Auscultation and percussion / by Frederick C. Shattuck.

AUSCULTATION AND PERCUSSION

SHATTUCK

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— AND —

PERCUSSION.

— BY —

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INTRODUCTION.

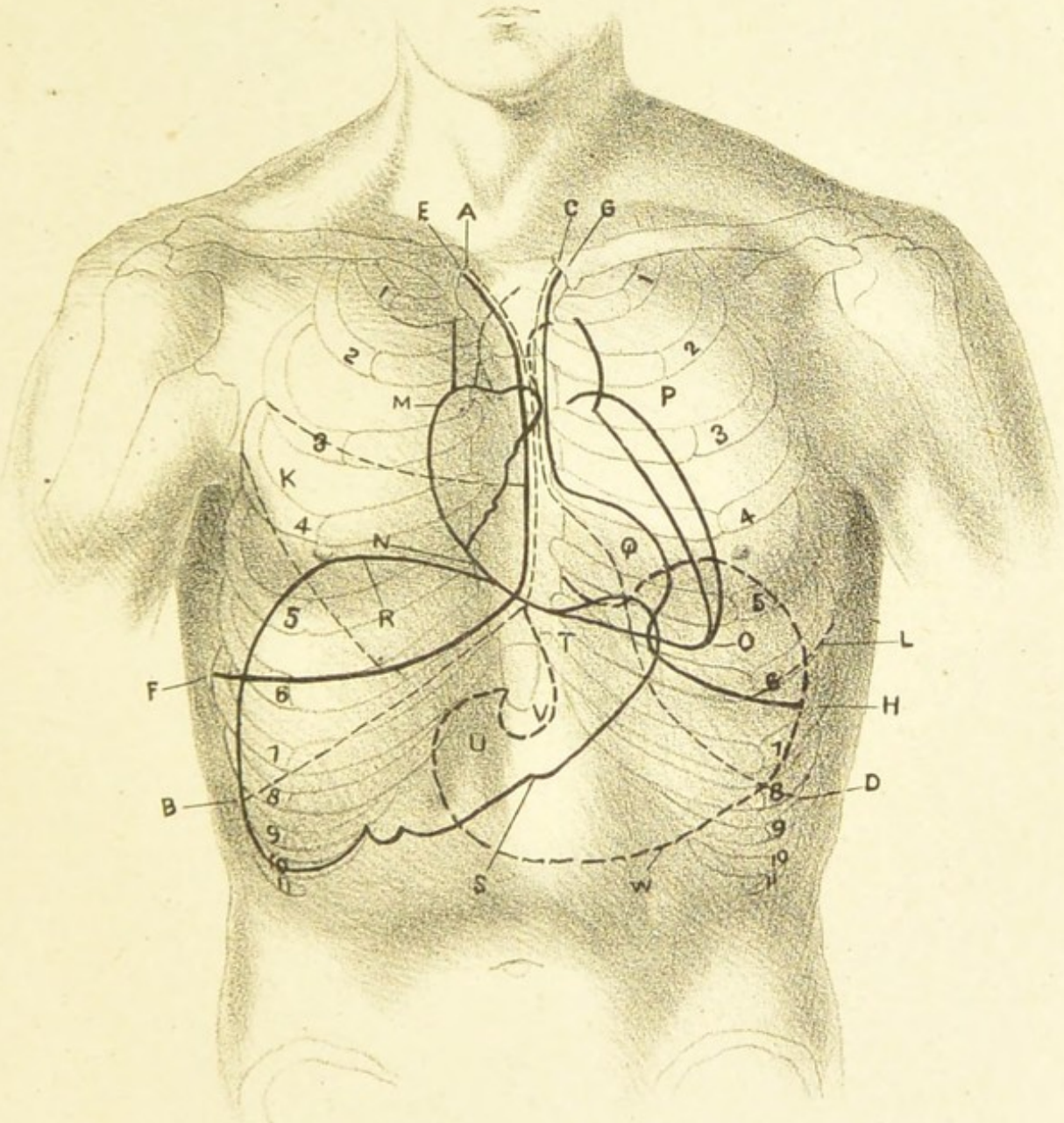
The primary object of the physical examination of the chest is the attainment of a knowledge of the physical condition of the important organs contained within it. Not until this knowledge has been obtained are we in a position to determine with all possible accuracy the cause or causes which have led to, or which underlie, those deviations from the normal physical condition revealed to us by the methods about to be described. Essentially the same physical condition may be encountered in widely different diseases—*i. e.*, as the result of quite different causes—to discriminate between which the family and previous history of the patient; the influences to which he has been exposed; the symptoms which he presents, with their mode of onset, progress, and sequence; a careful examination of the patient as a whole, of his other organs or systems of organs; and, finally, a thorough knowledge of the natural history of general and local morbid processes; must all likewise be duly noted and weighed. In a word, percussion and auscultation—the two chief modes of thoracic physical exploration—lead directly to the detection of diseased conditions, only indirectly to that of diseases.

Health precedes disease; it is, therefore, incumbent on us to master healthy conditions first. Perfect familiarity with the anatomy of the healthy chest and its contents, with the structure of each separately and the mutual relations of all is of vital importance. Furthermore, the variations of size and relation within the limits of health which may occur in the same person at different periods of life or under different conditions—as in activity and repose—as well as the limits of normal variation in different persons, must be

known. Finally, the physiology of respiration and circulation must be thoroughly understood. The possession of most of this knowledge is here presupposed, inasmuch as this series of Manuals is intended rather for physicians than for younger students. We may, consequently, now go on to consider the special methods of physical exploration and the results which they may be made to yield in health and disease. At the same time the accompanying plates, after Weil, may serve to refresh the memory as to the space occupied by the thoracic viscera and their mutual relations.

These methods in the order in which they should be practiced are: Inspection, Palpation, Mensuration, Percussion, Auscultation, Succussion.

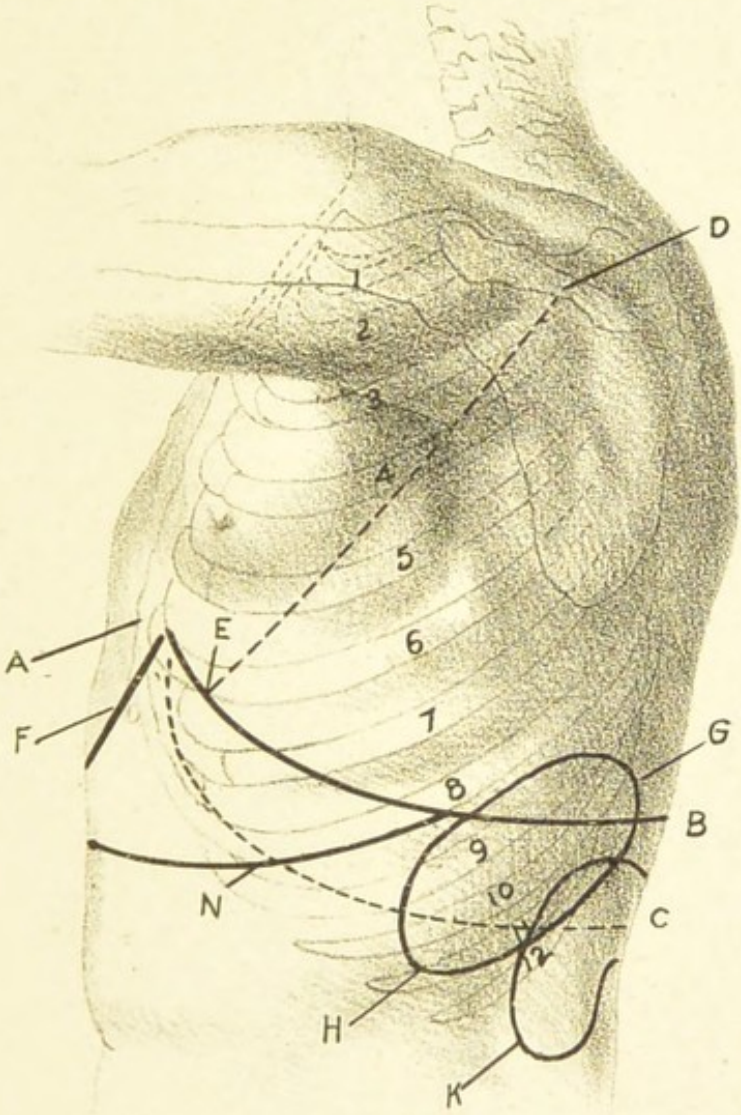
PLATE I.



Anatomical Borders—Anterior View. (Weil).

A B Border of the right pleural sac.
C D Border of the left pleural sac.
E F Edge of the right lung.
G H Edge of the left lung.
I Upper incisura interlobularis of the right lung.
K Lower incisura interlobularis of the right lung.
L Left incisura interlobularis.
M N Right border of the heart.
N O Lower border of the heart.
P O Left border of the heart.
Q Sinus mediastinocostalis, situated between the edge of the pleura and incisura cardiaca of the anterior border of the left lung.
R Highest point of the portion of liver covered by lung.
S Lower edge of the liver.
T Cardiac portion of the stomach.
U Pyloric portion of the stomach.
V Small curvature of the stomach.
W Greater curvature of the stomach.

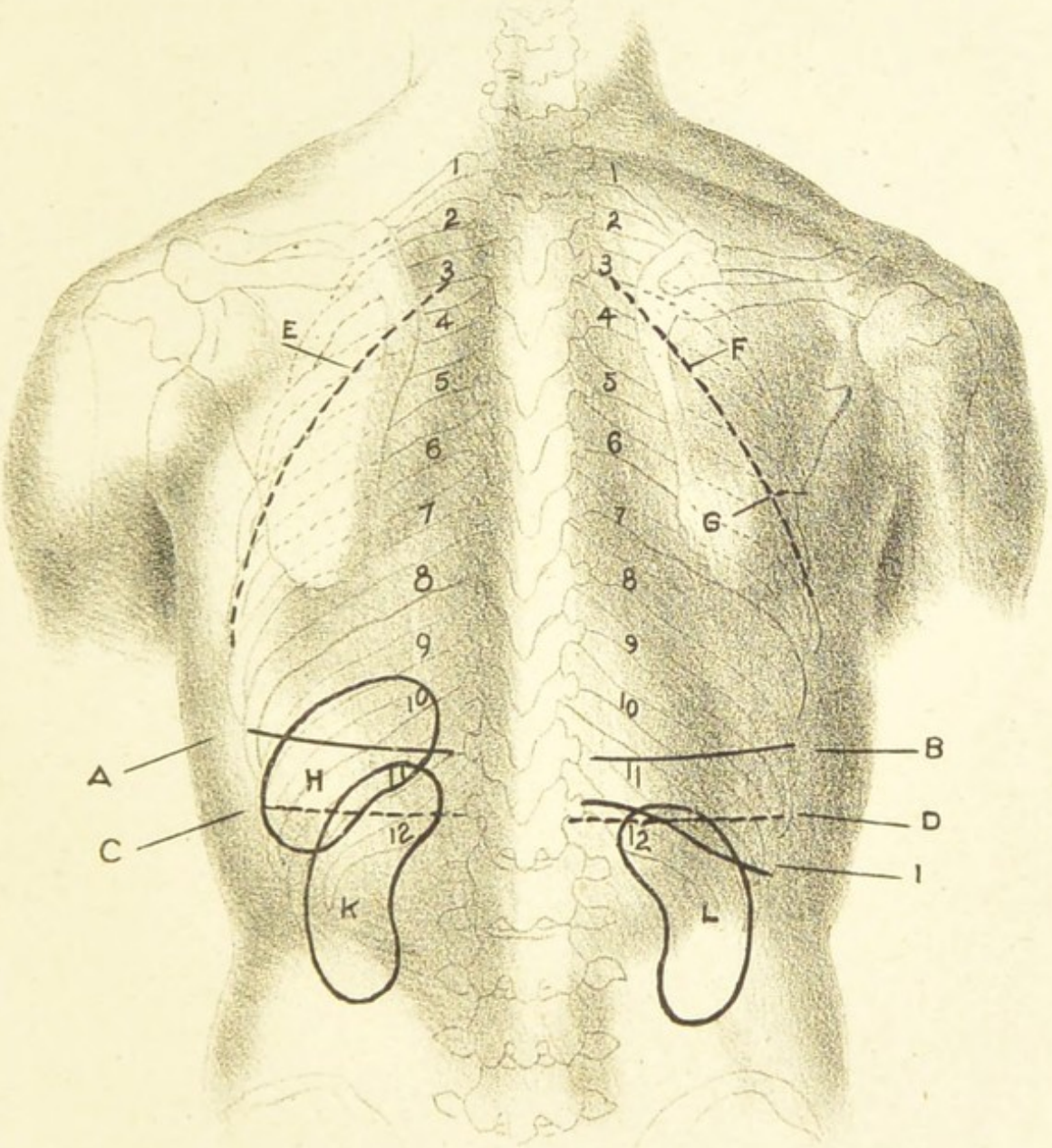
PLATE II.



Anatomical Borders on Left Side. (Weil).

A B Lower border of the left lung.
A C Lower boundary of the pleura.
D E Incisura interlobularis.
F Edge of the left lobe of the liver.
H G Anterior and posterior ends of the spleen.
K Kidney.
N Stomach in moderate distention.

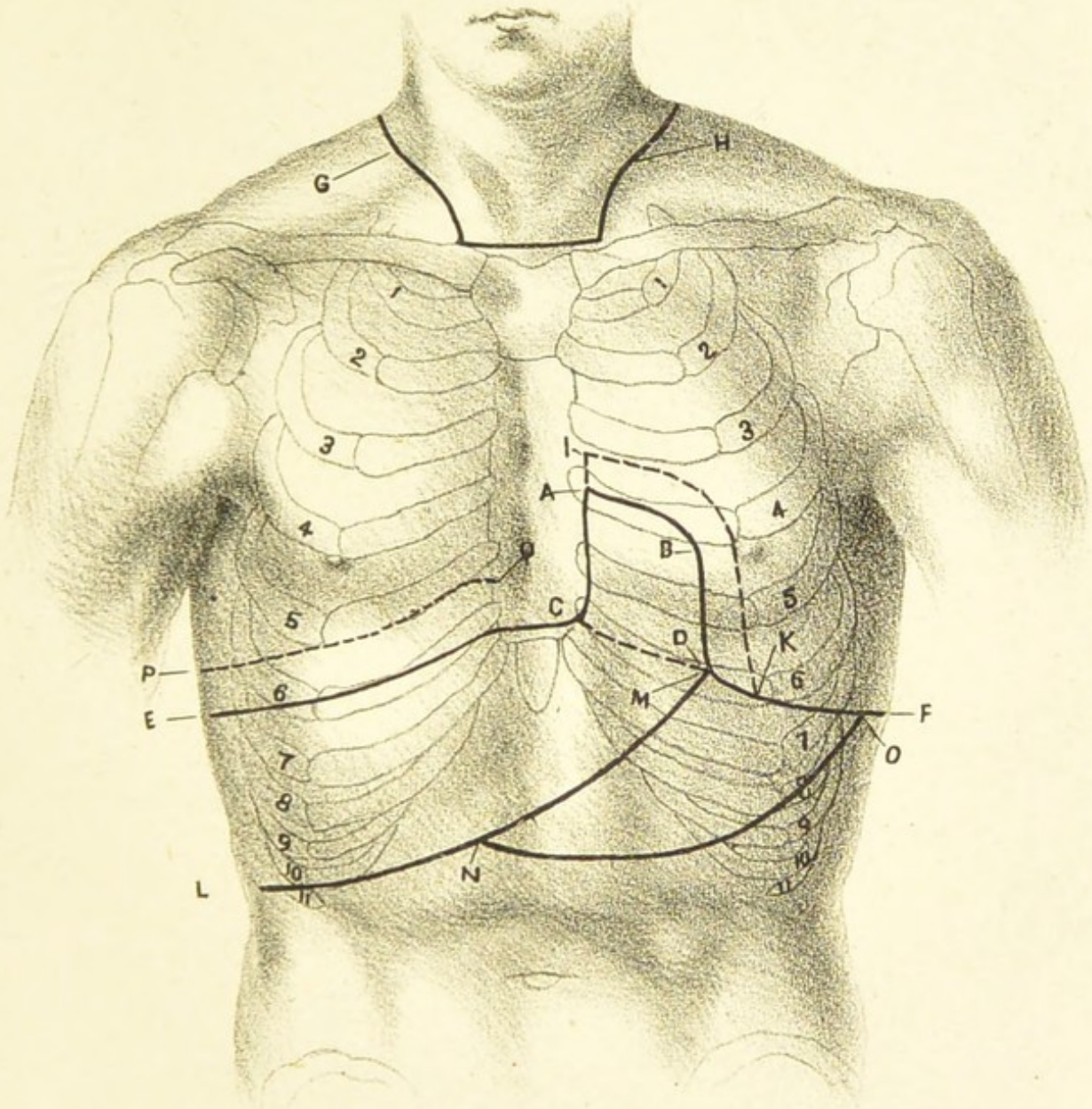
PLATE III.



