children a history of "grinding the teeth."

VI. **THE PALATE.**—First examine the mucosa of both the hard and soft palate and that of the uvula and faucial pillars for the presence of rashes, jaundice, tumors, ulcers, pigmentation, "mucous patches" or pseudo-membranes (diphtheritic, parasitic or pyogenic).

Reference will be found in the section on the nervous system to the movements and sensibility of the palate.

Next inspect the hard palate with regard to the height and shape of its vault, since a high, narrow, and pointed palatal arch is, especially if combined with other stigmata (page 28), suggestive of mental degeneracy.

Search also for congenital "clefts" and acquired perforations.

VII. **THE TONSILS.**—Their size and colour, evidences of acute follicular or diffuse inflammatory processes, crypts distended with plugs of cheesy secretion, ulcers and membranes if present must be noted and described.

The tonsils should also be palpated in order to detect the presence of abscesses or the location, mobility, and consistence of tumors. Note particularly if the upper pole is adherent and projecting. This condition usually points to a tonsillar infection.

VIII. **THE PHARYNX.**—The examination has already been discussed in connection with the upper air passages on page 62.

IX. **THE BREATH.**—Its odor may be referred to at this point, but before proceeding nasal, oral, laryngeal and pulmonary lesions must be excluded.

The odor is often characteristic of certain general diseases, especially *diabetes* which imparts to it a sweet, mawkish or "new-mown hay," or in the later stages an "ethereal" odor. The "ammonical" or "urinous" odor of *uremia* may aid in the diagnosis.

The distinctive odors of certain *drugs* such as turpentine, carbolic acid, alcohol, creosote, opium, paraldehyd, asafetida, chloroform, the "almond-like" odor of hydrocyanic acid, the "garlic" of phosphorous poisoning, and many others are easily recognized.

The fetid or putrid odors of *bronchiectasis and gangrene* of the lungs and the slighter degrees of offensiveness found in *gastrointestinal* disturbances and in patients taking large doses of the iodids may also be detected.

X. **THE ESOPHAGUS.**—The condition of this tube may be investigated by means of the esophageal bougie or the ordinary soft-rubber stomach-tube.

The *bougie* is of value chiefly in the diagnosis and localization of strictures and diverticula, the *tube* in removing the stomach contents and in performing gastric lavage. Killian's *esophagoscope* may be used in inspecting the interior of the esophagus, but requires considerable skill in its application. The latter is sometimes employed in the extraction of foreign bodies, for which purpose a sponge or "umbrella" *probang* or special "coin catcher" may also be used. The *x-rays* also are at times useful in the localization of foreign bodies.

Auscultation may be practised over the cervical region and the back of the trunk just to the left of the upper six thoracic vertebræ, or below this level to the right of the center line, as far down as the ninth thoracic spine.

Fluids may be heard as they pass through the gullet in cases of esophageal stenosis, but below the lesion the normal *deglutition murmur* becomes indistinct and delayed. This method is of no great practical value.

Having now completed the examination of the upper food passages, it is advisable to give a general account of the methods used in the investigation of the abdomen and its contents before proceeding to describe in detail those employed in the examination of the special organs.

B. THE ABDOMEN.

I. INSPECTION.

(1) **The Skin.**—The principles outlined in the section on the skin in Chapter IV may be applied to that of the abdominal wall, several additional points, however, should be particularly noted.

As regards the *technic* required for satisfactory work, it is necessary that the patient's abdomen be freely exposed, that the dorsal decubitus be assumed, and that he or she lie perfectly straight in the bed with a small pillow only under the head.

The examiner should see that the *light* falls in a slanting direction on the abdominal wall, from near the head or feet, it being frequently found advisable to move the couch so as to allow the rays to fall laterally. The clinician should inspect the surface from all aspects in order not to overlook any shadows or irregularities in movement or contour.

The color of the skin requires careful consideration, the abdominal wall being a favorite situation for the deposition of pigment (*linea nigra*, etc.); and the presence of edema, enlargement and tortuosity of the veins, especially those surrounding the umbilicus (*caput medusæ*), must be noted, as also should the presence of scars and the fine white cicatricial striæ (*lineæ albicantes*) found after the relief of prolonged abdominal distention, such as follows pregnancy and the removal of tumors or ascitic fluid.

The tense shining smooth appearance of the skin found where the abdominal walls are on the stretch (ascites, cysts, etc.) should be noted.

Protuberance of the *umbilicus*, due to the presence of a hernia or secondary to abdominal distention, and

occasionally a patent urachus, fecal fistula or purulent discharge may be present.

(2) **The Contour.**—The patient should whenever possible be examined both in the erect and supine positions, and instructed to allow as far as possible the abdominal wall to relax and to breathe with the mouth wide open.

In this section reference will be made only to general or symmetrical alterations in the size and contour, local abnormalities being discussed under that on abdominal palpation.

(a) **General or symmetrical enlargement** may be due to excessive omental or mural fat (obesity), ascites, tympanites (meteorism), massive neoplasms, pregnancy, pseudocyesis (page 201), or the presence of free gas in the abdominal cavity.

Determine at this point whether the enlargement is chiefly in the transverse diameter with bulging flanks and a flattened umbilical region, such as is found usually in ascites, or if the reverse is true; and it is well also in this connection to call in the aid of mensuration to corroborate our findings.

(b) **General retraction** (*scaphoid abdomen*) is found in most wasting diseases (emaciation) and occasionally in basilar meningitis, tuberculous peritonitis, hysteria, etc.

(c) **Local fullness or bulging** will be fully discussed in the section on palpation; but as fullness due to a dilated stomach may be visible but not palpable, it should be sought at this point.

(d) **Local retractions or depressions** are rare, but may occasionally be met with following chronic adhesive peritonitis.

(3) **Movements.**

(a) **Respiratory** movements are most marked in those conditions which cause the abdominal type of respiration (page 67); and are diminished in peritonitis, paralysis of the diaphragm and where abdominal distention is present, giving rise to the thoracic type of respiration.

Abdominal *tumors* connected with the diaphragm or influenced by its excursions, especially where the liver, spleen, and pylorus are involved, usually show vertical movements synchronous with the respiratory acts.

(b) **Peristaltic** movements, when excessive, suggest pyloric or intestinal obstruction, especially if traveling in an abnormal direction, as, for instance, when the stomach "waves" pass from right to left, or those of the transverse colon in the opposite direction (*retroperistalsis*). They may be exaggerated by gently flipping the abdominal wall with a wet towel.

It must not be forgotten that in very thin healthy individuals and in those whose abdominal walls are relaxed, peristaltic waves may be apparently excessive.

(c) **Pulsations** are most common in the epigastric region and may be due to enlargement of the right ventricle or displacement of the heart, in which cases the impulse will be strictly systolic in time; they may also owe their origin to movements in the thoracic aorta.

The impulse may follow the apex beat and be due to pulsations of the abdominal aorta (as seen in aneurysmal dilatations and in nervous subjects), to passive congestion of the liver (tricuspid regurgitation), or it may be transmitted to the parietes from a normal abdominal aorta by means of an overlying tumor.

In the case of the liver, and in aneurysms, the pulsations will be usually *expansile*, but in the other examples they are merely heaving in character.

II. **PALPATION.**

(1) **General Technic.**—Regarding the technic of abdominal palpation, several points require special notice.

It is first necessary to gain the patient's confidence, since nervousness is frequently responsible for the voluntary rigidity of the abdominal muscles so frequently found. To obviate this the subject's attention should be directed away from his abdomen, usually by conversation on the part of the examiner. It is well at the same time to instruct the patient to breathe slowly and deeply through the open mouth, and so facilitate the *relaxation* of the abdominal muscles, for unless the walls are "soft" or "relaxed" satisfactory examinations are impossible.

In some cases a general anesthetic will be found necessary before a thorough investigation can be accomplished.

The dorsal decubitus with the head slightly raised on a pillow is probably the best posture for routine examinations, but if the knees be "drawn up" they must be supported by a pillow Not infrequently, however, the lateral or prone position is most useful.

It is often an advantage to examine the patient sitting up in bed with the body bent forward and the elbows resting on the knees, as in this way the abdominal wall is relaxed so by standing behind the patient, one is enabled to pass the arms around his sides and so to palpate the abdominal contents. *Trendelenburg's posture* in which the patient is placed in the

dorsal decubitus with the pelvis raised well above the level of the shoulders, is also occasionally of value.

The *examiner's hands must be warm* and must never be suddenly applied to the surface, but be *gradually allowed to "sink in"* during the expiratory periods. They should also be allowed to "glide" from one area to another and *never be raised from the surface* during the manipulations.

(2) **Superficial Palpation.**—One or both hands should first be lightly applied to the abdominal wall, palms downward and with the fingers fully extended. Every portion of the abdominal surface, as well as *Scarpa's triangles*, should be subjected to light palpation, looking in the latter regions particularly for the presence of enlarged glands or femoral herniæ.

By this maneuver we are enabled also to detect the presence of subcutaneous emphysema, parietal tumors, the degree of rigidity of the muscles on the two sides, the tension of the abdominal contents, the presence of superficial tenderness, movements (respiratory, circulatory or peristaltic), retractions and certain "floating" abdominal tumors, which latter might escape notice if "deep" palpation alone were used.

Regarding *tumors of the wall*, the location, mobility, consistence, condition of surface (smooth or nodular), the presence of fluctuation, degree of sensitiveness and the presence or absence of movements coincident with respiration should be determined. For details, however, of the differentiation of mural abscesses, lipomata, herniæ, etc., reference must be made to works on surgical diagnosis.

Rigidity or the resistance offered by the abdominal muscles to palpation is a most important phenomenon,

pointing as it does to an underlying diseased process. It may be diffuse (general peritonitis) or confined to one locality (as, for example, over the appendix or gall-bladder). The muscles are said to be "on guard" and at times are contracted to such a degree as to warrant the use of the term "board-like" resistance.

Tenderness may be entirely superficial, and then is generally due to some mural lesion or neurosis (hyperesthesia); if present it must be accurately localized.

No further reference will be made to abnormal movements beyond the fact that pulsations should be carefully examined as to whether they are expansile (aneurysm) or merely heaving in character.

Tumors that spring from the intestines, mesentery, etc., may float free in the peritoneal cavity and may not infrequently be displaced and so overlooked by deep manipulations. Their location is usually uninfluenced by the respiratory excursions; but it is well to note that omental growths may show slight movements synchronous with those of the diaphragm.

(3) **Deep Palpation.**

(a) **Technic.**—The palmar surfaces of the fingers should first be applied to the abdomen and *gradually allowed to "sink in,"* and especially during expiration to follow in the respiratory recession of the anterior abdominal walls. The tips of the fingers should, as a rule, not be pressed directly backward, better results being usually obtained by palpating with their palmar surfaces. It is usual also where painful areas or tumors are suspected to *first palpate over the healthy quadrants.*

When, however, free fluid exists in the peritoneal cavity, and it becomes necessary to examine for deeply

lying organs, the finger tips may be placed over the organ and suddenly pressed backward until they, by displacing the supernatant fluid, come in contact with the underlying solid mass, this maneuver being termed "*dipping*."

Bimanual palpation with one hand over the anterior surface and the other under the loins, between the crest of the ilium and last rib, is frequently practised, especially in searching for the kidneys or spleen.

(*b*) **Tumors and Cysts.**—Tumors of the intestines, mesentery or omentum, enlarged glands (mesenteric or retroperitoneal), effusions, cysts or abscesses, may be found in any portion of the abdominal cavity. The more common of these will receive further attention under the section dealing with the special examination of the abdominal organs.

It is well to note that the condition designated *pseudocyesis* is occasionally encountered in females. This false tumor is principally of muscular origin and may closely simulate pregnancy or abdominal growths, differing from them in that it completely disappears under a general anesthetic.

Lastly, in the examination of every abdominal tumor it is necessary in determining its origin to endeavor to insert the fingers between it and the posterior abdominal wall and between it and the diaphragm. When situated in the lower quadrants one should endeavor to sink the fingers in below its level, in order to exclude tumors of pelvic origin. Mensuration should be employed and a vaginal and rectal examination made in all cases.

Deep tenderness must be carefully defined. In appendiceal inflammations this is usually most intense

over "*McBurney*'s *point*," which is situated at the intersection of a line drawn from the umbilicus to the anterior superior iliac spine with a second line corresponding to the outer edge of the right rectus muscle.

Pulsations and fluctuations should be sought for in every tumor and their mobility and respiratory excursion estimated.

The student is also urged to use every opportunity offered to practise abdominal palpation in order to familiarize himself with the various "types" of abdomen, the degree of muscular resistance offered by nervous patients; for, unless *a thorough conception of the normal* be acquired, an appreciation of diseased conditions is impossible.

Having now described the general methods employed in the examination of the abdomen it is advisable, before proceeding with that of the technic of the investigation of each individual organ, to refer to the more **common tumor masses** found in each of the four abdominal quadrants. The diagnosis between cysts and ascites will be discussed later.

(i) *Right Upper Quadrant.*—Liver, gall-bladder, hepatic flexure of colon, subphrenic effusion, pylorus, head of pancreas, right kidney.

(ii) *Left Upper Quadrant.*—Fundus of stomach, spleen, splenic flexure of colon, left kidney, tail of pancreas.

(iii) *Right Lower Quadrant.*—Appendix, ascending colon, psoas abscess, tumors of female pelvic organs.

(iv) *Left Lower Quadrant.*—Iliac colon, left kidney, psoas abscess, tumors of female pelvic organs.

(v) *Median Line.*—Aortic aneurysms, stomach, transverse colon, pancreas, bladder, uterus.

III. PERCUSSION.

(1) **Technic.**—The technic of abdominal percussion does not differ from that described on page 84, but usually the "light" method is the more useful.

The *normal abdominal note* (except over the liver, spleen, and distended bladder) is *tympanitic* in quality, its pitch depending on the size, wall, tension and character of the contents of the cavity percussed; so that it is obvious that the note elicited varies considerably from time to time.

Percussion is of greatest value in defining the lower border of the liver, differentiating stomach from bowel, tympanites from ascites, in outlining solid tumors or cysts, a distended bladder, and in demonstrating the relation of the colon to certain abdominal tumors (page 275).

(2) **Ascites.**—Since *free fluid always gravitates* to the most dependent portions of the abdominal cavity, when the patient is supine dullness will be found in the flanks and resonance over the umbilical and surrounding areas, on account of the floating intestines.

Again, since the fluid is free in the peritoneal sac, the phenomenon of **moveable dullness** may be demonstrated; that is to say, on altering the decubitus of the patient dullness will follow the fluid as it gravitates, and will hence always be present over the most dependent parts.

Moreover, if one hand be placed in contact with the dorsolateral surface of the abdominal wall between the crest of the ilium and last rib, and the corresponding point on the opposite side of the body be percussed or "flicked" with the fingers of the other hand, a **"thrill"** or "percussion wave" will be transmitted by the fluid

(providing sufficient free fluid be present to fill the flanks and cover the prominence of the vertebral bodies) and be felt by the palpating hand. In the obese it is necessary to request a third party to sink the ulnar border of his hand into the abdominal wall in the middle line in the vertical plane in order to interrupt the transmission of vibrations by the abdominal wall itself.

In *localized collections of fluid*, such as are found in chronic peritonitis and in cysts, areas of dullness with surrounding tympany are usually present, unless some coils of intestine intervene between the mass and the abdominal wall.

(3) **Cysts.**—Not infrequently cysts are of such a size as to render their differentiation from ascites a matter of practical difficulty. Those most commonly encountered arise from the ovary or parovarium, in which cases dullness will be found over the mass in the middle line anteriorly, and tympany in the flanks. Mensuration, vaginal and rectal examinations, etc., must also be employed.

The thrill felt over the hydatid cysts may occasionally be demonstrable.

(4) **Pneumoperitoneum** following perforations is shown by disappearance of normal liver dullness, "Peter's phenomenon," abdominal distention, etc.

IV. **AUSCULTATION** is seldom of any practical value except in the diagnosis of *pregnancy*, when the fetal or placental sounds may be heard. Occasionally *peritoneal friction* over inflamed areas or *vascular murmurs* over aneurysmal dilatations may be encountered.

The late *George Peters*, of Toronto, described a **"telephonic phenomenon"** heard in cases of general

peritonitis, in which the heart sounds become audible over the abdominal cavity He suggested that on account of intestinal paralysis the air in all the coils would be under the same pressure, and hence would more readily lend itself to the transmission of sounds than under normal conditions. At times, however, this is present to a slight extent in cases of marked tympanites. It is possible that the sounds may be transmitted by the tense abdominal wall alone, and not through the contents of the cavity.

V. **Auscultatory percussion** is of a certain value in the outlining of the abdominal organs, especially the stomach, liver, and spleen.

VI. **Mensuration** is frequently of the greatest importance in confirming the diagnosis of certain abdominal tumors.

First measure the *circumference* at the umbilicus and at two other levels—namely, half-way between the umbilicus and symphysis, and also midway between the former point and the sternum; these measurements may aid in settling the point of origin of abdominal tumors.

Determine the *distance from the umbilicus to the pubic symphysis and to the base of the sternum.* Normally the umbilicus should lie 1 inch nearer the latter. This measurement is of value in determining whether an intra-abdominal tumor originates in the epigastric region or is rising from the pelvis, in either of which cases the normal measurements will be disturbed.

The *distance from the umbilicus to each anterior superior iliac spine* must now be taken. In tumors confined to either of the lower abdominal quadrants, the distance between these points on the affected side

will be found to be greater than its fellow. In ascites and tympanites as a rule no asymmetry in these measurements will be found.

It is well in all examinations to ascertain the *ratio of the circumference of the thorax* (at the level of the nipples) *to that of the abdomen* at the umbilicus. This is especially insisted upon by life insurance companies.

C. **SPECIAL EXAMINATION OF THE ABDOMINAL ORGANS.**—As the general principles involved in the diagnosis of abdominal conditions and the examination of the upper food passages have been dealt with, we are in a position to proceed with a more detailed investigation of the remaining organs connected with the digestive system, taking up in order the stomach, small intestines, appendix, colon, rectum and anus, liver (gall-bladder), pancreas, and spleen.

I. **THE STOMACH.**

(1) **Inspection.**—In the normal individual there are no physical signs pointing to the presence of this organ; in pathological conditions, however, certain visible phenomena may be of the greatest value in diagnosis.

In *gastrectasis*, or dilatation of the stomach, the outlines of the distended organ are frequently visible, especially in thin-walled individuals, the greater curvature being found to lie below the level of the umbilicus. Not infrequently this condition is associated with *gastroptosis*, or displacement downward of the organ. If this latter be marked, great dislocation of the stomach may be present. Not infrequently the lesser curvature may be visible as a transverse shadow in the epigastric region.

To confirm the presence of these conditions we may **inflate** the organ by means of the "stomach-tube"

and a Higginson's syringe; or may administer by mouth the "blue" (alkaline) portion of a Seidlitz powder in solution, and then gradually that of the "white" (acid) part in solution. The visible distention that follows should be noted and confirmed by percussion. These maneuvers must not be used indiscriminately; excessive weakness of the subject, a recent history of gastric ulcer with hematemesis, marked arteriosclerosis, and cardiac disease should all be considered as contraindications to gastric inflation.

The above methods are of value in enabling us to determine the size and position of the stomach, and may frequently be of assistance in the diagnosis of gastric tumors, especially when combined with palpation and percussion.

Gastric **peristaltic waves** are not infrequently visible. Normally they should pass from left to right, but in pyloric stenosis they may pass in the reverse direction (*retroperistalsis*), in which case evidences of dilatation or a tumor mass may be seen.

In the place, however, of continuous waves traveling across the epigastric region, peristalsis may show itself as a *transient muscular tumor* of the stomach wall, appearing and then disappearing suddenly over different portions or traveling as a distinct mass across the epigastrium. These waves may be accentuated by flicking the skin with a towel moistened in cold water.

Gastric *tumors* unless large are seldom visible; but when seen they are usually subject to marked respiratory mobility.

(2) **Palpation** should be utilized to confirm inspection, especially regarding the diagnosis of *gastric tumors*. These must be differentiated from the "per-

istaltic tumors" above mentioned, from local spasms of the recti abdominalis; from hepatic, pancreatic, and retroperitontal cysts or growths, from tuberculous peritonitis, from abdominal aneurysms, and from tumors of the gall-bladder, duodenum, or colon.

Tenderness is a frequen. sign in disease of the stomach and is usually accompanied by the symptom pain. Especially is it found in inflammatory conditions and in ulcers (simple or malignant), being definitely localized in the case of the latter. Where tenderness is due o *peritonitis*, the recti remain rigid and contracted, even if steady pressure is exerted and the patient's attention be distracted from the abdominal manipulations. In most other conditions a certain amount of relaxation is found to occur. This method is of peculiar value in differentiating hyperesthesia rom severe pain due to organ lesions.

Succussion or splashing sounds are frequently demonstrable over dilated stomachs. When this phenomenon is detected three hours after a meal, it points to gastric atony, and, if present below the level of the umbilicus, to gastrectasis. It is elicited by making short, sudden "dipping" movements with the fingers over the epigastric region, or by employing palpatory percussion, using several fingers as a plessor.

The outlines of the stomach may be palpated during distention of the orgin either with gas or liquid. When for any reason inflation is undesirable the patient may be given a tumbler of cold water and his abdomen immediately palpated, a difference in temperature over the area occupied by the stomach will usually at once become recognizable, especially if the patient be in the erect posture.

(3) **Percussion** is of special value in connection with inflation in determining the size and position of the organ, and in localizing tumor masses of its anterior wall.

It is best directed in radiating lines away from a point just below the left costal margin in the parasternal line. Here, of course, in place of percussing from resonance to dullness, we are merely percussing from one "pitch" of tympany to another: in other words, from one hollow air-containing organ (stomach) to others (colon and small intestines), hence difficulty will frequently be encountered unless the stomach be simultaneously inflated.

The *gastric percussion note* varies greatly in health, according to the degree of tension of the stomach walls and the character of the contents, as also does that elicited over the colon and small bowel. Normally no stomach resonance should be found either below the umbilicus or to the right of the middle line. If the organ be distended, it may extend to the left as far as the area of splenic dullness, and upward to the fifth rib in the mid-clavicular line and the seventh rib in the anterior axillary line.

The lower border may also be demonstrated, when for any reason inflation is contraindicated, by percussion with the patient in the erect posture after drinking a glass of water.

Actual *diminution in the size* of the stomach is seldom seen apart from stenosis at the cardiac orifice or esophageal stricture. *Traube*'s *space* has already been referred to on page 96 and therefore requires no further mention.

(4) **Auscultatory percussion** is used for the same

purposes as simple percussion, but probably gives more accurate results. The stethoscope is placed over the center of the organ and percussion or "flipping" carried out by the finger in radiating lines toward the bell, starting well beyond the suspected limits of the stomach.

(5) **Auscultation** is of little value. Peritoneal friction sounds may occasionally be detected, and the heart sounds or even murmurs may at times be transmitted to the gastric area through a dilated stomach, although they are not propagated over the remainder of the abdomen as is sometimes found in general peritonitis (Peter's sign).

(6) **Other Methods.**—The interior of the stomach may be rendered visible by means of the gastroscope. Gastrodiaphany or transillumination by means of an electric lamp and flexible stomach-tube, and radioscopy (the use of the *x*-rays), especially when combined with the administration of bismuth subnitrate, are both of value at times in diagnosis.

(7) **Gastric Analysis** (see Appendix).

II. **THE INTESTINES.**

(1) **The Small Intestines.**—The general examination of these organs has already been described under the section on general abdominal examination, and the presence of peristaltic waves, tympanites or meteorism and tumor masses referred to. Several additional points, however, deserve attention.

Intestinal obstruction may be due to a number of causes, among the more important of which are tumors, scybala, strictures, volvulus, intussusception, hernia (internal and external), and strangulation by peritoneal bands or adhesions. The outstanding signs of obstruc-

tion consist in general abdominal distention, excessive peristalsis, constipation, fecal vomiting, and not infrequently the presence of a local mass.

Chronic peritoneal tuberculosis shows itself either by a general retraction of the abdominal wall with tenderness, localized collections of fluid, and the presence of irregular, firm intra-abdominal masses; or as a simple peritoneal effusion (ascites).

(2) **The Appendix.**—This organ when inflamed gives rise to tenderness over *McBurney's point* (page 202), and when enlarged or accompanied by perityphlitis gives rise to a palpable *mass* in the right iliac fossa. In certain cases it may hang over the brim of the pelvis or lie pointing upward behind the ascending colon and so complicate the matter of diagnosis.

Pressure over the inflamed organ, in addition to demonstrating the presence of tenderness, often gives rise to referred pain in the epigastric region.

(3) **The colon** is not infrequently the seat of *tumors* which are usually malignant in character. They may give rise to obstruction of the bowel, and not infrequently in this way cause oblong masses of impacted feces to form above them.

These fecal masses or *scybala* are doughy in consistence and must be carefully differentiated from new-growths, so that it is imperative before completing the examination of any case to completely empty the bowel of its contents by a purgative enema. It is also necessary to make a rectal examination.

Acute *colitis* shows itself by tenderness along the line of the large bowel, and cicatricial *stenosis* (usually syphilitic) by evidences of obstruction.

It is well to remember that an empty and contracted

colon may be distinctly palpable as a firm smooth tube, even if no disease be present.

The colon may be *inflated* with air or with warm water by means of high postural enemata, and so be rendered more easily palpable. This maneuver is of special value in the localization of strictures.

(4) **The Rectum and Anus.**—It is advisable first to *inspect the anus*, for which purpose the dorsal decubitus with both knees drawn up and abducted is the most convenient. The gluteal folds are now separated and hemorrhoids (piles), fistulæ, mucous patches, fissures, etc., are thus brought into view.

The *digital examination of the rectum* should reveal the degree of sphincter tone, the presence of "Houston's folds" of mucous membrane (which lie one on the anterior surface behind the prostate and the others slightly higher up and on the posterior wall of the rectum), the prostate, the female generative organs, and the degree of distention of the bladder. It is well either to wear a rubber finger cot, or fill the nail groove with soap and oil the examining finger. The lower bowel should always be emptied by means of an enema, as also should the bladder before making an examination.

Among the pathological conditions that may be met with, the following are the more important ones: hemorrhoids (not always palpable), fistulous openings, scybala, enlarged lymph glands, strictures, and tumors or ulcerated surfaces. For further details regarding these conditions reference must be made to surgical works.

The interior of the rectum can be rendered visible by means of a *proctoscope* and reflected light. The lo-

cation of tumors or strictures may also be determined by the use of a *rectal bougie.*

(5) **Examination of Feces** (see Appendix).

III. **THE LIVER.**

(1) **Inspection** is of little value and should not be relied upon as a diagnostic agent, since palpation gives much more accurate results.

(2) **Palpation.**—The general *technic* of palpation requires no further mention, but in applying this manipulation over the liver it is usual to place the patient in the dorsal decubitus and to first make a rough estimate of the thickness and resistance of the abdominal parietes.

After doing this the tips of several fingers are placed on a line parallel to the costal arch, about 1 inch below it and just external to the right rectus abdominus. The patient is now directed as far as possible to relax the abdominal walls and to breathe freely with the mouth widely opened. In some cases it is found advantageous to "draw up the knees," but if this is done they must be supported in order to insure complete relaxation.

The palpating fingers may now be sunk slowly under the costal arch, pushing before them a fold of skin, until the lower margin of the organ is felt. *Abrupt movements must always be avoided* on account of the contractions which they set up in the muscles of the abdominal wall.

The radial border of the hand placed parallel to the lower ribs may also be used in place of the method above described, the thumb being, of course, abducted and kept from contact with the skin.

Where *free fluid* exists in the peritoneum, "dipping"

may be used (page 201). When it is desired to feel for *hepatic pulsation*, the left hand palm upward is laid under the lower ribs posteriorly and the right one flat over the anterior surface of the enlarged organ. If marked tricuspid regurgitation is present, causing the hepatic pulsations, then overfilling of the jugular veins is liable to occur when the organ is compressed between the hands in the above manner.

It is well at this point to note the occasional presence of *Reidel's lobe*, which anomaly may at times be mistaken for an enlarged gall-bladder or neoplasm.

In the healthy adult the *lower edge of the liver* can only very rarely be felt and the gall-bladder cannot be palpated.

In some healthy individuals whose abdominal walls are thin and lax, especially women, the lower margin may be felt, particularly during inspiration which normally lowers the margin about 1/2 inch. In children, however, the margin can usually be felt with comparative ease.

When, therefore, the lower border of the liver is easily palpable, one of two conditions must necessarily be present: either the organ is enlarged or it is displaced downward. Recognition of the *position of the upper margin* of the liver by means of percussion will at once differentiate between these conditions, for in dislocation downward the upper border descends as well as the lower, and the normal amount of hepatic dullness remains unaltered; while in an enlargement, the upper margin is usually displaced upward and the area of the vertical liver dullness is increased.

The *character of the lower margin* must be determined; note whether the surface is smooth, sharp or rounded,

finely or coarsely granular, irregular, studded with nodules, or umbilicated. The presence of tenderness should also be noted.

The size of the organ having been determined and recorded by means of surface mensuration, its shape should be ascertained.

Asymmetry may occur as the result of "tight lacing," of the pressure of a tumor mass, or as a congenital anomaly (Reidel's lobe).

Suppose now that in any given case we have been able easily to palpate the lower border or edge of the liver and proved that the organ is not merely displaced, then we have to deal with an hepatic enlargement.

(a) **Symmetrical enlargement** may be due either to fatty infiltration, amyloid disease, passive congestion ("nutmeg" liver), leukemia, etc., in which cases the surface is usually smooth; or to hypertrophic cirrhosis (Hanot's), the earlier stages of the atrophic variety (Laennec's), or to fatty cirrhosis, under which circumstances the surface is more or less granular.

(b) **Asymmetrical or irregular masses** may be due to malignant disease (cancer or sarcoma), syphilitic hepatitis, abscess, hydatid cyst, etc. Umbilication if demonstrable points to the presence of a malignant growth.

Diminution in the size of the liver is found in the later stages of atrophic cirrhosis and in acute yellow atrophy; the former is a common chronic disease and the latter a rare and rapidly fatal one.

Enlargements of the gall-bladder are usually cystic in character and pyriform in shape; they must be differentiated from malignant nodules in the liver or neigh-

boring organs, from Reidel's lobe, and from a displaced or diseased kidney. The gall-bladder moves freely on respiration, and laterally on manipulation unless peritoneal adhesions be present. Other signs and symptoms will, of course, facilitate the diagnosis. Cholecystitis is shown by the presence of tenderness over the gall-bladder and not infrequently by referred pains in the left hypochondrium or region of the right scapula.

The *cystic duct* may become obstructed (generally by a stone or calculus), but as a rule no great enlargement of the gall-bladder occurs. When, however, the *common bile duct* becomes for any reason impervious, great distention of the bladder may ensue.

Courvosier's law refers to the fact that when the common duct is obstructed by gall-stones the gall-bladder is collapsed, but when it is obstructed by malignant disease the gall-bladder is frequently found distended. This is explained by the fact that chronic cholecystitis is a complicating condition when calculi are present, and this process results in thickening and contraction of the gall-bladder, thereby limiting distention.

(3) **Percussion** is of special value in outlining the upper margin of the liver.

Deep percussion must be carried out from above downward in the mid-clavicular, mid-axillary and scapular lines, starting well above what we know from our topographical anatomy to be the normal upper limit of the liver. Having thus marked out the upper margin of the liver, deep percussion is continued downward in the same vertical lines until the tympanitic intestinal note is reached. The area thus marked out is called that of the **"deep" or "relative liver dullness."**

On the other hand, it will be remembered that below the level at which lung resonance gives place to liver flatness lies the region of **"absolute liver dulness."**

The left lobe of the liver, since it lies in contact with the heart, cannot be demarcated from it as we have already shown; similarly posteriorly near the spine the nonresonant renal and hepatic areas fuse. In the epigastrium the dull liver note is lost or becomes indefinite on account of the bellies of the recti and the gastro-intestinal tympany.

In children the upper border is normally higher and the inferior somewhat lower than in adults; in the aged the opposite condition is frequently found. It is well to note that posture exerts a slight influence on the position of the liver, it being somewhat lower with the body in the erect than in the horizontal position.

As regards **pathological alterations in the liver dullness,** several points demand consideration.

The *upper border may be found higher than normal,* either due to a real increase in the size of the liver or its displacement upward. The latter results either from plus abdominal pressure (particularly ascites or large cysts) or from paralysis of the diaphragm when the elastic retraction of the lung draws the liver upward.

The *upper border may be lower* than normal in cases of emphysema and pneumothorax; the *lower border may be higher* in atrophic cirrhosis, acute yellow atrophy, and tympanites.

Liver dullness may to a great extent be *obliterated* by free gas in the peritoneal cavity, pointing usually to gastric or intestinal perforation. It occurs also in those rare cases of complete dislocation of the organ.

Percussion thus enables us to determine whether the

liver is enlarged or diminished in size, or merely dislocated. It must not be forgotten that certain conditions, such as a right hydrothorax, or a pneumonic or subphrenic abscess as well as displacing the liver downward, may give rise to an *apparent increase in the liver dullness*, because the nonresonant note given by them is difficult to differentiate from that due to the liver.

(4) **Auscultatory percussion** may be used in determining the outlines of the liver.

(5) **Auscultation** is of no value beyond aiding in the detection of perihepatitis by finding a friction rub. A continuous soft murmur may be heard in occasional cases of tricuspid incompetence.

IV. **THE SPLEEN.**

(1) **Inspection.**—In massive splenic enlargements or with a markedly dislocated or "floating" spleen, the tumor mass may be visible. In the former the tumor always moves with the respiratory excursion, in the latter only occasionally.

(2) **Palpation** is the most reliable method to employ in the detection of enlargements or displacements of the spleen. Normally the organ cannot be felt, so that when it is palpable the presence of one or other of the above conditions should be suspected.

The *most useful method* consists in standing on the right side of the patient (who should be supine, directed to relax the abdominal wall, and breathe deeply through the open mouth), in passing the left hand across the abdomen, and after placing it behind the normal position of the organ, gently lifting the lower ribs forward and toward the patient's right side; the examiner's right hand is then applied, palm down-

ward, with its radial border about 1 inch below the costal margin and parallel to its arch in the region of the anterior axillary line. The right hand is now allowed to slowly sink into the abdominal wall just below the costal margin and await the descent of the organ at the end of a deep inspiration. The anterior border of the spleen may thus readily be felt when the organ is enlarged or displaced.

It has been found that if the patient assumes the right lateral decubitus, palpation of the organ is often greatly facilitated, as the spleen then tends to drop forward toward the middle line.

When enlargements are present, the spleen passes downward and forward toward the navel. Occasionally the tumor is of sufficient size to pass beyond the middle line and even reach the right iliac fossa and pubes. If the costocolic fold, however, is greatly developed and tense and firm, it may prevent the downward displacement or growth of the organ.

(a) The spleen when symmetrically **enlarged** shows a smooth anterior border with one or more of the notches occasionally palpable; this edge is directed downward and inward. The mass moves downward with each inspiration, and usually the palpating fingers may be sunk into the space between its posterior edge and the left erector spinæ muscle.

These enlargements may be *uniform* and due to acute conditions (typhoid, tuberculosis, syphilis, or other infections) or passive (cardiopulmonary or portal) congestion; to malaria, leukemia, amyloid degeneration, Hodgkin's disease, splenic anemia, or rickets. They may occasionally be *irregular* as in malignant disease, hydatids, and abscess of the spleen.

(*b*) **Displacement** may be the result of thoracic deformity, a pleural effusion, or pneumothorax. It may be part of that condition known as enteroptosis (*Glenard's disease*), in which a general downward displacement of the abdominal viscera is found.

Differential diagnosis must be made from tumors of, or fecal accumulations in the splenic flexure, from scybala, from enlargement of the left hepatic lobe, from carcinoma of the fundus or cardiac end of the stomach, from abdominal cysts, and also from renal tumors or displacements.

(3) **Percussion.**—In outlining the spleen by percussion the same general technic is used as in the examination of other organs. Several points, however, should be noted. If possible the patient should be in the sitting posture and percussion carried out at the end of a deep expiration in order to eliminate as far as possible the influence of lung resonance which may tend to obliterate splenic dullness.

To define the *upper border*, moderately light percussion should be commenced just outside the left scapula and in the posterior and mid-axillary lines at about the level of the seventh rib and carried out, gradually passing downward until the splenic dullness is encountered. It is well to note that the upper and posterior splenic margins are covered by lung and that only a "relative" dullness is found here.

The *anterior border* is found by percussing along the eighth, ninth and tenth interspaces from before backward, commencing in the anterior axillary line; but it is often difficult to find on account of the presence of tympanites.

The *lower margin* is found by repeating the maneu-

ver from below upward in the posterior axillary line, commencing just below the iliac crest.

The *posterior edge*, however, cannot be defined on account of the presence of the dull note obtained over erector spinæ muscle.

Normally, an oval, dull area, 2 inches across by about 3 in its long axis, should be recognizable by percussion, lying between the ninth and eleventh ribs, and reaching as far forward as the midaxillary line.

Percussion is of the greatest value in differentiating dislocations from enlargements. In the case of "*floating spleen*" a band of resonance is found where the normal splenic dullness should be encountered, while the organ itself can be palpated or outlined by percussion in an abnormal position. Again, when the spleen is displaced by pressure from above, the presence of the causative factor can usually be easily demonstrated by percussion.

(4) **Auscultatory percussion** may also be used in defining splenic dullness.

(5) **Auscultation** is of little value except in the recognition of the friction rub of perisplenitis.

CHAPTER IX.

THE GENITO-URINARY SYSTEM.

A. THE KIDNEYS.

I. **INSPECTION.**—No visible evidence of the presence of the normal kidneys is to be found. Neoplasms and cystic kidneys, however, may occasionally be seen. As a rule, these masses are but slightly affected by the respiratory excursions.

A perinephritic abscess may show itself as a rounded, inflamed, edematous swelling in either loin.

II. **Palpation.**—The organs cannot easily be felt in the well-developed adult. In thin persons with lax abdominal walls and in some children, it is possible to feel their lower poles, this being especially true of that of the right organ during a deep inspiration.

The kidneys usually show a *slight downward movement during inspiration*, due to the contraction of the diaphragm.

(1) **Technic.**—Several methods are utilized in the palpation of the kidneys, the most useful being applied as follows, the subject being in the dorsal position with the head on a pillow and the abdomen relaxed:

To *palpate the right kidney* the examiner should take up a position on the corresponding side of the bed and place the left hand under the loin between the last rib and the crest of the ilium; his right hand should then be pressed firmly backward over the mid-clavicular line at the level of the umbilicus. While the patient is directed to breathe slowly and deeply the examiner's

right hand follows in the abdominal wall during each expiration and his left lifts forward the posterior parietes.

When the organ is palpable, a rounded mass is felt to descend slightly and separate the fingers of the two hands during inspiration and to recede as the diaphragm relaxes.

The same method is used in the examination of the left organ, the operator, however, standing on the left side and reversing the hands, or he may remain on the patient's right, pass his left hand across the body, place it under the loin, and with his right palpate the lumbar region.

The organ may also be palpated with the patient leaning forward over the back of a chair, while the examiner stands behind with his arms around the patient's side and applies his hands to the abdominal wall just below the costal margin.

(2) **Displacements.**—The kidneys are found to be freely movable in certain conditions, the right being that usually most affected.

The kidneys in these cases are freely *movable* in the loose retroperitoneal tissues and may be felt to slip up and down between the palpating hands during the respiratory acts. In marked cases it is often possible to sink both hands in above the displaced organ, and so prevent its ascent with expiration.

When the kidney, instead of simply being movable in the subperitoneal areolar tissues, has acquired a complete covering of peritoneum and is attached by its layers to the posterior abdominal wall as by a mesentery, it is called a *floating* kidney.

There may be all degrees of renal mobility, the organ

sometimes even reaching the pelvis. It can be distinguished from tumors of other parts by the "sickening" sensation felt as the organ is squeezed, by its smooth surface, rounded margins, and characteristic renal shape.

(3) **Enlargements** may be due to numerous causes. The smaller tumors cannot, as a rule, be felt unless they are accompanied by some displacement. The more common enlargements of the kidneys are due to cystic degeneration, hydro- or pyonephrosis, malignant tumors and tuberculosis.

Perinephric abscesses may be mistaken for renal tumors as also may tumors of the suprarenal capsules. In these conditions the organs lose their characteristic outline and move only slightly with respiration, which latter fact may be of importance in arriving at a diagnosis from tumors of surrounding organs, especially those of the liver, gall-bladder, and spleen. Occasionally "fetal lobulation" may be palpable.

Enlargements may easily be differentiated from those conditions in which the organ is merely movable.

The differentiation of renal growths from an enlarged and cystic gall-bladder, an enlarged spleen, tumors of the liver or colon, and psoas or perinephritic abscesses is at times difficult and occasionally almost impossible by simple palpation.

As regards the *splenic tumors*, a space can usually be demonstrated between the posterior margin and the erector spinæ muscle; and, since the colon lies behind these organs, it gives rise to an area of resonance on percussion posterior to the tumor.

III. **PERCUSSION** is of no value in localization of the kidneys, but at times is useful in *distinguishing*

between enlargements of the spleen and renal tumors. Since the colon always lies anterior to renal tumors, we are usually able to demonstrate the fact that no resonance is found between the posterior margin of the tumor and the lumbar muscles; whereas, in splenic enlargements a tympanitic bowel note is usually found in the latter situation. To faciltate this maneuver the colon may be inflated with air by means of an Higginson's syringe.

The use of the *x*-ray in the diagnosis and localization of calculus is often of the greatest value, especially from the standpoint of the surgeon.

IV. **THE URINE** (see Appendix).

B. **URETER, BLADDER AND URETHRA** (see works on surgery of the urinary organs).

C. **PROSTATE AND SEMINAL VESICLES** (see works on surgery of the male genital organs).

D. **FEMALE GENERATIVE ORGANS** (see works on gynecology).

CHAPTER X.

NEURO-MUSCULAR SYSTEM.

A. **THE PHYSIOLOGY.**

It is essential before proceeding with the examination of the nervous system to review in as concise a manner as possible the main points of its anatomy and physiology.

Both the brain and spinal cord are built up of two tissues, one active and composed of nerve cells capable of conducting impulses (*neurons*), the other passive and consisting of cells which act as a framework or connective tissue for the support of the former (*neuroglia*).

Each **true nerve cell** is composed of a cell body containing a nucleus and possessing certain branches, which latter are of two types. The main process or *axon*, which is generally single, conducts impulses centrifugally or peripherally and, after leaving the parent cell, acquires its sheaths, gives off short branches on its course (collaterals), and finally ends in specialized terminal sense organs, "end-plates", etc., or breaks up into branches which arborize with the dendritic processes of another cell body.

The second set of processes or *dendrons* are short and multiple in contrast to the axon which is usually single and of greater length; they arborize; but do not anastomose with those of the surrounding cells and with the axons of other cells. They conduct impulses centripetally or toward the cell body.

The primary nerve cell with its processes is termed

a **neuron,** and these neurons are of two types, termed either motor or sensory, depending on their function.

From the above it will be seen that the nervous system is composed of innumerable cells specially differentiated to perform the function of conduction (neurons) supported by a special type of connective tissue (neuroglia); also that each neuron is apparently not directly continuous with any of its fellows, but that their branches are merely contiguous. Hence we may consider each neuron as a separate anatomical unit and dependent for its vitality on its own cell body and nucleus.

The above description applies to a typical neuron, the size, number, and length, however, of the dendrons vary greatly, as do also the axons which in some instances are multiple.

These *neurons are collected together into bundles* in the central nervous system according to their functions; those specially differentiated for the transmission of motor or sensory impulses are grouped together in the brain and cord into what are termed, respectively, *motor or sensory tracts* and after leaving the central system into motor or sensory (efferent or afferent) peripheral *nerves*. In the latter fibers of both types may be present in the same trunk, forming what is termed a "mixed" nerve. Fibers governing vasomotor tone may also be present.

I. **THE MOTOR NEURONS.**—Motor inpulses are primarily generated in the cells of the cortex of either cerebral hemisphere and carried to their destination by the axons, the whole nerve unit constituting what is known as an **upper** or **central motor neuron.**

These fibers (axons) are gathered together into

bundles known as the **motor tracts,** which pass successively downward, one from each hemisphere, through the *corona radiata, internal capsule, crus, pons, and medulla,* in which the majority of fibers from the two sides of the brain cross to the opposite sides of the spinal cord at the *decussation of the pyramids,* and then proceed downward forming the *crossed pyramidal tracts.*

A few of these axons, however, do not decussate in the medulla, but travel downward in the cord forming what is known as the *direct pyramidal tract*; but they too ultimately cross in the gray matter to reach their destination on the opposite side.

It will now be seen that *all fibers from one cerebral hemisphere finally reach the opposite side of the spinal cord,* where their axons break up into numerous branches which arborize around the dendrons of the lower motor neuron cells found in the gray matter of the anterior horns.

This whole nerve unit has been termed the upper motor neuron to distinguish it from that next to be described whose function it is to carry on the impulses from the terminals of the upper neurons in the cord to the muscles themselves.

Each **lower or peripheral motor neuron** consists of a cell body and nucleus situated in the anterior horn of the cord (multipolar cells), whose *dendrites* arborize with the terminal branches of the axis-cylinder process (axon) of an upper neuron, and also with certain sensory fibers.

Its *axon* leaves the cord by the anterior root, acquires its sheaths, and forms a peripheral motor nerve whose terminal twigs end in the muscle fibers themselves.

Since every voluntary motor impulse generated in

the cells of the cerebral cortex must pass downward along one of the paths above described, it follows that any interference with the conductivity of either neuron, in any part of its course will result in a loss of motor power, and the patient will show signs of **paralysis or paresis.**

In other words, it is obvious that every patient complaining of a loss of voluntary muscular power (except in certain "functional" disorders, the myopathies, and in malingerers) must have an organic lesion in some part of his motor tract, either in the upper or lower neuron; and it is now our duty to discuss the differences in the signs produced, according to whether the upper or lower neuron be the one affected.

It has been found that the impulses which travel down the upper neuron in addition to giving rise to muscular contractions also exercise restraint over the action of the lower neurons so that, speaking generally, supposing the **upper neuron be injured** or destroyed in any part of its course, in addition to loss of voluntary power or *paralysis* of the muscles supplied, they will be found to be *spastic* or in a state of rigidity (hypertonus) from uncontrolled action of the lower neuron, the deep *reflexes* which will be described on page 272, are increased, and in as much as it has been shown that the voluntary muscles are dependent for their nutrition on the parent cells of the lower motor neurons, it follows that *no atrophy or wasting* except from disuse of the affected parts takes place, nor will there be any change in their *electrical reaction* (page 276).

On the other hand, supposing that the upper remains intact and that the **lower neuron shows a lesion** in any part of its course from the primary cell in the anterior

horn to the termination of its axon in the muscle, a *loss of voluntary power* will again be evident and *atrophy of the muscle* will take place, since the latter is cut off from the cells in the cord which govern its nutrition. The paralysis present will be *flaccid* (hypotonus), since no impulses whatever can reach the muscles involved, the deep *reflexes* will be lost, and the electrical excitability also will be found to be affected, the "*reaction of degeneration*" being present (page 277).

The importance of a thorough knowledge of what has been said above is, that in it lies the basis of the explanation of all the signs found where loss of muscular power is evident; so that instead of having to remember a different set of symptoms for each disease in which paralysis is a feature, the examiner can always apply certain general principles to the case under observation and vary the individual particulars as required.

II. **THE SENSORY NEURONS** are more numerous and complicated in their arrangement and communications than those of the motor system; they convey sensory impulses from the various parts of the body to the brain.

The **lowest neuron** of the chain has its parent cell body in the posterior root ganglion. Two processes leave this cell, one of which passes in a sensory nerve to the periphery, the other enters the spinal cord by the posterior root, in which its course will be traced later.

In the **peripheral sensory nerves** the fibers have been shown to be gathered into systems of three types, one is termed *protopathic* and is composed solely of fibers whose function is to carry impulses arising from the effects of pain and the extremes of heat and cold on the sensory nerve terminals.

A second system exists for "*deep sensibility*" through which sensations of deep pressure and of the movements of joints, tendons and muscles are carried; these fibers run with the nerves to the skin.

The third or *epicritic* system is that in which the fibers involved in the accurate cutaneous localization of light touch, the discrimination of the points of the compass (or esthesiometer), and the recognition of slight differences in temperature are situated (Head, Rivers, Sherren).

From what has been said it will be seen that since the fibers for deep sensibility run chiefly with the nerves to the muscles, this form of sensation may be intact even in the presence of cutaneous anesthesia, and also that great care must be exercised in testing superficial tactile sensibility in order that the fibers of the deeper system be not stimulated; the lightest substance, as cotton wool or a camel's-hair brush, should therefore be employed in the investigation of the sense of touch.

Also it has been found that the protopathic and epicritic fibers are not always equally affected by pathological processes.

Again, when a *divided nerve is undergoing reunion*, the returning painful and thermal sensations are the first to be recognized; the fibers governing the nutrition of the part are also early regenerated, as shown by the fact that any trophic disturbances rapidly improve as the sense of pain returns. Later the affected area becomes sensitive to light touch and the slighter differences of temperature.

It is to the researches of Frey and Head that we are indebted for the knowledge that touch and pain are subserved by separate fibers, and also that those which

conduct the latter impulses regenerate more quickly than those for touch.

The sensory fibers after entering the cord become rearranged anatomically and, as regards their function, those carrying impulses connected with the various forms of sensation unite into groups composed of those for touch, pain, etc., whose further course will now be traced.

The **chief sensory tracts in the cord** are:

(1) The *columns of Goll and Burdach*, composed of long fibers which have entered by the posterior roots and which pass upward in these tracts to arborize in the nucleus gracilis or cuneatus, of the same side of the medulla, around the cell bodies of the next set or relay of neurons.

In the gray matter of these nuclei lie the parent cells of another set of neurons, whose axons decussate in the medulla, pass upward in the fillet, and arborize similarly in the optic thalamus around a third set of cell bodies whose axons pass through the internal capsule and terminate in the cerebral cortex.

The function of this first set of long fibers of the posterior columns is to conduct impulses for **tactile discrimination** (compass test), also for **passive movements and sense of position** ("muscle sense").

A lesion in these columns therefore results in *ataxy and incoordination* of muscles, as well as in interference with the sense of *touch.*

A certain number of those *exogenous* fibers whose function also is to carry tactile impulses, pass a short distance up in these columns, cross in the gray matter of the cord to the opposite side, enter the ascending tracts of the anterior column, and finally terminate in

the optic thalamus. Also a few fibers of the posterior column are termed *endogenous* and merely pass between contiguous segments of the cord.

(2) Those exogenous fibers associated with the conduction or **painful and thermal sensations** enter by the posterior roots, pass a short distance up the cord, and arborize around certain cells in the gray matter of the same side.

From these cells a second set of axons arise, cross in the gray matter to the opposite side, enter the *antero-lateral column of Gowers* and terminate, as do those for tactile sensibility, by arborizing around the cell bodies of the optic thalamus; a few, however, enter the cerebellum by the superior peduncle.

(3) Certain fibers of the posterior roots end around a group of cells situated at the base of the posterior horn, known as *Clarke's column*, from which cells arise a second set of axis-cylinder processes which travel upward in the direct cerebellar, or dorso-lateral tracts, and terminate in the cerebellum.

They conduct impulses governing **coordination from bones, joints, muscles and ligaments,** loss of which results in ataxy.

(4) Lastly it has been shown that numerous sensory fibers pass through the gray matter to arborize around the lower motor neuron cells of the anterior horns. Their significance will be discussed with the *reflexes* (page 234).

We are now in a position to appreciate the results of a **complete destruction of the cord,** which are paralysis and loss of all forms of sensations below the lesion, and a complete absence of all reflexes (page 236).

It will also now be seen that on the side of a com-

plete **unilateral destruction of the cord** and below the level (*Brown-Sequard paralysis*) there will be in addition to paralysis on the side of the lesion a loss of painful and thermal sensations on the opposite and loss of muscle and tactile sensibility on the same side as the lesion. Touch and "muscle sense," however, are retained on the side opposite to that of the lesion, since decussation of these fibers does not occur until they enter the fillet.

When the **commisural gray matter** of the cord alone is involved by a diseased process (syringomyelia, etc.) loss of pain and temperature sense, from destruction of those fibers already described which cross in this region to enter Gower's tract, will follow, while "muscle sense" and that of touch is preserved, since their fibers which lie in the posterior columns escape.

Also it will now be obvious that as the sensory fibers of all kinds which supply one side of the body have crossed either in the gray matter of the cord or in the fillet and entered the optic thalamus of the opposite side, a lesion of that body will be associated with a **hemianesthesia** of the opposite side of the body, and that the same result will occur also when the posterior third of the posterior limb of the internal capsule is involved.

III. **THE REFLEXES.**—A reflex act consists in the contraction of a muscle or group of muscles in response to a sensory stimulus, and hence for its performance requires a healthy muscle, lower motor neuron (peripheral motor nerve and cell body in the anterior horn of the cord, or in the case of the cranial nerves in the brain itself), intact spinal gray matter, and a normal lower sensory neuron (peripheral sensory nerve and

posterior root ganglion cell) at the level of the segment of the cord under discussion.

A simple reflex act *may be carried out by a single segment of the cord* independently of the brain or remainder of the spinal marrow, but its *activity is modified by influences generated in the higher centers*, and it may to a certain extent be checked voluntarily.

The mechanism of a superficial reflex is as follows: Supposing that, for instance, the skin of the sole of the foot be tickled or otherwise stimulated, a sharp movement of its muscles will be seen to follow after a perceptible interval of time. This stimulation of the skin gave rise to sensory impulses which, traveling centripetally along the sensory nerve fibers supplying the part under examination and entering the cord by the posterior roots, were carried through the spinal gray matter to the anterior horns where the conducting fibers arborized around the parent cells of the corresponding lower motor neurons lying in this situation. These sensory impulses then irritated the anterior horn cells from which motor stimuli passed through the fibers of the anterior roots and peripheral motor nerves to the muscle itself, in which they produced a contraction.

This anatomical nerve path is termed a **"reflex arc."**

It will now be seen that any lesion which interrupts the continuity of any part of the anatomical neuromuscular arc above described will prevent the production of a reflex act, and hence all reflexes at the level of the arc involved will be absent.

Reflexes are divided into superficial (skin or mucous membrane, page 269), deep (tendon, bone, or muscle, page 272), and visceral. The first two classes may show

exaggeration, diminished activity, or may be completely lost. The visceral reflexes will be considered separately (page 276).

The *presence of a superficial or deep reflex* is proof-positive of the continuity of every part of the reflex arc under examination.

We have already stated that interference with the conductivity of the cells or fibers of the reflex arc will *diminish or abolish reflex acts* at the level of the lesion, but they may also occasionally be absent in certain diseased conditions of the upper motor neurons.

Reflexes may be *exaggerated* in nervous individuals, functional diseases, and in certain lesions of the upper motor neurons.

As we have already noted that the **reflexes are influenced by the higher centers,** it is now necessary to qualify that statement.

It seems that the *cerebrum*, through the upper motor neurons which travel down the pyramidal tracts, *exerts normally a restraining or sedative influence* over the lower neurons, so that when this restraint is removed, as in destructive lesions of the upper neuron, the reflexes involved will become exaggerated and the muscles become rigid or take on a state of spasticity.

Charles Bastian suggests that the *cerebellum exerts a stimulating or accelerating influence* over the lower neurons, and hence when the lateral tracts (upper neurons), with their sedative influence are destroyed, the unantagonized accelerating cerebellar stimuli are responsible for the increased reflexes present.

But these cerebellar impulses must be capable of being carried down the cord by innumerable paths since it requires a **complete transverse section of the**

cord to block them; thus, in this latter case, we find that as all influences from both cerebrum and cerebellum are cut off, the reflexes below the point of dissociation are permanently lost. Gowers, however, suggests that in these cases descending changes in the cord occur, which cause such nutritional disturbances as to abolish the function of the cord centers below without necessarily producing a recognizable change in their appearance.

In **partial destruction of the cord** where the conductivity of the pyramidal tracts above the lumbar enlargement is destroyed the lower reflexes will show exaggeration, since the cerebellar stimuli are not cut off. The importance of examining the reflexes in cases of spinal injury to determine if complete destruction of the cord has occurred is now obvious.

IV. **CEREBRAL LOCALIZATION.**—As certain regions of the brain control certain functions, and as in disease these areas are not infrequently involved and the corresponding functions disturbed, we are often enabled by a knowledge of cerebral topography to localize the primary lesion.

In view of the importance of *topical diagnosis* the position of a few of the more important centers will be described and the result of lesions in some of the areas more commonly involved in disease will be discussed.

(1) **Frontal Lobes.**—The main functions of these areas seem to be connected with the higher intellectual faculties; motor and sensory disturbances are absent, but occasionally a fine vibratory tremor of the limbs and diminished activity or loss of the superficial abdominal reflexes on the affected side may be found (page 271).

(2) The **Motor Area of Cortex** which, according to Sherrington, lies entirely anterior to the fissure of Rolando, and is limited to the ascending frontal convolution, contains from above downward the motor areas for the legs, trunk, arms, neck and face; hence lesions in this region will be accompanied by paralytic or spasmodic muscular disturbances (monoplegia, Jacksonian epilepsy, etc.).