Anatomical Borders—Posterior View. (Weil).

A B Lower borders of the lungs.
C D Lower borders of the pleuræ.
E F Incisuræ interlobulares.
G Point where the right incisura divides into the sulc., interlob., dext, super., and infer.
H Spleen.
I Lower border of the liver.
K L Kidneys.

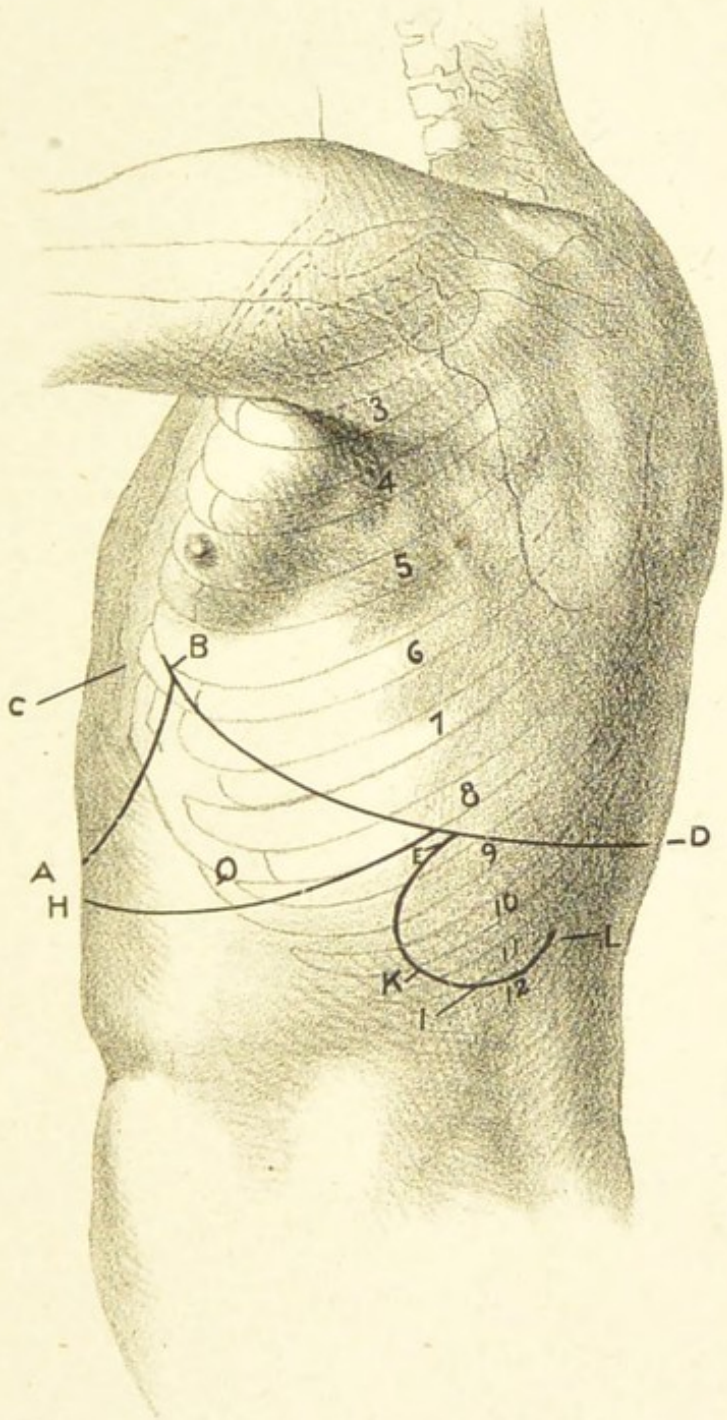
PLATE IV.



Percussion Borders in Middle Age. (Weil).

A B C D Area of cardiac flatness.
A I K Area of cardiac dulness.
C E Lower border of right lung.
D F Lower border of left lung.
G H Upper borders of lungs.
P Q Upper border of hepatic dulness.
L M Lower border of hepatic flatness.
N O Lower border of stomach in moderate distention.

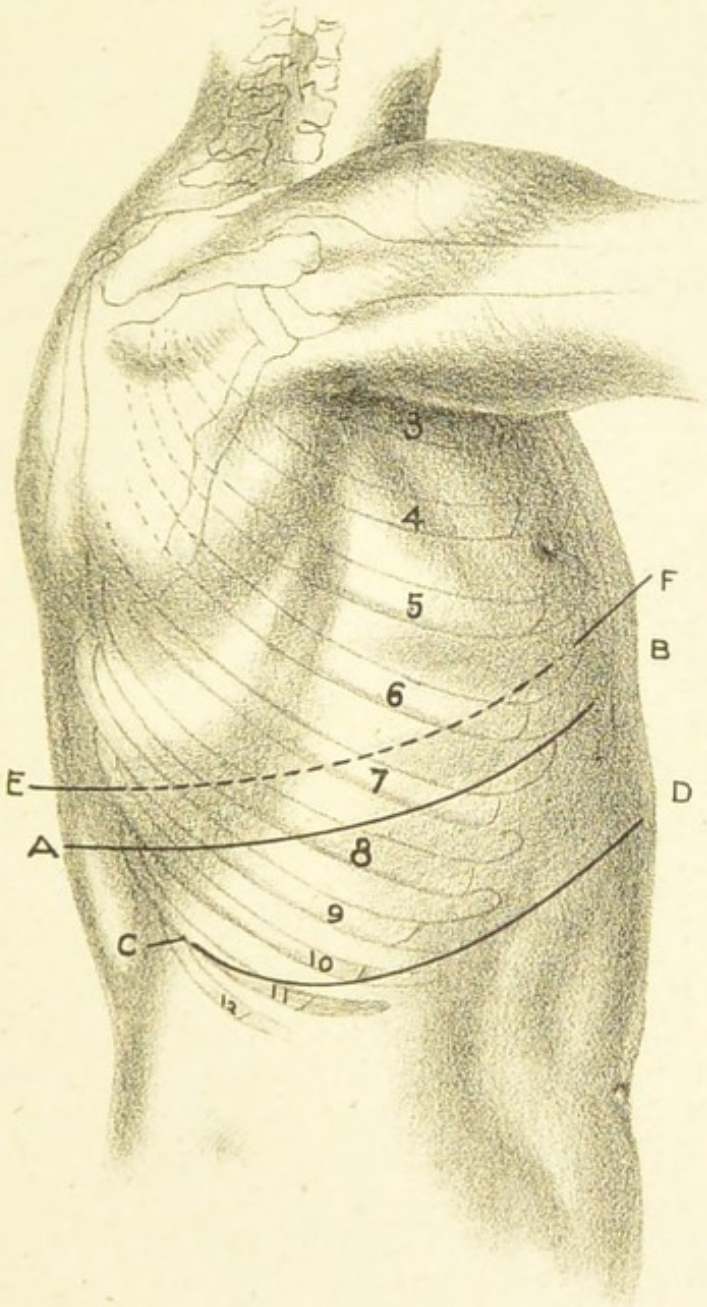
PLATE V.



PERCUSSION BORDERS ON LEFT SIDE. (WEIL).

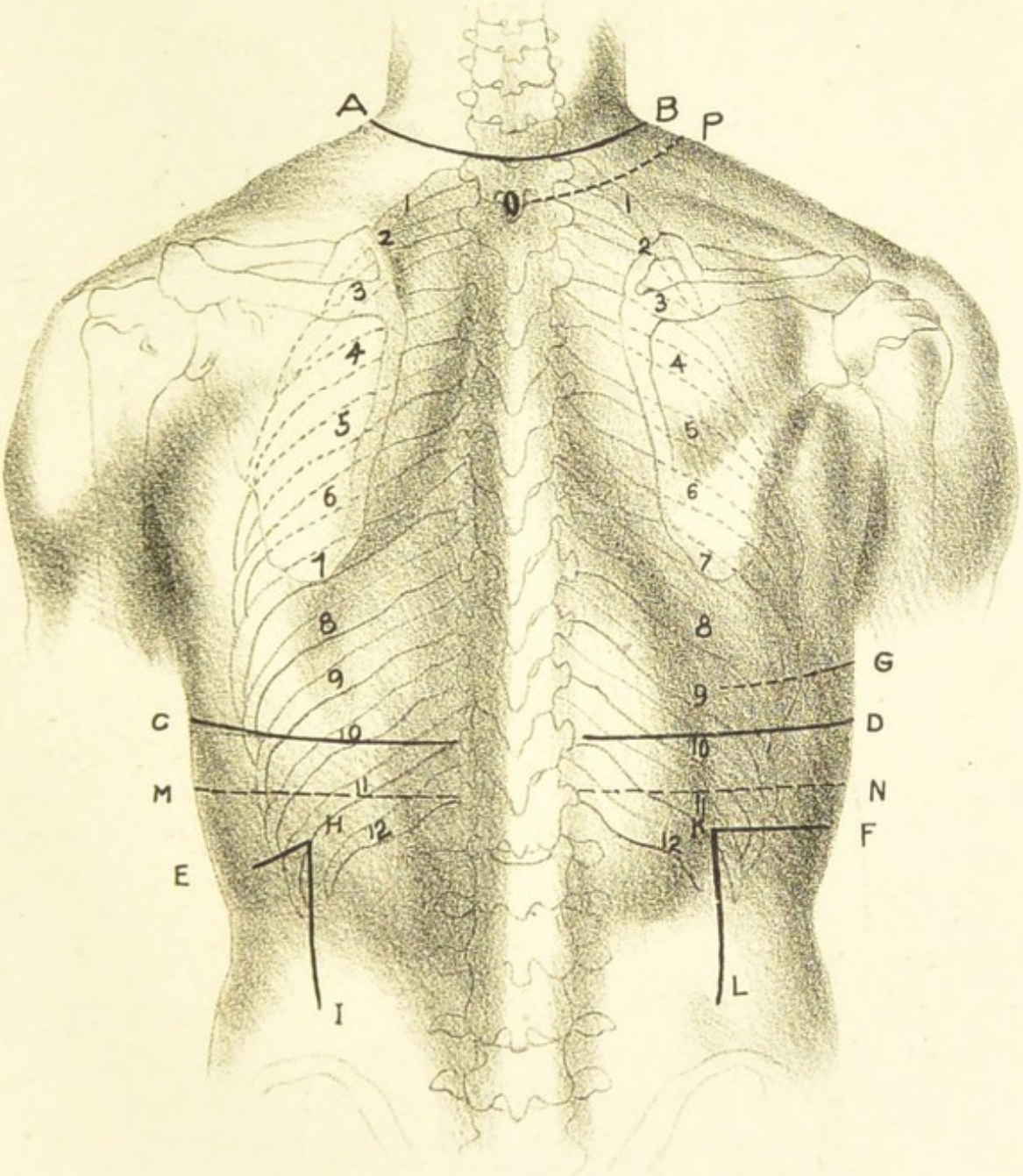
A B Lower border of hepatic flatness.
C D Lower border of left lung.
E I L Splenic dullness.
Q Lower border of stomach.

PLATE VI.



PERCUSSION BORDERS ON RIGHT SIDE. (WEIL).

A B Lower border of the right lung. C D Lower border of hepatic flatness.
E F Upper border of hepatic dullness.



Percussion Borders on the Back. (Weil).

A B Upper border of lungs.
C D Lower border of lungs.
E H Lower border of spleen.
K F Lower border of hepatic flatness.
H I K L Outer borders of the so-called renal dullness.
M N Lower borders of the lungs in deepest inspiration.
O P Shrinkage of upper border of lung in phthisis.

PART I.—THE LUNGS.

CHAPTER I.

PHYSICAL SIGNS IN HEALTH.

1. Inspection.—The patient should stand at ease, stripped to the waist, with the heels together and the weight equally borne on both feet, facing the light. If he is confined to the bed, or unable to sit up, similar precautions should be observed; the aim being to avoid the artificial production of any disparity between the two sides of the chest.

The facies of the patient, his general nutrition and muscular development, cyanosis and œdema or their absence, are to be noted; also the size, form, and symmetry of his chest; the rate and character of his breathing, both as a whole and relatively on the two sides; the seat and character of the cardiac movements, and their area of distribution; the presence or absence of abnormal pulsations in the chest or neck, and arterial trunks of the upper extremities.

2. Palpation.—The chief application of this method of examination is in confirmation of the results of auscultation. The vibrations of the voice are in most men transmitted to the chest walls unless this

is covered by a thick fat layer; in women and children they are often not so transmitted unless the voice be raised as in singing or crying. The tension of the chest wall has an important influence on the transmission of vibrations originating in the lungs or upper air passages to the parietes; thus the tactile vocal fremitus is more marked, other things being equal, if the wall is relatively tense than if it is relatively lax. Palpation is also of service in localizing the apex of the heart.

3. MENSURATION is less used than any of the other methods except succussion; it may be made to yield information as to the extent of respiratory excursion of either or both sides, and symmetry or deviations therefrom may be more accurately determined by the stethometer than by the unaided eye.

4. PERCUSSION.—This important method of examination is best practiced simply with the fingers; a great variety of hammers and pleximeters has been devised, but they have no notable advantages over the fingers in ordinary clinical work. It is possible for one who percusses very much to excite periostitis of the phalanx used as a pleximeter; and, in class demonstration, louder sounds can be elicited by many through artificial than through natural means.

To obtain satisfactory results certain precautions are absolutely necessary, and some practice is needed to acquire a good technique. The finger which is struck should be firmly and accurately applied to the

part, and the striking finger should deliver a perpendicular and quick blow, not remaining in contact with the other. The manner in which the hammer of a piano strikes the wire may be regarded as the type of percussion. Forcible percussion is rarely necessary or desirable. A gentle blow properly delivered yields a better note, does not hurt or alarm the patient, especially important in children, and elicits the note belonging to the part struck. A hard blow tends to set more or less distant parts in vibration, particularly if the chest wall is thin and elastic. In determining the line of separation between a solid and an air-containing body, far better results are obtained by gentle than by forcible percussion. The idea of many authorities, that the dullness of a deep-lying solid body is best brought out by hard percussion, is believed by the writer to be fallacious. It is to be remembered that there is no absolute standard .of resonance to which all chests must conform. Speaking broadly, each person furnishes his own standard. Consequently similar points on the two sides should ordinarily be struck successively and with equal force. Percussion should usually also be in straight lines, especially when it is desired to map out accurately a dull area. Finally, the lessons of the regional anatomy of the chest are to be borne in mind, and one standard is not to be applied to the chest as a whole. Percussion results which are normal in one portion of the thorax are abnormal in another.

Percussion is either non-resonant or resonant. Non-resonance is termed flatness, and signifies the entire absence of air in the part struck. Dullness is a relative term, and denotes diminished resonance. Resonance implies the presence of air, differs greatly in degree, and, as encountered in the chest, also differs in kind. It is, consequently, desirable to analyze resonance in order that we may be the better able to appreciate its shades and modifications. The elements into which resonance may be resolved are *intensity*, *pitch*, *quality*, *and duration.*

Intensity denotes simply the loudness of the note, and may be made to vary with the force of the blow and the skill with which it is given. The thinner and more elastic the chest wall, the larger the amount of air, and the thicker the layer of tissue containing it beneath the part struck, other things being equal, the greater the intensity of the note.

Pitch also varies in different normal chests, and in different parts of the same chest. We speak of it as being either high or low. It is relatively low over healthy lung, and is apt to rise with diminution in intensity or change in quality. A well-trained musical ear is an advantage in the detection of shades of pitch, but is not absolutely necessary.

Quality is that which gives to the sound its peculiar character and chiefly distinguishes one sound from another. In physical examination we meet with only two qualities—the vesicular, that which is produced

by percussion over normal lung; and the tympanitic, produced over relatively large bodies of air, as in the stomach or intestine. The quality is more or less marked in different persons and in different parts of the same persons, in accordance with obvious laws. A pure tympanitic note is rarely met with over the healthy chest; but a mixture of the two qualities is not very rare, and is termed, after Flint, vesiculo-tympanitic. This mixed note is heard in health chiefly over the trachea and primary bronchi, and over the chest of a child which has temporarily somewhat over-distended its lungs by crying. When any doubt is felt as to the quality of a note, it is well to seek out typical notes of the two qualities, which can nearly always be found somewhere in the chest or abdomen, even of those with advanced disease. Intensity and quality are in no way related; but low pitch and vesicular quality are closely associated.

The *Duration* of the note is nearly related to the pitch, and is, by an unmusical ear, sometimes more easily appreciated than is the latter. The higher the pitch, the shorter the note.

In percussing, it is also well to note the sense of resistance under the finger; the more solid and less elastic the part percussed, the greater is the resistance.

5. AUSCULTATION.—This is spoken of as *immediate* when practiced with the ear applied to the chest wall, with or without the intervention of a towel or

clothing; *mediate* when a stethoscope is used. The former is a rougher method, but, especially when the ear is held to the naked skin, may be made to yield valuable results. A dirty patient and the female sex preclude this method to a certain extent; but immediate bears somewhat the same relation to mediate auscultation that the low does to the high power of the microscope. A good auscultator is not dependent on the stethoscope, which, however, apart from its convenience and æsthetic qualities, is of great value in bringing out and localizing fine shades and changes. The binaural stethoscope is the best, and that which is generally used in this country; but the straight instrument is the more convenient to carry about and one should be able to use all kinds.

The breath sounds vary not only in different persons but also in the same person; in the latter case to a less extent comparatively on the two sides, to a greater extent in different regions of the chest. As in percussion, so here each person must furnish his own standard to some degree, and similar points on the two sides should, as a rule, be successively examined.

The sounds produced in ordinary superficia respiration are apt to be so feeble that we wish to induce forced breathing in order to intensify them. But care must be exercised that in forcing the breathing we do not alter its rhythm, and much patience must often be practiced to attain this result, the ausculta-

tor illustrating the desired method himself. Remember that expiration is normally a passive act; patients must be taught to *let go*, not to expire forcibly.

Auscultation enables us to study the sounds produced by the air entering and leaving the air passages from the larynx to the base of the chest, and also those produced by the voice, spoken and whispered. These sounds are quite different as heard over the trachea and over a distant portion of the lung. Over the former we hear normally a kind of respiration—called bronchial—which may serve as a type of a respiration often heard in abnormal conditions over the lungs themselves. But, before going further, we must analyze the respiratory murmur. We divide it in the first place into the inspiratory and expiratory acts; and each of these acts is then studied with reference to intensity, pitch, and quality; the relative duration of the two, or the rhythm, being also noted.

Tracheal Respiration.—Inspiration is of variable, but generally marked, intensity, high pitch, and bronchial or tubular quality. Expiration is also of variable, but usually rather greater, intensity, high pitch, and tubular quality; both sounds are long, expiration somewhat the longer of the two, and they are separated by a slight pause.

Tracheal Voice-sounds.—These are intense, concentrated, with marked resonance and fremitus.

Pulmonary Respiration.—To listen to this in its

typical form one should select a point as far removed as possible from the trachea and primary bronchi, the lower back, for instance. By bearing in mind the broad principle that the nearer we approach these large tubes the more bronchial in quality does the respiration normally become, much confusion may be avoided. As in percussion, so in auscultation, we have to deal with practically only two qualities; the vesicular, that which is proper to healthy lung tissue; and the bronchial, such as is heard normally over the trachea and, in pathological conditions of the chest, is especially characteristic of condensation of the lung. As we shall see later, there are sub-varieties of bronchial respiration which are usually pathological. Mixed respiration, called broncho-vesicular, is also frequently heard; in health over and near the trachea and large bronchi in front and behind, in various situations in disease. There is normally a disparity between the two sides of the chest, especially in the upper portions; the murmur being less purely vesicular over the right apex—and, indeed, over the whole right upper lobe—than over the left. Fixing these principles and facts in mind let us then proceed to analyze the vesicular murmur.

Inspiration is very variable in intensity, age having an important influence; in children it is especially loud, and may be somewhat harsh. It is, in general, louder the thinner and more elastic the chest wall. It should be always remembered that intensity is in

no way distinctive; marked feebleness may go with a purely bronchial as well as with a purely vesicular quality. This is a stumbling-block to many students. The pitch is low; the quality soft and breezy – vesicular; the duration is nearly as long as the inspiratory act, which is immediately followed by :—

Expiration. The sound produced by this act is often inaudible, especially at points far removed from the large tubes and in highly developed lungs; when heard, it is of variable but relatively slight intensity, still lower in pitch than inspiration, rather blowing in character, of short duration—often not more than a fifth as long as inspiration.

The Thoracic Voice-Sounds vary in different parts of the chest along the same lines as the breath-sounds, and in different persons, much as does the tactile vocal fremitus. The voice, whether spoken or whispered, produces, as a rule, but little effect in women, and also in children, unless they cry. Indeed, in children, diagnosis must often rest largely upon the auscultatory phenomena of crying.

Over a thick layer of normal lung the loud voice is appreciated as a distant and diffuse resonance and fremitus, and it is difficult or impossible to distinguish the spoken words; the nearer we approach the large tubes the more intense and concentrated are the sounds. The whispered voice corresponds in most cases to a forced expiration; is inaudible, or very faint and distant, over the lower portions of the chest,

becoming louder, more concentrated, and of higher pitch, near the large bronchi. It is often of great value to study the whispered voice for diagnostic purposes.

CHAPTER II.

PHYSICAL SIGNS OF DISEASE.

1. INSPECTION.—Too much importance is not to be attached to changes in form and symmetry, especially if these are slight. The long shallow chest, with wide intercostal spaces, vertical sternum, and feeble muscular development—the paralytic thorax—is not so suggestive of phthisis as was formerly supposed. The barrel-shaped thorax which moves as one piece in respiration, has been so much and so carefully described as to lead many students to believe its absence excludes emphysema—a serious error. Marked examples of this form of chest are, at least in the experience of the writer, not common. Its presence is more distinctive than its absence, and the superficial appearance varies a good deal with the amount of fat and muscle covering its owner. In thin persons the prominence of the accessory muscles of respiration, and the rounded high shoulders, are striking; in the well nourished, one notes the shortness of the neck, depth and shortness of the thorax as a whole, the slowness of movement, and the turgescence of the general integument. The pigeon-breast in its several varieties is always characterized by flattening of the sides, with prominence of the sternum and also, perhaps, of the upper costal cartilages; it is in the angle formed by the

sternum and the cartilages that the essence of the variations usually lies. This form of chest is suggestive of rickets, with which severe bronchitis and atelectasis, or broncho-pneumonia, were probably associated, in early life. It throws light on the previous history rather than on the present condition. The distortion of the chest due to angular spinal curvature may be very great, and lateral curvature may, primarily, cause a great disparity in the two sides. But lateral curvature is often secondary to such affections as chronic pleurisy or fibroid phthisis, which seriously cripple one lung; in these cases the contraction of the side with close approximation of the ribs, the droop of the corresponding shoulder, and the double curve of the vertebral column, at once strike the eye.

A quickened or labored respiration shows either that emotional causes are at work, or that blood-aëration is accomplished with difficulty or imperfectly; if imperfectly, cyanosis is also present to a greater or less degree. A disparity between the movements of the two sides, seen better when the respiration is forced, indicates that the expansion of one lung is more or less interfered with from some cause, and that the cause is, at least mainly, unilateral. The eye, too, often puts the careful observer on the track of modifications in the circulatory apparatus, and even may suggest a shrewd guess as to their nature.

2. PALPATION and MENSURATION call for no extended remarks here. Of the two palpation is far

the more important, enabling us to confirm or reject suspicions of unilateral or local increase or decrease in the transmission of vibrations from the larynx to the chest wall; often to appreciate with the hand the presence of secretion in the larger bronchial tubes, or of roughening of a serous membrane; and yielding valuable information as to the seat, character, and rhythm of abnormal pulsations in the neck, chest, abdomen, and peripheral vessels.

3. Percussion.—As the physical condition of the chest or its contents becomes altered by disease or accident, the percussion note proper to the affected individual is frequently changed, either over the thorax as a whole or locally, according as the changes are general or circumscribed. The intensity of the note, other things being equal, is proportional to the amount of air beneath the part struck and the thickness of the layer of tissue containing it. The quality of the note, on the other hand, is, speaking broadly, rather indicative of the manner in which the air is held in the part. Flatness, absence of resonance, means absence of air, and is not found normally above the diaphragm except on very gentle percussion over a limited area near the heart's apex, the superficial cardiac space. But dullness—diminished resonance—is a purely relative term, and may approach closely to flatness on the one hand, or deviate very little from the normal resonance of the part on the other. It is indicative of a diminution from one

or more of many causes of the proper amount of air beneath the part percussed, or of an increase in the solids, or of both; and is, consequently, associated with a great variety of conditions and diseases. According to its degree and the skill of the examiner, dullness is very easy or very difficult of appreciation. If the note is dull the intensity is less, the pitch higher, and the duration shorter; the quality may remain vesicular, though of less intensity, or it may lose entirely its vesicular quality and become tympanitic; in other words, dullness does not determine quality.

Any resonance which is non-vesicular must necessarily be tympanitic; but the two qualities may be mixed, giving rise to the note called vesiculo-tympanitic. As has been already stated, there are regions of the normal chest over which this combined note is often found; elsewhere, however, it is apt to be a sign of some deviation from perfect health. The pitch rises as the tympanitic element comes to the fore. A pure tympanitic resonance over the chest or a part thereof is practically always indicative of an abnormal condition, whether it arises within the thorax or is conducted from without—the hollow abdominal viscera. It is the quality which is especially distinctive of tympanitic resonance; the intensity may be great or slight; the pitch varies, but is usually higher than normal vesicular resonance. There are two sub-varieties of the tympanitic resonance, the cracked-pot and the amphoric. The former can be imitated by clasp-

ing the hands together, palm to palm, in such a way as to leave between them an air space communicating in one direction with the outside air, and then by striking the back of the under hand sharply against the thigh; the air between the thumbs is thus forcibly compressed and produces a "chink" in its escape under pressure through the narrow opening. To elicit this sign tolerably forcible percussion is often required, and it may be necessary to hold the ear or the chest-piece of the stethoscope near the open mouth of the patient. If the chest is very elastic or the lungs are somewhat over distended with air, both of which conditions may be filled in a crying child, this sign may be met with in a healthy chest. It is, however, commonly heard only over a relatively large air-space, as, for instance, over a pulmonary cavity, but is also sometimes heard in pleurisy and pneumonia; in the former over the condensed lung just above the level of the fluid, in the latter in the immediate vicinity of complete consolidation. The illustration given above affords a hint as to the mechanism of its production. A sound closely resembling the cracked-pot, and liable to lead to error, may arise on a hairy chest, as well as on a chest which is not hairy if the pleximeter is not accurately applied to the skin. These sources of error are far more common if artificial pleximeters are used. Wetting the hair and a careful technique will eliminate them. Amphoric resonance is that which is obtained by striking on an empty cask or

pitcher, and may be imitated by percussing the cheek distended with air, while percussion on the undistended cheek gives rise to a simple tympanitic note. Amphoric as contrasted with tympanitic notes are metallic, and are followed by a metallic echo which prolongs their duration and raises their pitch. Even more than the cracked-pot, this sound is indicative of a large air space such as is furnished in the chest only by lung cavities or by air in the pleural sac. But other conditions are needed; the cavity must be, according to Wintrich, at least six centimeters in the direction in which the force is applied; and it must also be superficial, bounded by homogeneous walls, free from an excess of fluid contents, and covered by a chest-wall thin enough not to be too resistant. If the cavity communicates with a large bronchus, the note is intensified by opening the mouth, but such communication is not necessary for its production. It is, therefore, seen that while the presence of this sign is very distinctive, its absence is very far from excluding cavity formation. It is over the upper fronts that the conditions for the production of the cracked-pot and amphoric notes are usually met; the former is rarely, if ever, found over the lower lobes, the latter in this situation only in cases of pneumo-thorax.

4. Auscultation.—The auscultatory signs of disease fall into two main classes; those in which the breath and voice sounds, one or both, are changed from those proper to the person and the part in

health; and those sounds called "adventitious," not heard at all in perfect health. The change of the first class are as follows:

Vesicular Respiration may be *increased in intensity* without any change of pitch or quality, and is then often called supplementary or puerile; supplementary because when heard in adults it indicates that the part over which it is heard is doing extra or supplementary work; puerile because it resembles the respiratory murmur normally heard over the chest of a child. It is not an uncommon error for students to mistake puerile for bronchial respiration, an error easily avoided by paying careful attention to the pitch. It may perhaps be stated here as well as elsewhere, that when one is in doubt as to the quality of respiration it is often well to study the type qualities, the bronchial over the trachea, while the vesicular can usually be found at some portion of even the most diseased chest. By comparing the doubtful respiration with the types its true characteristic can generally be made out.

Diminished intensity is incident to a great variety of conditions. In simple diminution the quality remains vesicular or, from very feebleness, cannot be determined. The sign may be due to any affection of the chest wall, the diaphragm, or the abdomen and its contents which, often by involving pain, induces shallow respiration; to thickening of or slight accumulation in the pleura; to any morbid condition of

the lung tissue involving deficient aëration of the part; to affections of the bronchial mucous membrane and the lodgment of foreign bodies; to the pressure of tumors in the lungs, larger bronchi, or upper air-passages, and the like.

Absence or *suppression* of the respiratory murmur, as will be readily understood, occurs under much the same conditions as simple enfeeblement; but denotes that these have reached a higher degree.

Bronchial Respiration is to be heard in the healthy body only over the trachea and larynx, or in the region of the malar bone, and its characters can here always be studied; it is, however, to be remembered that the murmur seldom, if ever, is as intense over the chest as in the above mentioned localities. It is not necessary to repeat here the analysis of this variety of respiration. The sign indicates consolidation of the lung of a certain extent and at or not too far removed from the outer surface. In cases of pleural accumulation, or of more or less central consolidation, the sound may convey the impression to the ear of coming from a distance. As to the nature of the solidification the sound in itself affords no information. The sign is sometimes incomplete in that only one of the respiratory acts may be audible, more frequently inspiration. Its suggestiveness is not, however, lessened thereby. Or, again, inspiration may be vesicular but expiration bronchial, the latter being more intense and consequently further transmitted.

Broncho-Vesicular Respiration is heard in many healthy chests about the apices of the lungs and near the primary bronchi in front and behind. As the name indicates it implies a combination of the bronchial and vesicular qualities, either of which may predominate in any given case. It is called by some rude or harsh respiration. In proportion as the bronchial element is marked the pitch of both acts rises and expiration increases in length. The sign denotes partial solidification of lung, a degree not sufficient to produce bronchial respiration. It will be readily understood that there is room here for wide variation and nice shades of difference. According to the extent and completeness of the consolidation, its proximity to the chest wall, the thickness of this medium, and other factors, the bronchial element is more or less well marked and easy of recognition. It is sometimes only after taking into consideration the other physical signs that one can rightly estimate this form of respiratory murmur. Once more the fact must be alluded to that respiration over the right apex is normally less vesicular, or more broncho-vesicular, than over the left.