A variable amount of loss of sensation is usually also a concomitant phenomenon, that of touch being most affected. The extent of this anesthesia roughly corresponds to that of the motor disturbances.

(3) **Parietal Region.**—Mills is of the opinion that the main cortical sensory areas lie in the ascending parietal convolution, immediately behind the fissure of Rolando, arranged in the same order from above downward as the corresponding motor areas and on the same level.

Other sensory fibers also probably terminate in different areas of the parietal cortex; their course and termination, however, is not so clearly understood as those of the motor system.

(4) **Cuneate Lobes.**—Homonymous or simple hemianopia, in which loss of the corresponding halves of the two visual fields occurs, may be present where these regions are diseased (page 246).

(5) **Angular Gyrus.**—In addition to the presence of sensory aphasia or "word blindness" in disease of this area (page 243) if the lesion is in the left hemisphere (in right-handed individuals); simple hemianopia may also be found.

(6) **Uncinate or Hippocampal Gyrus.**—Alterations of the normal sense of smell and the presence of

Hughlings Jackson's "dreamy states" suggest disease of this region.

(7) **Pituitary Body.**—Lesions in this region which involve the chiasma are usually accompanied by double temporal hemianopia (page 246). There is also a close association between acromegaly and pituitary disease.

(8) **Internal Capsule.**—Hemip egia (complete unilateral paralysis), and hemianesthesia (loss of sensation over one-half of the body) of the opposite side, with homonymous hemianopia, resulting from interference with the fibers connnecting the occipital lobe with the lower visual centers, are usually found.

(9) **Optic Thalamus.**—Hemianesthesia of the opposite side, loss of control over emotions, and the presence of athetoid movements are usually present in lesions of this body.

(10) **Crus Cerebri.**—"Crossed paralysis" with loss of muscular power of the leg, arm, and face are present on the opposite side to a unilateral lesion, and that of the third cranial nerve on the same side. Hemianesthesia and tremors are also not infrequently present.

(11) **Pons.**—Irregular "crossed paralyses" involving the third, sixth, seventh, and eighth cranial nerves of the same side, with the leg and arm of the side opposite to that of the lesion, may occur.

"Crossed hemianesthesia" with the fifth cranial nerve involved on the same side and all other sensory nerves on the opposite side to that on which the lesion is situated is occasionally met with.

Contracted pupils, glycosuria, interference with the bulbar muscles (with the presence of dysphagia and difficulty in articulation), and hyperpyrexia may be

present, especially if the upper part of the medulla be subjected to pressure or disease.

(12) **Corpora Quadrigemina.**—Ocular paralyses, nystagmus, vertigo, and altered pupil reactions to light and accommodation are suggestive of disease of these bodies.

(13) **Cerebellum.**—Loss of tone, paresis and incoordination in the limbs on the same side as lesion, the peculiar gait (page 18), tremors, nystagmus, and "skew" deviation of the eyes are all suggestive of cerebellar disease, especially if accompanied by vertigo and forced rotatory movements of the body.

If the *middle lobe* is involved the "cerebellar attitude" of Hughlings Jackson, with retraction of the head, opisthotonos, and extension of the legs, may be present. A general tendency to fall backward is usually also noted.

On account of the close connection of certain parts of the remaining areas of the cerebral cortex with the subject of *aphasia*, we will for the sake of convenience under the section on speech indicate the cortical centers involved. (Page 241.)

B. **THE CLINICAL EXAMINATION.**—Having now completed our review of the physiology of the nervous system, including as we have under this heading the subject of topical diagnosis and that of the reflexes, we are in a position to proceed with the practical clinical examination of this system with a view to eliminate or demonstrate the presence of pathological phenomena.

In the investigation of the neuro-muscular system it is usual, as we have already noted any outstanding peculiarities in the gait and the presence or absence

of tremors, to proceed with a detailed examination of the speech, cranial nerves, motor and sensory functions (brain, spinal cord, peripheral nerves and muscles), the reflexes, the subject of electro-diagnosis, the mentality, and the analysis of the cerebrospinal fluid in the order above named.

I. THE SPEECH.

(1) **Development.**—It is necessary before discussing the abnormalities of speech to trace briefly its development in the infant.

A cry is the first sound uttered after birth, and it is of reflex origin; at about the end of the first month variations occur indicative of pain or hunger, gradually that of anger is also added.

Smiling occurs about the end of the second month, but laughter seldom until the sixth, "babbling" or "crowing" is, however, the first attempt at using the organs of speech to any purpose. About the end of the first year the simple consonants such as "da-da," "ma-ma" are used, but in an unintelligible manner.

The sounds produced by familiar animals and their application to that animal usually follow in sequence, "gesture-language" is also observed about this period; the vocabulary then rapidly enlarges, so that by the end of the second year a few nouns and adjectives can be correlated and even short phrases expressed.

The understanding of spoken words, however, precedes by many weeks the ability to form the same ideas into articulate speech, usually also until the fourth year pronunciation is indistinct, there being a special tendency to the dropping of consonants.

To learn to speak intelligibly it is necessary that the organs of hearing and the receptive apparatus be

perfect, that the perceptive or higher auditory centers can transform the vibrations (sound) received into "ideas," that the motor centers be unimpaired, and finally that the apparatus for converting the impulses received from this center into articulate speech be complete.

Congenital **"deaf-mutes"** are those who cannot speak because they cannot hear and, as it is only during the third and fourth months that an infant recognizes voices and sounds, it is rarely possible before the end of the sixth to detect evidences of deafness. An infant who at the end of its first year of life has never uttered an articulate sound, but who is otherwise healthy, can be safely said to be deaf or mentally defective. Lastly, a child who has lost the power of hearing before the seventh year, after having once been able to speak, will then begin gradually to lose the power of intelligible articulation and will acquire "deaf mutism," unless instruction in "lip-reading" be commenced without delay.

(2) **Mechanism.**—Let us remember that we have in connection with the speech two mechanisms, a productive (consisting of two divisions, the one connected with spoken, the other with written speech) and a receptive (for the receiving of spoken or written facts).

The "center" for spoken speech is claimed by some to be contained in Broca's and in the lower end of the ascending frontal convolutions and by others to be situated somewhat farther back; that for written speech is situated in the posterior extremity of the second frontal; the receptive apparatus for spoken speech is found in the posterior half of the temperosphenoidal, and the visual speech center is in the angular gyrus. This latter

has also a connection with the primary visual center in the occipital lobes.

It should be remembered that productive speech centers are not actually motor centers, but really centers of association.

(3) **Aphasia.**

(a) *Motor aphasia* refers to the inability to express spoken language. It is usually due to organic lesions but may be merely "functional" (as is sometimes seen after exhausting diseases such as enteric fever), its duration in the latter case, however, being usually only temporary.

(b) *Aphemia* is the loss of power to carry on a conversation and *agraphia* the inability to write; these two are classed as motor aphasias.

(c) *Auditory and visual aphasia*, or "word-deafness" and "word-blindness," respectively, are designated sensory aphasias. These terms refer to an inability to understand the meaning of sounds or writing, although the auditory and visual organs are in themselves intact, the defect lying in the "higher centers."

(d) *Amimia* is a loss of "gesture-language" and *paramimia* the term applied to cases where this function is perverted.

(e) *Paragraphia* describes a condition in which the patient, while able to write, makes use of wrong words.

(f) *Paraphasia* refers to the mistaken use of spoken words.

(g) *Amnesia verbalis* to forgetfulness of words, and

(h) "Recurring utterance" or *eccholalia* to the repetition of certain words in a sentence.

To determine the presence of abnormalities, such as

TABLE OF SIMPLE APHASIAS (*Church*).

Variety	Topography	Understanding of speech		Expression of speech		Repetition of dictation	Copies writing	Writes from dictation	Incidental
		Spoken	Written	Spoken	Written				
Motor aphasia, "dumbness"	Foot of III left frontal gyrus	Yes	Partly	Dumb	Exceptionally	No	Yes	May	Paramimia. Amimia.
Auditory aphasia, "word deafness"	Posterior thirds of I and II left temporal gyræ	No	Yes	Paraphasia	Paragraphia Agraphia	No	Yes	No	Amusia.
Visual aphasia, "word blindness"	Angular gyrus of left parietal lobe	Yes	No	Yes	No	Yes	Yes	Partly but cannot then read it	Homonymous hemianopia if lesion in optic radiation. "Mind-blindness" if lesion bilateral.
Conduction aphasia (Wernicke)	Island of Reil. Lesion usually must cut off fibers between auditory and motor "word-memory" centers	Yes	Yes	Paraphasia	Paragraphia	Paraphasia	Yes	Paragraphia	Mental confusion.

we have described, it is first necessary to *test spoken speech* and its reception, thus, where these functions are normal, eliminating at once auditory deafness, aphonia (a loss of power to produce speech) and "word-deafness."

Test next written speech and the patient's understanding of it, thus investigating visual power and "word-blindness," or the inability to recognize objects or written speech, with apparent retention of each of the various functions necessary for its performance.

On account of the complexity of the aphasias the author has thought it advisable to insert here a table compiled by Church, which indicates in a concise manner the various forms and their anatomical relations to the higher centers in the cerebral cortex.

For further details regarding the examination of an aphasic the reader must refer to "text-books" on the nervous system, since they lie beyond the scope of this "manual."

(4) **Special Types.**—Lastly, it is necessary to refer to some special peculiarities noted in the speech in certain diseases. The following are those most commonly met with:

(a) **Scanning or staccato** speech is found typically in most cases of multiple sclerosis, the speech comes slowly and the syllables are separated as, for example, "de-cus-sa-tion" for decussation.

(b) **Slurring** speech is found in general paresis and resembles that heard in cases of intoxication.

(c) **Lalling** or "baby-speech;" here consonants are dropped, especially c and l.

(d) **Stammering.**—No difficulty will be experienced in its detection. It is usually seen before the period of

the second dentition and is apt also to occur in those who inherit neurotic tendencies, especially in boys.

II. **CRANIAL NERVES.**

(1) **Olfactory** (*I Cranial*).—The cortical center for smell, as has already been noted, is situated probably in the uncinate or hippocampal gyrus. From here the exact course of the fibers which pass forward to form the olfactory nerves themselves is not definitely known.

The condition of the centers, continuity of the nerve paths, and the state of the nasal mucosa are simultaneously investigated by applying the *oil of cloves or peppermint* to each nostril separately; so it is obvious that local nasal disease must be in all cases excluded by rhinoscopy in the routine examination. Ammonia must not be used since it also stimulates the fifth cranial nerve.

Anosmia refers to the loss; and *parosmia* to the perversion of the sense of smell. Olfactory *hallucinations* are occasionally encountered, especially as auræ of an epileptic convulsion and in certain cases of insanity. Hysteria is also not infrequently responsible for perversion or loss of this function.

(2) **Optic Nerve** (*II Cranial*).—From their terminals in the rods and cones of the retinæ all the fibers of the optic nerves pass backward to the chiasma. A unilateral lesion anterior to the chiasma will therefore interfere with all the visual fields of the corresponding eye.

(a) **Hemianopia.**—Those fibers which supply the mesial or nasal halves of the two retinæ decussate in the chiasma and enter the optic tract of the opposite side, while those from the outer or temporal halves proceed backward in the tracts of the same side. It

will now be seen that the nasal half of one and the temporal half of the other retina are represented in the optic tract of the same side and hence also in the corresponding occipital lobe.

To apply this fact clinically, supposing a lesion involves one optic tract, optic radiation or the occipital lobe, then the temporal half of the retina of the same and the nasal half of that of the opposite eye will be affected; in other words, the patient will be found to be blind in the nasal field of vision of the eye on the same side, and in the temporal field of its fellow; in other words, to suffer from *homonymous hemianopia or hemiopia.*

But as the central or commisural portion of the chiasma is almost completely composed of decussating fibers from the nasal halves of the two retinæ, a lesion destroying this portion will result clinically in blindness of both temporal fields of vision—*bitemporal or heteronymous hemianopia.*

Again, in those rare cases where symmetrical lesions interfere with the conductivity of the uncrossed fibers which run in the outer parts of the chiasma, defective vision in both nasal fields of vision must necessarily ensue and *binasal or heteronymous hemianopia* results.

A few commisural fibers run in the posterior part of the chiasma, connecting the two internal geniculate bodies through the mesial roots of the tracts, but, however, they probably have no direct connection with vision.

(b) **Vision.**—The lateral roots of the tracts pass backward to terminate in the lower visual centers, which are situated in the posterior quadrigeminal, external geniculate bodies and in the pulvinar of the

optic thalamus. From these lower centers fibers proceed backward in the optic radiation, passing close to the retroventricular part of the internal capsule, to the primary or higher visual centers in the occipital lobes.

Certain fibers also connect these lower centers with the nuclei of the oculomotor nerves, interference with which is probably responsible for the Argyll-Robertson pupil (page 250).

The acuity of sight must now be investigated, and also the condition of the fields of vision, the importance of which latter investigation will now be obvious, since the detection of hemianopia is frequently a means of locating cerebral lesions.

Examine each eye separately for **visual acuity** (myopia or "short" and hypermetropia or "long sight"), then both eyes together to detect *diplopia* ("double vision"). This latter is sometimes transient and it may be necessary to rely on the patient's history of having at one time "seen double."

The **field of vision** of each organ should be roughly tested by directing the patient to keep the eye looking directly before him with the globe stationary. An object is then advanced along the side of the head on a level with the orbit, and the patient requested to say "now" at the moment it becomes visible. This maneuver is repeated in each quadrant of the eye.

Another method is applied as follows: Place patient with his back to a window, before the examiner. Bandage one eye and direct him to look with the other into the eye of the clinician (40 cm. distant). The examiner now closes one eye, holds a small object (1 cm.) midway between his eye and that of the patient's and

controls by the extent of his own visual field the examination of that of the patient's.

In cases where the fields of vision are markedly and *concentrically contracted*, as in hysteria, glaucoma, and diseases of the optic nerve, the object may not become visible until brought directly in front of the pupil; in other words, the patient is as though he were looking through a small hole in a screen held before the eyes. For accurate and permanent records the use of a *perimeter* is essential.

In addition to the concentric diminution of the fields just referred to, by this maneuver we are able to detect clinically hemianopia, or that condition alluded to in which *asymmetric disturbances* in the visual fields occur.

The fields of vision for various colors should also be tested in order to render this examination complete.

Further, we are enabled by means of *Wernicke's hemiopic pupil reaction* to differentiate between lesions lying before and behind the corpora quadrigemina. A narrow ray of light should be thrown on the blind section of the retina under observation; if the lesion be anterior to the corpora, no alteration in the pupil occurs, but if it be posterior contraction occurs since the reflex arc is uninterrupted. It is, however, extremely difficult to demonstrate the reaction.

(c) **Pupil Reflexes.**—Although the pupillary reactions are dependent both on the third cranial and cervical sympathetic nerves, as well as on the optic for their presence, it is probably better to consider them at this stage.

The *anatomical reflex arc* involved in the reaction to light consists in the retina, optic nerve, optic tract,

corpus quadrigeminus, Meynert's fibers (which connect the two latter), the nucleus of the third cranial nerve and the main trunk of the oculo-motor nerve, which passes forward to supply the iris.

The pupils are now tested for **reaction to light,** each eye being examined separately by covering or shading the ball of the opposite organ and allowing a ray of light to fall on the pupil of the one under examination. A sharp contraction normally follows.

To test the **reaction of accommodation** the patient is asked to observe a distinct object, and immediately afterward one placed at a distance of several inches from the face. Normally, contraction of the pupil (sphincter iridis) occurs and also convergence of the axes of the globes, from an associated movement of the internal recti, when the eyes are focussed on a near object.

A pupil which reacts to accommodation but fails to contract on being exposed to light (**Argyll-Robertson pupil**) is found in tabes dorsalis and general paresis. The explanation advanced to account for this latter phenomenon is that a lesion either in the fibers of Meynert or degeneration in the ciliary ganglion has interfered with the reflex arc involved.

It is also found normally that if light be thrown on one pupil, both it and its fellow contract under the stimulus; this is known as the "*consensual reaction*" and depends on fibers which connect the nuclei of the two third cranial nerves.

Lastly, it is found that irritation of the skin of the neck stimulates the cervical sympathetic nerve, and that a reflex enlargement of the pupil from the action of its dilator muscular fibers occurs (*ciliospinal reflex*);

variations in the pupils may also occur when these nerves are interfered with by diseased processes (aneurysm, etc).

To complete this investigation it is necessary to inquire for the presence of flashes of light, dark spots or floating specks before the eyes (muscæ volitantes), and also to determine the perception of color (Daltonism or "color-blindness"), and the acuity of vision in the dark.

(d) **Ophthalmoscopy.**—Direct examination of the optic nerves as they enter the retinæ by means of an ophthalmoscope should now be undertaken. For further details, however, the reader is referred to special works on this subject, since they lie beyond the scope of this volume.

(3) **Oculomotor, Trochlear, and Abducent Nerves** (*III, IV, VI Cranial*) for convenience are now to be studied together, and since, as they are all purely motor in function, their condition may be deduced from that of the muscles they supply.

The nuclei of these nerves lie in the substance of the pons and medulla below the level of the anterior quadrigeminal bodies and above that of the eminentia teres.

The trunks of the third nerves emerge from the inner aspect of the crura, those of the fourth from the roof of the fourth ventricle, and the sixth from the groove between the medulla and pons, whence they pursue their various courses across the base of the brain to enter the orbit, and hence are liable to pressure at any part of their route, especially from tumors of the base of the brain. In basal meningitis also they are often involved.

The third nerve supplies all the external muscles of the globe except the superior oblique and the external rectus which derive their innervation from the fourth and sixth respectively. The third nerve also supplies the levator palpebræ superioris and the constrictor fibers of the iris.

(a) **Examination.**—We may now utilize the above facts clinically by putting into action the various muscles supplied by these three nerves. The patient is first requested to raise the upper eyelids, and the resulting movement is noted. *Ptosis* (drooping) is found when the muscle involved is paralyzed; *blepharospasm* may also at times be present.

The patient is now asked to follow the movements of an object held about 12 inches from the face; by this means each of the muscles in turn can be brought into action and their condition noted.

Next the power of "*convergence*" is tested by advancing the object toward the patient's nose, and noticing the contraction of the internal recti which should normally occur.

(b) **Squint.**—Deviation of the normal axis of the globe from loss of power or abnormal action of any of the external muscles is termed squint or *strabismus*, which may be permanent or periodic. In the transient forms diplopia or double-vision is usually a symptom; in permanent squint this phenomenon is not often complained of, since the mind learns to disregard one of the retinal images. For further details regarding the subject of strabismus reference must be made to special works on diseases of the eye.

(c) **Nystagmus,** or short regular jerking movements of the eyeball, may be demonstrated in certain diseases,

particularly in multiple sclerosis, by bringing into action the external muscles of the ball (*e. g.*, lateral nystagmus, on looking to one side); they may also be present even if the globes be stationary, and the patient looking directly forward.

Nystagmoid or irregular movements of a similar type are frequently seen in "functional" diseases and must be differentiated from those due to organic lesions.

(d) **Conjugate deviation** of the eyes may be due to either an irritating or destructive cerebral lesion. In the latter case the eyes and often the head are turned toward the side of the lesion; an irritative lesion, on the other hand, causes deviation toward the healthy side. These rules do not apply when the lesion is situated in the *pons*, as in this situation lesions produce directly opposite results.

(4) **Trigeminal** (*V Cranial*).—It is a mixed nerve composed of both motor and sensory fibers. The *motor root* has its origin in the gray matter at the lateral angle of the IV ventricle, and also in certain cells lying close to the Sylvian aqueduct. The fibers from these nuclei join, emerge from the sides of the pons, pass beneath the Gasserian ganglion and become incorporated in the inferior maxillary division of the trigeminal. The following muscles derive their nerve supply from this root, the temporals, masseters, pterygoids, mylohyoids, the anterior bellies of the digastrics, the levator palati and azygos uvulæ (Meckel's ganglion), and the tensor palati and tympani (otic ganglion).

The *sensory root* terminates in nuclei in the floor of the IV ventricle, but also has a spinal root connected with the substantia gelatinosa of Rolando. The fibers

are gathered together, pass across the base of the brain in company with the motor division, and enter the posterior edge of the gasserian ganglion.

From this ganglion three sensory divisions arise, the ophthalmic, maxillary, and mandibular, and just beyond this point the motor root joins the latter nerve.

These three sensory trunks supply fibers of sensation to the anterior portion of the scalp, half the face (including the orbit and globe), the mucosa of the nasal and oral cavities, salivary glands, teeth, and anterior two-thirds of the tongue.

The anterior two-thirds of the tongue is thus supplied by *taste fibers* from the lingual branch (through the corda tympani of the seventh nerve) and, although those for the posterior third (vallate papillæ) are derived apparently from the ninth cranial nerve, it is generally believed that these fibers also ultimately join the trigeminal through the nerve of Jacobson, the tympanic plexus, and the small superficial petrosal nerves which enter the otic ganglion and thus establish a connection with the fifth, so that it is probable that all the taste fibers ultimately enter the brain through the latter trunk.

(a) **Motor Functions.**—To test the motor fibers the patient is first requested to slowly open the mouth and to thrust the jaw forward; a unilateral paralysis shows itself in a deviation of the lower jaw toward the paralyzed side, on account of the healthy external pterygoid pushing the ramus beyond the middle line.

Next he is directed to tightly clench the jaws, the examiner meanwhile placing his fingers on the temporals and masseters of each side in order to estimate and compare their power. "Lock-jaw," or a tetanic contraction of

the muscles which close the lower jaw, is met with in tetanus, strychnin poisoning, etc.

(b) **Sensory Functions.**—As regards lesions of the sensory root, facial anesthesia is the predominant feature, the degree of sensibility possessed by the conjunctiva and oral mucosa must also be investigated. For full details of the examination of sensory functions of the skin reference should be made to page 266.

Trophic disturbances, such as ulceration of the cornea and destruction of the eyeball, are not uncommon following section or disease of the ophthalmic division. It is also well to note that the rare condition termed facial hemiatrophy is apparently due to a pure trophoneurosis of the trigeminal nerve, to the exclusion of its motor and sensory functions, and, lastly, that this nerve is frequently the seat of neuralgic pains which in their most severe form constitute *tic douloureux*.

(c) **Taste.**—To test the sense of taste small quantities of a solution of sugar, salt, a "bitter" and dilute acetic acid are applied to either side of the protruded tongue, both on its anterior and posterior portions. The patient is then requested to point to the taste experienced in the four quadrants of the tongue on a card ruled into four squares, in each of which the words *sweet*, *salt*, *bitter*, and *sour* are written.

(5) **Facial** (*VII Cranial*).—Arises from a nucleus situated in the pons below the floor of the fourth ventricle. The fibers wind around the nucleus of the sixth nerve, which lies a little nearer the middle line, emerge in company with the eighth from the angle between the pons and the cerebellum, and with the latter trunk enter the internal auditory meatus. The

facial then passes into the aqueductus Fallopii, and leaves the skull by the stylomastoid foramen.

During its passage through the aqueduct it gives off the petrosal nerves, a branch to the stapedius muscle, and also the chorda tympani, which latter nerve after communicating with the otic ganglion joins the lingual branch of the fifth cranial and supplies taste fibers to the anterior two-thirds of the tongue.

This nerve is motor in function (except for the fibers of the chorda tympani incorporated in its trunk) and supplies all the facial muscles with the exception of the levator palpebræ superioris, in addition to the branch it gives off to the stapedius muscle.

The facial nerve is frequently involved in diseased conditions which may attack the trunk in any part of its course, the nucleus itself, or those fibers which connect it with the cortex.

If the conductivity of the **peripheral nerve trunk** be interfered with after leaving the styloid foramen, a complete lower motor neuron, unilateral facial, or *Bell's paralysis* results (infranuclear). The patient has difficulty in whistling, smiling, and closing the eyes; he cannot wrinkle the forehead or show the teeth, and also finds that there is a tendency for particles of food to accumulate between the cheek and gums on the affected side. The reader is referred to page 27, for a full description of the characteristic facial expression.

Suppose now that the **lesion has occurred in the aqueduct** (between the geniculate ganglion and a point just above the foramen), the patient in addition to showing a *facial paralysis* of the peripheral type above described will be found to have *lost the sense of taste in the anterior two-thirds of the tongue.*

Again, when the diseased process involves **the nucleus itself or the nerve trunk before it has entered the aqueduct,** paralysis of the stapedius muscle will usually be detected by the *hyperacuteness of hearing* complained of by the patient. This results from loss of the controlling or "damper" action of this muscle on the vibration of the ossicles or from the unantagonized action of the tensor tympani tightening the drum. A *peripheral nerve paralysis* will, of course, also be present.

Suppose, however, that the lesion involve those fibers which pass from the cortex to the nucleus (**upper motor neuron or supranuclear paralysis**), the loss of muscular power resulting will be found to involve chiefly the *lower two-thirds of the face.* The explanation of this lies in the fact that as the forehead and orbicular muscles so frequently act together, each set derives a nerve supply from both sides of the brain, and hence a bilateral lesion is necessary to destroy all the fibers. "Crossed paralysis" has already been described (page 239).

Not infrequently also the facial muscles are subject to spasmodic contractions, which may be tonic or clonic and due to cortical or peripheral lesions, and functional disturbances.

(6) **Auditory** (*VIII Cranial*) consists of two elements.

(a) *The cochlear fibers* which supply the auditory apparatus arise in the spiral ganglia beneath the floor of the fourth ventricle close to the restiform body and form the dorsal part of the main nerve trunk. The succession of neurons which connect the cortex and the organs of hearing are the bipolar cells of the spiral ganglion, the neurons of the terminal cochlear nuclei, of the superior olive, the nuclei of the lateral fillet

and of the corpus geniculatum internum (*Thomson*). Through the superior olive communication is established with the nuclei of the third, fourth and sixth cranial nerves.

The *cortical centers for hearing* are situated in the first and second temporal convolutions of the opposite side to that on which the corresponding labyrinth lies; the exact course of the connecting fibers is not definitely known.

(b) *The vestibular or equilibriation fibers* arise just external to the cochlear, emerge from the brain and form the ventral part of the auditory nerve, terminating primarily in the eighth nerve nucleus.

The *cortical centers for equilibriation* are probably situated in the cerebellum. Before testing the function of the fibers of the eighth nerve it is necessary to exclude disease of the middle ear and of the external auditory canal.

The *acuity of hearing* is determined by means of a watch or tuning-fork, each ear being examined separately, the meatus of its fellow meanwhile being kept closed. In disease of both the middle ear and the auditory nerve, deafness is the outstanding feature.

When impairment in this function is detected it will be found that the vibrations of the tuning-fork, if placed in contact with the bones of the forehead in the middle line, are heard loudest on the diseased side if the lesion be limited to the middle ear, whereas if "*nerve deafness*" be present the sound will only be audible on the healthy side.

For further details regarding deafness reference must be made to special works on diseases of the ear. Suffice it to say that in addition to deafness, tinnitus

and hyperacusis are not infrequently concomitant subjective phenomena.

On the other hand, *disorders of the mechanism* of *equilibration* are shown clinically by true vertigo (dizziness), in which visible objects seem to rotate around the patient; but it must not be forgotten that vertigo is frequently due to other causes (gastric, ocular, anesthesia of the lower limbs, cerebral tumors, etc.).

(7) **Glossopharyngeal and Vagus** (*IX, X Cranial*) are so frequently involved simultaneously in disease, owing to their close anatomical relations, that for convenience they will be considered together.

Their efferent or motor fibers arise in the dorsal motor and ambiguus nucleus both of which lie deep in the medulla, fibers from which, along with certain afferent twigs terminating in the dorsal nucleus and funiculus solitarius, go to form the two nerve trunks.

The nerve fibers emerge by numerous roots from the upper part of the medulla, between the olivary and restiform bodies, join to form the main nerve trunks, cross the base of the brain, and leave the skull by the jugular foramen. The vagus in particular has numerous connections with certain other cranial nerves.

(a) The **Glossopharyngeal** nerve supplies sensory fibers to the mucosa of the pharynx, middle ear, posterior third of the tongue, and carries taste fibers to this latter area; it is the motor nerve also for the stylopharyngeus and the middle pharyngeal constrictor muscle.

Lesions of this nerve may be accompanied by loss of palate reflex, defective sensation of the mucous mem-

brane it supplies, loss of taste over the back of the tongue, and sometimes by slight dysphagia or difficulty in swallowing.

In "root" affections taste is not affected on account of the connection of the taste fibers with the fifth cranial nerve.

(b) The **Vagus** supplies motor twigs to the soft palate, larynx and pharynx, and also sensori-motor fibers to the respiratory passages, heart, and certain abdominal organs.

Lesions may affect one or both nuclei or the nerve trunks themselves; the vagus, however, is seldom involved without the ninth being at the same time affected.

Defective movements (paralysis) of the soft palate with regurgitation of fluids through the nose and defective action of the vocal cords resulting in altered speech or aphonia may be found, as also may certain alterations in the rhythm of the cardio-respiratory excursions. Details of vocal cord paralysis and spasms will be found in text-books on laryngology.

(8) **Spinal Accessory** (*XI Cranial*).—Consists of two divisions. The *spinal* set of fibers arise from anterior horn cells of the first to fifth cervical segments, leave the cord, pass upward through the foramen magnum, and join the accessory portion. Finally, the nerve trunk, after crossing the base of the skull, leaves its cavity by the jugular foramen and terminates in twigs which supply the sternomastoid and upper part of the trapezius muscles.

The *accessory* division whose nuclei of origin lie in close proximity to those of the vagus, of which it is usually regarded as an extension, also ultimately sup-

plies fibers to the pharyngeal and superior laryngeal nerves.

To test the spinal division of this nerve the patient is requested to shrug his shoulders and to rotate the head.

(9) **Hypoglossal** (*XII Cranial*).—Takes origin in a nucleus on the floor of the fourth ventricle, its fibers, which are purely motor in function, emerge on the anterior surface of the medulla from between the olive and the anterior pyramid and leave the cranial cavity by the anterior condyloid foramen.

To *test* its functions direct the subject to protrude the tongue and then, with the mouth open, to thrust it into either cheek; a unilateral *paralysis* is shown by deviation of that organ toward the paralyzed side on protrusion. Not infrequently an *apparent deviation* occurs in healthy individuals; which fact must never be forgotten.

If the lesion be nuclear or involve the peripheral nerve trunk, *atrophy* will be a concomitant sign and *fibrillary tremors* will also usually be present.

Before leaving the subject of the cranial nerves it is necessary to remember that as those having motor functions all arise in certain nuclei; as their axis-cylinder processes form peripheral motor nerve trunks, and as they are governed by certain cortical centers, it is obvious that they belong to the class of lower motor neurons, and hence it follows that the muscles they supply may show evidences of either central (supranuclear or upper motor neuron), nuclear, or peripheral nerve paralysis (lower motor neuron.)

We must also remember that as the parent cells and trunks of the cranial nerves are analogous to the cells of the anterior horns of the spinal cord and their

peripheral nerves, they are liable to similar diseased processes (for example progressive muscular atrophy); and that when the nuclei of certain of these cranial nerves become diseased the resulting phenomenon is termed *bulbar paralysis*.

III. **MOTOR FUNCTIONS.**

(1) **Paralyses.**—Since normal voluntary muscular action depends directly on an intact cortical motor area, upper and lower motor neuron and a healthy muscle, any subject who can voluntarily move a certain muscle to the normal extent can safely be said to have no lesion in any part of that portion of the neuromuscular system under investigation.

Supposing, however, a lesion occurs in the cortex, corona radiata, internal capsule, crus, pons or medulla above the level of the pyramidal decussation, which interferes with the function of any of the motor fibers, a loss of voluntary movement or *paralysis of the upper motor neuron type* (page 229) will be found on the opposite side to that occupied by the lesion.

If, also, a unilateral lesion lies in the pyramidal tract of the cord, an *upper type paralysis* of the same side will be found, but if the disease be bilateral and involve both lateral columns a *spastic paraplegia* will result.

Again, where lesions attack the anterior horn cells or pure motor nerve fibers, loss of voluntary power of the *lower motor neuron type* (page 229), will be found. It is frequently impossible therefore at times to differentiate anterior poliomyelitis from a true motor neuritis.

Lastly, in certain primary muscular degenerations or **myopathies,** in which no nerve, cord, or brain lesions can be detected, the patient loses to a greater or lesser extent the power of voluntary action, not on account

of any defect of innervation but merely from the loss of power due to the diseased muscle fibers themselves, and this will be proportionate to the amount of muscle degeneration present.

To test clinically the neuromuscular functions it is necessary to instruct the patient to voluntarily put into action certain muscles or groups of muscles and to note the results obtained. Theoretically, each muscle should be put separately into action, but for ordinary examinations certain individual muscles or groups are selected, the most important of which are the flexors of the fingers, the interossei and lumbricals (the "grip" may be estimated by means of the dynamometer) muscles of the thumb, flexors and extensors of the wrist, supinator longus, biceps, triceps, deltoid, pectoralis major, sternomastoid, trapezius, serratus magnus, latissimus dorsi, erector spinæ, and the muscles of the neck and jaws, in every case carefully comparing the amount of contraction found on the two sides.

In the lower limbs the muscles of the feet, flexors and extensors of the thighs and knees, and also the adductors, abductors and rotators of the thighs are successively tested.

For the technic of putting into action these and the remaining muscles the student is referred to a text-book on anatomy.

Suppose now that we find a complete **paralysis** or partial loss of voluntary power (*paresis*) of the **lower motor neuron type** in certain muscles, it is next necessary to determine whether the causative lesion lies in the anterior horns of the cord, in the nerve roots, or in the peripheral nerves.

If the *anterior horn cells* alone be involved (anterior poliomyelitis, etc.), the grouping of the paralyzed muscles will correspond to a cord segment distribution, and there will be no alterations in the sensory functions.

In *radicular or root palsies* the distribution will be found to correspond to that of the root involved; in *plexus palsies* to that of the particular cords; and in *peripheral nerve lesions* to that of the diseased nerve itself.

If, however, sensory fibers are included in the nerve cords involved or if the diseased nerve be a "mixed" one there will be, in addition to the paralysis, defective sensation over the skin areas which correspond to the distribution of the sensory fibers involved.

Lastly, the **myopathies** or primary muscular degenerations must be differentiated from paralyses due to defective neuron control, by the history of the case, peculiar distribution, absence of sensory changes, the condition of the deep reflexes (which are merely diminished in proportion to the amount of loss of muscular power) and the electrical reactions.

Where a complete unilateral paralysis of the leg, arm, face, body, and leg exists the condition is termed **hemiplegia** ("crossed hemiplegia," page 239); a bilateral paralysis of the lower limbs is termed **paraplegia,** and that of single sections of the body a **monoplegia.**

So far we have discussed only paralysis of the lower motor neuron type; it is hardly necessary to describe at length those in which the causative lesion interferes with the upper nerve relay, since its general characteristics have already been referred to on page 229, and its distribution under the section on cerebral localization.

Having now eliminated or demonstrated the presence of paralysis, noted its "type," the electrical and reflex reactions, and the presence of tremors, we must next test muscular coordination and examine for spasmodic or convulsive movements and fibrillary twitchings.

(2) **Incoordination** (*ataxy or ataxia*) refers to the defective cooperation or inharmonious action of groups of muscles.

Since defective sight and altered sensibility of the skin may be factors in its production, it is necessary to eliminate the influence of the former by bandaging the eyes before testing for its presence, and as far as possible also that of touch.

Let the patient stand with closed eyes and *the arms* stretched away from his sides and direct him to touch the point of his nose with the ends of the fingers or to bring the tips together before him; any ataxy will during this maneuver at once be rendered evident.

As regards the legs, the *gait* will usually demonstrate its presence. The subject therefore is requested to walk a crack in the floor, or as he lies in bed to follow with his leg lifted and toes in the air certain movements of the examiner's finger; ataxy, if present, will in this manner at once be rendered evident, as it also is if the patient be directed to place one heel on the opposite knee and to "run" it down the shin.

Romberg's sign refers to an inability to stand in the dark or with the eyes closed (tabes, general paresis); it may be partly due to plantar anesthesia, but is usually associated with lesions in the posterior columns of the spinal cord (loss of muscle sense as in tabes dorsalis, etc.).

(3) **Spasmodic Movements** occur in all degrees from

generalized convulsions to local or Jacksonian epileptiform attacks and may be due to the action of organic disease on the central nervous system, to functional or hysterical seizures, or to the action of certain toxins (uremia, strychnia, etc.). In these cases they are usually of the "*clonic*" or intermittent type; they may also assume, however, a "*tonic*" type as seen particularly in tetany and tetanus.

The various forms of *tremors* are described on page 47. For description of the "occupation cramps," the "tics," etc., reference must be made to special treatises on diseases of the nervous system.

(4) **Fibrillary Twitching** is seen best in the tongue and thumb muscles, especially in progressive muscular atrophy. It points to disease of the nuclei of origin of the spinal or cerebral motor nerves.

(5) **Hysterical manifestations** are frequently found in the muscular system, both *paralyses* and *contractures* of all types. They are recognized by the *history* of the case, the *absence of any of the reflex changes, atrophy, or electrical reactions* characteristic of organic disease and also by a *distribution* which does not correspond with the cortical or cord segment areas, or to the groups of muscles supplied by any peripheral motor nerve. Hysterical *stigmata* (globus and clavus hystericus, hysterogenetic zones, mental changes, contracted visual fields, anesthesia, etc.) are also frequently present, and when found will clinch the diagnosis.

IV. **SENSORY FUNCTIONS.**—In order properly to examine the sensory functions it is essential to apply the following tests to every part of the cutaneous surface and where abnormalities are found to outline the affected areas with a dermographic pencil.

It is first necessary to remember that certain *types of distribution may be met with in cases of altered sensibility;* in one the areas affected correspond to those represented in certain portions of the brain or the cord; the next will be found to coincide with those areas supplied by the sensory nerve trunks; and, lastly, the peculiar forms of distribution found in hysteria, in which, for instance, the regions showing altered sensibility do not correspond to any anatomical distribution and hence are comparatively easy to recognize; for example, a complete total, an accurate hemianesthesia, the "glove and stocking" type, etc.

(1) **Touch** is tested by means of a piece of cotton wool or light camel's-hair brush (in order to eliminate the possibility of the sense of pressure being at the same time stimulated); every portion of the skin should be subjected to this test. The patient being directed to say "now" each time he feels the stimulus, and also to locate the area touched, the eyes meanwhile being closed.

To prevent error it is a good plan frequently to ask if he feels anything when no stimulus is being applied. For accurate results the parts if hairy should be shaved.

The sense of touch may be abolished (*anesthesia*) or exaggerated (*hyperesthesia*), delay may occur in the conduction of the stimulus to the cerebrum (*delayed sensation*), or the patient may be unable to localize it (*allocheiria*).

(2) Discrimination of the **compass points.**

(3) Appreciation of differences of size and shape and consistence of objects by touching and feeling them is termed **Stereognostic Sense.**

(4) **Pressure Sense** is tested, after directing the sub-

ject to thoroughly relax the muscles underlying the parts under investigation, by laying coins of different weights on the skin, and

(5) **Muscle Sense,** by placing a limb in various positions and requesting the patient (with closed eyes) to describe them, or to put its fellow into a corresponding position.

(6) **Bone Sensibility** is investigated by means of the "tuning-fork."

(7) **Pain** is estimated by means of a pin or faradic current; the disadvantage of the former methods lies in the fact that on account of its small size the nerve terminals may be missed.

Absence of the sense of pain is termed *analgesia* and increased sensitiveness *hyperalgesia.*

(8) **Temperature** is tested by means of hot and cold objects (for example, water of different temperature in test tubes).

(9) **Paresthesias** are certain abnormal subjective sensations, the chief of these being tingling, numbness, formication (crawling of insects), pruritus (itching), burning, "pins and needles," etc.; they are entirely spontaneous and have no relation to any applied stimulus.

V. **THE REFLEXES.**—As we have already discussed the reflexes and principles involved, we are now in a position to describe the more important ones from a practical standpoint and to appreciate their significance.

It will be seen after studying the various reflexes below described that as each is inseparably connected with certain definite segments of the spinal cord and dependent on the integrity of these segments for its presence, it is often possible from a careful investigation

of abnormal phenomena present in a given case to localize the level of the cord lesion.

(1) **Superficial Reflexes.**

(a) **Plantar.**—Is induced by stimulating or irritating the sole of the foot; on firmly stroking its skin, movements of the toes, foot, and leg should normally ensue, the most important of which phenomena being the plantar flexion or **"flexor response"** of all the toes.

Where organic disease of certain of the upper motor neurons exists, it will be found that plantar flexion takes place in all but the great toe, which now becomes slowly extended or, in other words, moves deliberately upward and constitutes (**Babinski's sign or "extensor response"**).

Oppenheim has shown that the same result may also be obtained by stroking the inner side of the leg.

To obtain a satisfactory reaction the feet should be warm and dry, the leg firmly held so as to limit its movements, which by their briskness may tend to obscure the plantar response, and the maneuver should be repeated with the legs both in the flexed and extended positions, as sometimes a better reaction is obtained in the one than in the other; it is also necessary to eliminate the possibility of an extensor response being purely voluntary in origin.

When a typical extensor response occurs, a simultaneous contraction of the *tensor fascia lata* may usually also be demonstrated.

A definite **extensor response therefore is indicative of organic disease of the upper motor neuron; it is never found in functional disease,** hence its importance from a diagnostic standpoint. But it must not be forgotten that, although this sign when present is proof-positive of organic disease, a flexor response or absence of any

plantar reaction whatever does not necessarily exclude it.

Since a lesion of the upper motor neuron is essential for the production of Babinski's sign, it necessarily follows from what has already been said that the deep reflexes will show exaggeration and, as will be seen later, "ankle clonus" will usually also be a concomitant phenomenon.

In addition, it is well to note that in some cases of epilepsy this extensor reaction may be temporarily present after a convulsion and also that occasionally, according to Byrom Bramwell, in hemiplegia (a disease in which an extensor response is usually present on the paralyzed side) stimulation of the sole of the foot on the unaffected side results not only in the normal flexor reaction, but also in the flexor response of the toe of the opposite or paralyzed side ("*crossed plantar reflex*").

It is also necessary to be aware of the fact that the plantar responses in *children* under two years are clinically of no value.

No movement whatever may occur on stimulating the skin of the soles. This total absence of reflex in certain circumstances is suggestive of the presence of functional disease, but as a temporary absence is often found with organic lesions (in which later on the typical extensor response may make its appearance); a permanent loss is generally of less importance than the presence of a normal or an extensor response, both of which phenomena give us definite information.

Dorsi-flexion of the great toe is not infrequently accompanied by a spreading movement of the other toes, especially by abduction of the little toe, constituting the "*fan sign.*"

The normal plantar reaction requires integrity of the *I, II, III sacral segments* of the cord for its production.

Mendel's sign consists in a plantar flexion of the toes, sometimes with spreading, found in spastic states on tapping the lateral part of the proximal half of the back of the foot. Dorsal flexion is the normal reaction.

(b) **Anal.**—Contraction of the sphincter ani on stimulation of the perineal skin (*III, IV sacral*).

(c) **Gluteal.**—Contraction of the gluteal muscles on stimulating the skin of the buttock (*IV, V lumbar*).

(d) **Cremasteric.**—Retraction of the testicle on stroking the skin over the inner and upper aspects of the thigh (*I, II lumbar*).

(e) **Abdominal.**—Contraction of the abdominal muscles on stimulation of the overlying skin, the umbilicus being drawn toward the side stimulated (*VIII to XII thoracic*).

(f) **Epigastric.**—A similar contraction of the epigastric muscles occurs on stimulating the skin over the lower part of the side of the thorax (*IV to VI thoracic*).

In connection with these and the abdominal reflexes it is found that their loss does not necessarily mean that the corresponding reflex arc is broken, as in some cases of upper neuron disease (disseminate sclerosis, on the paralyzed side in hemiplegia and in tumors of frontal lobes) they may become abolished.

There also seems to be a tendency for these superficial reflexes to be diminished or lost in the same conditions which cause an increase in the activity of the deep tendon reflexes of the lower limbs.

(g) **Scapular.**—Contraction of the muscles clothing the scapulæ on stimulation of the overlying skin (*IV to VII thoracic*).

(2) **Deep Reflexes.**—The majority of the deep reflexes are obtained by percussion of certain tendons. These will be described individually in detail. If certain muscles be put slightly on the stretch and their tendons tapped by a plessor, the muscle itself immediately undergoes contraction. In the case of the superficial reflexes, however, an interval of time was noted to elapse between the application of the stimulus to the sensory nerve and the muscle reaction.

Since insufficient time elapses in the case of the "deep" reactions for a stimulus to travel around the arc, it is probable that they are not "true" reflexes. The *explanation* advanced is that the preliminary stretching of the muscle has to a certain extent increased its tone and that now, when its tendon is struck, the muscle is directly stimulated to contract.

Since *muscular tone* is dependent on the cells of the anterior cornua of the cord, it follows that any condition which interferes with the normal influences of the lower motor neuron cell body or its axon reaching the muscle will induce a flaccid condition of the same; and, therefore, since a relaxed muscle will not respond to direct stimulation, the deep reflex under consideration will be lost.

Conversely, when the control of the upper motor neuron is removed from any cause (disease of pyramidal tracts, etc.) an exaggeration of reflexes will ensue. In nervous individuals and in the neuroses frequently also increase in the reflexes is found.

(a) **Knee-jerk** depends on the *II, III, IV lumbar segments* for its existence, hence interruption of any part of the reflex arcs connected with these segments will result in their abolition.

To elicit this reaction the quadriceps is put on the stretch by flexing the leg to near a right angle and then striking the ligamentum patellæ sharply with a plessor. The patient's leg, providing he is in the dorsal decubitus, may be raised from the bed by a hand placed behind the knee-joint, or if in the sitting posture may be allowed to hang down loosely, the foot in this case being kept clear of the floor.

Jendrassik suggests that if the reflex be difficult to elicit, it may be "*reinforced*" by directing the subject to look at the ceiling and at the same time to hook the fingers of his hands together before him and pull one against the other.

Occasionally, as a result of extreme spasticity of the legs and the thigh muscles, great difficulty may be encountered in producing a reaction, even if no pathological condition be present.

Great variation in the briskness of the knee-jerks is found in different individuals, even if no disease be present. *Unequal jerks* on the two sides or total bilateral absence is, however, of more significance than mere exaggeration.

The knee-jerks, from what has already been said, may be found to be *exaggerated* from interruption in the conductivity of the lateral columns above the lumbar region (traumatic, inflammatory or degenerative lesions), in functional disease and in nervous subjects, with the one exception that they are lost in complete division or destruction of the cord.

They are *lost* when the reflex arc involved is destroyed in any part of its course (neuritis or disease of the cord, either in the anterior horns, gray matter, or the posterior roots), also in cases where irritation

of the pyramidal fibers exists (during the early stages of cerebral hemorrhage, meningitis, etc.).

(b) **Ankle-jerk.**—The patient may be directed to kneel on a chair or the tendon achillis may be stretched slightly by dorsi-flexing the foot. The tendon is then percussed on its posterior surface. Sudden extension of the foot normally ensues (*I, II, sacral*).

(c) **Ankle Clonus.**—Slightly bend the knee, grasp the foot firmly, suddenly produce a dorsi-flexion and keep up continued pressure on the sole. When this phenomenon is present a series of clonic *rhythmical contractions* of the calf muscles ensues, lasting so long as the plantar pressure is exerted.

The mechanism of this reflex is unexplained; it depends on the *I, II, III sacral segments* for its existence and is usually a sign of organic disease of the upper motor neurons.

It must be differentiated from the irregular "*false clonus*" often found in nervous subjects and which may occasionally be produced at will even in a healthy individual.

(d) **Knee Clonus** is elicited by fully extending the leg, grasping the patella lightly between the fingers and suddenly pushing it downward toward the foot. A series of clonic contractions similar to those found in ankle clonus occurs in similar diseased conditions (*II, III, IV lumbar*).

(e) **Adductor-jerk.**—Abduct the thigh and tap the tendon of the adductor magnus. In the "*crossed adductor jerk*" stimulation of the one ligament patellæ produces an adductor jerk in the opposite limb. It is sometimes found in cases exhibiting great reflex excitability.

(f) **Triceps or Elbow-jerk** is demonstrated by percussing the triceps tendon just above its insertion, the arm meanwhile being flexed to a little over 90 degrees (*VI cervical*). Extension at the elbow-joint should result.

(g) **Supinator-jerk.**—Slightly pronate the forearm, flex the elbow and strike the tendon of the supinator longus where it crosses the radius just below the styloid process (*VI cervical*). Contraction of the muscle normally occurs.

(h) **Wrist-jerk.**—Percuss the extensor tendons as they cross the posterior surface of the radius, the hand meanwhile being allowed to hang down (*VI cervical*). Extension of the hand should follow.

(i) **Periosteal Radial Reflex** refers to the contraction of the M. brachioradialis seen at the bend of the elbow or tapping the lower end of the radius. It results in flexion and slight pronation of the forearm and hand.

(j) **Jaw-jerk** *is not present in normal* individuals. It is elicited by percussing the chin while the mouth is held partly open (*V cranial nerve*).

(k) **Kernig's sign** is found in cases exhibiting evidences of cerebral irritation, especially meningitis, in which disease it is demonstrable in over 75 per cent. of all acute cases.

One direct result of the cerebral irritability is evidenced by increased muscular tone (hypertonus), and it is on its presence that the sign depends.

The patient should be in the dorsal decubitus, one leg fully extended and held flat on the bed, the opposite thigh meanwhile is flexed to a right angle at the hip-joint with the knee flexed on the leg. The leg is now slowly extended, and it is found in cases where this

sign is present the degree to which this latter movement can be performed will be greatly lessened.

(3) **Visceral Organic Reflexes.**

(a) **The Pharyngeal or Faucial and Laryngeal** reflexes are usually involved together. There is difficulty in swallowing and the food may regurgitate through the nose or enter the larynx. When the former reflex is absent tickling of the soft palate produces no reaction.

(b) **Vesical and Rectal Reflexes.**—The cord centers for the bladder and rectum lie in the *III, IV, V sacral segments.*

Destruction of these centers therefore interferes with the continuity of the reflex arc, and as a result the bladder and rectal walls lose their tone, and continuous or *true incontinence* of urine with constipation and involuntary passage of feces ensues.

On the other hand, after destructive lesions of the cord above the lumbar enlargement, the bladder is compelled to rely on its own cord center and reflex arc for its innervation; it then fills and empties itself automatically and involuntarily. Not infrequently however, if the discharge is incomplete the organ becomes distended, overflows, and a *false incontinence* is produced.

The *rectal sphincter reflex* may be tested by the passage of a finger within the anus and the strength of the resulting contraction noted.

(c) The **Pupil and Ciliospinal** reflexes are described on pages 249 and 250.

VI. **ELECTRO-DIAGNOSIS.**—Normally a muscle can be made to contract by stimulating its nerve or the fibers themselves by a faradic or galvanic electrical current.

(1) **The faradic current** can be made through its

application to a motor nerve to bring about contractions in the muscles supplied.

One pole of the battery is applied over the nerve and the other to some distant part of the body; it is a matter of indifference which pole is supplied to the nerve. It is found, however, that the maximum amount of muscular contraction occurs when the electrode is placed upon certain spots known as "*motor points.*" Diagrams showing their location are found in most textbooks which deal particularly with the nervous system, and the reader is referred to these for further details.

The faradic current *stimulates muscles to contract only through their nerves.* If the faradic stimuli succeed one another with sufficient rapidity the muscle will become tetanized or, in other words, remain contracted so long as the current is applied.

(2) The **galvanic or continuous current** has the property of causing muscular contractions *through the nerve and also when applied directly to the muscle* itself, thus differing in its action from that of the faradic.

But it is found that the muscular contractions occur *only when the electrical contact is "made" or "broken"* (completed or interrupted) and not during the passage of the uniform current, and also that they are more easily obtained on making contact between the muscles and kathode (negative pole) than when the anode is applied as expressed by the statement that KCC is greater than ACC (*kathode closing contraction* greater than *anode closing contraction*).

(3) The **Reaction of Degeneration** occurs when the nerve communication between the muscles and their corresponding motor cells in the anterior horns of the cord is cut off (lower motor neuron lesion).

The main features of the change are, as regard the stimulation of the *nerve*, partial or complete loss of excitability to both galvanic and faradic currents.

When, however, the galvanic current is applied to the *muscle* itself, for a time the latter remains unduly irritable but contracts in an abnormal manner, contact with the positive pole (anode) now giving a stronger contraction than when its fellow is applied to the muscle, as shown by ACC greater than KCC (*anode closing contraction* greater than *kathode closing contraction*), graphically ACC > KCC.

Finally no contractions whatever to either current, whether applied to nerve or muscle, can be elicited.

VII. **THE MENTALITY.**—Having now completed our examination of the patient, having talked with him and watched his behavior, we are in a position to form some idea of his mental condition, but let us never draw absolute conclusions until the friends and relatives have been interrogated, for we must remember that many who are mentally deranged have but one subject on which their insanity hinges, and in our examination this vital point may have escaped notice.

A thorough conception of the temperament will be of the greatest assistance in dealing with any given case; with certain patients infinite kindness and sympathy is essential; with others firmness of demeanor to the verge of harshness is necessary; to some we may fully explain their physical defects, to others similar frankness would be disastrous.

At this point it is usually found advisable to endeavor to determine what reliance can be placed on our subject's statements regarding his symptoms and history, remembering that with those of a taciturn frame

of mind a searching interrogation may be necessary to elicit the information required, while in the garrulous much of the history may be irrelevant and the value of many statements discounted.

It is well now to inquire as to sleep, memory, *delusions*, or misinterpreted sense impressions (for example, believing a stump seen to be a dog, which idea cannot be shaken by pointing out the facts), and *hallucinations* (in which, for instance, the patient hears imaginary voices, the impression here being purely intellectual and without a material basis, thus differing from the former affection).

Under this heading may also be noted the shifting restless glance of those who have something to conceal, the haunted look characteristic of certain "phobias," or that profound expression of despair seen in the melancholic.

It is impossible in the space at my disposal to do more than indicate a few of the headings under which the investigation of the mental condition of any subject must be carried out. The more important anomalies to be sought for and analyzed are those connected with the attention ideation, reproduction of memory, affective and emotional states, the anomalies of volition, etc. For further information regarding these functions reference must be made to works on psychiatry.

Details of the methods used in the investigation of the psychoneuroses also lie beyond the scope of this work, the reader is therefore referred to special treatises on these subjects for further information.

VIII. **CEREBRO SPINAL FLUID** (see Appendix).

APPENDIX.

THE URINE.

A. PHYSICAL EXAMINATION.

I. **Collection.**—The total quantity voided in twenty-four hours should in all cases be collected in a clean bottle, thoroughly mixed and measured. In making this twenty-four-hour collection, the patient is instructed to empty the bladder at a specified time, preferably 7 A. M.; this portion is rejected and the urine passed from that hour until the bladder is again emptied at 7 A. M. the day following is retained for examination. It is well also to keep the specimen corked and in a cool place, in order to limit decomposition. Where renal disease is suspected, the day and night samples should be examined separately.

If the presence of calculi in the urinary passages is probable it is well to examine microscopically the urine passed shortly after physical exertion, especially in regard to the presence of erythrocytes.

If only one sample can be obtained that voided between the third and fourth hour after a meal should be selected. In the case of life insurance examinations the urine must be voided in the presence of the physician.

It is frequently of advantage to collect the urine in conical glass receivers; by this means we are enabled to appreciate the amount and character of sediments

when present. This is of special value when centrifugalization is impossible. It is also often well to have the urine voided in three separate glasses, the act of micturition being voluntarily interrupted, particularly in cases where the presence of prostatic disease or urethritis is suspected.

In females we must never forget the probability of vaginal or menstrual discharge contaminating the urine during micturition; for this reason it is well if the urine shows any abnormal ingredients to examine that withdrawn by the catheter; this method also must of necessity be employed when any urine is to be subjected to bacteriological examination.

When unilateral renal disease is suspected the secretion from the two kidneys may be collected separately, either by means of segregation or by ureteral catheterization; the latter is, however, obviously the more accurate method.

II. **Preservation.**—In order to prevent decomposition due to bacterial growth various preservatives may be employed. Among these are camphor, chloral, salicylic acid, boracic acid or thymol; the latter may give a reaction similar to those for bile pigments or albumin. A few drops of formalin to the pint may be added; this, however, may give a pseudocarbohydrate reaction. Chloroform is probably the best for general use, as it may be driven off by gentle heat preparatory to examination of the specimen. Toluol is also used.

It must not be forgotten that an excess of certain of these reagents may show itself in a crystalline deposit and so lead to confusion if its origin be not recognized.

III. **Quantity.**—The amount of urine passed by any one individual must necessarily be subject to

considerable daily variation, influenced as the kidney functions are by perspiration, exercise, the amount of fluid imbibed, and many other factors; so also each different individual has his or her own daily average.

The following table, however, gives an approximate idea of the total quantity of urine excreted at various ages (*Holt*).