The cavernous respiration of Flint and its modifications are not sufficiently common or important to require description in a book of this size.

Amphoric Respiration, however, is a sub-division of the bronchial which is so distinctive and peculiar as to merit brief mention. Its character is precisely

that which belongs to the sound produced by blowing into a somewhat narrow necked and, at least partially, empty vessel; and that which especially stamps it is the metallic echo and musical sound which accompanies and follows the bronchial quality. It may be heard in both respiratory acts or in one only; if only in one more commonly in expiration. When heard it is absolute proof of the presence of a large cavity with walls of uniform density and in free communication with the bronchial tract, whether the cavity be seated in the lung or outside of it. In the former case its favorite seat is the upper front, in the latter the lower and middle regions of the chest. If a pulmonary cavity is full of secretion the conditions for its production are not fully met, and the sign may, consequently, be present at one and absent at the next examination, or *vice versa.*

Under the head of modifications of rhythm we distinguish:

1. *Shortened Inspiration*, a sign of deficient aëration of the part and encountered alike in the opposite conditions of partial obliteration and dilatation of the air vesicles. To distinguish between the two, pitch and quality must be studied.

2. *Prolonged Expiration* is, like shortened inspiration, sufficiently described by its name; it indicates that some kind of impediment to the free escape of air from the part exists, and is met with in the various degrees of condensation as well as in those

of dilatation of the lung tissue. In the first case the pitch and quality are changed, in the second they are not. Here too, the regional anatomy of the chest must be remembered and the normal difference between the apices.

3. *Interrupted*, *Jerky*, or *Cogged-wheel Respiration* is often due simply to lack of skill or want of training in breathing on the part of the patient. It is worthy of no special regard when it stands alone, unassociated with other signs.

Adventitious Sounds or *Râles*, never heard in absolutely normal conditions, may originate in any portion of the respiratory tract and are often classified on the basis of their seat of origin. They are also divided into coarse and fine, moist and dry, according to the notion conveyed to the ear as to their mode of production. They arise either within the air passages or in the pleura, and are due in the former situation to narrowing of the tubes from swelling of the mucous membrane, spasm, pressure from without, or similar cause, or to the presence of mucus, pus, blood, serum, fibrin, and other abnormal matters in the respiratory tract; in the latter to roughening or partial agglutination of the pleural surfaces.

Tracheal râles are usually audible without the stethoscope; of the moist ones the death rattle is most typical; the dry ones are suggestive of stenosis from whatever cause and produce a respiration termed stridulous or, simply, stridor.

The *moist bronchial râles* are bubbling in character and vary in size with that of the tubes within which they arise; the *dry bronchial râles* are whistling —sibilant,—or snoring—sonorous,—and are believed to originate only in the larger bronchi; moist râles indicate the presence of a relatively thin fluid, dry râles of partial occlusion from one or more of various causes; swelling, constriction, or very tough secretion, being the most frequent of these. Bronchitis is the chief underlying cause of bronchial râles, the special character of which is determined by the character and seat of the secretion or swelling, as well as by its extent. The finer bronchial râles, however, classed as sub-crepitant and arising in the bronchioles, are incident to many different diseases, as we shall see later, though they generally indicate essentially the same physical condition, namely, the presence of a thinnish fluid. All the bronchial râles may be heard in either or both of the respiratory acts. Coarse and fine, moist and dry, may be heard in different parts of the same chest simultaneously or at brief intervals of time. It is well to train the ear to note the pitch of these and other adventitious sounds as they sometimes so obscure the breath sounds that the character of the latter cannot be determined. Lung consolidation may be betrayed by high pitched and more or less intensely loud râles when the respiratory murmur cannot be heard. These are the consonating râles of Skoda.

The only râle which is believed to arise within the air vesicles is the *crepitant*, the finest of all, uniform in size, dry in character, heard only in inspiration, sometimes in the latter part of that act alone. It can be artificially imitated by rolling the hair near the ear between the thumb and finger, among other ways. The crepitant râle is especially characteristic of pneumonia in its first and third stages: the first stage frequently does not come under medical observation, and in the third stage the moist sub-crepitant is quite as common as the dry crepitant râles, consequently there are many cases of pneumonia in which this characteristic sound is not heard. It also occurs, especially about the apex, in some cases of phthisical consolidation. In other conditions it is, to say the least, rare.

Cavities containing liquid and communicating with a bronchus, if sufficiently large and near the surface, often give rise to râles which are very suggestive, either by their coarse and bubbling nature and their marked intensity, or by their ringing, musical, or metallic quality. The latter occurs under practically the same conditions as the amphoric percussion note and respiration. Besides the above mentioned râles which admit of more or less accurate classification, others are sometimes met with in the chest, especially the apices, which can be characterized as crumpling, creaking, or crackling. In that situation they awaken suspicion of phthisis.

Before leaving the subject of pulmonary râles, one or two remarks are in order. The act of coughing often has a marked effect on their production, the coarser moist râles sometimes promptly disappearing as the secretion which caused them is dislodged, the finer râles becoming more distinct, or, indeed, appearing only on cough—forced expiration—or during the relatively deep and quick inspiration which must almost necessarily follow it. In cases of suspected limited consolidation, especially about the apex and of phthisical origin, an examination is not complete unless this method is practiced.

It is not uncommon to hear during the first deep inspirations of a person who has been lying on his back for some time and breathing superficially, fine râles over the lower lobes behind. When heard thus, and thus only, they have no pathological significance, and are due simply to the unfolding of partially collapsed lung tissue.

Finally, one should always be careful not to mistake sounds produced by the rubbing of clothing, or of the chest piece of the stethoscope, against the skin —very liable to occur if the latter is hairy— for true râles. This remark applies to pleural as well as to pulmonary adventitious sounds.

The typical *pleural adventitious sound* is called friction rub, or sound, a term which is highly descriptive. In health the pleural surfaces glide noiselessly over one another; but when they are roughened a

sound is produced of extremely variable intensity, sometimes scarcely audible to the skilled auscultator, occasionally so loud as to be heard by the unaided ear at some distance from the chest, often easily felt by the hand applied over the part. Pleural friction may be heard during either or both acts, is near the ear, can sometimes be intensified by pressure with the stethoscope, and is apt to be broken or interrupted —*i. e.*, not to be continuous throughout the whole of inspiration, for instance. When typical, friction can hardly be mistaken for anything else except the rubbing of the stethoscope against the skin.

That sounds indistinguishable from medium and fine râles of intra-pulmonary origin may, and frequently do, arise within the pleura, the writer of this little book is strongly inclined to believe, although he is not prepared to adopt in full the views of Leaming, who holds that "all of the smaller râles have an interpleural origin, and also most of the larger."

The *voice sounds*, loud and whispered, in disease are well worthy of careful study. As with the breath sounds, the changes which they undergo are to be regarded rather as modifications of the normal sounds than as entirely new sounds, like râles. Making due allowances for sex, age, depth of voice, and thickness and tension of chest wall, they are *diminished in intensity*, or entirely suppressed under all conditions which impair or cut off the transmission of vibration from the larynx to the thorax.

Thus, if a primary bronchus is occluded they are entirely absent over the whole corresponding side of the chest; if air, fluid, or a solid tumor is interposed between the lung and the parietes they are lost or diminished over the seat of interposition, in a measure largely proportional to the thickness of the interposed layer, while they may be markedly increased over other portions of the chest. The limits of variation extend from the slightest possible decrease to total loss. In like manner an *increase* in resonance and fremitus is indicative of a condition facilitating conduction, and such condition is in the great majority of cases lung consolidation due to one or more of its many causes. Such increase, if more than slight in degree, is termed *bronchophony*, loud or whispered, from its identity with the vocal sound normal to the region of the tracheal bifurcation, although the type is better derived from the trachea or larynx direct. The slight degrees of increase have no special term applied to them. *Ægophony* is a peculiar form of bronchophony, to which distance from the ear and a nasal element resembling the bleating of a goat—hence the term—are superadded. It occurs often in cases of pleural effusion, moderate in size but sufficient to permit some condensation of the lung, and is heard near the level of the fluid. It is not a sign of great importance. *Pectoriloquy* signifies the distinct transmission of the words to the ear of the auscultator, and is an unimportant subdivision of broncho-

phony. Amphoric voice sounds are heard under the same conditions as the amphoric breath sound, over large pleural or lung cavities in direct connection with a bronchus.

6. Succussion, the last method of physical exploration of the chest, never yields results in the normal thorax, though such are often obtainable over the stomach more or less distended with air and liquid. When, in like manner, air and liquid are both contained in a large cavity in the chest, as in pneumo-hydro-, or pneumo-pyo-thorax, splashing sounds may be heard if the patient is shaken or shakes himself; the agitation produced by cough may be sufficient to give rise to them, and they may or may not have a metallic ring.

CHAPTER III.

THE PHYSICAL CONDITIONS AND DISEASES OF THE PLEURÆ AND LUNGS.

Inasmuch as physical signs lead directly to the detection of conditions, not of diseases, the natural order will be followed in treating of pathological states of the respiratory organs.

A.—OF THE PLEURÆ.

1. *Roughening* of the pleural surface, if acute, is apt to give rise to pain, and consequently to a catching or superficial respiration, which may or may not be accompanied by short, suppressed, or painful cough. Respiration being superficial, the murmur is feeble, but the distinctive sign is the friction rub, in the absence of which, acute dry pleurisy can be surely differentiated from pleurodynia and intercostal neuralgia only by rise of temperature. In subacute and chronic conditions, the subjective symptoms are generally very slight or absent, and friction is the only manifestation. Roughening is usually local, but may be diffuse, especially on one side. It may stand alone, or play a rôle (usually a secondary one) in connection with a great variety of diseases of constitutional or local origin. Thus, every inflammatory disease of the lung which approaches or reaches the surface is almost sure to set up pleurisy.

2. *Thickening*, in that it limits the expansion of the lung, and also separates it from the chest wall, gives rise to characteristic signs. Expansion of the affected side is diminished; the side may be shrunken; the percussion note is diminished in intensity, as is also the respiratory murmur; the tactile vocal fremitus and the resonance of the voice are generally not lessened, and may be somewhat increased. Friction sounds and pleural râles may or may not be heard, according to the density of the adhesions and the presence or absence of exudation in their meshes. In chronic cases the shoulder of the affected side is often decidedly lower than its fellow and there is lateral curvature of the spine.

3. *Liquid in the Pleural Cavity* may be either free or encapsulated, large or trifling in amount, serous, sero-fibrinous, purulent, or hæmorrhagic in character. Encapsulated collections are comparatively rare, and the notoriously great variation in the signs of the condition under consideration depends, in the main, on the widely varying amount of the collection. This variation makes the comprehension of the subject difficult unless one clearly understands the intra-thoracic changes which are wrought during the rise and decline of an accumulation. The experiments of Garland would seem to indicate that fluid thrown out into the chest is at first moulded by the lung—unless adhesions complicate matters—and does not compress that organ until the retractile force is exhausted; not

until then, also, can the diaphragm begin to lose the upward arch. With the descent of the diaphragm descend also the organs which lie immediately beneath it, notably the liver, spleen, and stomach. The heart swings on the great vessels arising from it as on a pivot, and toward the sound side, but never moves as far to the left as it may to the right. A moderate-sized collection may dislocate the heart, but a decidedly larger amount is required to depress the diaphragm. The lung itself is more or less atelectatic in moderate collections, especially near the level of the fluid, and is therefore capable of being inflated unless adhesions bind it down or there is coëxistent disease in its tissue.

(*a*) The signs of a very small effusion are nearly the same as those of simple thickening, except that the tactile fremitus and the vocal resonance are diminished in the former, while they may be unchanged or increased in the latter, false membranes often conducting sound fairly well. A distinction between the two conditions is not always easy to draw. Friction may be heard in both alike. The smallest amount of fluid which, in the average adult, can be detected with certainty is said to be about twelve ounces.

(*b*) In moderate collections impaired motion of the affected side is seen on inspection, especially with deep inspiration; percussion over the fluid is flat if, as should always be done, it is practiced gently; and the

line of flatness often follows a curved line which is lowest near the spine, rising at first sharply and then more gradually to the axillary region where it reaches its highest point, advancing to the sternum with a slight inclination downward. A space, termed by Garland "the dull triangle," is enclosed between the ascending portion of the curve and the spinal column; percussion over this space may be flat until the patient has deeply inspired several times and thus distended this portion of lung. Without this precaution it is often impossible to mark out the curved line of flatness, which is also obscured by adhesions and complications. Percussion over the relaxed lung above the fluid often yields a vesiculo-tympanitic note, and a flat or dull area may clear in a measure with a change in the position of the patient, the fluid tending to gravitate to the most dependent part of the chest; a few minutes must often be allowed for this change in the site of the fluid to take place. Displacement of the heart is often seen on inspection; but, especially if the apex lies under the sternum, the cardiac movements may not be visible and it is then desirable to determine the right border of the heart by percussion and the seat of maximum intensity of the first sound by auscultation. The breath and voice sounds over the fluid are markedly diminished or lost, as is the tactile fremitus; at the level of the fluid there may be ægophony; over the lung above, an enfeebled but broncho-vesicular respiration and increased resonance

of the voice are common. Subcrepitant, according to some authorities even crepitant, râles may be heard over this partially collapsed portion of lung as it unfolds with forced inspiration.

The signs of a decreasing pass through the same series of changes as those of an increasing effusion, but in an inverse order of sequence. Friction is apt to be more marked and more persistent in the declining stage, and dulness on percussion, due to pleural thickening, may last for a long time.

(*c*) In great collections the immobility of the affected side is striking, and the side may appear to be distended, while the intercostal spaces are flush with the ribs. An actual bulging of, or fluctuation in, the interspaces is very rare, and denotes a very large effusion limited by a thin wall; it is perhaps more liable to occur if for any reason space cannot be won for the fluid by descent of the diaphragm; it is less rare in children than in adults. The cardiac movements are seen in the left anterior axillary line if the right side is affected, over a large area extending even beyond the right nipple if the left side is affected. The tactile fremitus is lost, and the edge of the liver may be felt below the level of the navel with right, that of the spleen well below the rib margin with left effusions. If the inspiratory force is sufficient to inflate the compressed lung somewhat, these organs may be felt to descend with the diaphragm. Percussion is flat with great resistance under the finger over

nearly the whole of one side of the chest, more or less tympanitic in the front near the primary bronchus. The breath sounds are entirely absent, or more or less feeble and distant, and bronchial. Occasionally, however, bronchial respiration of a surprising intensity is heard all over the affected side, rendering it hard to believe that the lung is compressed rather than consolidated. This phenomenon seems to depend chiefly on the tension of thc chest wall and is therefore more common in children. Vocal fremitus and resonance are usually completely wanting—though the spoken words may be very distinctly transmitted to the ear—except about the root of the compressed lung where there may be bronchophony. The mediastinum may be pushed toward the sound side as is demonstrable by percussion. Over the healthy lung the resonance and breathing are increased in intensity. The degree of dyspnœa depends mainly on the rapidity with which the collection has taken place. The tolerance of a very large effusion which has come on insidiously and gradually, thus giving time for the system to adapt itself to the changed conditions, leads the careless to neglect a physical examination and thus to overlook a condition which mere inspection of the naked chest may diagnosticate with almost absolute certainty.

Fluid in the pleural cavity is usually the result of pleurisy, when it is generally unilateral; or of passive transudation—hydrothorax—when it is bilateral,

sometimes nearly equal, but often markedly different, in amount on the two sides. Hydrothorax is, of course, not an inflammatory process, and often therefore does not involve pleural roughening or adhesions. The fluid is thus more free to obey the laws of gravity and changes its seat more constantly and rapidly with change in the position of the patient; friction too is absent. But pleurisy may be secondary to hydrothorax, causing both adhesion and friction. For hydrothorax a cause is generally found in either the heart or kidneys, and there is also dropsy elsewhere. Pleurisy with effusion may complicate many other conditions, the signs characteristic of each more or less obscuring one another. Encapsulated collections of fluid are more difficult of detection, but should not ordinarily escape a careful examiner. The essence of all alike is the separation of the lung from the chest-wall by an airless and relatively non-conducting medium; the rest is detail.