First twenty-four hours of life	℥ii		
Three to six days	℥iii	–	viii
One week to two months	℥v	–	xiii
Two to six months	℥vii	–	xvi
Six months to two years	℥viii	–	xx
Two to five years	℥xvi	–	xxvi
Five to eight years	℥xx	–	xl
Eight to fourteen years	℥xxxii	–	xlviii
Over fifteen years	℥l		

Average, 1450 c.c.

Normally the quantity excreted during the day exceeds that during the night : : 100 : 25— 60; where this condition is reversed (*nycturia*) the presence of some pathological condition should be suspected.

(1) *Polyuria.*—Refers to a pathological increase in the quantity of the renal secretion; an output of over 2500 c.c. per diem is usually considered to constitute this condition.

Diabetes mellitus, diabetes insipidus, chronic interstitial nephritis, amyloid degeneration of the kidneys, and certain of the neuroses are among the more common conditions in which it is found.

(2) *Oliguria.*—Is the term applied to a diminution in the daily renal secretion; usually an amount below 800 c.c. per diem constitutes this condition. Acute nephritis, chronic parenchymatous nephritis, febrile reactions, broken compensation in valvular disease

of the heart, cerebral irritation, and many other conditions may be responsible for this altered function.

(3) *Anuria*, or complete suppression of urine, may be a further stage of oliguria; in some cases of acute nephritis, however, and in certain hysterical attacks the cessation in the renal function may be abrupt. It may be obstructive and then is termed retention of urine, or it may be of reflex or renal origin.

III. **Color.**—The color of normal urine varies from amber to canary, the exact tint depending on the reaction and degree of concentration. As a rule, an acid urine of high specific gravity being the more highly colored, in diabetes, however, we find a pale urine of high specific gravity.

(1) Colorless urine is found after imbibing large quantities of water, in certain cases of hysteria, diabetes mellitus or insipidus, and occasionally in amyloid disease of the kidney.

(2) A milky appearance may be due to chyluria, or to the addition of cow's milk to the specimen by hysterical individuals or malingerers. Large quantities of pus from the urinary tract may give a similar picture.

(3) Orange-colored urine is found after the administration of rhubarb, senna or chrysophanic acid. A small amount of bile will also give a similar tint.

(4) A dark reddish-brown color may be due to the presence of bile or blood. Suppuration, gangrene, and intestinal putrefaction may through the presence of indican give a dark brown, greenish-black or even bluish tint to the secretion. In cases of melanotic sarcoma while the urine may be clear when fresh, it occasionally turns dark brown or even black on standing. The condition known as *alkaptonuria* gives

rise to a urine syrupy in consistence and brownish in color, which also becomes darker on standing.

(5) Greenish-black urine may be seen in individuals taking carbolic acid, salol, guaiacol or resorcin, and is then due to the presence of hydroquinone and of pyrocatechin. Bile also may give this same discoloration.

(6) A blue or green-blue color is found in patients taking methylene blue by mouth, in indigo workers and occasionally in indicanuria.

(7) Port-wine color, due to the presence of hematophoryn is seen after the use of trional, sulphonal, and tetronal. Antipyrin may produce a distinctly red urine.

(8) Opalescent urines may be due to the presence of bacteria or small quantities of blood; if merely a surface pellicle is found it is probably due to the presence of phosphate of lime in an alkaline urine.

V. **Odor.**—Normal urine possesses the characteristic aromatic odor, due to the presence of certain volatile acids. If allowed to undergo decomposition it acquires the so-called ammoniacal odor; if freshly voided urine has this character it is an indication of the presence of chronic cystitis.

A fecal odor is at times noticed in cases where perforation of an abscess into the urinary tract has occurred, providing the abscess cavity also communicates with the lumen of an intestinal coil.

Certain drugs impart their peculiar odors to the urine, especially turpentine which gives rise to a distinct odor of violets; menthol, cubebs, sandal-wood oil, asafetida and others may also be excreted in the urine and so impart to it their characteristic odor. It is well to

remember that asparagus gives a peculiar odor due to the presence of methyl mercaptan.

In diabetes mellitus a "new-mown hay" or a "fruity" odor is often noticed (due probably to the presence of acetone). It may also be detected in some febrile states and occasionally in gastrointestinal disorders.

VI. The **density or specific gravity** varies normally from 1015 to 1025 and is determined by means of a urinometer. In diabetes insipidus it may fall to that of water and may in urine containing sugar (diabetes mellitus) rise as high as 1075.

As the specific gravity varies to a great extent at different hours of the day, since it depends on the quantity of water imbibed, the activity of the skin and other factors, it is obvious that for the result obtained to be of any value a sample of the twenty-four-hour specimen must be procured for examination.

It is well to allow the urine to cool before examination, to remove air-bubbles from its surface, and to remember that neither the presence of albumin nor suspended particles will affect the specific gravity.

If the quantity of urine available be too small to allow the urinometer to float, dilute the specimen with a known amount of water, take the specific gravity of the mixed fluids, and multiply the last two figures of the result by the degree of dilution; this will give the density of the original sample.

In normal urines a rough estimate of the *total solids* may be made by multiplying the last two figures of the specific gravity by 2.33 (Haeser's coefficient). The result obtained will be the approximate number of grams of total solids in each 1000 c.c. of urine; the total twenty-four-hour excretion being known, a simple

calculation will yield the total twenty-four-hour excretion of solids. The average daily output averages 60 grams of solids in 1500 c.c. of urine, one-half of this being composed of urea.

It is well to note here that urines containing large quantities of bile, blood, sugar or albumin are less mobile than normal, and that in cases of fibrinuria the specimen may exhibit more or less coagulation.

VII. **Sediments** and turbidity are frequently seen in urine; the macroscopic appearance of the more common therefore demand a short description. If a centrifuge is not at hand sedimentation may be allowed to take place in conical glass receivers.

(1) Bacteria give rise to a diffuse cloudiness which cannot be removed by filtration or dissolved by acids.

(2) Mucus forms a "woolly" cloud, which slowly settles on standing; if, however, the specfic gravity of the urine be high it may float in the specimen. The chemical composition of this "mucus" is not definitely known; it is thought to be a nucleo-albumin.

(3) Phosphates separate out in neutral or alkaline urines as a white deposit; they dissolve on the addition of a few drops of acetic acid, but are unaffected by heat.

(4) Urates appear frequently in concentrated, cold, acid urines as a heavy "brick-dust" deposit, which dissolves on heating and is unaffected by the addition of acetic acid. Strong nitric acid, however, will dissolve urates with effervescence. If the urinary pigments be scanty this precipitate may be merely yellowish.

(5) Uric acid shows itself as a "cayenne-pepper" deposit which is composed of crystalline grains (Fig. 28).

(6) Oxalates may form an abundant sediment

they are usually mixed with and held partially in suspension by a cloud of mucus (Fig. 25).

(7) Pus when present in appreciable quantities forms a deposit which somewhat resembles that produced by phosphates; if in an alkaline urine it is of a "ropy" consistence, this appearance becoming more marked on adding a solution of caustic potash. It is insoluble in an excess of acetic acid and unaffected by heat. Pus and phosphates, however, frequently occur in the same urine.

A rough quantitative estimation may be made by noting the amount of deposit after three minutes centrifugalization. This method is a useful one for comparing the quantity passed by any individual from time to time.

VIII. **Urinary Calculi.**—The analytical method described on the following page is that suggested by *Salkowski*.

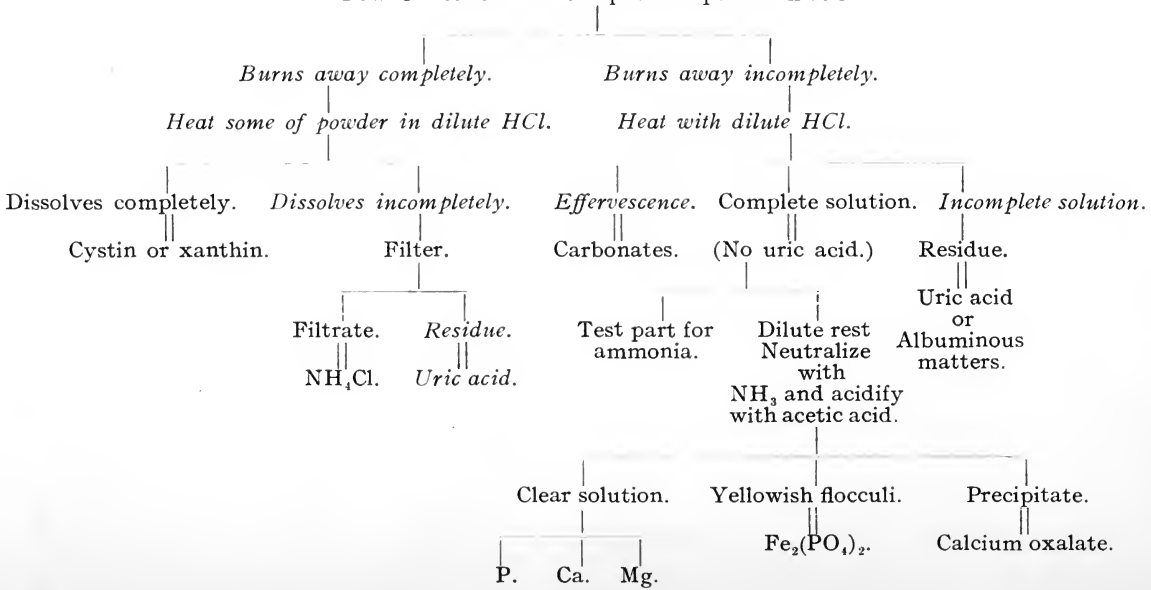
B. **CHEMICAL ANALYSIS.**

I. **Reaction.**—Normal urine gives an acid reaction due probably to the presence of sodium dihydrogen phosphate (NaH_2PO_4), as shown by turning blue litmus red; an alkaline urine of course giving the opposite result. Occasionally the reaction is blue to red, and also red to blue litmus; this *amphoteric* reaction has no clinical significance.

The reaction varies at different times of the day, the acidity being highest in the morning on waking and least after a meal; in fact, what is known as an "*alkaline tide*" frequently occurs after meals, in which case a precipitation of phosphates will often occur

Alkalies and organic acids of the fatty series in moderate doses will lessen the acidity of the urine,

ANALYSIS OF URINARY CALCULI.



mineral acids, on the other hand, having an opposite effect.

If in alkaline urines the altered red litmus remains permanently blue the term "*fixed alkalinity*" is applied; in cases, however, in which decomposition in the urinary tract has occurred through the influence of bacteria, the alkaline reaction may also be shown by merely holding the litmus paper just above the surface. If now the litmus be allowed to dry it will be found that the red color returns; this reaction is called "*volatile alkalinity*" and in fresh urines is suggestive of chronic cystitis.

A urine showing "fixed alkalinity" may be found during rapid absorption of large exudates, after intestinal hemorrhages, in pneumonia, chronic nephritis, during hypersecretion of gastric juice, in some cases of nervous disease and in the anemias; on the other hand, during febrile reactions in diabetes and subjects of "uric acid diathesis" highly acid urines are common.

II. **Chlorids.**—Under normal conditions from 10 to 15 grams of sodium chlorid are excreted in twenty-four hours, and since the chlorids are derived chiefly from the food their output will under normal conditions bear a direct ratio to the intake.

The normal elimination of chlorids bears to that of the total nitrogen output a ratio of as one is to one; in cases of renal insufficiency, however, this may be much disturbed owing to chlorid retention.

(1) A marked *diminution* in the chlorid output is met with in most febrile conditions, where a large exudate or transudate is present in the serous sacs, in pneumonia, in renal disease associated with albuminuria, severe diarrhea, marked gastrectasis,

rickets, the anemias and numerous other less common diseases.

(2) An *increase* in the chlorid excretion is found most commonly in patients who are recovering from conditions in which a retention has been noted, also in diabetes insipidus after epileptic seizures and chloroform anesthesia, and in malaria during the pyretic periods.

(3) *Qualitative Determination.*—Heat a few c.c. of urine acidulated with acetic acid to remove albumin, filter, add a few drops of pure nitric acid, next a few drops of a 10 per cent. solution of silver nitrate. Normally a white flocculent precipitate should separate out; if the chlorid output is increased a dense curdy sediment will form; if the solution merely turns slightly milky a diminished excretion is present.

(4) *Quantitative Estimation.*—Arnold's modification of Volhard's method is the most accurate (*Webster*). For a description of the technic the reader is referred to special works on urinalysis.

III. **Phosphates.**—The normal excretion of phosphates amounts to from 1 to 5 grams per diem, physiological variations depend chiefly on the amount ingested with the food. About 75 per cent. of this amount is combined with sodium, potassium and ammonium, the remainder with magnesium, calcium and glycerin.

Speaking generally, they are eliminated in larger quantities on an animal than on a vegetable diet, and their relation to the total nitrogen output is as one is to seven.

Not infrequently in fresh urine a rapid phosphatic precipitate appears, which condition has been termed phosphaturia; this is usually due not to an increased

output of the salts of phosphoric acid, but to a change in the urinary reaction from acid to alkaline.

(1) *Increased* elimination is found in the leukemias, during inflammatory processes in the urinary tracts, and in that condition termed "phosphatic diabetes," in which, although the clinical picture is that of diabetes mellitus, the urine contains no sugar, showing only a marked increase in the quantity of phosphates.

(2) *Diminished* elimination is found in many conditions, notably in acute fevers, in most chronic diseases and in all pathological renal conditions, in pregnancy, hystero-epilepsy, Addison's disease, hepatic cirrhosis, etc. Alterations in the quantity of the excretion of phosphates, however, have little clinical significance.

(3) *Qualitative Determination.*—Render 10 c.c. of urine alkaline with ammonia. The earthy phosphates are precipitated as a flocculent cloud and the quantity may be roughly estimated by its density. Filter this specimen, add a few drops of acetic acid, then a few drops of ferric chlorid solution, and the alkaline phosphates will now separate out.

(4) *Quantitative Estimation.*—Is usually made by means of uranium nitrate, for details of which procedure reference must be made to special works.

IV. **Sulphates.**—Normally from 2 to 3 grains of sulphuric acid are excreted daily. The technic of a quantitative estimation is too complicated for ordinary clinical use, but may be found in special works on urinalysis.

Qualitative determination may be made by adding a few drops of hydrochloric acid and 3 c.c. of a 10 per cent. barium chlorid solution to about 10 c.c. of urine. Normally a definite milky appearance results; a heavy precipitate will indicate roughly an increased output

of the salts of sulphuric acid, and a mere opalescence diminution.

V. **Oxalates.**—The amount of oxalic acid excreted in twenty-four hours varies from 10 to 20 mg. It occurs in the urine in combination with calcium and is usually kept in solution by means of acid sodium phosphate; it is, however, found as a precipitate in one urine out of three, but this does not necessarily mean that the salt is being excreted in excess, as its precipitation frequently is only due to the absence of a sufficient amount of the sodium phosphate to keep it in solution.

(1) *Increased* elimination of calcium oxalate is found after eating certain foods (rhubarb, tomatoes, strawberries, sugar, etc.), drinking waters charged with carbon-dioxide, gastrointestinal disturbances, a spermatorrhea, neurasthenia and other diseases, constituting that condition known as *oxaluria*.

(2) *Diminished* excretion has apparently little clinical significance.

(3) *Quantitative estimation* of the output is best made by means of Baldwin's method (*Webster*).

VI. **Uric Acid.**—From 0.2 to 2 grams of uric acid are excreted daily. Being a dibasic acid it forms two classes of salts, the "normal" and the "acid" urates.

(1) *Increased* output is found when a marked destruction of nuclein is proceeding as in acute fevers, in diabetes, leukemia, etc.; in other words, during increased protein catabolism.

(2) *Diminished* excretion is encountered in simple anemia, chronic nephritis, plumbism, and following large dose of quinin or opium.

(3) *Qualitative Determination.*—The *murexid test* is applied as follows: Evaporate some of the urine to a

small bulk, add to five drops of this fluid in a porcelain dish one drop of nitric acid. Evaporate this mixture slowly and when almost dry add to the orange-colored residue a drop of ammonia; a purple or rose-red color appears in the presence of the uric acid, which may be intensified by gentle heating.

(4) *Quantitative estimation* is best made by means of Folin's method (*Webster*).

VII. **Urea.**—The daily amount of urea excreted on an ordinary diet varies from 15 to 40 grams; on a nitrogen-free diet the quantity falls to about 2 grams. The relation of the urea output to protein metabolism is too complicated for discussion in this manual, the reader should refer to Von Noorden's hand-book of pathology of metabolism and to Leathes' lectures on "problems in animal metabolism."

(1) *Increased* output is met with in febrile states, diabetes, during absorption of serous exudates, exophthalmic goitre, and when large quantities of food and water are being taken.

(2) *Diminished* excretion is found in association with hepatic degenerations, certain nervous diseases, etc.

(3) *Qualitative Determination.*—Add a drop of nitric acid to several drops of the suspected fluid on a glass slide, warm gently, and on evaporation crystals of urea nitrate will appear.

A rough estimate of its amount may be arrived at in urine free from albumin or sugar by dividing the last two figures of the specific gravity by ten; which result will give approximately the percentage present in the specimen.

(4) *Quantitative Estimation.*—This may be roughly made by means of a Doremus', or of Hind's modifica-

tion of Doremus' *ureometer*, but for accurate results Folin's or the Mörner-Sjoqvist methods should be used, for the technic of which reference must be made to special text-books.

Doremus' Test.

Sodium hydroxide, 100 grammes.
Distilled water, 250 c.c.

To 10 c.c. of solution add ½ to 1 c.c. of bromine. Shake thoroughly and dilate ½ with water, add secundem artem, 1 c.c. of urine to solution in Doremus tube and read off percentage.

Normal amount of urea in urine is .02 grammes per c.m., 10 grs. per oz., or about 500 grains per diem.

VIII. **Ammonia** represents 4 to 5 per cent. of the total nitrogen output, the average quantity excreted in twenty-four hours being 0.85 gram; this will vary directly with the intake of nitrogen or, in other words, with the diet.

(1) *Diminished Output.*—With pronounced diminution in the protein metabolism, as shown by the total nitrogen output, there is usually a decrease in the absolute quantity of ammonia eliminated.

It is observed in many cases of nephritis, in some gastric carcinomata, and during the administration of alkalies in bulk.

(2) *Increased* elimination is observed in febrile and hepatic disturbances, uremia, dyspnea from any cause, delayed chloroform poisoning, the vomiting of pregnancy, diabetes mellitus and in acid intoxications.

(3) *Quantitative Estimation.*—Schlösin's or Webster's modification of Folin's method should be used where accurate results are essential.

IX. **Purin Bases.**—Numerous nitrogenous bodies have been isolated from the urine, but since their isolation and estimation are as yet of little clinical value, no description will be attempted; suffice it to say that the more important are creatinin and hippuric acid.

X. **Total Nitrogen.**—Of the total nitrogen excreted in the urine urea comprises about 85 per cent., ammonia 5 per cent., uric acid 2 per cent., the purin bases and undetermined nitrogenous compounds about 8 per cent. of the whole. The normal amount on an ordinary diet varies from 10 to 15 grams per diem.

From the metabolic standpoint the estimation of the total nitrogen of the urine is one of the most important features of its chemical examination; it varies with the intake of nitrogenous foods and with the degree of tissue metabolism, and may be taken as a direct indication of protein metabolism.

(1) *Increased* excretion is found in cancers, pernicious anemia, chronic tuberculosis, leukemia, exophthalmic goitre and in fevers; in other words, when a toxogenic decomposition of protein is found. In both forms of diabetes and during the absorption of large exudates a similar condition is found.

(2) *Diminished* excretion occurs in convalescence from both acute and chronic illnesses, and is here probably an attempt to make up for the losses incurred during the active progress of the disease. It is also found in nephritis and when the absorptive power of the intestine is reduced.

(3) *Estimation* is best made by means of Kjeldahl's nitrogen apparatus.

XI. **Pigments.**

(1) *Urochrome* is the chief pigment found in normal urine; it has, however, not been isolated in its pure state and, moreover, is of little clinical importance.

(2) *Urobilin* appears in the urine as urobilinogen and not as a free pigment; it appears to be identical with stercobilin and is probably chiefly of enterogenous origin.

Clinically urobilin is of little importance. Its presence is best detected by means of the spectroscope. For quantitative estimation a text-book on physiologic chemistry should be consulted.

(3) *Indican.*—In the decomposition of protein in the intestinal canal skatol and indol are formed, the latter is absorbed into the blood, oxidized, and excreted as indoxylpotassium sulphate, to which compound the term indican has been applied.

On oxidation indican yields a colored substance termed indigo blue, and hence is spoken of as a urinary indigogen.

An *excess* of indican is found in the urine in diseases associated with increased intestinal decomposition, constipation, intestinal obstruction, etc.; traces, however, are normally present in the urine, and a moderate increase may even be noted after an excessive meat diet.

The urine when passed is normal in color; occasionally however, if oxidation of this chromogen has occurred in the system, the urine may appear greenish-blue when voided and if the specimen be allowed to stand a reddish or blue metallic scum may separate out.

(a) *Qualitative Determination.—Jaffé's test* is applied (after removing any albumin present by boiling) by adding to a few c.c. urine in a test-tube an equal volume of concentrated hydrochloric acid, a few drops of a fresh, strong solution of calcium hypochlorite, mix well, add 2 c.c. of chloroform, shake tube, allow to stand for a few minutes, and note the blue color taken up by the chloroform in the presence of indican.

Obermayer's test is applied by mixing a few c.c. urine with an equal volume of a 0.4 per cent. solution of ferric

chlorid in hydrochloric acid, mix well, add a little chloroform, agitate test-tube, set aside, and note the dark blue discoloration produced by the presence of indican.

(b) *Quantitative* estimation is best made by means of Wang's or Folin's methods.

(4) *Blood Pigments.*

(a) *Heller's Test.*—Render a few c.c. urine strongly alkaline with sodium hydrate, boil. This test will detect one part of oxyhemoglobin in four thousand of urine. A positive reaction is shown by a brownish-red precipitate and a greenish discoloration of the supernatant fluid.

(b) *Donogany's Test.*—Add 1 c.c. of ammonium sulphid solution and 1 c.c. of pyridin to 10 c.c. urine; in the presence of blood a deep orange color appears.

(c) *Spectrum-Analysis* (see special works).

(d) *Guaiac Tests.*—Add 2 drops of tincture of guaiac to a few c.c. urine, a white precipitate will form due to the separation of guaiac resin; add now a few c.c. of ozonic ether by the supernatant method, in the presence of blood a blue ring appears at the junction of the two liquids. (Ozonic ether is a solution of peroxid of hydrogen in sulphuric ether. Oil of eucalyptus or ozonized oil of turpentine may be used as a substitute.)

Hematoporphyrin does not give this reaction, but when present in urine lends to it a port-wine color; it is best recognized on spectroscopic analysis. Clinically, it is most frequently seen in patients taking sulphonal, in which cases its appearance is of grave significance and is an indication for the free administration of alkalies. (See also page 344.)

(5) *Bile Pigments.*—Usually the bile pigments and acids occur together in the urine, the former, however, being much the more abundant; normally neither are present.

Bilirubin is the most important of the pigments found; it may, however, become oxidized into numerous derivitaves.

Urine containing bile may show various color shadings, greenish-yellow or brown to dark green. This is rendered more conspicuous if the specimen be shaken and the color of the resulting foam examined.

(a) *Smith's test* is applied by acidulating if necessary a few c.c. urine with acetic acid, add a 1 per cent. alcoholic solution of iodin by the supernatant method; in the presence of bile an emerald-green color will appear at the junction of the liquids.

(b) *Gmelin's Test.*—To a few c.c. of nitric acid add the urine to be examined by the supernatant method, oxidation of any bile pigment present will occur and a series of colored rings appear at the junction of the fluids, from above downward green, violet, and red. Several modifications of this test may be found in books on physiological chemistry.

Since the clinical significance of the bile acids is similar to that of the pigments and as their detection is a matter of difficulty, no attempt will be made here to describe the necessary technic.

(6) *Melanin.*—Melanogen is not infrequently found in the urines of patients suffering from melanotic tumors. These urines when freshly voided are normal in color, but on exposure to air darken until a black discoloration may be seen.

This pigment may also be found in some cases of chronic malaria, and so is not diagnostic of melanotic growths.

The addition of ferric chlorid produces a black precipitate which is soluble in sodium carbonate solution, and may be reprecipitated by the addition of a mineral acid.

XII. **Mucin.**—True mucin is a normal constituent of urine; it is found both as an insoluble portion which forms the nubecula, and as a soluble portion much smaller in amount which can be precipitated by acetic acid, but is soluble in a slight excess of the reagent; it is thus easily distinguished from albumin.

This form of protein is derived from the urinary passages and has practically no pathological significance, although in severe catarrhal conditions it may appear as a gelatinous deposit. In alkaline urines the mucin may, however, pass either partly or completely into solution.

XIII. **Nucleo-albumin.**—A large number of urines contain a substance other than mucin, which is precipitated by the addition of acetic acid, insoluble, however, in an excess of acids, thus differing from true mucin.

It is doubtful if the substance usually termed nucleo-albumin is chemically a true nucleo-albumin. Morner believes it to be a compound of true serum-albumin with an albumin-precipitating body formed on addition of acetic acid.

Since its chemistry is so little understood and its clinical significance uncertain, no further discussion will be attempted beyond stating that it may be distinguished from mucin by the fact that, unlike this com-

pound, it contains phosphorus and will not reduce copper solutions after heating with acids.

Although it gives most of the ordinary tests for albumin, it, however, may also be precipitated on saturation of the urine with sulphate of magnesium.

XIV. **Serium-albumin** is that substance usually referred to clinically as "albumin." Serum-globulin is practically always present in urines showing the serum-albumin actions, though in lesser quantities, the ratio between their amounts has no clinical significance.

Morner considers an excretion of from 25 to 75 mg. per liter as a normal output; this amount, however, is insufficient to show itself by the ordinary clinical tests, and therefore when a reaction is obtained by the methods described below a pathological secretion may be inferred.

It is necessary also to note that before the presence of a "*true albuminuria*" can be diagnosed the possibility of inflammatory exudates, pus, prostatic or spermatic fluid, blood or lymph, becoming mixed with the urine in the passages and so giving rise to a "*false or accidental albuminuria*" must be considered.

The amount excreted in disease is variable and does not necessarily bear any relation to the severity of the causative kidney lesion; from 5 to 10 grams per diem is considered a moderate pathological secretion.

It is impossible to discuss the various forms of "*functional albuminurias*" in this work; suffice it to say they are termed alimentary, cyclic, orthostatic, intermittent, lordotic, and hypostatic. The albuminurias of adolesence also come under this heading. Other forms are the febrile, traumatic, hematogenous,

toxic, and neurotic albuminurias, none of which are constantly associated with definite renal lesions.

(1) *Heat Test.*—Before applying any of the following reactions filter, render faintly acid with acetic acid if necessary, and if bacteria are present add caustic soda until a precipitate of phosphates occurs carrying with it the organisms which may now be removed by filtration.

(a) Gently heat to the boiling-point the upper third of a test-tube filled nearly to its top with urine; the appearance of a cloud indicates the presence of albumin, calcium phosphate or carbonate. Acidulate with a few drops of a 5 per cent. acetic acid phosphate's dissolve, and effervescence will occur if the turbidity be due to the carbonates. It is well to boil after the addition of each drop of acid in order to avoid an excessive acidity.

Nucleo-albumin it will be remembered is precipitated in the cold by acetic acid and may thus be distinguished from true albumin.

(b) Boil as before, then add a few drops of nitric acid; this must never be added before boiling, nor must the urine be boiled after its addition. The phosphates and carbonates never give a precipitate, so that a flocculent precipitate is indicative of albumin. Nucleo-albumin may be eliminated since it is soluble in an excess of nitric acid. Allow the specimen to cool; albumoses if present will then separate out as a white precipitate. Uric acid may also appear as a deposit, but this is nearly always colored and can easily be recognized microscopically.

(2) *Heller's Test.*—Add a few c.c. of urine by the "overlying" method to about 1/2 inch of concentrated nitric acid in a test-tube or conical glass receiver; in the

presence of any urinary proteid, with the exception of peptone, a white haze will develop at the junction of the liquids.

Albumoses give a white ring, soluble on heating, and which reappears as the urine cools. Globulin a similar ring which can be differentiated only by separating these proteins from that of serum-albumin, but for clinical purposes since these two bodies are practically always associated this is unnecessary. For the reactions of the nucleo-albumin see page 299. Resins may also give a precipitate closely resembling that of albumin.

In the presence of bile the play of colors referred to on page 298, will appear; and of indican if in excess a bluish-black ring may appear; these rings, however, may be present in addition to that of albumin, in which cases they will lie below the level of the white precipitate.

It is well to note that even in normal urine a reddish discoloration may appear where the fluids meet, due merely to the reaction of the acids with the normal pigments; that a faint cloud well above the line of contact may be due to the presence of mucin; and that in the presence of an excess of uric acid, urea nitrate crystals may separate out in the urine above the level of the nitric acid.