The distinctive signs of liquid in the pleural cavity are the combination of percussion dullness, impaired transmission of vibrations, and dislocation of organs. The first of these is common to lung consolidation; the other two are more characteristic but still open to sources of error. In true consolidation the transmission of all vibrations may be cut off by bronchial occlusion. Dislocation of the heart and liver may be simulated by diseases involving enlargement of those organs, or by more or less complete

congenital transposition of the viscera; while genuine dislocation may be caused by new formations, pericardial effusion, or a retraction of the lung due to fibroid phthisis. The latter may be secondary to pleurisy attended by marked thickening, the interstitial tissue gradually penetrating the lung, condensing and destroying its vesicular structure. Again, a large effusion may be present without any dislocation, adhesions preventing its occurrence.

While the diagnosis of fluid in the pleural cavity generally presents no serious difficulties, and may be extremely easy, even uncomplicated cases occur in which a positive diagnosis from the combined physical and rational signs is of the greatest difficulty. Consolidation of the lung and intra-thoracic tumor are the two conditions most liable to involve error. The latter is as rare as the former is common, but both will be more fully considered later.

In case of doubt, puncture with an irreproachable needle will settle alike the presence and nature of fluid, the latter not being accurately determinable by the ordinary physical signs. Œdema of the chest-wall, a jagged temperature chart, to a less degree childhood and an African descent, are suggestive of empyema. A pulsating pleurisy, almost invariably left-sided, is nearly sure to be purulent. An hæmorrhagic effusion is far more apt to accompany tuberculosis or cancer than more innocent affections.

4. *Air in the Pleural Cavity* is rarely found with-

out some important complication. When found, the physical condition to which it gives rise is almost identical with that arising from liquid in the pleura; the main difference is that of the medium separating the lung from the throax. This in pneumo-thorax, being air, is resonant; and this air, being enclosed in a large space, yields a purely tympanitic or amphoric note on percussion. The physical signs other than those derived from the percussion note are practically the same in the two cases. Pneumo-thorax arises from rupture or perforation of the lung, or from perforation—traumatic—of the chest-wall.

5. *Air and Liquid in the Pleural Cavity.*—The physical condition as regards the lung is, in cases without notable complication, the same as in the two preceding divisions. It is retracted or compressed, unless adherent or consolidated, in proportion to the amount of air and liquid just as when only one of these is present; the liquid, of course, gravitates to the lower portion of the chest, while the upper is occupied by the lighter air. The recognition of the condition here again depends chiefly on the results of percussion; the note being dull or flat over the liquid, tympanitic over the air, and the relative positions of the flatness and tympany changing strikingly with the position of the patient. The relative proportion of the two media varies, of course, widely in different cases, somewhat in the same case from time to time. Succussion sounds are in the highest degree diagnos-

tic. Amphoric respiration denotes that there is a perforation of the lung through which the air passes to and fro. Metallic tinkling may also be present. Pleural adhesions and lung consolidation, if present, will modify the signs in ways which are readily understood. Again, the signs in themselves do not inform us whether we have to deal with pneumo-hydro-, or pneumo-pyo-thorax.

B.—OF THE UPPER AIR PASSAGES AND LUNGS.

1. *Of the Larynx and Trachea.* The conditions which here give rise to the sort of physical signs under consideration, are those involving more or less obstruction to the free access of air to the lungs; inspiration alone, or both acts, may be impeded; a purely expiratory dyspnœa of laryngeal or tracheal origin is conceivable, but practically does not occur. If the obstruction is of high degree simple inspection is fruitful in results; the dyspnœa is great, and heightened by the least exertion; cyanosis may be marked, the lungs fail to expand, and the soft parts above and below the chest, as well as the lower and lateral portions of the same, are retracted in inspiration. The respiratory murmur is weakened proportionally to the degree of obstruction; the respiration is more or less stridulous. If the upper air passages are obstructed, the feeble respiration is bilateral; if a primary bronchus, it is confined to the affected side, while on the other the murmur is exaggerated.

The diseases giving rise to obstruction of the larynx and trachea are, œdema of the glottis, paralysis of its dilators, spasm, false membranes, tumors within or pressing on the parts, foreign bodies.

2. *Of the Bronchi.*—(*a*) *Of the Larger Tubes.* The presence and character of the physical signs depend mainly on whether respiration is or is not obstructed, partly on the nature of the obstruction and its degree. The signs are unilateral if the cause be such; as with foreign bodies, the pressure of a tumor, aneurismal, glandular, or neoplasmic. Bilateral conditions are, however, far more common. If there be no notable obstruction, physical signs are absent, and diagnosis rests on the negative result of examination associated with positive symptoms. Obstruction is due generally to swelling, or secretion, neither of which can, in this situation, produce dullness or a change in the voice sounds. They may weaken the breath sounds more or less, but do so bilaterally and thus deprive us of the advantage to be gained by a comparison of the two sides. Otherwise the signs are negative unless swelling causes sibilant or sonorous, secretion or other liquid product coarse moist râles. By far the most frequent condition underlying the condition here described is bronchitis, which may or may not, of course, be complicated with asthma, emphysema, or lung consolidation.

(*b*) *Of the Smaller Tubes.* It will be readily understood that changes do not need to attain such a

high degree here to produce physical signs as in tubes of large calibre. Obstruction to the free access of air to the vesicles may be caused by swelling, spasm, liquid products, or compression; involving both or only one side of the chest. The distinctive signs are: weakening of the respiratory murmur over the affected area or areas, and fine moist—sub-crepitant—râles if secretion or other liquid is present; the râles may entirely obscure the vesicular murmur. Notable dyspnœa and cyanosis are seen if the obstruction is widespread, and the soft parts above and below the chest may exhibit inspiratory retraction; this dyspnœa being distinguished from that due to laryngeal and tracheal obstruction by the presence of signs in the chest. The number of diseases to which this condition is incident is large, including, as it does, practically, the very slight degrees and early stages of affections involving consolidation of the lung tissue to a greater or less extent. Thus it is present in many cases of phthisis, especially about foci of active inflammation; but the two bilateral affections which interest us most here are capillary bronchitis and pulmonary œdema. The latter is usually non-inflammatory, is generally traceable to heart or kidney disease and affects by preference the dependent portions of the chest; it is, moreover, often accompanied by dullness on percussion. The former befalls chiefly the extremes of age, and is often fatal, either directly by suffocating and exhausting the patient, or by inducing

its dreaded sequels—atelectasis and broncho-pneumonia. These both really involve consolidation, but the areas may be so small and so scattered as to give rise to no signs distinctive of that condition. The recognition of their presence, then, must be a matter of inference, chiefly from the symptoms. Fibrinous bronchitis may impair resonance and cut off the breath and voice sounds, to a greater or less degree. It is rare, and can be diagnosticated with certainty only by the expectoration of casts of the bronchi. It is, of course, the medium and finer tubes which are most liable to be occluded by the fibrinous exudation.

Before we leave this portion of our subject, a few words must be said about *Asthma*, invariably a symptom rather than a disease, as is held by some; in its pure spasmodic form an independent affection, as is held by others. Whether we affirm or deny that spasm of the muscular layer of the smaller non-cartilaginous bronchi is the cause of the symptoms, that an impediment of some nature to the normal to-and-fro movement of the air between the larger bronchi and the air vesicles exists during the asthmatic paroxysm, cannot be disputed. The positive physical signs are: Dyspnœa, perhaps cyanosis; a thorax nearly motionless in spite of great efforts, and fixed in the position of full inspiration; general hyper-resonance on percussion; and the universal presence of sibilant and sonorous râles masking an enfeebled vesicular murmur; the breathing is not quickened, but labored—a

point of contrast with capillary bronchitis. The signs of emphysema, temporary and permanent, will be considered later.

3. *Bronchial Dilatation*, if sufficient to be recognized, gives rise to cavitary signs which, in themselves, throw no light on the manner of production of the cavity. The signs of cavities belong more appropriately in another place, but it may be stated here that bronchiectasis is more common in the lower lobes, and is apt to be accompanied by more or less emphysema and solidification.

4. *Dilatation of the Air Vesicles.*—This condition is associated with but one disease, emphysema of the lungs, which may be general or local, temporary or permanent. In any case the affected air cells cannot be emptied in expiration and there is consequently no room for fresh air; or they can be emptied only by the aid of unusual respiratory exertion, a difference of degree. General temporary emphysema is met with in severe pertussis and during the paroxysm of pure spasmodic asthma; local temporary emphysema exists vicariously in the healthy parts when other and considerable areas of the lungs are thrown out of work. The general permanent change is usually associated with chronic bronchitis and often with secondary asthma; the local with the same conditions as the temporary variety but, in this case, of a chronic nature. There is, however, a point of essential difference between temporary and permanent emphysema; over-distention

of the air vesicles is a common factor in both; but in the latter the elasticity of the lung tissue is seriously impaired or lost, while in the former it is not.

The destruction of the pulmonary capillaries and the defective interchange of gases throw extra work on the right heart which becomes gradually dilated and hypertrophied. If compensation fails we get the usual signs of general venous stasis.

The physical signs depend in large measure on whether the changes are local or general. The latter, being the more characteristic, will be considered first.

Inspection yields results which may be in themselves highly characteristic. In a typical case the patient is apt to be pale and somewhat cyanotic, sluggish in his motions and mental processes, slow and labored in breathing even while at rest, but far more so after the exertion involved in undressing. All the diameters of the chest are increased but especially the antero-posterior, so that a transverse section, as shown by a stethometer, is much rounder than normal —the barrel shaped chest. The shoulders are raised and rounded, and the clavicles as well as the accessory muscles of respiration are prominent. The thorax is more or less fixed in the position of inspiration and moves as one piece, especially if the costal cartilages are ossified. There may be marked swellings above the clavicles, visibly increased by cough. If there is a thick layer of subcutaneous fat, the tur-

gid countenance, hyperæmic conjunctivæ, short neck, and general form of the chest are still noticeable; but the muscular attachments to the clavicles and the distinction between the ribs and the interspaces are hidden. It must not be thought that the above described form of chest is always present in extensive emphysema; the affection may be general and far advanced in a thorax of the paralytic type. The cardiac impulse is feeble or wanting in the normal site while epigastric pulsation may be marked, the dilated lung occupying the space where the heart ought to lie in immediate contact with the chest wall, and the right ventricle being dilated and hypertrophied.

Percussion gives exaggerated or vesiculo-tympanitic resonance as well as an increase in the area of pulmonary resonance; the cardiac dulness is diminished or lost and percussion gives about the same results in that region, as well as over the lower limits of the lungs, at the height of either respiratory act; the liver dulness and flatness begin lower down than is normal, and the liver itself may be pushed below the rib margin, as can be proved by palpation and percussion; resonance may extend unusually low in the backs. The hyper-resonant condition of the lung prevents determination of the right border of the heart although it extends farther to the right than in health.

On auscultation we are struck by the weakening and shortening of inspiration and the prolongation of

the low pitched expirations; a change of rhythm for the most part, and, in the main, proportional to the impairment of function of the elastic tissue which, in health, renders expiration chiefly a passive act. Râles, sibilant and sonorous or coarse moist, are usually heard; but are traceable rather to the chronic bronchitis which almost always accompanies emphysema than to the latter process itself. The heart sounds may be more or less indistinct; the pulmonic second is apt to be accentuated by reason of the increased resistance in the lesser circuit. The tactile fremitus and the voice sounds, of course, undergo no modification as a rule. In the lesser degrees of emphysema the signs are essentially the same in kind, though simple enfeeblement of the vesicular murmur may be more striking than a change in rhythm.

There is a rarer form of general emphysema called "atrophic," "small-lunged," or "senile" in contradistinction to the ordinary "large-lunged," or "hypertrophic" form. As the name indicates it is an affection of advanced life and involves no enlargement of the chest; the lungs, though emphysematous, being atrophied and diminished in volume. The superficial cardiac space may be increased in size. The physical signs are not distinctive. The physical signs of permanent local emphysema depend much on its extent and the condition to which it is secondary. It is apt to occupy the upper lobes and is often associated with the more chronic forms of phthisis,

the consolidation of which it may mask more or less completely. Its detection may demand a thorough knowledge of principles on the part of the observer.

Interstitial or interlobular emphysema has no direct physical signs and can scarcely be diagnosticated during life unless the air escapes from the chest through the mediastinum into the subcutaneous tissue of the neck and thorax.

5. *Consolidation of the Lung.*—This important condition is incident to a great variety of diseases, and differs so widely in degree, extent, and situation, that its detection may be impossible; more or less difficult, partly according to the skill of the observer; or so easy as to present little or no difficulty to the merest tyro in physical examination. An area of consolidation must be of notable size, lie in contact with or nearly approach the chest wall, and be pretty complete, in order to give rise to distinctive signs. Small areas, and those larger but deep-seated, of complete consolidation, as well as those relatively diffuse but imperfect, however near the surface, may be either impossible to detect at all, or may have their presence betrayed by the rational signs in combination with the localization of indistinctive physical signs. Solidification of the lung, as the term implies, involves a diminution in the amount of air proper to the part, which diminution is generally combined with, if not dependent on, an increase of the solids; it is, therefore, the opposite of emphysema, though the two con-

ditions may be associated as has already been seen. There are two chief ways in which solidification may be brought about, from without or from within; the first is compression, and is incident especially to large pleural accumulation; the second is due to obliteration of the air cells by interstitial or other growth; to filling of the cells with morbid products, as in pneumonia lobar or lobular, infarction of the lung, generally to a less degree in pulmonary œdema; to collapse of the lung, as in atelectasis due to capillary bronchitis; or to a combination of these processes. By any or all of these means the amount of air in the part is diminished, and its access to the vesicles is impeded or prevented, while the atmospheric circulation in the upper air passages and larger bronchi remains essentially unchanged. The condensed lung tissue, moreover, conducts sounds produced in the larger bronchi, trachea, and at the rima glottidis comparatively unchanged to the periphery, these sounds being in health modified and broken up in the ramifications of the bronchioles, and in the air cells themselves. Consequently, many of the physicial signs of consolidation are the same in kind as those found in health over the upper and larger air passages.

The physical signs, then, of complete consolidation of considerable extent are as follows:

Inspection may or may not show notable dyspnœa and cyanosis, according to the proportion of lung involved in the solidification and the rapidity with

which this has come on; there may, also, be a disparity in the respiratory movements of the two sides if the change is unilateral, though this sign is never as marked as in pleural accumulation. The tactile fremitus is increased over the solidified area, which is very dull or flat on percussion with increased resistance under the finger. But the tympanitic resonance of the stomach may be conducted over the left chest if the left lung or its lower portion is solidified.

The respiratory murmur is bronchial over the affected area, and usually intense, and bronchophony and whispering bronchophony are marked. Râles are generally, though not necessarily, present, their abundance and character depending largely on the cause of the consolidation. In the second stage of lobar pneumonia they may be entirely absent, or coarse râles due to secretion in the larger bronchi may be heard. Medium and fine moist râles are common, at least over portions of the solid area; and, in general, the râles over solid lung are apt to be loud and somewhat ringing—the consonating râles of Skoda. They may be heard only after cough, or at the end of deep inspiration. Complete and extensive consolidation is more often associated with lobar pneumonia than with any other one disease, perhaps than with all others put together.

Vesicular resonance and respiration over the sound lung may be exaggerated.

The condition is also incident to large pleural

accumulation, some cases of phthisis and gangrene, and to some new growths.