(3) *Purdy's Test.*—Add to a test-tube half filled with filtered urine one-fifth its volume of a saturated aqueous solution of sodium chlorid, boil the upper third; the presence of a precipitate insoluble in a few drops of 50 per cent. acetic acid, even after again boiling, indicates the presence of albumin.

(4) *Ferrocyanid Test.*—Acidulate a few c.c. urine with

acetic acid, add a few drops of a 10 per cent. solution of potassium ferrocyanid. A white precipitate indicates the presence of albumin, albumoses, or nucleo-albumin. This test may be also applied, utilizing the "overlying" method.

Albumoses dissolve on heating; nucleo-albumin is detected by the presence of a precipitate on adding the acetic acid unless the acid be in excess.

(5) *Esbach's albuminometer* affords the most convenient method for the quantitative clinical estimation of albumin. This tube is filled with acidulated urine to the point U and Esbach's reagent (10 grams picric acid, 20 grams nitric acid, 1 liter aqua distillata) to the mark R. Close the tube with a rubber cork, agitate well, set aside for twenty-four hours.

The amount of precipitated albumin is then read off the graduations in the tube which represent the number of grams per liter; to obtain the percentage divide the result by ten; and to estimate the number of grains to the ounce multiply the result by 4.375.

If the specific gravity of the specimen be above 1010 it is necessary to dilute the urine with water to bring it below this point; if the urine has required dilution, the result obtained must be multiplied by the required number of times.

An average degree of albuminuria reaches about 1/2 per cent. or, in other words, about 8 grams per diem.

(6) A rapid but fairly accurate quantitative estimate of albumen may be made by using a graduated centrifuge tube in which are placed 10 c.c. urine with 3.5 c.c. 10 per cent. potassium ferrocyanide and 1.5 c.c. acetic acid, and revolving for five minutes, after which the percentage may be read off the scale.

XV. **Serum globulin** is practically always found in association with serum-albumin; its composition is, however, not definitely known, but it is probably a mixture of euglobulin and pseudoglobulin.

As the qualitative and quantitative tests are seldom of use clinically no description of the technic employed will be given.

XVI. **Primary Proteoses.**—*Bence-Jones protein* is excreted in the urine in cases associated with the formation of multiple myelomata of the bones and is practically pathognomonic of this condition.

Apply *Heller's test;* a white cloud which disappears on heating and reappears as the urine cools indicates the presence of primary albumoses. This cloud lies above the line of contact of the fluids.

Boston suggests the following reaction: Filter 15 c.c. urine, mix with equal volume of saturated solution of sodium chlorid, add 2 c.c. of a 30 per cent. solution of sodium hydrate, mix thoroughly, slowly heat to the boiling-point the upper one-fourth of the mixture in a test-tube, adding drop by drop a 10 per cent. solution of lead acetate, continuing the heating after each addition. When the drop of lead solution comes in contact with the liquid a pearly cloud appears at the surface, becoming less dense as the boiling-point is reached; if ebullition is prolonged for one minute the upper portion turns a brown to black color. This reaction depends on the presence of sulphur.

XVII. **Secondary Proteoses.**—Deuteroalbumose differs in its reactions from the above albumin; it may occur in the urine alone or in association with serum-albumin. It has been noted in a great variety of conditions; the more important of which are the presence of large ac-

cumulations of pus, disturbed hepatic functions, certain blood and intestinal disorders, and in practically all febrile states. Its presence is probably due to certain toxins which produce protein disintegration.

Test.—First remove ordinary albumin by adding 10 c.c. of a 40 per cent. solution of sodium acetate and an equal amount of 10 per cent. ferric chlorid (which will color the urine bright red); the albumin separates out and is then filtered off. Take a few c.c. of this urine, add one-fifth its volume of concentrated acetic acid, then a 10 per cent. solution of phosphotungstic acid; a milky precipitate will be observed in about five minutes in the presence of this form of albumin. Filter off the precipitate, wash the filtrate with distilled water, dissolve on the filter with a very dilute solution of sodium hydrate, the solution will turn blue. Warm the solution until clear, adding more sodium hydrate if necessary. Cool and apply the *biuret test* by adding a few drops of sodium hydrate and a few drops of 2 per cent. copper sulphate solution, on warming a violet-red color will appear. (Page 344.)

XVIII. **Glucose.**—The excretion of this carbohydrate in the urine is termed *glycosuria* and presupposes a hyperglycemia. It is impossible in this work to undertake a discussion on the various factors which influence the excretion of sugar in the urine. Suffice it to say that in an ordinary case of diabetes mellitus from 1 to 8 per cent. of sugar are excreted in twenty-four hours.

(1) Qualitative Determination.

(a) *Trommer's Test.*—To a few c.c. of urine add one-third its volume of a 10 per cent. solution of sodium hydrate, add then drop by drop a 10 per cent. solution of copper sulphate. The latter should be

added, shaking the tube constantly, until a slight excess of the precipitated cupric hydrate remains undissolved. Warm the upper layers of the urine, in the presence of sugar a yellowish-red precipitate appears.

(b) *Haine's Test.*—Gently boil 2 c.c. of Haine's fluid (copper sulphate 12 grams, potassium hydrate 45 grams, glycerine 90 c.c., water q. s. ad 1000 c.c.) and add about 5 drops of suspected urine, boil again for a few seconds only, and in the presence of sugar an abundant yellowish-red precipitate will form.

(c) *Fermentation Test.*—Render urine acid if necessary, drive off air by boiling, add a small piece of German yeast to about 10 c.c. of urine in a fermentation tube (or in a Daremus' ureometer tube), set aside in an incubator for twelve hours. In the presence of glucose gas will accumulate at the top of the tube. This method should be controlled by placing a sample of a normal urine and yeast in a similar tube and noting the result under the same conditions.

A rough estimate of the percentage of sugar present may be made my multiplying the difference in the specific gravities of the urine before and after complete fermentation by 0.23; the result obtained will indicate the percentage of sugar present.

If a copper-reducing substance is found in any urine it is well in all cases to apply this test, as glucose alone will give rise to fermentation.

(d) *Phenylhydrazin Test.*—After removing any albumin present by boiling and filtering, acidulate urine if necessary with acetic acid, add 1 gram phenylhydrazin hydrochlorate and 2 grams sodium acetate; mix well, place in boiling "water-bath" for an hour, filter while

still hot, allow filtarate to cool slowly. If glucose be present a deposit of yellowish, needle-shaped bundles or sheaves of phenylglucosazone will appear; these are detected microscopically.

The test may be varied by taking 5 minims of a solution of phenylhydrazin, 10 minims of acetic acid, 1 c.c. of a saturated solution of sodium chlorid and 5 c.c. urine, boil gently for two minutes, cool and examine precipitate microscopically.

(2) Quantitative Estimation.

(a) *Fehling's* method:

Solution A.

Copper sulphate	34.64 grams.
Aqua distillata	q. s. ad. 500 c.c.

Solution B.

Rochelle salt	175 grams.
Sodium hydrate	125 grams.
Aqua distillata	q. s. ad. 500 c.c.

Place 5 c.c. each of solutions A and B in a glass flask of 250 c.c. capacity, add 50 c.c. of distilled water, boil. Add urine, about 1 c.c. at a time, from a graduated buret until all blue color has disappeared from the original solution. The urine should be diluted five times (by adding 4 volumes of water) if the specific gravity reach 1030, and ten times if over 1035, as the test is not accurate in urines of high concentrations.

The amount of urine used to decolorize 10 c.c. of the copper solution is equivalent to 0.05 grams of glucose; the calculation is therefore simple if the total quantity of urine passed in twenty-four hours be known.

(b) The *Polariscope* test is made by means of a

polarimeter, the technic of which procedure lies beyond the scope of this manual.

XIX. **Lactose** is sometimes found during lactation. It gives no reaction with the fermentation test, yields broad yellow crystals of phenyl-lactosazone with phenylhydrazin, and reduces Fehling's solution in proportion to glucose as 7 to 10; that is, if 7 parts of glucose reduce a certain amount of Fehling's fluid it will require 10 of lactose.

XX. **Levulose** is occasionally found in urines; it is best recognized by the polariscope. In urines which show both the reduction and fermentation tests, Selimanoff's reaction should be employed in order to determine if part of the sugar present be levulose.

XXI. **Pentose** and **maltose** have little clinical significance and are rarely found in urines.

XXII. **Glycuronic acid** may be mistaken for glucose; it does not, however, yield the fermentation test.

XXIII. **β-oxybutyric acid** is closely related to diacetic acid into which it may be oxidized; the latter in its turn splits up into acetone and carbon dioxid. Fat is the probable source of these bodies.

A certain amount of carbohydrate food is necessary for proper normal metabolism, and so long as the body is supplied with sufficient carbohydrate, or is able to oxidize a sufficient amount, the urinary acetone bodies remain low; when, on the other hand, the system is no longer capable of oxidizing the carbohydrates the acetone group increases to a marked degree, as seen in diabetes.

The condition arising from a surcharging of the blood with these bodies is termed *acidosis*, and since oxybutyric acid is the mother-substance of the acetone

group, we should expect and usually do find that the more severe the acidosis the greater will be the amount of the acid present in proportion to that of its derivatives.

Again, since diacetic and oxybutyric acids combine with ammonia and are excreted as ammonium compounds, an accurate method of determining their output is to estimate the total ammonia output, 1 gram of ammonia equaling 6.12 grams of the latter acid. When the ammonia excretion approaches 10 per cent. of the total nitrogen the onset of coma is probable. β-oxybutyric acid is now assumed to be the direct cause of the diabetic coma.

For a quantitative estimation the author recommends Black's and Schaffer's methods. If the ferric chlorid reaction for diacetic acid is strongly positive, β-oxybutyric acid is in all probability present. (Page 310.)

XXIV. **Acetone.**—From a clinical standpoint this substance and diacetic acid bear the same significance. It lends to urine a fruity odor and reduces Fehling's solution.

(1) It may be detected by adding 5 drops of a 10 per cent. solution of caustic soda or potash to a few c.c. urine, heat gently, then drop in a saturated solution of iodin in potassium iodid until a yellow-brown color is noticed; add a few more drops of the alkali, a yellow crystalline precipitate of iodoform appears in the presence of acetone.

(2) *Rothera's Test* for acetone in urine.—To the urine in a test-tube is added a few drops of sodium nitroprussid solution, ammonia till alkaline, and then solid ammonium sulphate to saturation. A color similar to that of potassium permanganate develops in a few

minutes, and then slowly fades. The test is sensitive to 1 in 20,000 acetone and is distinctive for ketones.

(3) Add to a few c.c. urine a crystal of sodium nitroprussid, render strongly alkaline with sodium hydrate, shake; the addition now of a few drops of acetic acid gives a purple color to the foam if it contains acetone and the urine be again agitated.

(4) For quantitative estimation *Folin's* method is probably the most reliable (*Webster*).

XXV. **Diacetic Acid.**—If acetone be present in large amounts diacetic acid is probably also present and when so found is indicative of an even graver condition.

As it is volatile the suspected specimen should be examined fresh.

(1) *Gerhardt's Test.*—Add by drops a 10 per cent. ferric chlorid solution to 10 c.c. fresh urine as long as a precipitate of phosphates occurs, filter, to the filtrate add a few more drops of the iron solution, and in the presence of diacetic acid the urine will show a bordeaux-red color. Other substances may give this reaction; if, however, the specimen be now heated the color if due to diacetic acid will either become paler or disappear; the color produced by all other substances is unaffected by heat.

XXVI. **Cammidge's "Reaction C."**—The following reaction is frequently found in patients suffering from organic pancreatic disease.

Free a portion of a twenty-four-hour specimen of urine from albumin by boiling and filtration, and sugar by fermentation, boil for ten minutes 40 c.c. of the filtered urine to which 2 c.c.; of hydrochloric acid has been added, cool, and then add distilled water to make up to the original 40 c.c. neutralize any excess of acid

by adding 8 grams of lead carbonate, cool. Filter, treat filtrate with 8 grams of powdered tribasic lead acetate to remove glycuronic acid. Filter, treat filtrate with 4 grams of powdered sodium sulphate, boil, cool and filter. Ten c.c. of the filtrate are now made up to 17 c.c. with distilled water, 8 grams of phenylhydrazin, 2 grams of sodium acetate, and 1 c.c. of 50 per cent. acetic acid are added; boil for ten minutes, filter while hot, make up filtrate to 15 c.c. with warm water on cooling yellow crystaline sheaves and rosettes may be observed under the microscope.

XXVI. **Ehrlich's Diazo Reaction** is found in most cases of severe typhoid fever and is of especial value in the diagnosis of a relapse; it is, however, found in many other diseases and during the administration of numerous drugs.

To 5 c.c. sulphanilic acid add 2 drops of a 5 per cent. solution of sodium nitrate, add an equal volume of urine, shake, add 2 c.c. of ammonium hydrate. A carmine color in the foam indicates a positive reaction. If now the specimen be allowed to stand for twenty-four hours a greenish-black precipitate will separate out. The reagents must be fresh.

XXVIII. **Russo's test** is present as early as the second day in cases of typhoid fever, it is also given, however, by measles, variola, and suppurative tuberculosis, but is negative in varicella, scarlet fever, and miliary tuberculosis. The author, however, doubts its reliability.

Add 4 drops of a 1 to 1000 aqueous solution of methylene blue to 5 c.c. of urine, a positive reaction is shown by an emerald or mint-green color. A light or blue-green tint is negative.

XXIX.—**Drug Reactions.**

(1) Antipyrin may color the urine red and partially reduce Fehling's solution. If a little diluted perchlorid of iron solution be added a purple color appears, which is unaffected by heat but soluble in acids.

(2) Carbolic acid gives a white precipitate with bromine water; the urine on standing also becomes darker in color.

(3) Bromids are detected by adding a few drops of a dilute solution of bleaching powder, a few minims of hydrochloric acid, and some chloroform; shake and the latter reagent will turn red, since it dissolves the freshly liberated bromine.

(4) Salts of salicylic acid partially reduce Fehling's solution and give a blue color on the addition of tincture of iron.

C. **MICROSCOPIC EXAMINATION.**

The urine for this examination should be fresh and preferably removed by catheter; sedimentation may then be allowed to proceed in a conical glass or the centrifuge may be utilized. If the urine cannot be examined at once it is well to add preservatives, as certain organic deposits may undergo solution in the presence of decomposition, especially casts.

I. *Calcium carbonate* crystallizes in colorless, glistening, concentrically striated spheroids which usually present indentations resulting in a dumb-bell appearance or may resemble crosses or rosets. It is rarely found in human urine, but when seen is usually deposited along with a phosphatic precipitate in old, alkaline urine (Fig. 24).

II. *Calcium oxalate* is found as colorless, transparent, highly refractive, quadratic, crystalline octahedra

of varying size, forming the so-called "envelope" crystals. Occasionally oval or spheroidal forms (hour-glass, dumb-bell, spectacle, biscuit-shaped) or square

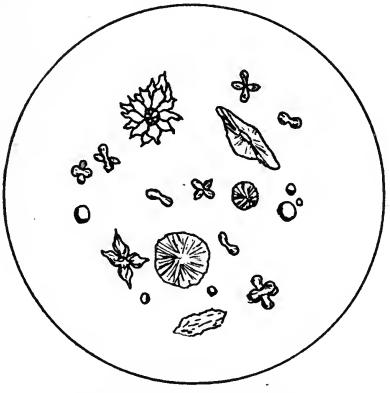


FIG. 24.—Calcium carbonate.

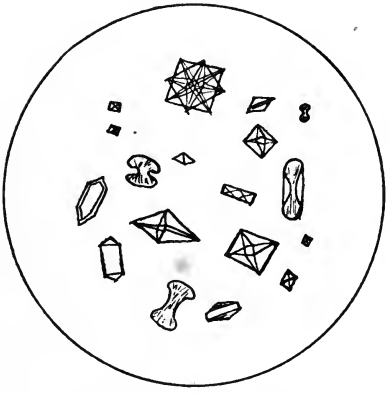


FIG. 25.—Calcium oxalate.

prisms with terminal pyramids are seen. They occur in 25 per cent. of all urines which have stood for eighteen hours (Fig. 25).

III. *Calcium sulphate* is extremely rare; the crystals appear as long, thin, colorless needles or prisms or as elongated crystalline plates.

IV. *Phosphates.*

(1) Calcium and magnesium form colorless, amorphous masses composed of fine granules or spherules. This deposit has no pathognomonic significance.

(2) Neutral magnesium phosphate is of very rare occurrence, appearing as large, refractive, elongated

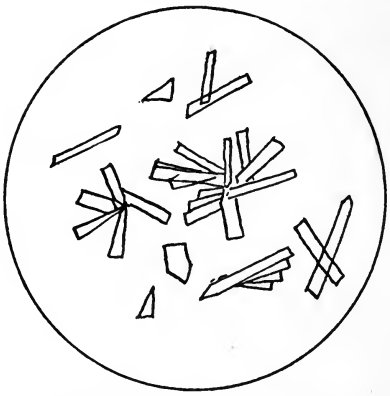


FIG. 26.—Neutral calcium phosphate (stellar phosphate).

plates which may show obliquely-cut ends; bunches of acicular crystals may appear at the poles.

(3) Neutral calcium phosphate, is also called dicalcium or "stellar" phosphate. The crystals are small colorless wedge-shaped or pointed, arranged usually in rosets, fans or flowers. This substance may form form a pellicle in the urine on standing (Fig. 26).

(4) Ammonium magnesium phosphate is the commonest form found; it is also termed "triple" phosphate. It occurs as transparent, colorless, refractive, three-,

four-, or six-sided prisms, having oblique terminal surfaces resembling coffin-lids in shape. They may, however, deviate considerably from the standard type. Occasionally the crystals assume a feathery appearance and closely resemble fern-leaves in their arrangement (Fig. 27).

V. *Uric Acid.*—These crystals appear as yellowish rhombic prisms, whetstone or spindle-shaped; also as sheaves, rosets and barrel-shaped, dumb-bell or

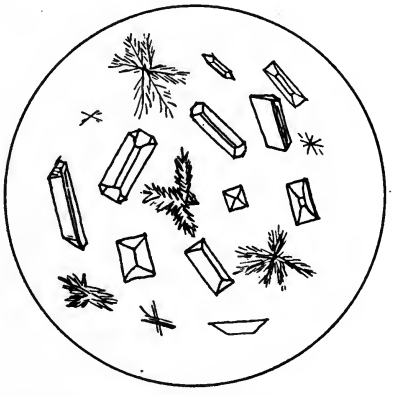


FIG. 27.—Ammonium magnesium phosphate (triple phosphate).

hour-glass forms. These crystals have a tendency also to become grouped or adherent to other materials in the sediment (Fig. 28).

VI. *Urates.*

(1) Acid sodium urate is the principal constituent of the urate deposits, it is usually of a reddish color due to urobilin, and is composed of minute amphorus granules arranged in clumps. These when viewed separately seem almost colorless, but *en masse* show the characteristic yellowish-red tint. These granules

closely resemble those of the amphorous phosphates. The latter, however, do not dissolve on heating.

(2) Acid ammonium urate is an infrequent deposit.

FIG. 28.—Uric acid.

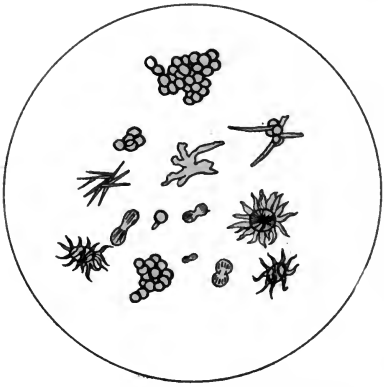


FIG. 29.—Acid ammonium urate.

It crystallizes in opaque yellowish spheroids which may present radiating spines and are sometimes termed "hedgehog" crystals; occasionally "rhyzome" forms are also seen (Fig. 29).

VII. *Hippuric Acid.*—The pathological significance of this rare deposit is as yet unknown. It appears as colorless needles, rhombic plates, and four-sided prisms which are terminated by two or four oblique surfaces (Fig. 30*A*).

VIII. *Cystin* occasionally forms urinary calculi; it crystallizes as thin, colorless, frequently imbricated, hexagonal plates (Fig. 30*B*).

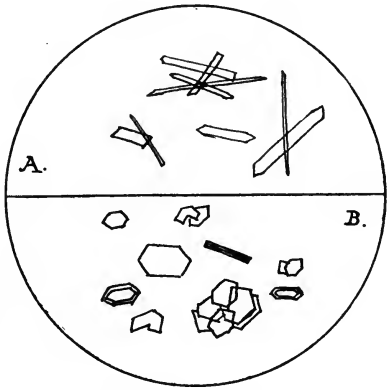


FIG. 30.—A, Hippuric acid. B, Cystin.

IX. *Leucin and Tyrosin* are met with particularly in acute yellowish atrophy of the liver, phosphorus poisoning, leukemia and pernicious anemia.

Leucin appears as glistening, yellowish radial or concentrically striated spherules (Fig. 31*A*).

Tyrosin as fine, colorless, silky needles arranged in tufts, sheaves or stars, but they are frequently colored yellow from the presence of urinary pigments (Fig. 31*B*).

X. *Cholesterin* is occasionally met with in lipuria,

amyloid, fatty and hydatid disease of the kidneys, sometimes also in cystitis and nephrolithiasis.

It crystallizes in thin, transparent, frequently imbricated, rhomboid plates, which plates may show rhomboid notching of one angle (Fig. 31*C*).

XI. *Fat.*—The presence of fat in the urine is termed lipuria and is also present in that condition known as chyluria.

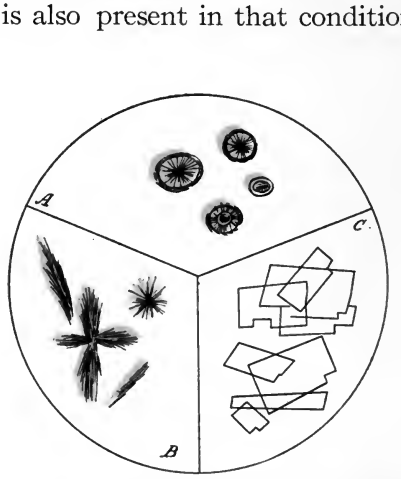


FIG. 31.—A, Leucin. B, Tyrosin. C, Cholesterin.

Free fat globules vary considerably in size, are highly refractive, and turn black after treatment with 1 per cent. solution of osmic acid, and scarlet with "Soudan III." Fat globules may also be present in degenerating epithelial cells, casts, and leukocytes.

XII. *Indigo, melanin, and hematoidin* are occasionally observed.

Indigo may appear as amphorous blue scales; rhombic, lanceolate crystals, or as stellate groups of acicular crystals,

Melanin as fine black granules, and

Hematoidin or bilirubin as reddish needles, plates, or prisms.

XIII. *Epithelium.*

(1) Renal epithelial cells are rarely seen. They are cubical, larger than leukocytes, and present large round nuclei (Fig. 32*A*).

(2) From the urinary passages and vagina the size and shape will vary greatly according to their origin.

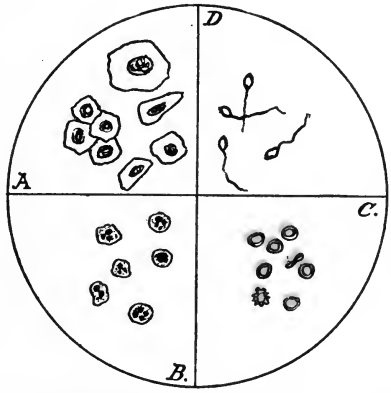


FIG. 32.—A, Epithelial cells. B, Leukocytes. C, Erythrocytes. D, Spermatozoa.

Evidences of degeneration as shown by granulations are frequent (Fig. 32*A*).

XIV. *Spermatozoa* are frequently found after seminal emissions. The occurrence of a few has usually little clinical significance (Fig. 32*D*).

XV. *Cylindroids* consist of long, pale, ribbon-like bodies composed of mucus, frequently striated longitudinally and showing frayed ends; unlike casts, from which they must be differentiated, they are insoluble in acetic acid. They are also of greater length, often extending over several microscopic fields, have a

variable diameter and usually exhibit sharp twists and bends (Fig. 33*A*).

In inflammations of the mucous membrane of the urinary passages they appear in large numbers; a few have little significance.

XVI. *Urethral threads* are formed of mucin derived from the glands of the urethra or prostate, are long and thin, visible to the naked eye and are frequently seen floating in the morning urine of patients suffering from

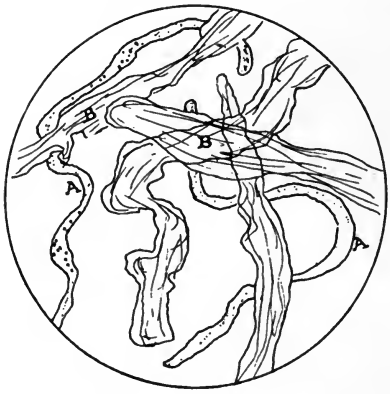


FIG. 33.—A, Cylindroids. B, Urethral threads.

urethritis following gonorrhea, in which case they may contain gonococci (Fig. 33*B*).

XVII. *Erythrocytes* are recognized by their characteristic round biconcave form and faint yellow color. In concentrated urines small and crenated cells are frequently found. In females they not infrequently owe their origin to the menstrual flow, and unless this source be recognized their presence may lead to erroneous deductions (Fig. 32*C*).

XVIII. *Leukocytes.*—These cells in acid urines generally appear granular, in an alkaline medium may

become swollen and transparent, not infrequently they may show evidences of fatty degeneration (Fig. 32*B*).

At times they closely resemble small epithelial cells; the addition of a drop of iodopotassium iodid solution stains the leukocytes a gray-brown color; the epithelial cells assume a light yellow tint.

A few leukocytes, however, are found in even normal urines; if pus be present to any extent a grayish deposit will separate out on centrifugalization. In an excessively alkaline urine they may become gelatinized and lose their morphology.

XIX. *Casts* are coagulated albuminoid deposits formed in the renal tubules and are almost invariably associated with recognizable albuminuria.

(1) Hyaline casts occasionally appear in the absence of renal disease; they are straight or convoluted, homogeneous, transparent cylinders, varying considerably in length and diameter (Fig. 34*A*).

In urine containing bile or blood they may appear yellow in color. It is also well to remember that no definite relation exists between the severity of the primary lesion and the number of casts present.

(2) Waxy or colloid casts are supposed to result from renal epithelial degeneration and are frequently found in chronic interstitial nephritis and amyloid disease. They are highly refractive, homogenous, often exhibiting lateral indentations; they are usually more bulky and broader than the hyaline variety (Fig. 34*B* (*a*)).

In amyloid disease they may give the characteristic reaction, a pink color with a methyl-violet stain and a brown with iodin solution, but the same reaction may appear even in the absence of this lesion.

(3) Granular casts are usually shorter and thicker than the above varieties, are somewhat darker and generally granular throughout, the granules consisting of albumin or fat globules; they are, as a rule, composed of degenerated epithelium (Fig. 34*C*).

A hyaline cast may be studded with amphorous urinary salts; it then differs from the true granular cast in being lighter, longer, narrower, and not so

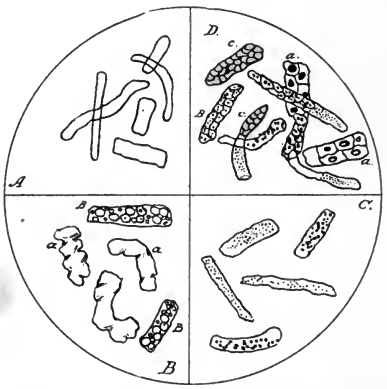


FIG. 34.—A, Hyaline casts. B, (*a*) Waxy casts, (*b*) fatty casts. C, Granular casts. D, (*a*) Epithelial casts, (*b*) leukocytic casts, (*c*) blood casts.

densely granular, it frequently also shows transparent areas.

(4) Epithelial casts are indicative of desquamation in the renal tubes. They are met with particularly in acute and chronic parenchymatous nephritis, the renal cells being either adherent to or being imbedded in a granular or hyaline cast (Fig. 34*D* (*a*)).

Occasionally the epithelial lining of the renal tubules may be shed *en masse*. "Epithelial tubes" may thus appear.

(5) Fatty casts are simply hyaline or granular varieties beset with fat globules, and are met with in fatty degeneration of the kidneys (Fig. 34*B* (*b*)).

(6) Leukocytic casts consist of the ordinary hyaline or granular forms coated with leukocytes (Fig. 34*D* (*b*)).

(7) Blood casts are those coated with erythrocytes; they are indicative of renal hemorrhage or acute nephritis (Fig. 34 *D* (*c*)).

XX. *Tumor cells*, elastic fibers, spermatozoa, air-bubbles, foreign bodies, and the ova of certain parasites may also be found in the urine.

XXI. *Bacteria.*—Numerous organisms may be found in the urine, of the pathological forms the tubercle bacillus and gonococcus demand consideration.

Whenever the bacilluria is present and it is necessary to isolate the organisms, the urine must be removed by catheter under aseptic precautions. For details of bacteriological technic reference must be made to text-books on bacteriology.

To detect tubercle bacilli or gonococci centrifugalize urine, decant, dilute with water, recentrifugalize, make a cover-glass preparation from the precipitate, fix by passing three times through the flame of a Bunsen flame, and stain by the special methods described on page 355.

Direct *inoculation* of several c.c. of urinary sediment may be made into the peritoneal cavity of a guinea-pig in order to detect the presence of the tubercule bacillus. Progressive loss of weight, adenitis, and at the end of from four to six weeks on postmortem examination a generalized tubercular infection of the animal will be found.

THE BLOOD.

I **ENUMERATION OF ERYTHROCYTES.** — The Thoma-Zeiss or Gower's hemocytometer may be used. The blood is usually obtained from the lobe of the ear. The part is thoroughly cleansed with a solution of corrosive sublimate, 1 in 1000, dried with sterile wool, and washed in alcohol which is then allowed to evaporate. Puncture with a cutting needle or lance, allowing the blood to flow freely. Never attempt to squeeze the blood from the ear, and never use the first drop exuded.

(1) **The Thoma-Zeiss' apparatus** is composed of a graduated capillary pipet with a reservoir and a glass counting slide.

Draw up blood in the tube to the mark 0.5 exactly, wipe off tip of instrument carefully, plunge it immediately into a diluting fluid (Toisson's or Hayem's solutions) and draw this up until the mark 101 is reached, thus diluting the blood 1 in 200; grasp the tube between the thumb and finger so as to close its ends and shake for one minute.

It is necessary that these directions be carried out most carefully, for if the blood happens to be drawn beyond the 0.5 mark the procedure must be repeated after a thorough cleansing of the instrument.

It will now be seen that the blood in the reservoir of the pipet has been diluted 1 in 200 times, also that the fluid which remains in the capillary tube must not be used on the counting stage as it contains no blood and should be blown out and discarded.

Next place a small drop of the mixture on the round stage of the slide and cover with a glass slip (both

having been previously cleansed and freed from grease). The drop must be of such a size that on application of the cover-slip it spreads out exactly to the size of the round platform. If any overflow into the trench, if the quantity is insufficient to cover the platform, or if air bubbles are present, the slide must be again cleansed and the procedure repeated.

This degree of accuracy is necessary since the depth of the space between cover and platform is 1/10 mm. and our calculations are based on the assumption that the depth of the layer of diluted blood is of precisely that measurement.

Examine the specimen under the low power of the microscope and it will be seen that in the center of the round platform 400 small squares are ruled on the glass, and it will also be noted that a series of double lines divide these 400 small squares into a set of 16 large squares, in each of which there will be 25 of the smaller. The whole area thus delineated covers exactly 1 sq. mm., and since the layer of supernatant fluid is 1/10 mm. in depth, we see that we have practically isolated 1/10 cu. mm. of diluted blood and also that each of the smaller squares must necessarily contain 1/10 x 1/400 of a cu. mm.

Now turn on the high-power lens, count the number of red cells in at least five of the sixteen large squares, or in other words the number lying in 125 small squares, and make allowance for the dilution as follows:

$$\frac{A \times C \times 4000}{B} = \text{number of erythrocytes per cu. mm.}$$

A. Total number of corpuscles counted.

B. Total number of small squares counted.

C. Number of times blood diluted.

In counting the cells take each small square separately. It is usual to include in the count any cells touching the upper and right sides of the squares and disregard those overlapping the remaining sides.

(2) **Turck's** counting stage may also be used; the same pipets, diluting fluids and technic are employed as with the Thoma-Zeiss method. This stage is divided into nine large squares, each having a capacity of 1/10 cu. mm., the centre square, however, being subdivided as is that of the Thoma-Zeiss stage for the purpose of enumerating the erythrocytes. The advantage of this apparatus is that the leukocytes may be counted in the same preparation, which in this case contains 9/10 cu. mm. of diluted blood.

(3) **The number of red blood cells** per cubic millimeter of normal blood ranges from 4,500,000 in females to 5,000,000 in males. In concentrated states of the blood (following diarrhea, etc.) this number may be considerably exceeded, while in certain anemias it may fall to even below 500,000 per cubic millimeter.

Toisson's Fluid.—	Methyl violet (5 B)....	0.025 grams.
	Sod. chlor.	1.000 grams.
	Sod. sulph.....	8.000 grams.
	Glycerin (neut.).....	30.000 grams.
	Aq. distill.....	160.000 grams.
Hayem's Fluid.—	Sod. chlor.....	1.0 grams.
	Sod. sulph.....	5.0 grams.
	Mercuric chlorid.....	0.5 grams.
	Aq. distill.....	200.0 c.c.

II. ENUMERATION OF LEUKOCYTES.

(1) With the *Thoma-Zeiss instrument* is supplied a special graduated pipet for the estimation of the white cells. The preliminary technic is the same as for the red counting, but the blood is drawn up to the

mark 0.5 and the diluting fluid to 11.00. The dilution therefore will be 1 to 20.

Enumerate the leukocytes in the whole field of 400 squares. The result will be the number in 1/10 cu. mm. of the fluid. Now multiply by ten, and then by the dilution in order to obtain the number of leukocytes per cubic millimeter. The dilutent used, contains acetic acid which dissolves the red cells and renders the white more distinct. Gower's fluid may be used, or simply a 2 per cent. solution of acetic acid.

For accurate results at least three slide preparations and counts should be made; the same statement applies in the estimation of the red cells.

The leukocyte count may be made by means of the same pipet as was employed for the red cells, but draw the blood up to the point 1.0, thus diluting the blood 1 to 100. This method, however, does not give such accurate results. Turck's slide may also be used.

(2) The **normal number of leukocytes** varies from 5000 to 7000 per cubic millimeter. Shortly after meals a physiological increase of several thousand may occur.

(3) **A leukocytosis,** or increase in the total number of white cells, is found in many diseases. In some cases of leukemia over 1,000,000 per cubic millimeter may be found; an ordinary inflammatory leukocytosis, however, seldom exceeds 40,000.

(4) **Leukopenia** is the term used to express the fact that there are present in the blood less than the normal number of white cells.

III. **BLOOD PLATELETS** are seldom enumerated clinically; the student is referred to special works on hematology for the necessary technic.

IV. **ESTIMATION OF THE HEMOGLOBIN.**

Numerous instruments have been devised for the estimation of the hemoglobin, chief of which are Gower's, Sahli's, Haldane's, Oliver's, Dare's hemoglobinometers and Tallqvist's scale. These are all graduated in a percentage scale, 100 per cent. being the normal reading.

The *hemoglobin index* is obtained by estimating the percentage of coloring matter in terms of the amount contained in each cell. Thus if the red blood cell count in a case be 1,000,000 and the percentage of hemoglobin be twenty or, in other words, if both the cells and pigment be reduced in the same proportion, the "index" will be read as "one"; again, if the cells number, say 2,500,000 (in other words, 50 per cent. less than the normal count), and the hemoglobin read 25 per cent., the index will be "one-half of one."

V. **FRESH BLOOD SMEARS** are used if the presence of the spirillum of relapsing fever or of the filaria sanguinis hominis (Fig. 35) is suspected, and also if it is desired to observe the movements of the malarial parasites.

The blood is obtained in the usual manner, a drop being placed on a slide, a ring of vaselin painted around the spot and a cover glass placed in position. The vaselin must not be allowed to come in contact with the blood, it is employed only to exclude air and so limit evaporation.

VI. **FIXED BLOOD SMEARS.**

(1) **Preparation.**—Touch the drop of blood with the middle of one of the narrow ground edges of a microscopic slide, draw this rapidly across a clean coverslip free from grease and an evenly distributed film will be obtained.

Allow the films to dry in the air. Fix by rapidly passing the slips, film upward, three times through the flame of the spirit lamp, or by immersing them for five minutes in a mixture of equal parts of alcohol and ether, or by heating on a specially arranged copper platform.

(a) They may be stained with *eosin* until the film is pink, then washed and counterstained with a saturated aqueous solution of *methylene blue*. Note the depth of the staining under the low power of the microscope as it is proceeding, wash in water, dry and mount in canada balsam, covering with a glass slip.

(b) *Jenner's stain* is applied to the dried smear, cover with a watch-glass to prevent evaporation for about seven minutes, wash in distilled water until pink (five to ten seconds), dry rapidly over a flame, mount.

The erythrocytes should stain a copper color, platelets mauve, nuclear material blue, neutrophilic granules purple, basophilic ("mast-cells") dark violet, and eosinophilic a bright pink color.

(c) *Ehrlich*'s *triacid stain* is applied after fixing the film by heat on a copper bar, staining for two to ten minutes, wash, dry and mount in neutral balsam. The staining resembles closely that obtained by Jenner's method, but the basophilic mast-cell granules are unstained, the nucleus of these cells staining a light green color.

(d) *Wright's Method.*—Add 5 to 8 drops of stain to dried smear on cover glass, allow to remain from one-half to one and one-half minutes, add 4 or 5 drops distilled water or until a metallic sheen appears, leave for 3 to 5 minutes, wash in distilled water for 3 minutes and dry.

Numerous other methods are employed for details of which the student is referred to treatises on hematology.

(2) The **clinical value** of these fixed and stained blood films will now be discussed.

(a) **Erythrocytes.**—Note the size, shape, color, presence of abnormal colored granules in the cells, and of nuclei (Plate IV).

Erythrocyte.—A normal red blood cell.

Microcyte.—A small form of red blood cell.

Macrocyte.—A large form of red blood cell.

Normoblast.—A normal sized, nucleated red blood cell.

Microblast.—A small sized, nucleated red blood cell.

Megaloblast or *Gigantoblast.*—A large sized, nucleated red blood cell, (12–20 microns).

Poikilocyte.—A misshapen, red blood cell.

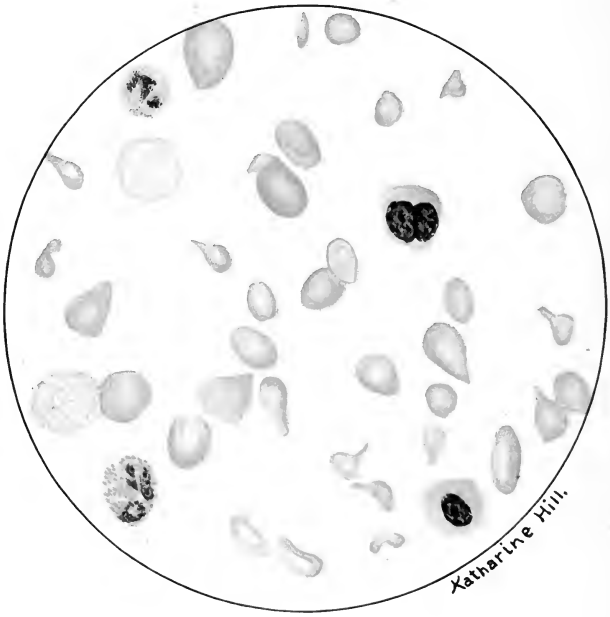
Polychromatophilia.—Is the term applied to an abnormal staining reaction of the erythrocytes, in which they take up the nuclear stains in patches or granules. This is believed to be an indication of degeneration.

It is well for the student to remember that two forms of stains are used, the acid or "protoplasmic", and the alkaline or "nuclear", and that staining is a chemical reaction and not a mere "dyeing." Nuclei always take up the alkaline, and protoplasm the acid stains if the chemical composition of the cells be normal, remember also that therefore the color of even the stained red cells will be an indication of their hemoglobin index, they being paler in color when this is low.

(b) **Leucocytes.**

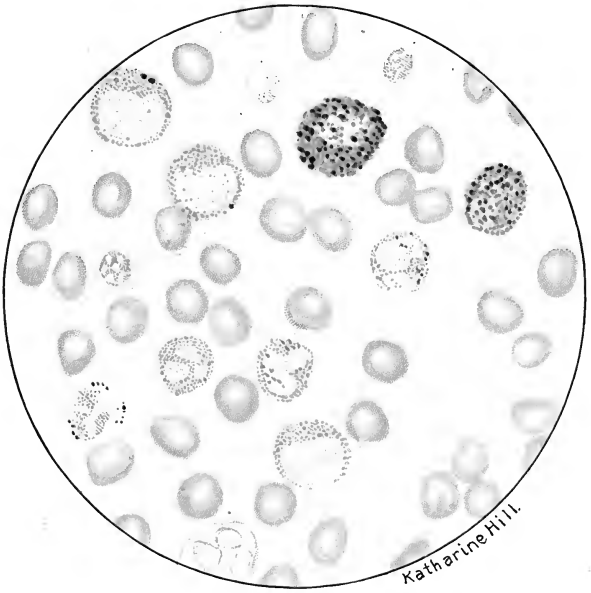
(i) *The polymorphonuclear* forms comprise about 70 per cent. of the total number of leukocytes present.

PLATE IV.



Blood in Pernicious Anemia. Wright's Stain.
(*From Webster's Diagnostic Methods.*)

PLATE V.



Blood in Spleno-myelogenous Leukemia. Tri-acid Stain.
(*From Webster's Diagnostic Methods.*)

The nucleus is multipartite and the protoplasm contains numerous fine neutrophilic granules, which stain a purple color.

In an ordinary inflammatory leukocytosis it is this form that is increased out of all proportion to the others.

(ii) *Eosinophiles* are cells in size closely approximating the polynuclear cells. They contain a lobed nucleus and large refractive, bright pink, eosinophilic granules and form from 2 to 4 per cent. of the leukocytes.

They are increased out of proportion to the other cells especially in cases of asthma, pemphigus, trichinosis and certain other parasitic diseases.

(iii) "*Mast cells*" or basophilic polynuclear leukocytes comprise 0.5 per cent. They are not stained by Ehrlich's mixture.

(iv) *Small mononuclear leukocytes* with a deeply staining, round nucleus surrounded by a ring of clear protoplasm, form from 20 to 25 per cent. of the total. They may in lymphatic leukemia form 95 per cent. of the total leukocytes present (Plate VI). Of these cells there are two varieties, the "large lymphocytes" which have a diameter of 8 microns or over and the "small lymphocytes" which closely approximate erythrocytes in size.

(v) *Large mononuclear* cells with a less deeply staining nucleus and hyaline protoplasm, along with occasional other cells of a similar size, often with a lobed or horse-shoe shaped nucleus and occasionally a few neutrophilic granules, form from 2 to 4 per cent. of the total leukocytes.

(vi) *Myelocytes* are never found in normal blood,

they are present in spleno-myelogenous leukemia and form about 30 per cent. of the leukocytes (Plate V).

There are three forms, all being considerably larger than an ordinary leukocyte, the *nuclei* are always large and single, the *protoplasm* granular, and these granules either neutrophilic, basophilic or eosinophilic.

(c) **Differential count of leukocytes.**—At least two hundred corpuscles should be counted, and the percentage of the different varieties noted. A "*movable stage*" will greatly facilitate this work.

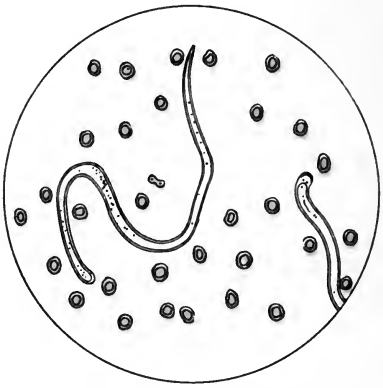
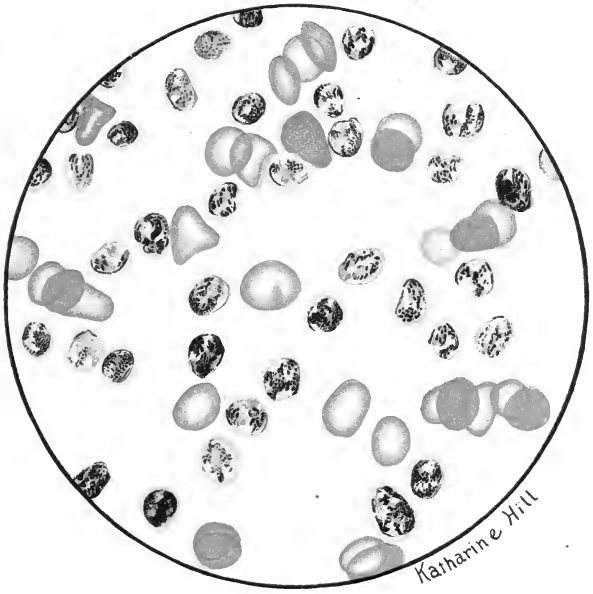


FIG. 35.—Filaria sanguinis hominis.

(vii) **Coagulability** of the blood is estimated by means of Wright's coagulometer. Directions for its use accompany this instrument so no description will be given here. The normal "coagulation time" at a temperature of 18.5 C. is close to four minutes.

(viii) **The Alkalinity** of the blood may be estimated by either Hutchison's or Wright's methods, which are too complicated to be of use in ordinary routine examinations. The same statement applies to the

PLATE VI



Lymphatic Leukemia Tri-acid Stain.

(*From Webster's Diagnostic Methods.*)

determination of its *specific gravity*, and also that of the presence of *carbon dioxide*.

(ix) **Parasites,** etc. The filaria sanguinis hominis (Fig. 35), the protozoa of the malarial fevers, trypanosomes, and the spirillum Obermeiri may be seen in

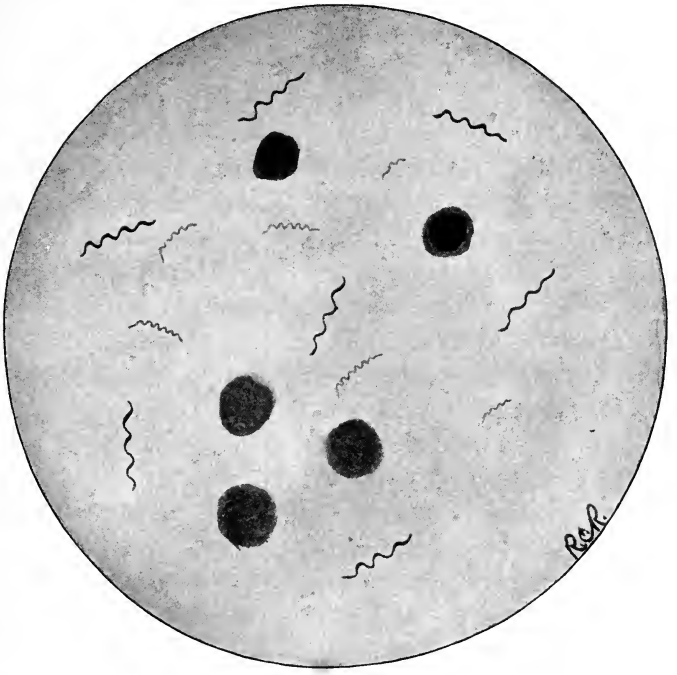


FIG. 36.—Spirochæte pallidæ and refringens. (*Pitfield.*)
The darker ones are the refringens.

ordinary fresh blood smears. The spirocheta pallida is only visible when stained (Fig. 36). The other bodies also may be fixed and stained by special methods.

(x) **Bacteria.**—*Blood cultures* must be made under the most rigid precautions in order to guard against contamination of the cultures and infection of the patients.

The blood is taken from one of the large veins of the forearm by means of hypodermic syringe, and the blood so removed inoculated on various media. For details regarding the technique and the isolation of any organisms present the reader should consult a text-book on bacteriology.

(xi) **Opsonic Index** (see special works).

THE SPUTUM.

A. MACROSCOPIC EXAMINATION.

I. **Amount.**—The total quantity expectorated depends on the type of bronchitis present, in the "dry" form for example practically none may be obtained; while on the other hand, in bronchiectasis and during the formation of vomicæ the quantity may be enormous. Also it is well to note if the expectoration occurs in paroxysms, as is found when from change of posture a cavity is allowed to discharge its contents into the bronchial tubes.

It is necessary to determine the *viscidity*, markedly tenaceous sputa being common in "dry" bronchitis and lobar pneumonia; also the *reaction* which though usually alkaline may, if the sputum be retained for some time in the lungs, acquire an acid reaction.

II. **The color** varies greatly; blood, for instance, shows itself in the sputum either as bright red streaks, "*rusty*" granules, (pneumonia) or as bright, frothy blood (recent hemoptysis).

Bile may be present after perforation of an hepatic abscess into the lung; this tints the sputum usually red, green, or bluish. The bacillus pyocyaneus also gives a green or blue color to the discharge. It is well to

remember that a brownish color is often due to the use of chewing tobacco, that in workers in brass and bronze a reddish color (*siderosis*) may be present, and that in coal-miners the sputum may be distinctly black (*anthracosis*).

III. **The Odor.**—With bronchiectasis and tubercular vomicæ a sweetish offensive odor is commonly found, and with perforating empyemata one resembling that of stale cheese.

IV. **The Character.**—The sputum is best examined macroscopically by placing a small quantity between two glass plates, and thus spreading it out into a thin layer.

Sputum unmixed with air, such as comes from deep-seated cavities, sinks in water, it is called *sputum fundum petens*. *Mucoid sputum* is glairy, tenaceous, transparent and is seen most frequently in asthma and acute bronchitis.

Muco-purulent sputa are those containing a mixture of mucous and pus, while the term *purulent* is applied to specimens containing pus in large quantities as from ruptured empyemata, pulmonary abscesses, etc.

Nummular sputum refers to that type of muco-purulent discharge which arranges itself flat like a coin on the bottom of the receiver, It is usually seen with tubercular vomicae.

In edema of the lungs a *serous* sputum may be obtained; it is usually frothy and colorless. The mixture of blood with sputum is described by the term *sanguinous*.

In putrid bronchitis, bronchiectasis, and gangrene of the lung not infrequently the specimen on standing will separate into three layers, the uppermost being

mucoid and frothy, the middle opaque, watery, and consisting of sero-pus, while the lowest contains tissue shreds, bacteria, pus, etc.

Rusty sputum is characteristic of lobar pneumonia, in it the blood is intimately mixed with the sputum which is usually very tenaceous.

Cheesy masses formed of fragments of necrotic tissue, varying in color from yellow to black, are not infrequent with gangrene, tuberculosis or abscess formation.

Dittrich's plugs which resemble the masses above described may be expectorated even by normal persons. They vary in size from a pin-point to that of a large pea, in color are grayish-yellow, and are often expectorated without any accompanying mucous. They are composed of bacteria, fatty-acid crystals, fat globules, and cellular detrius; possess a disagreeable odor, and not infrequently form casts of the bronchioles.

Curschmann's spirals are present in nearly all cases of true bronchial asthma, but may also be seen in acute bronchitis, lobar pneumonia, and chronic tuberculosis.

They are composed of a spiral network of fine fibrils wrapped around a central cord; often eosinophiles, epithelial cells, and Charcot-Leyden crystals are adherent to them. In length they vary from 1 to 2 cm., but they are usually wound into a tiny ball which should be unravelled before subjecting them to microscopic examination.

Fibrinous casts of the bronchi vary in length from 1 to 15 cm., the larger forms often possessing a lumen. They are occasionally found in certain types of bronchitis.

Bronchioliths are merely concretions formed in vomicæ by the precipitation of calcium salts, and may reach the size of a walnut.

B. **MICROSCOPIC EXAMINATION.**

I. **Leukocytes** are found in every specimen, the eosinophilic forms being particularly numerous in asthma.

II. **Erythrocytes** unless present as a contamination are usually of serious import. They point to pulmonary congestion or destruction of lung tissue.

III. **Epithelial cells** of all types may be present. They may contain coal pigment in the form of granules, fat or myelin globules. The latter are irregular in shape, show concentric striation, a greenish-blue color, and are but faintly refractile. Myelin may appear as homogenous masses which resemble boiled sago grains, no cell bodies being visible.

"*Heart failure cells*" are alveolar epithelial cells containing reddish-brown pigment granules derived from hemoglobin. They are found in cases exhibiting chronic pulmonary congestion.

IV. **Elastic tissue** may be seen macroscopically and when present is indicative of destruction of the lung parenchyma. It is best detected by placing the two layers of glass with the intervening layer of sputum against a dark surface; the fibers which often form a distinct net-work may thus be isolated and then examined microscopically.

V. *Crystals of cholesterin* (Fig. 31*C*), those described by *Charcot-Leyden*, those of *hematoidin*, *leucin* and *tyrosin* (Fig. 31), are occasionally also encountered.

VI. **Bacteria.**

(1) *Saprophytic* forms are very numerous, especially those of the strepto- and leptothrix groups, yeast fungi and moulds. Almost any forms of the pus-forming organisms may be present as saprophytes.

sarcinæ, the micrococcus tetragenus, and many others may also be present as nonpathogenic germs.

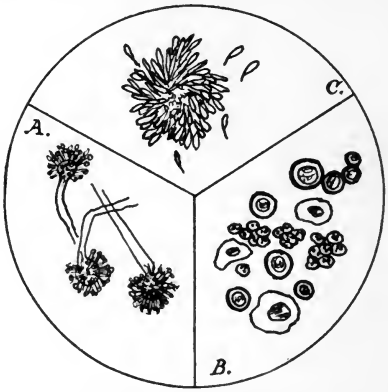


FIG. 37.—A, Aspergillus fumigatus. B, Blastomycetes. C, Actinomyces.

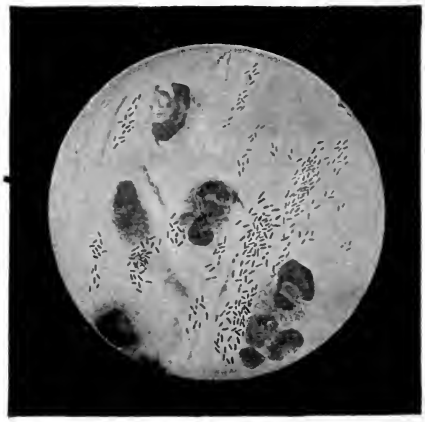


FIG. 38.—Influenza Bacillus. (*Abbott.*)

The aspergillus fumigatus (Fig. 37*A*), oidium albicans (Fig. 23), actinomyces (Fig. 36*C*), and blastomycetes (Fig. 36*B*) may also be found; they are at times patho-

genic and produce lesions resembling those of ulcerative tuberculosis.

(2) *Pathogenic Bacteria.*

(a) *Tubercle Bacillus* (Fig. 46).—Isolate one of the small cheesy particles and smear well on the cover-slip. For method of staining see page 355.

It may be necessary to differentiate them from the smegma, timothy and lepra bacillus, for details of which reference should be made to text-books on bacteriology.

(b) *Influenza bacilli* (Fig. 38) are "Gram-negative" organisms, but may be also stained by ordinary methods.

(c) *Pneumococcus of Fraenkel* and *Friedlander's* organisms are frequently found and may be stained by the ordinary dyes (Fig. 49).

GASTRIC CONTENTS.

The object of examining the contents of the stomach is to determine the digestive, motor, and absorptive powers of that organ.

A. **MACROSCOPIC EXAMINATION.**—In order to obtain accurate information regarding the gastric digestive functions, *test-meals* have been devised. These must be taken on a fasting stomach, or on one emptied and recently washed out.

I. **Test-meals.**

(1) *Ewald's test-meal* is the one most frequently made use of. It consists of a roll or a slice of toast without butter, and a cup of water or weak tea without milk or sugar.

In all meals the food should be well masticated and the contents withdrawn at the end of one hour exactly.

The normal amount obtained ranges from 30 to 50 c.c. In hypermotility the amount will be decreased, in hypomotility it will be increased.

It is well also to remember that the bread may contain a small amount of lactic acid.

(2) *Boas' test-meal* consists of a dish of oatmeal made by adding a tablespoonful of oatmeal to 500 c.c. of water and concentrating this by heat. The amount of lactic acid is small, less than in the Ewald meal. It is similarly withdrawn one hour after ingestion. Normally the amount obtained is very small. A large quantity would indicate dilatation, hypomotility, or pyloric obstruction.

An objection to either of the preceding forms is that the meal is inconsistent with that usually taken by people living in America, being far less liberal in amount. Nor do either of these meals adequately stimulate the flow of gastric juice.

In order to overcome these difficulties a meal has been contrived consisting of Ewald's meal with an addition of one-quarter pound of finely chopped lean beef, boiled and seasoned. The contents are withdrawn in three hours.