Partial consolidation may give rise to dyspnœa and cyanosis; percussion is dull, and may also be tympanitic, as in the first and third stages of fibrinous pneumonia, and over the relaxed and somewhat condensed lung in pleural effusion. Auscultation gives a broncho-vesicular respiration, often of slight intensity, in which one or the other quality predominates according to the degree and extent of the solidification. The heart sounds are heard with remarkable distinctness over a solidified lung in or about the cardiac area. The tactile fremitus, and vocal fremitus and resonance, are increased. Râles are practically always present, usually of the medium and fine moist varieties, sometimes so abundant as to mask the respiratory murmur; in the first and third stages of lobar pneumonia the fine dry, or crepitant, râle may be heard. Coarse râles, moist or dry, are accidental, and depend on the presence of secretion or inflammation in the larger bronchi. In lobar pneumonia an opportunity may be afforded the observer of studying the whole process of extensive consolidation from start to finish, though cases are usually not seen until solidification is marked. The waning gives rise to the same signs as the waxing process, except that their development is generally slower in the former, and that the order of their appearance is reversed.

Partial consolidation is, or may be, met with in

the first and third stages of pneumonia, in broncho-pneumonia and atelectasis, phthisis, moderate pleural accumulation, infarction—hæmorrhagic and embolic—œdema and hypostatic congestion, pulmonary hæmorrhage, gangrene and infiltrating neoplasms. These processes are to be differentiated by a careful consideration of the localization, mode of onset, course, and progress of the disease, character of the sputum, presence or absence of disease in other organs or parts—in short, by putting together and weighing all the information which can be gained by questioning the patient or his friends, as well as by an examination of all his organs, and his body as a whole. Even then it is sometimes necessary to hold one's opinion in reserve. Consolidation, complete or partial, may be variously combined, as with effusion, emphysema, bronchial obstruction, or cavity.

The more important physical signs of each of the above affections are as follows:

1. *Phthisis.* (*a*) First stage.—If consolidation is very slight the sole physical sign may be the presence, especially at one apex, of a dry crumpling or creaking sound, or of subcrepitant râles, more or less abundant, and heard sometimes only after cough or during the quick inspiration which should follow that act. If consolidation is more advanced there is dulness on percussion, broncho-vesicular respiration, increased fremitus, and a degree of bronchophony, in addition to fine and medium râles.

The commencing process is usually, though not necessarily, unilateral; and involves the apex by preference. Hence any signs in that region should be regarded with suspicion. A sputum examination may settle the diagnosis before any physical signs can be obtained.

(*b*) Second stage. Here the signs are the same except that the consolidation is usually more marked and extensive while the breaking down lung tissue gives rise to more abundant and more varied râles.

(*c*) The criterion of the third stage is generally considered to be cavity formation, the distinctive signs of which will be set forth later. A cavity may form in the midst of a single and very circumscribed area of disease; and, on the other hand, no cavity which can be detected by physical signs may be formed in cases of wide spread disease. As a rule, however, cavities are associated with a more or less extensive and chronic process. It is, of course, understood that such a classification as the above is artificial rather than natural.

Fibroid phthisis, so-called, is a very chronic process the main pathological characteristic of which is the substitution of connective tissue for vesicular structure. It is generally unilateral, may begin at the base as a result apparently of pneumonia or pleurisy, often contracts and distorts the chest, and gives rise to well marked signs of consolidation. It may draw the heart out of place toward itself and leave an undue portion

of the organ uncovered by lung. At the same time the heightened resistance in the pulmonary circulation induces dilatation and hypertrophy of the right ventricle, the fingers and toes are often clubbed, and the usual phenomena of failure of the right heart may supervene. The strongest diagnostic feature lies in the association of such physical signs as the foregoing, and of great dyspnœa on exertion, with a surprisingly good condition of general nutrition and an afebrile state.

2. *Lobar Pneumonia* is ordinarily an acute febrile process but may in the aged be latent as far as symptoms are concerned.

(*a*) The first stage, that of engorgement, so-called, gives at first slight dulness and feeble respiration with, in typical cases, the crepitant râle; the favorite localization of these signs being over a lower lobe. Pleural friction, especially in the absence of the crepitant râle, may obscure diagnosis at this time.

(*b*) As the first passes into the second stage, dullness increases in intensity and area; respiration becomes broncho-vesicular, and then bronchial; the tactile fremitus is increased, and bronchophony appears. Crepitant and subcrepitant râles may be heard over incomplete areas of solidification; over complete areas no râles are heard except those attributable to secretion in the larger bronchi.

(*c*) In the third stage, as the exudation is absorbed and air begins to re-enter the alveoli, the lung

finally quite recovering its normal condition, the same signs return, but in an inverse order.

3. *Broncho-pneumonia* is an acute febrile process, secondary to bronchitis, bilateral, and incident especially to the extremes of life. One form is the so-called "inhalation pneumonia," excited particularly during the course of severe adynamic fevers by the inhalation of particles of food, or of secretion from the mouth and upper air passages. The diagnosis rests on the association of such circumstances as the above, with more or less diffused bilateral fine moist râles, and the evidences of scattered areas of solidification of varying size.

4. *Atelectasis* gives rise to signs which, in themselves, may be identical with those of broncho-pneumonia, and which never present any sharp distinction therefrom except that in atelectasis the areas of consolidation may change their seat from day to day. It is a condition rather than a disease, and the chief clinical peculiarity is that it is not febrile. It is often combined with lobular pneumonia, and is then incapable of diagnosis.

5. *Hypostatic Pneumonia*, or *Congestion*, affects the dependent portions of the lungs of those whose hearts are weak, whose breathing is superficial, and who lie much in one position—postulates which are most frequently met in the severe infectious diseases. It is bilateral and, naturally, most common in the posterior bases.

Its signs are: Dullness on percussion; feeble respiration, sometimes partly bronchial in quality; and coarse and fine moist râles.

6. *Œdema of the Lung* may be associated with, and constitute an important part of, hypostatic congestion. But transudation of pure serum into the lung tissue and alveoli may occur in obstructive disease or failure of the heart from any cause, as well as in Bright's disease. The physical condition and localization are much the same as those of hypostatic pneumonia, though the process seldom reaches so high a degree, and the diagnosis must therefore depend largely on the associated conditions.

A rare form of acute primary and general pulmonary œdema is described, which is characterized by relatively sudden and intense dyspnœa, the universal presence of subcrepitant mixed with coarser moist râles all over both sides of the chest, and a very copious serous expectoration, generally pink or pinkish from admixture with blood.

6. *Gangrene of the Lung* has no distinctive physical signs, and the diagnosis rests entirely on the horrible fœtor of the breath and sputum, in combination with signs of limited or diffuse consolidation. It is to be differentiated only from putrid bronchitis, and a bronchiectatic cavity with decomposing contents. Lung consolidation and elastic fibres in the sputum distinguish it from the pure cases of the former; with the latter it may be combined, and in this case the history of the patient can alone remove doubt.

8. In *Hæmoptysis*, some of the blood may be retained and give rise to the signs of partial consolidation, or to those of a more or less extensive capillary bronchitis. The history of the case, or the actual observation of the hæmorrhage, are requisite for diagnosis.

9. *Hæmorrhagic* and *Embolic Infarction* may be followed by bloody expectoration and give rise to solidification large or complete enough to produce the physical signs of that condition. The former can be diagnosticated only by excluding every other cause for the above combination of rational and physical signs—a task of the greatest difficulty unless there is a history of trauma. The latter requires a source for the embolus either in the right heart (especially thrombosis in the auricular appendage) or in the peripheral venous circulation. Pleurisy may be excited at the base of the wedge-shaped mass. Given a clear source of the embolus, sudden dyspnœa and pleuritic pain may awaken strong suspicion, which hæmoptysis and the signs of consolidation convert into certainty; but the process may not reach the surface and cause pleurisy, nor does hæmoptysis always occur. Diagnosis may therefore be impossible.

6. *Pulmonary Cavities.*—These must be of a certain size to give rise to distinctive signs, and it stands to reason that a cavity in the deeper portions of the lung surrounded by more or less healthy tissue will be less easily detected than a smaller one near or at

the outer surface. Even distinctive signs are materially influenced by the presence or absence of free communication with the bronchial tract, the amount of secretion or other fluid in the cavity, the smoothness and tension of its walls, and the amount of consolidated lung surrounding them.

Distinctive signs are cracked-pot or amphoric percussion note; bronchial, cavernous, or amphoric, respiration; coarse gurgling, or amphoric râles; bronchial, cavernous, or amphoric, voice sounds and whisper; and greatly increased fremitus, which may be even painful to the ear.

Cavities are incident especially to phthisis, in which case their favorite seat is the upper lobes; or to bronchiectasis, gangrene, and abscess of the lung; in the three latter the lower lobe is more frequently the seat. Which cause is operative in a given case must be ascertained by sputum examination, the history and course of the disease, and the associated signs, rational and physical.

7. *Thoracic Tumors, Non-Aneurismal.*—These are all solid, but have little else in common. Of course the physical signs must vary with the starting point of the growth, its size, whether it infiltrates or compresses the lung, is single or multiple, compresses a large bronchial, arterial, or venous trunk, the œsophagus, or an important nerve. The indirect or pressure effects will be better discussed under the head of aneurism which far surpasses in frequency

other thoracic tumors. The pathological nature of the tumor has less influence on the physical signs than its origin and size. There are, indeed, no distinctive signs of tumors in this situation; they may simulate solidification of the lung, deep or superficial, or pleural effusion. Ecchinococcus cysts, projecting from the upper surface of the liver into the space normally occupied by lung, are especially liable to give rise to the last named error, an error which tapping may fortify unless the fluid is examined chemically or microscopically. Enlargement of the bronchial glands, cancer, and sarcoma, are the other least rare forms of intra-thoracic new growths. The writer has seen a case of myxo-chondroma. In general the diagnosis must be reached by exclusion. Growths which start from or approach the pleura are apt to excite inflammation of that membrane with fibrinous, serous, or hæmorrhagic, product.

PART II.—HEART AND AORTA.

CHAPTER I.

THE HEART IN HEALTH.

Experience as a teacher has led the author to believe that the chief difficulties encountered by students in the physical diagnosis of cardiac disease depend on the lack of a sufficiently intimate and accurate knowledge of the anatomy and physiology of the heart. Therefore the structure of the organ itself as well as its relation to surrounding organs and parts, and the mechanism of the circulation, should be first thoroughly mastered. Speaking broadly, the diagnosis of valvular disease of the heart, to say the least, is less difficult than is that of pulmonary disorders. Yet students seem to find it more so, as I believe for the reason stated above; their knowledge may be fairly accurate, but is usually not quickly enough at command. We must, however, here presuppose the possession of most of this knowledge, and shall dwell upon it only, as it were, incidentally in describing the methods employed in cardiac examination. It is hoped that the little plates may be of service.

1. *Inspection* may, in chests well covered by fat or muscle, show little or nothing, especially if the

heart is acting quietly; in women the mamma is very apt to mask the impulse. In thin persons, or in the stouter if the heart is excited by exercise or emotion, the movement communicated to the chest-wall by the ventricular contraction is generally seen in the fifth, occasionally in the fourth, left interspace and inside the mammillary line. This is called the apex beat. In children it lies rather farther to the left than in adults, and often occupies the fourth space. It is apt to be less distinct, or absent, with a dorsal decubitus; moves somewhat to the right, often as far to the left as the anterior axillary line, with the right and left lateral decubitus respectively. In very thin persons, especially if there be retraction or solidification of the corresponding portion of lung, a basic movement may be seen.

2. *Palpation* in health is chiefly of value in localizing the apex beat in case this cannot be done by the eye, or as confirmatory of the results of inspection. The shock of the systole can sometimes be felt when it cannot be seen. But palpation may also give negative results, and under much the same circumstances as inspection. A change of position on the part of the patient may yield results quite analogous to those above alluded to under inspection. A basic pulsation may also be felt in some persons.

3. *Percussion* enables us in most cases to map out the heart's area with great accuracy provided that the method is skillfully practiced. Gentle percussion

is flat over the small and somewhat triangular space where the pericardium is uncovered by lung and lies in immediate contact with the chest wall. This space corresponds to the body of the right ventricle, the lingula of the left lung covering with a thin layer the very apex, which is formed, as is well known, by the left ventricle. The lower boundary of this space cannot be determined by percussion as the heart and liver, both solid and non-resonant bodies, here come together separated only by the diaphragm. The right border of the space is the left edge of the sternum; its upper edge near the sternum is the fourth costal cartilage, and the left border runs thence outward and downward toward the apex. This is called the superficial cardiac space. It varies appreciably in size with forced inspiration or expiration. The deep cardiac space comprises the greater portion of the heart, which is covered by a layer of lung and by the sternum, and therefore extends chiefly to the right and upward, though also somewhat to the left, of the superficial space. The deep space corresponds to a portion of the right ventricle, a strip of the left, and to the auricles. Percussion over it is dull, more or less. The accuracy with which it can be mapped out by percussion varies in different persons. In some the extreme right border can be made out by careful percussion about three centimeters to the right of the right sternal border; in others it cannot. The thickish layer of the lung and the resonance of the sternum

are disturbing factors. But the negative evidence of a failure to detect dulness to the right of the sternum between the third and fifth cartilages is important in determining the absence of enlargement of the right cavities, dislocation of the heart, or notable pericardial effusion. Above, the dulness begins at the third rib; the left border follows a line which passes slowly downward to the fourth rib, and then rapidly downward to the extreme apex. The lines are but little modified by a change from the recumbent to the vertical position or *vice versa*; but the lateral borders move somewhat to the right or left in one or the other lateral decubitus.

4. *Auscultation* enables us to listen to the sounds produced by the heart in action and to determine their nature. These sounds are two in number and are distinguished as the first and second. The first corresponds in time to the ventricular systole and has a double origin; the change of tension which the auriculo-ventricular valves undergo when they close the mitral and tricuspid orifices, and the muscle sound caused by the contraction of the ventricles. That the impingement of the heart against the chest wall enters into the production of the first sound is open to more question. This sound is low in pitch, long in duration, and may be described as booming in quality. It is heard all over, and often even far beyond the limits of the heart, but is loudest at the apex. Here that portion of the sound which is attri-

butable to the mitral valve is most marked, while to estimate the tricuspid element we listen over the middle of the lower part of the sternum. In health this differentiation is not much practiced but it may become important in some diseased conditions. The second sound follows almost immediately on the first, and is attributable to vibrations set up in the aortic and pulmonic valves by the change of tension to which they are subjected at their time of closure. As compared with the first sound it may be characterized as sharp, short, and valvular. It also is heard all over and beyond the heart's area, but is loudest at the base of the organ. To determine the relative value of the two elements composing it we listen over the second right interspace at the sternal border for the aortic portion, a point somewhat above and to the right of the seat of the valves themselves, but that at which the aorta nearest approaches the external surface. The sound is in health somewhat louder and sharper here than it is over the corresponding point on the left side where we are accustomed to study that portion of the second sound due to the pulmonic valves. If there is any difficulty in distinguishing the sounds from one another the finger may be placed on the apex, or, if this cannot be felt, on the carotid; the sound which is synchronous with the apex beat or the dilatation of the artery is, of course, the first. The second sound, marking the closure of the semilunar valves, is followed by the long pause of

the heart, as it is called, during which the blood is passing under gentle pressure and in a noiseless stream from the auricles to the ventricles—the ventricular diastole. Toward the end of the ventricular diastole the auricles contract to empty themselves and complete the filling of the ventricles, and this is promptly followed by the ventricular systole and the commencement of another cardiac revolution. In health the auricular systole gives rise to no sound which we can hear, and one revolution succeeds another, equal in force and regular in rhythm. The rate is slower or faster from individual peculiarity, rest or exercise, and the absence or presence of emotional excitement. The distance to which the sounds are propagated depends in a measure on the tension of the chest wall; the greater the tension the more distant the propagation.

To sum up, the first sound marks the ventricular systole, the second the beginning ventricular diastole which continues up to the next ventricular systole but, just before its close, is synchronous with the auricular systole.