II. **Collection of the specimen** is usually accomplished by means of the *stomach-tube*.

Several points must not be overlooked regarding the passage of the tube. Thus great care must be used in sterilizing it, as for example, one patient might easily be infected from another with syphilis, etc. False teeth must be removed and the mouth and pharynx be inspected. Should a marked degree of irritability be met with, swabbing the pharynx with a weak solution of cocain is indicated.

Should the patient become greatly excited or cyanosed during the manipulations, it is better to withdraw the tube and make a second or third attempt, or even to desist till some future time.

To pass the tube seat the patient leaning well forward. The tube is passed quickly directly backward into the pharynx, while at the same time he is told to swallow repeatedly and to breathe deeply. The esophagus grasps the tube and the effort of swallowing carries it downward as it is payed out from above.

The position of the mark on the tube indicating the normal distance from the teeth to the cardiac orifice should be closely observed, and also the moment any resistance is met with.

Should a flow not occur spontaneously as a result of the expulsive efforts, either pay out a little more tubing or withdraw a short distance. Aspiration by means of the bulb may be resorted to; slight pressure may also be made over the epigastrium at the same time.

Should, however, expulsion or siphonage of the contents prove unsuccessful a measured quantity of water is allowed to run in and the maneuver repeated. This factor must be taken into consideration when the quantitative examinations are made.

III. **Physical Examination.**

(1) The *amount* has already been discussed.

(2) *Color.*—Normally the fluid obtained is colorless or yellowish-brown.

Bright red indicates recent hemorrhage, as seen in gastric ulcer, etc. When blood remains in the stomach for some time in contact with the gastric juice it becomes dark red or brownish-black. This is the so-called *coffee-ground* material.

Bile mixed with the stomach contents imparts various tints to the fluid and answers to the tests for bile as given on page 298.

(3) *Odor.*—It is sour normally, but may be aromatic from the presence of the fatty acids. Butyric acid yields an odor like that of rancid butter, bile and feces also give their characteristic odors when present.

A putrid odor is found in cases of dilated stomach.

The nature of an ingested poison may also often be detected from the odor of the vomitus.

(4) *Consistence.*—Normally the contents are watery in character; they may be, however, tough and slimy owing to an excessive amount of mucus.

(5) *The Vomitus.*—In cases of dilatation of the stomach or of stenosis of the pylorus the amount vomited at one time may reach 2 or even 3 quarts. The vomitus may also show the characteristics mentioned above under color of the contents.

Fecal vomitus may be green or brown in color. It is characterized mainly by the odor and its neutral reaction. Blood if in large quantity is usually clotted and is dark in color.

B. **CHEMICAL EXAMINATION.**—From it a definite knowledge of the acidity of the stomach contents, the presence or absence of certain ferments such as pepsin and rennin, the degree of digestion as far as the carbohydrates and proteins are concerned, and the presence of bile, blood or other foreign material is determined.

(1) **Reaction.**—Normally the reaction is acid. After a severe hemorrhage it may be alkaline; in some cases of gastritis it may be neutral. Free acids, combined acids, and acid salts turn blue *litmus* red.

(2) **Free Acids.**—Strips of filter-paper are soaked in a saturated solution of *congo red* and allowed to dry. Free acid turns this dark red color to a deep blue. A positive reaction indicates the presence of free hydrochloric, lactic, butyric, or acetic acids.

(3) **Free Hydrochloric Acid.**

(a) *Günzburg's Test.*—Two grams of phloroglucin and 1 gram vanellin are dissolved in 30 c.c. of absolute alcohol. The solution decomposes readily, so should be freshly prepared. A few drops of the above reagent are added to a portion of the stomach contents. On gently heating a clear pink color is obtained providing free hydrochloric acid is present.

(b) *Dimethyl-amido-azo-benzol.*—A cherry-red color is given with free hydrochloric acid. Weak organic acids (under 1/2 per cent. solutions) do not give a positive reaction, but produce an orange or yellowish color.

(c) *Tropæolinin Test.*—Tropæolinin in a saturated alcoholic solution gives a yellow orange color. A few drops are added to an equal quantity of stomach contents, heat gently, a blue or violet color indicates free hydrochloric acid. No organic acid will give this reaction. Vary the test by evorporating to dryness before adding the stomach contents.

(4) **Organic Acids.**—*Uffelmann's Reagent* consists of a solution of 1 in 20 of carbolic acid solution with a few drops of tincture of the perchlorid of iron added, and a further dilution with water is made till an amethyst blue color is obtained. With organic acids (lactic) a canary-yellow color develops.

(5) **Gastric Ferments.**

(a) *Pepsin.*—If albumoses are present then pepsin

is proven to be present, because pepsin must be present before albumose is produced.

The detection of albumose is accomplished by adding 2 drops of a 10 per cent. copper sulphate solution to a test-tube and inverting so as to allow the greater part of it to escape. Add to this a column of gastric contents 1 inch in height and an equal quantity of 10 per cent. sodium hydrate solution. A red or reddish-purple color indicates albumose (*biuret reaction*, page 305).

If albumose is absent another test may be applied. Small strips of hard-boiled egg are placed in a few c.c. of the stomach contents and the digestive action upon the egg albumin noted.

(b) *Rennin.*—Two or three drops of the gastric contents are added to 5 c.c. of fresh amphoteric milk and kept at 98 to 99° F. for fifteen minutes. If the milk sets into a mass rennin is present.

(6) **Carbohydrate Digestion.**

Lugol's Solution.—A few drops of this solution are added to some of the gastric contents. If starch is present the reaction is blue. Erythrodextrin causes a brown color.

If all the starch has been changed to dextrose at the end of one hour there will be no reaction.

Lugol's Solution.

Iodin	1 gram.
Potass. iodid	2 grams.
Aq. distill	30 c.c.

(7) **Blood.**

(a) A small portion of the dark brown material is taken and a small amount of powdered potassium chlorate is added to it. To this is added a few drops of hydrochloric acid and the whole is heated till the

powder is dissolved. A blue color on the addition of potassium ferrocyanid indicates the presence of blood. The reaction is due to the presence of the iron of the blood.

(b) *Teischmann's test* may also be used. Thoroughly mix a small portion of the contents with a few grains of common salt, evoporate gently till dry. Moisten with a drop of glacial acetic acid, add a cover-glass, and heat just short of boiling. On cooling rhombic crystals seen with the high power lens of the microscope indicate the presence of blood.

(c) The Benzidin test is applied as follows: Boil a portion of the suspected material in a test-tube, allow to cool, add 3 to 10 drops of this material diluted with an equal part of water, to 1 c.c. of a concentrated benzidin solution, then add 1 to 3 c.c. of H_2O_2 (3 per cent.) and shake. A green or blue color will indicate the presence of blood (*Goodman*).

(d) See also page 297.

(8) **Quantitative Estimations.**

The acidity of the gastric contents is expressed quantitatively in degrees of acidity or acidity per cent. (not %). The degree of acidity or acidity per cent. of a gastric contents is the number of c.c. of N/10 NaOH required to neutralize the acid in 100 c.c. of that gastric contents.

The acidity of the gastric contents is due to free HCl, to HCl combined with food, to organic acids or acid salts.

(1) *First Titration.*—Take 10 c.c. of the filtered contents in a breaker and add 2 or 3 drops of dimethyl-amido-azo-benzol as indicated. The mixture becomes cherry red if free HCl be present. Fill the buret with

N/10 NaOH, and run this in until the indicator shows neutralization, the red color changing to orange-yellow. The number of c.c. of N/10 NaOH used, multiplied by 10 gives the acidity due to free HCl in 100 c.c., or the acidity per cent.; this is normally from 15 to 30.

Now add a few drops of phenolphthalein to the contents of the beaker and run in more of the alkali solution until a red color appears. The total number of c.c. of alkali solution used from the first gives the degree of total acidity, normally 50 to 70.

(2) *Second Titration.*—To 10 c.c. of filtrate add 2 or 3 drops of alizarine solution, and add the alkaline solution from the buret till a violet color appears. This amount of soda solution multiplied by 10 gives the acidity due to free HCl, acid salts, and organic acids.

The difference between the total acidity and this last result gives the acidity due to HCl combined (with food). This is normally 45.

C. **MICROSCOPIC AND BACTERIOLOGIC EXAMINATION.**—Minute shreds of ingested food, large flat epithelial cells, bacilli. and yeast cells are to be seen even in normal cases, but when found only in small amounts no note need be made of them. A few erythrocytes may occasionally be due to hemorrhage following an abrasion caused by the stomach-tube.

Various varieties of food may be recognized. Muscle fibers have transverse striæ. Starch granules are oval in contour, show concentric striation, and are turned blue by dilute iodine solution. Fat globules are highly refractile of different sizes but usually spherical in outline (osmic acid, Soudan III).

Sarcinæ ventriculi may occur normally in small numbers, but are usually found in cases of dilatation with excessive fermentation. They are found arranged in tetrahedra or large cubes, and are said to resemble bales of cotton.

The *Oppler-Boas bacillus* is generally found in cases of carcinoma of the stomach associated with a large quantity of lactic acid. This latter body, however, is

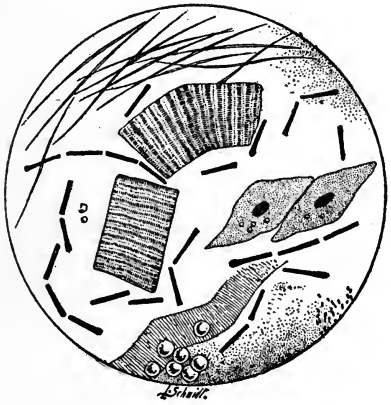


FIG. 39.—Oppler-Boas bacillus. (*Hemmeter.*)

usually not found in large amounts, in early cases; the organism also may frequently be absent.

They are large bacilli, non-motile, 5 to 10 microns in length and 1 in width (Fig. 39), and are often found in long chains joined end to end. Iodin solution stains the Oppler-Boas a brown color. It is a "Gram-positive" organism.

This organism is present in about 85 per cent. of all cases of cancer. It is rare to find it in simple dilatation of the stomach.

THE FECES.

I. **Amount.**—Note if copious or scanty, also the number of stools per diem. Average quantity passed ℥iv in twenty-four hours.

II. **Color.**—Black stools may be due to the administration of iron, bismuth, etc., or to the presence of altered blood.

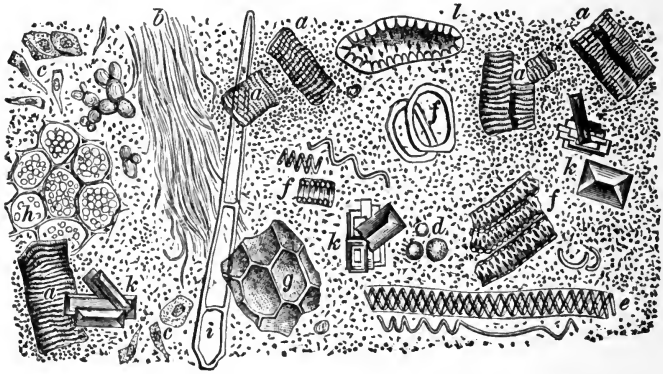


FIG. 40.—Normal Feces. (*Landois.*)

a, Muscle fibers; *b*, tendon; *c*, epithelial cells; *d*, leucocytes; *e-i*, various forms of plant-cells, among which are large numbers of bacteria; between *h* and *b* are yeast-cells; *k*, ammonium-magnesium phosphate.

If blood is present in large quantities "tarry" masses may be found. It also may give rise either to a somewhat diffuse reddish-brown or black color, or may appear as bright red streaks on the surface of formed motions. For tests see pages 297, 344.

"Clay-colored" movements are found in the presence of an excessive quantity of fat, and in the absence of bile pigment from the intestine.

Green motions are due to numerous causes, the

principal ones being the presence of biliverdin as found in abnormal decomposition processes in infants, and of unaltered bile as seen after the administration of calomel.

FIG. 41.—Uncinaria (anchylostoma) duodenale. (A) female, (B) male, (C) eggs, (D) male and female of natural size. (*Greene after Pribram, slightly modified.*)

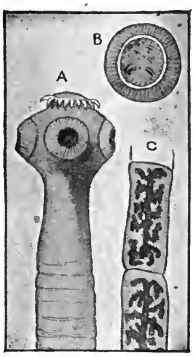


FIG. 42.—(A) Scolex, (B) egg and (C) segments of tenia solium. (*Greene after Pribram and Wood, modified segments.*)

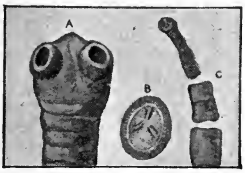


FIG. 43.—Tenia Saginata. (Mediocanellata.) (*Greene after Pribram and Wood.*)

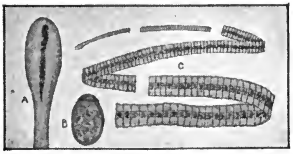


FIG. 44.—Bothriocephalus Latus. (A) head (magnified), (B) egg, (C) head, neck and proglottides. (*Greene.*)

III. **Consistence and form** of a stool may be of importance. For instance, in obstinate constipation scybala or small hard masses of dry feces may be passed; the motion may be ribbon-like as when a stricture of the colon is present; or the mass may be grooved from the action of a rectal polyp.

IV. **Mucus** is recognized as a colorless, slimy coating to the stool, or it may be intimately mixed with the feces if diarrhea be also present.

V. **Pus** shows itself as grayish-yellow masses or streaks. It often closely resembles mucus in appearance.

VI. **Curdled milk** may be voided undigested. It must be distinguished from fat.

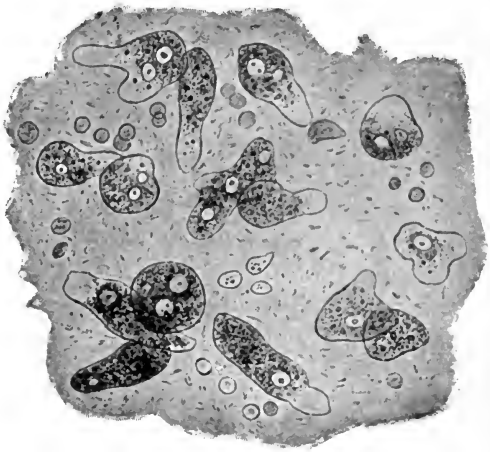


FIG. 45.—Ameba coli. (*Hemmeter.*)

VII. **Gall-stones** are easy of recognition if the stool be first strained.

VIII. **Parasites.**—(1) The *Oxyuris vermicularis* or thread worm appears as fine white thread-like structures, varying from 1/16 to 1/2 inch in length.

(2) The *Ascaris lumbricoides* closely resembles an earth worm; it may reach a length of 8 inches.

(3) The *Anchylostoma duodenale* is rarely seen in the adult form, which is about 1/2 inch in length.

Diagnosis is reached by means of detection of the ova in the feces (Fig. 41).

(4) The proglottides and heads of the *Tenia solium*, *Tenia saginata* and *Bothriocephalus latus* are easily recognized (Figs. 42, 43, 44). The ova, however, of the first two are indistinguishable.

(5) The *Ameba dysenteriæ* has a somewhat greenish tint. The organisms are best seen if a portion of the stool be treated with Muller's fluid, and then sectioned and stained (Fig. 45).

SEROUS FLUIDS.

I. PERICARDIAL, PLEURAL AND ASCITIC FLUIDS.

Excessive collections of serous fluids in the lymphatic spaces and sacs are known as *transudates*. If the etiological factor, however, be inflammatory the fluid is termed an *exudate*.

(a) **Transudates** are as a rule transparent, colorless or straw-colored, alkaline, noncoagulable fluids; at times, however, they may take on a reddish or greenish tinge or may be lactescent in appearance. The specific gravity seldom falls below 1010 or rises above 1015, and the amount of albumin is practically never over 3 per cent.

(b) **Exudates,** on the other hand, are usually yellow in color, the depth of tint depending on the degree of the inflammatory action. They may, however, be hemorrhagic or purulent in character, and at times bile-stained.

The specific gravity is usually over 1015, and the percentage of albumin over three. Not infrequently they coagulate spontaneously on standing.

Chylous exudates are white or milky and contain considerable quantities of fat. *Chyloid* fluids are found in certain cases of carcinoma, tuberculosis, hepatic and cardiovascular disturbances, puerperal septicemia and filariasis (Fig. 35); they contain less fat and accumulate much less rapidly than does the true chylous form.

(c) **Bacteriology of Exudates.**—A few c.c. of the fluid are transferred under aseptic precautions into a flask containing 50 c.c. of nutrient bouillon and the mixture incubated. The technic of further procedures of isolation will be found in works on bacteriology.

It is necessary also to centrifugalize some of the exudate and make smear preparations from the precipitate, as some organisms are very difficult to cultivate by the ordinary methods, especially the gonococcus and the tubercle bacillus.

Inoscopy as recommended by Jousset is applied by coagulating the fluid, thus imprisoning the organisms in the clot, digesting the clot by means of pepsin, centrifugalizing the residue and preparing smears from the precipitate. By this means the organisms are concentrated in a comparatively small quantity of fluid and so rendered more easily detectable.

(d) The **Cytology** of serous fluids, or the examination of the cellular elements, is usually only applied to the nonpurulent forms, since in the latter the bacteria are the more important.

The fluid is first shaken to ensure coagulation, the clear fluid is then centrifugalized, smear preparations are made and stained, preferably by Wright's method (page 329); 100 of the cellular elements are now counted and the relative proportion of the various

types noted. Erythrocytes, leukocytes, and endothelial cells derived from malignant growths may be detected.

It is found that in the presence of a tubercular infection the lymphocytes are usually the predominating cell, while most other organisms seem to attract the polynuclear neutrophiles; also that in nephritic and cardiac secondary exudates an endotheliosis is common.

II. THE CEREBROSPINAL FLUID.

(1) **Technic.**—Place the patient on his left side near the edge of the bed, with the knees drawn up and the back arched. The most rigid antiseptic precautions must be taken.

The needle which should be from 5 to 10 cm. in length and have a lumen of from 1 to 2 mm. is inserted to a distance of 4 to 6 cm. in adults, at which depth the needle should have entered the subdural space, and fluid should exude. The point should be directed slightly upward and inward, 1 cm. to the upper side of the median line, on the level of the junction of the third and fourth lumbar vertebræ.

The needle may be provided with a mandrin which may be removed after the point has penetrated the dura, and a rubber tube (40 cm. long and having a diameter of 2 mm.) is then attached to the end of the needle, and to the peripheral end of the tube is attached a glass tube (12 cm. in length and 2 mm. in diameter) and held vertically, the free end being bent into a short curve whose concavity is directed downward.

(2) **Pressure.**—It will be found now that as the fluid exudes it fills the glass tube which should be held close to the patient and in a vertical position, the free

end being uppermost. The lower extremity should lie on the same level as the spinal canal.

With a tape measure the height to which the fluid rises in the tube. Any rise over 500 mm. should be considered as a pathological increase, providing the patient has "settled down" and is breathing quietly.

Excessive pressures, up to 1000 mm., are usually indicative of intracranial hypertension; but a low lumbar pressure with undoubted evidence of a plus intracranial pressure is sometimes found, as, for instance, when the foramen of Magendie is obliterated.

(3) **Composition.**—Normal cerebrospinal fluid is a colorless, alkaline liquid, varies in specific gravity from 1002 to 1010, and contains no cellular elements or bacteria.

(4) **Bacteriology.**—The tubercle bacillus, the pneumococcus, and the diplococcus intracellularis meningitidis are the commonest organisms found in inflammatory conditions. The fluid should be centrifugalized and smears made from the precipitate, cultures also should be prepared.

(5) **Cytology.**—Erythrocytes and leukocytes may be found. A differential count should be made of the latter, remembering that a lymphocytosis is the rule in tubercular infections.

(6) **Wassermann's reaction,** for details of which reference must be made to larger works, is found in 90 per cent. of cases of general paralysis of the insane and in practically every case of untreated syphilis. It, however, usually does not appear for some weeks after injection.

SPECIAL STAINS AND REACTIONS.

I. **TUBERCLE BACILLUS.**—Prepare and fix a film as already described. Drop carbolic fuchsin solution upon the film so as to cover it without running over; heat so that the stain just steams gently, and continue for quite three minutes but avoid boiling. Drain off the surplus stain, wash well and place for ten seconds in 25 per cent. hydrochloric acid, or 3 per cent. HCl in alcohol. Wash in methylated alcohol until no more red color comes away; rinse in water, then drop an aqueous solution of methylene blue upon the film, allowing it to stain for thirty seconds. Drain off the stain, wash rapidly in water, and allow it to dry in the warm air above

FIG. 46.—Tubercle bacilli in sputum. (*Greene.*)

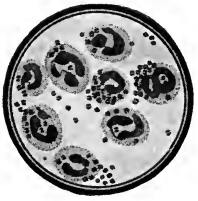


FIG. 47.—Gonococcus. (*Greene.*)

the flame. Mount in xylol balsam. Tubercle bacilli will be stained red by the fuchsin; all other organisms will be colored blue (Fig. 46).

In the examination of sputum or feces the material may be mixed with "*antiformin*," in the proportion of one to three, set aside in a warm room for twenty-four hours, or for three hours in an incubator. Centrifuge and stain the precipitate in the ordinary way.

II. **GONOCOCCUS.**—This organism may be stained by any ordinary method, but for differentiation from other somewhat similar organisms use *Gram's method* as follows. This organism is "Gram-negative" (Fig. 47).

(1) Cover smear with gentian-violet stain, heat to steaming point, wash. (Gentian violet 3 grams, aqua 100 c.c., add 9 c.c. of this stain to 1 c.c. of the following; gentian violet 2 grams, analin gentian oil 9 c.c., absolute alcohol 63 c.c. The above are "stock" solutions; the mixture should be freshly prepared as it will not keep.)

(2) Cover with a solution of 1 part iodin, 2 parts of potassium iodid, and 300 parts water for 1 minute, or until smear is dark brown, wash in 95 per cent. alcohol until blue color ceases to run.

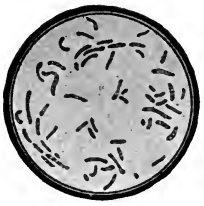


FIG. 48.—Diphtheria bacillus (bacillus diphtheriæ) of Lœffler. Pleo-morphic, non-motile, non-sporogenous, non-flagellate, non-liquefying, non-chromogenic, aërobic, readily cultivated and stained by all methods. F. F. Westbrook has described (a) a virulent type with clubbed extremities and polar granules, (b) granular type, (c) barred type, (d) solid type. (*Greene.*)

(3) Stain with Bismarck brown or safranin for thirty seconds, wash, dry and mount.

Diplococci lying within pus cells, which take ordinary stains but are decolorized by Gram's method and then take the brown counter-stain are to be considered as gonococci.

The following important organisms do not stain by Gram's method ("*Gram-negative*"): Neisser's gonococcus, Friedlander's pneumobacillus, bacillus typhosus, bacillus coil communis, influenza, diplococcus intracellularis mengitidis and numerous others.

III. **KLEBS-LOEFFLER BACILLUS** of Diphtheria.—Take a swab from the suspected pseudomembrane by means of a sterile whisp of absorbent cotton wound on the end of a probe, smear over the surface of a culture tube containing a "slant" preparation of Loeffler's blood-serum mixture, and place in thermostat for twelve

to eighteen hours. Take a smear from the bacterial colonies, fix stain for half a minute in Loeffler's methylene blue, wash, dry and mount (Fig. 48).

Diphtheria bacilli outgrow all other forms of bacteria during the first eighteen hours if inoculated on Loeffler's mixture. This method therefore greatly facilitates their recognition.

IV. **THE SPIROCHETA PALLIDA** is present in syphilitic lesions, both primary and secondary. Films should be prepared from the exudate obtained from the lesion, or a blood film may be prepared and stained after fixing by Leishman's, Giemsa's, or Wright's methods.

(1) *Leishman's Method.*—A solution of eosinate of methylene blue in methyl alcohol is used as a combined stain and fixative. Stain film for two minutes then add a quantity of distilled water double the volume of the stain. This differentiates the ruby tint of the nuclei from the blue of the protoplasm. Allow to remain for five minutes, wash in distilled water, dry and mount.

(2) *Giemsa's Method.*—Fix smears for fifteen minutes in alcohol, stain for at least thirty minutes in the solution, wash, dry and mount. The slide should be fully immersed in the solution which should be freshly prepared.

Better results may be obtained by diluting Giemsa's stain with 20 parts distilled water and allowing the specimen to remain immersed for at least twelve hours.

(3) *Wright's Stain* (see page 329), or *Indian Ink* may also be used (Fig. 36).

V. **CEREBROSPINAL MENINGITIS** (Diplococcus Meningitidis Intracellularis).—Films are prepared in the same manner as in the case of the blood, from the

fluid obtained by lumbar puncture. The stain employed is made up as follows:

Mix 20 c.c. of water with 8 drops of saturated methylene-blue solution, then add 45 to 50 drops of carbol-fuchsin stain. The films are stained for five minutes; the cocci alone will appear blue, all else being red. It is a *gram-negative* organism.

VI. **FRANKEL'S PNEUMOCOCCUS** may be stained by any of the ordinary bacterial stains. It is a "*Gram-positive*" organism (Fig. 49).

FIG. 49.—Diplococcus Pneumoniæ. (Frankel-Weichselbaum) and the pneumo-bacillus of Friedlander. Upper segment shows former, lower segment shows latter. (*Greene.*)

VII. **RINGWORM.**—Soak the portions of hair in ether for five minutes to remove fat. Fix their distal ends on to a slide by means of a little melted paraffin.

Skin, after being soaked in ether, may be fixed with egg-albumin.

A few drops of 10 per cent. caustic potash are now poured on the slide and allowed to remain for ten minutes. Wash with ether, mount in 50 per cent. glycerin solution, and examine under a high power for mycelial threads and spores. They may also be stained by Gram's method, modifying the time to five to ten minutes in the carbol genetian violet and to half a minute in the iodin. Clear with anilin oil, wash with xylol, and mount in balsam.

VIII. **WIDAL'S SERUM REACTION** is of the greatest value in the diagnosis of typhoid fever; the two methods in use are:

(1) *The Macroscopic.*—Take 0.1 c.c. of patient's serum, add 2.4 c.c. physiological salt solution thus ob-

taining a dilution of 1–25. Then take 0.5 c.c. of a formolized culture of B. Typhosus and 0.5 c.c. of the diluted serum, thus obtaining a dilution of 1–50. Mix thoroughly, incubate at 37° C. for one hour, or allow to stand twelve hours in a warm room. A clear supernatent fluid indicates the presence of a positive reaction, and turbidity a negative result.

(2) *Microscopic.*—Allow a large drop of blood to dry on a piece of glazed paper, then add to this a platinum loopful of distilled water; a certain amount of the serum will pass into solution in the water. When this becomes a faint straw color the desired result is obtained, (a dilution of 1 to 20).

Take now a loop of a twelve-hour broth culture of the typhoid bacillus, add to it and mix well 1 drop of the diluted serum (1 to 20), and make a hanging-drop preparation. In the presence of a positive reaction, agglutination and cessation of mobility, should take place within fifty minutes, in a dilution of 1–40.

Pure serum may be obtained by drawing blood into a small glass tube, sealing, and allowing coagulation to take place after which the end may be broken off the tube and the serum obtained.

IX. **THE DIAGNOSIS OF TUBERCULOSIS.**

(1) *Koch's old method* depends on the febrile reaction produced by the hypodermic injection of "old tuberculin."

In cases of suspected tuberculosis, a dose of 0.001 c.c. of old tuberculin is given by hypodermic injection. For children above five give half this quantity and a quarter of the dose or less for children under five.

A positive reaction will follow within forty-eight hours, shown by rise of temperature 1° to 2° F., malaise

and constitutional disturbances, if the patient be the subject of tuberculosis.

Should no reaction follow, a second dose of 0.002 c.c. is given on the third day. If after a third injection of 0.005 c.c. no reaction follows it may be concluded that the patient is not tuberculous.

The following methods have been recently introduced:

(2) The *Cutaneous Reaction* (*Von Pirquet*).—The skin of a tuberculous patient should be gently scarified by means of a special scarifier, and tuberculin be applied to the scratched surface. A reaction should occur within twenty-four hours, as shown by the appearance of a vivid red papule. These signs disappear in a few days, leaving a small area of pigmentation which may remain for weeks. A "control" should always be made.

Von Pirquet recommends the use of Koch's original tuberculin in 25 per cent. solution, the diluting medium consisting of 1 part of a 5 per cent. solution of carbolic acid and glycerin, and 2 parts of normal saline solution.

The reaction is often not always given in the presence of a febrile state.

(3) *The Cutaneous Reaction* (*Moro*).—For this reaction an ointment consisting of equal parts of "old tuberculin" and lanolin is employed.

A piece the size of a pea is rubbed into the clean skin, with the finger covered with a rubber "cot." A positive reaction in the form of a papular eruption should appear within twenty-four to thirty-six hours in tuberculous patients.

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