It should always be remembered that changes in the lungs may cause an apparent modification in many of the physical signs of the healthy heart. Thus, air or liquid in the pleural cavity frequently produces striking changes in the position of the central organ of the circulation, which, fixed at the base by the great vessels, can on them be swung from side to side

and also rotated in a measure; it can be pushed out of place by mediastinal or other tumors, or by the healthy lung when its fellow is markedly retracted; it can descend with the diaphragm when that muscle is depressed by large lunged emphysema, the dilated lung at the same time effacing the superficial cardiac space, though emphysema of this degree is nearly sure to cause secondary dilatation and hypertrophy of the right cavities; the seat of maximum intensity of its sounds may be changed by solidified lung; or adventitious sounds arising in the lung or pleura may simulate those of endo- or pericardial origin. Again any abdominal disease or process which unduly forces up the diaphragm is also liable to alter the normal relations of the healthy heart. Transposition of the viscera must also be mentioned. In every examination these sources of error must be thought of and allowed due weight. Of course cardiac is often coexistent with pulmonary or abdominal disease, and a state of affairs may thus be produced the unravelling of which demands the greatest acumen and the most accurate knowledge.

CHAPTER II.

THE HEART IN DISEASE.

Just as in pulmonary, so in cardiac examination physical signs lead directly to physical condition, indirectly to disease. But the number of diseases which in the heart have a similar underlying physical condition is much smaller than obtains with the organs of respiration. It consequently seems wiser to pursue a somewhat different order in dealing with the heart from that which was followed with the lungs.

ENLARGEMENT OF THE HEART.—Enlargement may be either general or local, involving the whole organ or only one or more of its component divisions. It may, moreover, be due either to dilatation or to hypertrophy; or, as is usually the case, to a combination of the two. The physical signs of these several conditions vary so that they must be classified and discussed separately as far as truth to nature will permit.

1. *Simple hypertrophy* means an increase in the bulk, and possibly in the number, of the muscular fibres without an increase in the size of the cavities which they enclose; a condition which, in its pure, and especially in its general, form, is rare.

The physical signs to which it gives rise are: an apex beat unusually evident to the eye, situated lower and farther to the left, and slightly more diffuse than

is normal, and communicating a strong shock to the finger applied to the apex region; a slight general increase in the area of cardiac dullness; and unusually loud and distinct sounds which preserve alike their relative, and seat of maximum, intensity. It may be stated here that hypertrophy is a less important factor in producing an increase in the size of the heart than is dilatation. It consequently cannot be expected by itself, to markedly increase the area of percussion dullness. General simple hypertrophy is apt to be associated with general plethora.

It is in the left ventricle that local simple hypertrophy is most common, and in this case the apex beat is lowered, marked alike to the hand and eye; there is little if any increase in percussion dullness, but that little is to the left; and the first sound in the mitral area is loud and prolonged while the aortic second, as heard in the second right interspace, is more or less sharply accentuated. The pulse is strong, slow, regular, usually incompressible and full between the beats—of high tension. Pure, or concentric, hypertrophy of the left ventricle is met with especially in cases of chronic, and, above all, of interstitial nephritis, and is to be regarded as the result of and means of compensation for the heightened pressure in the peripheral arterial circulation so characteristic of that disease. In those very rare cases of pure aortic stenosis it is through simple hypertrophy of the left ventricle that compensation may be secured.

This condition of the right ventricle is very rare. The signs are: An apex beat moved to the left rather than lowered, and diffused toward the epigastrium; a slight increase in the transverse dullness to the right of the sternum; and an unusually loud first sound in the tricuspid area with accentuation of the pulmonic second, as heard at the second left interspace near the sternal border. The condition is incident to and compensatory for increased pressure in the lesser, or pulmonic, circuit, as in emphysema.

Simple hypertrophy of the auricles can scarcely be said to exist. Their walls are so thin and feeble as compared to those of the ventricles that heightened pressure is sure to result in dilatation, with which hypertrophy may or may not be combined.

2. *Simple Dilatation* denotes a condition in which one or more of the cavities is increased in size without thickening of the wall. In its pure form it is an even rarer condition than simple hypertrophy. General dilatation is manifested by absence or great feebleness of the apex beat and radial pulse, for the former of which a thick chest wall will not account; general increase in the cardiac dullness; and feebleness of the sounds, the first being shorter and higher pitched than in health and approximating in character to the second. Outside of the heart there are usually the evidences of venous stasis, such as dropsy, internal, external, or both. The condition is to be differentiated from pericardial effusion, and in a manner

which will be discussed later. The usual cause is defective blood formation and consequent failure of tissue nutrition, under which the heart wall yields to internal pressure. It would occur oftener than it does were it not that in these very cases the internal pressure is generally far lower than in health and the patients are incapable of increasing it by exercise.

Local dilatation is more apt to befall the auricles than the ventricles, and the right ventricle more than the left. The only positive signs of auricular dilatation are increased dullness and perhaps pulsation at the base of the heart; in the second left interspace if the left auricle be affected, in the third right interspace if the right auricle; corroborative evidence is afforded by finding in another chamber of the organ a cause to which the auricular dilatation can be attributed, and secondary it practically always is.

Dilatation of the right ventricle brings that chamber still more to the front than in health and, through rotation of the organ, the left ventricle may cease to form the apex. The visible pulsation, if any is visible, thus becomes diffuse, short, weak, and rapid, perhaps irregular; entirely different from the slow, heaving, powerful, and usually regular impulse of simple hypertrophy; the area of cardiac dullness, especially transversely and to the right of the sternum, is increased; the first sound is short and high pitched —valvular,—resembling greatly the second and distinguishable from it with difficulty. There may be a

marked discrepancy between the beats over the heart and in the radial artery, many of the imperfect contractions failing to reach the periphery. Tricuspid incompetency is apt to result from the dilatation alone, and may be betrayed by a systolic murmur, loudest over the seat of the tricuspid valves, the lower mid-sternal region; dilatation of the right auricle may be determinable by percussion. Evidences of venous stasis in and behind the lungs are usually not wanting. The causes are, in a word, increased resistance in the pulmonary circuit and a failure to establish compensatory hypertrophy.

The signs of simple dilatation of the left ventricle differ from those above described, in that the area of dullness is increased downwards and to the left; the impulse, if visible or palpable, is less diffuse and farther to the left; and the murmur, if heard, belongs to the mitral area, while percussion may show enlargement of the left auricle. The cause is undue resistance in the systemic circulation, or at the aortic orifice, and a failure to establish compensatory hypertrophy.

The above more or less special and somewhat theoretic considerations must be supplemented by some of a more general character.

As has been already stated, hypertrophy and dilatation are generally combined and dependent on a common cause, namely, increased resistance beyond the seat of the enlargement. Augmented internal

pressure tends at first to stretch the walls of the cavity, and the integrity of the circulation can then only be maintained by a proportional increase in the force of the muscular contraction of the chamber, or chambers, behind the cause augmenting the pressure. If this cause be in operation but a short time the resulting dilatation may subside without the supervention of hypertrophy, the demand for heart power being lessened by rest, and, perhaps, artificial means —acute dilatation. But if the cause be longer operative, the heightened demand can be met only by an increase in the muscular bulk—hypertrophy. If this is sufficient to dominate the dilatation, and thus reestablish the balance of the circulation, we speak of the lesion as "compensated." But compensation cannot take place unless there be a reserve nutritive power in the organism in general, and in the heart wall in particular; and it is obvious enough that all degrees of compensation are encountered in different cases, or even in successive stages of the same case. Whether hypertrophy or dilatation is predominant, depends on several factors, such as the rapidity with which the cause has come into operation, its permanency, its degree, the part on which it operates, its time of operation, the general and local reparative power of the individual, and the judiciousness of the treatment which he receives, or allows himself to receive. Other things being equal, a cause of insidious onset and very gradual increase, such as the high ten-

sion of chronic Bright's disease is apt to be, offers a better chance for compensatory hypertrophy; acute dilatation may subside without requiring much, if any, hypertrophy; great resistance demands more than slight; the ventricles, particularly the left, are capable of an increase in muscular power which is quite impossible for the thin-walled auricles to attain in even proportional measure; a hollow muscle will yield more readily to a strain applied while it is weakest than while it is strongest—during dilatation than during contraction; a muscle cannot increase in size and power unless the blood is good and can gain free access to it, unless the nervous centres which influence its nutrition are in fair condition and uninterrupted connection with it, and unless the muscular tissue itself is so far free from fatty, interstitial, or other pathological change as to be still capable of sufficient reparative growth.

It will thus be seen that, speaking broadly, hypertrophy is a conservative, dilatation a destructive, process. At the same time, in many a condition, a certain amount of dilatation is salutary, nay essential. The all-important thing is that hypertrophy be predominant.

The diagnosis of hypertrophy is easier than that of dilatation, in that there are fewer sources of error in determining the presence of enlargement with an increase in power than obtain in enlargement with diminished power. The powerful, heaving, and dis-

placed apex beat, and strong, slow, regular pulse are striking and distinctive enough; but changes in the lungs and in the pericardium may closely simulate the dilated heart. The writer has been asked to tap the pericardium in a case in which, after careful examination, he believed the signs to depend on dilatation.

B. DISEASES OF THE MUSCULAR STRUCTURE OF THE HEART NOT NECESSARILY ASSOCIATED WITH APPRECIABLE ENLARGEMENT OF THE ORGAN.

These processes offer some of the most difficult diagnostic problems which we are called upon to solve, and are oftentimes impossible of accurate solution with the means which we now have at command. They are: Myocarditis, acute and chronic; fatty infiltration and degeneration; aneurism of the heart; and new growths. All are liable to produce weakened heart action, and may, but by no means inevitably, cause modifications in the size of the heart.

Acute Myocarditis may be febrile, occurring, as it does, chiefly in the course of acute rheumatism or other infectious diseases. It may be associated with endo- and peri-carditis and can be diagnosticated only by relatively sudden cardiac weakness occurring under conditions known to predispose to acute inflammation of the heart muscle and as a cause for which endocarditis and pericarditis can be either confidently excluded or determined to be insufficient.

Chronic Myocarditis, or *Fibroid Degeneration*, may also produce the local and general signs of cardiac weakness; but is chronic and persistent, not acute. The diagnosis is alike surrounded with difficulties, and can only be made when other causes of chronic weakness, such as valvular disease and fatty degeneration can be excluded or fairly judged to be insufficient for the production of the signs and symptoms. It is well known that the connective tissue growth in adherent pericardium frequently invades the heart wall; if the latter condition can, therefore, be made out the existence of chronic myocarditis can be pretty confidently affirmed.

Aneurism of the Heart is due to a local giving way of a portion of the heart wall weakened or thinned by disease and consequently unable to withstand a pressure which the rest of the muscle can still support. It may arise acutely as the result of acute local myocarditis, ulcerative endocarditis, or more or less sudden impairment of blood supply; the chronic form is generally the result of chronic myocarditis. In either case rupture of the heart wall and speedy death may ensue. The cases are rare at the best, and there are no distinctive signs whatever on which the diagnosis can be based. It is, therefore, unnecessary to say more about them here.

New Growths of the Heart can be dismissed as quickly, having, if possible, even less clinical interest than aneurisms. The occurrence of pericarditis in a

cancerous patient for which no other reasonable explanation can be given might warrant a shrewd guess as to the presence of a secondary deposit in the substance of the heart.

Fatty Infiltration of the Heart, Fatty Overgrowth or Obesity of the Heart, is, in typical cases, to be sharply distinguished from fatty degeneration both clinically and pathologically. The muscular fibres are intact unless atrophy is brought about by the pressure of the fat layer between the bundles. This permeation of the wall with fatty tissue, which also envelopes it in a thicker or thinner layer, heightens the embarrassment of the circulation caused by the abundant subcutaneous, omental, and other fatty deposits. The physical signs are, for the most part, negative or indistinctive, the enlargement of the heart being often masked by the subcutaneous fat which may render percussion valueless. The diagnosis must consequently rest mainly on the general and local signs and symptoms of cardiac weakness or insufficiency in an obese person who usually is addicted to the excessive comsumption of malt liquors and leads a sedentary life. The cardiac insufficiency may be so great as to cause general dropsy. Cases are encountered in which fatty infiltration is combined with the graver condition next to be described.

Fatty Degeneration of the Heart is a term descriptive enough of the condition to which it is applied, and it is obvious that the contractile power of

the affected muscle must be impaired proportionally to the extent and degree of the change. Here, again, diagnosis must be largely indirect, if it can be made at all. We must have the signs of a chronically weak heart and exclude all other causes of that condition, such as dilatation, chronic myocarditis, adherent pericardium, and the like. Late middle or advanced life, atheroma, arcus senilis, and alcoholic excess are factors to be taken into account in making the diagnosis. A failure of suitable treatment by rest and cardiac tonics is considered by some a diagnostic point; this simply shows, however, that the lesion, whatever its nature, is beyond repair. Slowness of the pulse is not considered of as much diagnostic value as it was formerly. In grave anæmia, knowledge derived from autopsies enables us to diagnosticate fatty degeneration with certainty. All sorts of combinations of fatty degeneration with other pathological changes are encountered.

It will be readily seen that the positive diagnosis of the conditions comprised in the above division is nearly always very difficult and often impossible. Murmurs, especially in the mitral area, are often heard in them and may depend either on co-existent valvular disease or simply on dilatation and such inefficiency of the muscular contractions that relatively or absolutely healthy valve-curtains fail to close an auriculo-ventricular orifice during the ventricular systole. These latter murmurs will receive further con-

sideration under the head of functional disorders of the heart. So much has been said about cardiac weakness that it is, perhaps, well to sum up the signs of its presence. These are, in brief, an apex beat more or less evident to the eye, and more or less difused toward the right or displaced to the left, according to the amount of dilatation and the part of the heart most affected; or the apex beat may be entirely absent; the impulse to the finger is short and feeble, usually rapid, often irregular; the first sound is weak, short, and high pitched, and may be accompanied or replaced by a murmur; the second sound is generally also feeble, but may be accentuated in either the aortic or pulmonic area if the weakness is predominant in one or the other side of the heart rather than general. Basic murmurs, unless of hæmic origin, are less common than those in the mitral area. The pulse is feeble and some of the heart beats may fail to reach the wrist; the patient is incapable of much exertion, physical or mental; syncopal attacks are common; there may be cyanosis and other evidences of venous stasis.

We must be ever on our guard not to mistake a mere temporary or accidental weakness due to emotional or other causes for one that is real, chronic, or more or less permanent. One should hesitate, therefore, to make a positive diagnosis of one of the above described conditions, all of which are serious, while some are of the utmost gravity, after a single examination however thorough it may be.

C. VALVULAR DISEASE OF THE HEART.

The two main causes of valvular disease are endocarditis, largely of rheumatic origin, and atheroma. The first is chiefly operative in early life and affects the mitral valve by preference, though the others, and especially the aortic, are far from enjoying immunity; the latter is a senile change to which strain, syphilis, and alcohol predispose; and is more apt to extend to the aortic valves from the aorta itself than to originate in the former situation. The actual valvular damage is, in a sense, of less importance than the results which it entails to the heart-wall and to the organism as a whole, results largely of a mechanical nature. The immediate effects of damage to a valve are one of two, which may be found either singly or both in combination. That is to say, an impediment may be placed in the way of the onward passage of blood through a valve; or, from failure to close an orifice completely, leakage and a backward current are established. The former condition is called stenosis or obstruction; the latter regurgitation, incompetency, or insufficiency. Just as only one, or both of these conditions simultaneously, may be present at any orifice, so they may be confined to one orifice, or involve two or more at the same time. Whatever the nature or seat of the lesion, its broad results are the same, the differences being mainly in details. Whether a valve is stenosed or incompetent, the tendency is necessarily toward an

over-accumulation of blood immediately behind the lesion. In stenosis, the chamber behind cannot empty itself in systole with the same ease and in the same time as in health, and is thus ill-prepared for diastole; in regurgitation it receives a double instead of a single blood-supply during diastole, and starts in on the systole with an extra load of work to perform. In either case the tendency is for the part beyond the lesion to get less, the part behind to get more, than its share of the nutritive fluid, and is consequently toward over-accumulation in the pulmonic or systemic veins, or both, and a proportional deficiency in the arteries.

The differences depend on the seat of the lesion, and the period of the cardiac revolution during which the accumulation takes place. Aortic lesions manifest their effects primarily on the left ventricle—slightly, if at all, on the left auricle, lungs, and right cavities as long as the mitral continues to do its work. Mitral lesions react at first on the left auricle, the thin muscular walls of which can neither withstand notable pressure nor furnish compensation; the effects, consequently, are promptly reflected back through the lungs to the right ventricle, on which more hope can be placed; the work of the left heart is at first lightened, though it may ultimately be somewhat increased if the mitral disease involve regurgitation. Pulmonary valve lesions affect the right ventricle exclusively as long as the tricuspid holds tight; tricuspid lesions fall

upon the very weakest chamber of the whole heart, but are, fortunately, singly and alone, of the greatest rarity. In general, the thicker walled ventricles are better able to withstand increased pressure than the auricles. If the increased pressure comes during diastole dilatation is sure to occur, the strain coming on the relaxing muscles.

Now, in practice, while the above more or less dire results occasionally ensue promptly on the establishment of the lesion, they are, as a rule, delayed for a longer or shorter period, or may never appear at all, thongh the patient reach or surpass three-score years and ten. The reason for this is that the heart, like the other viscera, is rarely or never called upon to do continuously its maximum amount of work, and has a certain reserve power of growth for emergencies, such as a valvular lesion. If the reserve nutritive power of the heart in particular, and of the body as a whole, has been already exhausted through any cause or causes, or if advanced and irremediable tissue changes have very seriously compromised the heart muscle, repair is either impossible or can be but slight. Suppose, however, that the condition of the patient permits reasonable repair, and that he becomes the possessor of a valvular lesion. The overplus of blood in a heart cavity seems to act as a stimulus to contraction, which, in its turn, leads to more active circulation and consequently to improved nutrition and increased growth. Thus the dilatation, which

tends to be the immediate local effect in the cavity behind the lesion, is limited by hypertrophy; which, again, by overcoming the obstruction or pumping a larger quantity of blood so that the distal circulation still gets its needful supply, though some escapes backwards, restores a proper balance of blood-distribution. The lesion is then, as we say, compensated. The heart is not a normal heart, but it accomplishes a more or less normal amount of work nevertheless. How long it continues to do so, depends upon many circumstances, among which may be mentioned the degree of the lesion, its seat, its progressive or non-progressive character, the age, pecuniary resources, and character of the patient, who may or may not choose a wise adviser and follow the advice which he receives. Of course there is a wide variation in the degree of obstruction or regurgitation at an orifice in different cases, and there is also a difference in gravity according to which valve is affected, how it is affected, and how rapid has been the injury. It is not enough to diagnosticate the seat and character of the valve lesion itself; the degree of compensation is also to be determined in each case as far as possible, and, for prognosis perhaps even more than for diagnosis, the origin of the lesion is of great importance. We are now in a position to take up the lesions of the valves one by one, and begin with the valve most frequently affected.

Mitral Regurgitation, very common and frequently

uncombined, involves a back current during the ventricular systole from the left ventricle to the left auricle, which thus receives a double blood supply, and is subjected to increased internal pressure during its diastole, with dilatation as a result. A certain amount of hypertrophy can take place in the auricle, but not enough by itself to overcome the effects of any notable degree of leakage; the heightened pressure is thus thrown back on the right ventricle, in hypertrophy of which the main part of compensation must lie. The left ventricle, in cases of some standing, is usually somewhat dilated and hypertrophied, although its work would at first sight appear to be lessened. But the blood is delivered to it under increased pressure from the hypertrophied right ventricle, tending to dilate its cavity, to stimulate contraction and nutrition, and thus cause hypertrophy. Some increase in the size and power of the left ventricle is truly conservative inasmuch as all its contents are not passed on to the aorta, and more or less general arterial anæmia must result unless the whole amount dealt with is greater than in the normal condition.

In young subjects there may be marked prominence of the præcordial region, but this is rarely noticeable when the lesion was not established until the bony framework of the thorax had ceased active growth. A diffuse and more or less well-marked pulsation is to be seen and felt in the apex region,

a larger portion of the right ventricle lying in contact with the chest wall; and this pulsation may be found in the sixth space and well beyond the nipple line, the displacement outward depending mainly on changes in the size of the right, that downward of the left ventricle. A systolic thrill is felt by the hand in the mitral area in the smaller proportion of cases. The chief increase in the percussion dullness is transverse and toward the right, extending in extreme cases nearly or quite to the right nipple. Auscultation gives a systolic murmur, replacing or accompanying the sound, often heard all over the præcordia, but of maximum intensity in the mitral area; occasionally loudest about the third left interspace; in very rare cases, loudest in the back; transmitted to the left into the axilla, as far as the vertebral column, even completely round the chest; generally soft and blowing in character. Exertion may be necessary to develop the murmur. The pulmonic second sound is accentuated to a degree which is of some value in determining the freedom of regurgitation and the perfection of compensation. If the right ventricle is unequal to the extra work thrown upon it, it does not distend the pulmonary artery forcibly in systole, and the arterial recoil is relatively slight. The pulse is soft, and the patient pale from relative arterial anæmia. The growth of children thus affected may be markedly retarded and limited.

If compensation is defective or fails, the murmur

may diminish in intensity, or even disappear, a certain amount of force being requisite for its production, and pulmonic accentuation becomes less marked or absent. The action of the heart becomes rapid, often irregular and intermittent; cyanosis appears on the scene with other evidences of pulmonary and general venous engorgement. Ultimately the tricuspid may give way from dilatation and weakness of the right ventricle, giving rise to the signs of that condition to be later described. Freedom from symptoms and ability to withstand exertion are the clearest evidences of good compensation. It will be understood that the amount of leakage varies widely in different cases, with a corresponding variation in the degree of secondary changes. The murmur of mitral regurgitation due to structural lesion can be confused with that of a purely functional or more curable condition. The points of distinction between the two come better later.

Mitral Stenosis is quite common, especially in females, but is seldom uncombined with incompetency. In the pure state its mechanical effects behind the lesion are the same in kind, though greater in degree, as in regurgitation; those in front are somewhat different. The left ventricle tends to be ill-supplied with blood, which it can receive in the normal amount only by such increase in the rapidity of the current and such lengthening of its duration as will make up for the decrease in volume. Enlargement of the left ventricle is consequently absent.

Here, again, prominence of the præcordial region depends chiefly on the age of the patient when the damage was done, and on the elasticity of the thorax. The impulse is more or less diffused from or beyond its ordinary seat, especially toward the left sternal border, and a presystolic thrill in the apex region is often very marked. If the thrill is less distinct it may be felt only when the finger tips are applied very gently to the interspace, anything like firm pressure seeming to prevent its detection. The increased percussion dullness of the enlarged right ventricle, and perhaps also of the left auricle, can be readily made out; and auscultation in typical cases, gives striking results. The first sound is shorter and sharper than in health, and may be accompanied by a thump or roll which is not easily described but is suggestive to the practiced ear. Vibration of the blood as it passes the constricted orifice of the mitral flaps, and of the curtains themselves is heard by the ear as a murmur; usually rough, rasping, or blubbering, in character; of greatest intensity in, or a little to the right of, the seat of the normal apex; never propagated far and sometimes limited to a spot not much if at all larger than the chest piece of the stethoscope; prolonged diastolic or presystolic in time – that is to say it may occupy a large portion of the diastole or be limited to that part of it which corresponds to the auricular systole—presystolic—,the contraction of the cavity so increasing the blood pressure that a murmur, for

which the pressure was insufficient during the earlier part of the diastole, is caused. The second sound is often unusually distinct at or even beyond the apex, and may be reduplicated. In many cases all auscultatory evidence of stenosis is absent, or the murmur may come and go. If the stenosis is slight, or if the pressure in the pulmonary circuit is low from weakness of the right ventricle, direct signs are apt to be wanting. Accentuation of the second pulmonic at the base obeys the same laws as in mitral regurgitation. Deranged innervation, as shown by irregularity and intermittency of the heart's action, is very common, often extreme, variable from time to time; the rhythm may, however, be perfect. Patients are pale and, if the lesion was well established in early life, ill developed. The pulse is small and soft. The compensation is more easily deranged than in incompetency, but the signs of its derangement or rupture are essentially the same in kind, and the lesion is less compatible with longevity. Regurgitation is usually co-existent and the signs of that lesion may be perfectly manifest while those of the stenosis are ill defined or absent. The only murmurs with which that of mitral stenosis can be confused are those of aortic regurgitation and of tricuspid stenosis. The latter is rare and is always combined with mitral stenosis; the left ventricular enlargement and vascular signs of the former should promptly clear up any doubt which might at first be felt.

Aortic Regurgitation is a common lesion and often uncombined. It involves a backward passage of blood from the aorta into the left ventricle during diastole, the arterial supply being thus lessened while the internal pressure on the relaxing ventricle is increased by a double current which dilates its cavity and demands hypertrophy for compensation. A large blood wave is thrown with much force into the aorta, the coats of which are distended thereby; but, as soon as the pressure is relieved by the completion of the systole, a recoil proportional in strength to the distending force follows—unless the elasticity is impaired—and becomes still greater from the fact that the blood delivered to the aorta is not all retained within it or its branches, some flowing back to the ventricle. Thus the mechanical effects of the lesion are confined to the left ventricle and the arteries, if, and as long as, compensation is established. The cardiac physical signs are: prominence of the præcordia, sometimes very marked; a powerful heaving impulse; and an apex beat decidedly lower than and more or less to the left of the normal seat, perhaps in the seventh space and anterior axillary line. Sometimes a diastolic thrill can be felt at the base. The change in the size of the left ventricle brings this portion of the heart forward and downward in the main, increasing the vertical diameter. Percussion confirms the results of inspection and palpation and shows that the right border is little, if at all, changed. Ausculta-

tion gives a prolonged, generally soft and blowing, murmur throughout the diastole over more or less of the cardiac and immediately adjoining regions. The seat of maximum intensity varies, being sometimes in the aortic area, sometimes below this point over mid-sternum, rarely not far below the clavicle; not infrequently the murmur is comparatively feeble, or even inaudible, in the aortic area, but loud and distinct near the fourth left costal cartilage; these latter are the cases in which mitral stenosis might be confused with aortic insufficiency by a superficial observer. The murmur is always propagated downwards in the direction of the current producing it, toward the ensiform cartilage. It may replace or only obscure the second sound, according to its intensity, the transmission of the pulmonic second, and the involvement of all the aortic segments, or of only one or two; it is occasionally heard in the back. The first sound at the apex also varies in character. It may be very loud and booming; rather impure; accompanied by a murmur not dependent on mitral regurgitation apparently, but on vibrations in the valves set up by the very powerful ventricular contractions; or it may be lost altogether. The vascular signs are striking and distinctive, perhaps sufficiently so to permit diagnosis at the first glance without removal of the clothing from the chest. The visible arteries, and also some which are usually not visible, rise rapidly in systole and fall with equal rapidity in diastole.

If, as is apt to be the case when the lesion is of high degree and long standing, the arteries have become lengthened and tortuous, a lateral movement takes place in them, as may perhaps be most distinctly seen in the brachial, the temporal, or with the ophthalmoscope in the central artery of the retina. The aorta may then become permanently distended and easily felt in the supra-sternal notch, while atheromatous changes in the arteries generally, as well as in the aorta itself, are favored by the abnormal differences of tension to which they are subjected. The sensation conveyed to the finger placed on an artery corresponds exactly with the above. The pulse is quick, large, rises and falls rapidly—the water-hammer, or Corrigan's pulse. By laying the fingers across the wrist firmly the radial and ulnar, and even the interosseous, may simultaneously be felt strongly pulsating. These peculiar features of the pulse may be intensified by raising the arm vertically above the head of the patient, thus favoring the collapse by the addition of the force of gravity exerted on the column of blood between the wrist and the heart. Over the carotids and subclavians a systolic thrill may often be felt, and heard as a murmur; this does not necessarily indicate stenosis, but may be caused simply by the violence of the systole, just as an apex systolic murmur, under similar circumstances, may or may not be due to mitral regurgitation. Over the peripheral arteries generally, often even those of relatively small size,

a systolic sound is to be heard, and in strongly marked cases a double sound may be present in the crural. Capillary pulsation can often be beautifully seen by pressing a glass microscope slide against the mucous membrane of the everted lower lip; or after successive strokes with the finger on the skin, say of the forehead. It will be understood that many of the signs depend directly on the compensatory hypertrophy, and grow less distinct, or disappear with its decline and failure. A patient with aortic regurgitation has often a ruddy appearance and is capable of great muscular exertion without distress. Compensation is lost through failure of the left ventricle when dilatation advances and the mitral gives way; downward progress may be arrested for a time by hypertrophy of the right ventricle. Finally the tricuspid may begin to yield and the picture is identical with that of loss of compensation in mitral lesions. When compensation is ruptured in aortic lesions the outlook for its restoration is not as good as in mitral cases, though this rule, like all others, has its exceptions.

Aortic Stenosis is rarely found alone, being combined in varying proportion with regurgitation. When the valve segments are so fused that they form a sort of diaphragm with a central orifice, the amount of blood which flows back into the left ventricle cannot be very large. The lesion involving an impeded passage of blood from the left ventricle into the aorta, the increased pressure within the cavity of the former

tends to dilate it; but this dilating force is exerted against the contracting, not as in regurgitation against the relaxing muscle, and is consequently at a relative disadvantage. This tendency can be overcome solely by hypertrophy, which, to be truly compensatory, must be in exact proportion to the degree of obstruction. Thus the wall of the left ventricle may attain the thickness of an inch. Dilatation is measured rather by the freedom of a combined incompetency.

Inspection and palpation may show some prominence of the cardiac region—an apex beat lower than, and somewhat to the left of, the normal seat; a strong and heaving impulse; occasionally a systolic thrill in the aortic area. Percussion is chiefly important as showing that the right heart is not enlarged. Auscultation gives a systolic murmur, usually harsh and sometimes musical, loudest at and above the aortic area, transmitted into the aorta and great arterial trunks given off from the arch, sometimes to be followed down the left vertebral groove a greater or less distance in the dorsal region. The aortic second sound is feeble, if not replaced by a regurgitant murmur. The first sound at the apex is prolonged and intensified. Owing to the constriction at the outlet, a longer period is required for the emptying of the ventricle; the pulse is therefore slow, rises gradually under the finger, is rather hard, and usually regular. The steps of failing compensation are essentially the same as in aortic regurgitation.

Mere roughening of the cusps, or of the aorta itself, may give rise to a murmur and other signs indistinguishable from those of stenosis, especially if increased resistance in the peripheral circulation, due to arterial degeneration, renal cirrhosis, or other cause, has induced enlargement of the left ventricle. Even careful weighing of all the features of a case may not clear away all doubt as to the presence of a moderate degree of stenosis.

Lesions of the Right Heart can be discussed more briefly, in that they are not only far more rare than those of the left, but underlying principles should be so thoroughly understood by this time as to permit the taking of much for granted. As combined lesions they are generally secondary to those on the left side. Congenital defects will receive brief separate consideration.

Tricuspid Regurgitation is of considerable clinical importance, forming, as it does, one of the last steps of compensatory loss in left-sided muscular failure, of valvular or other origin. Behind this valve is practically no compensatory force. The valve may or may not be structurally altered; usually it is not, leakage being due to failure of the flaps to close, simply from dilatation of their ring of insertion, and feebleness of the muscular contraction. Long-standing and advanced lung disease, emphysema, and fibroid phthisis, may underlie the condition, as well as valvular disease of the left heart.

The apex beat is usually diffused to the right and left, and is often undulatory; percussion shows enlargement, especially toward the right and over the right auricle; the murmur is systolic, loudest at the right apex, not far propagated or heard in the back. It may be distinguished from a coexistent mitral regurgitant murmur by a difference in pitch and quality; the pulmonic second is not generally accentuated. The clinching evidence, however, is to be looked for outside the heart. The systolic blood-wave is transmitted through the incompetent orifice to the auricle, and thence to the contents of the veins emptying therein. The hepatic veins, being devoid of valves, offer no impediment to the wave, and the liver itself may then be felt to pulsate synchronously with the heart; this sign must not, however, be confounded with the motion which an enlarged right ventricle, separated from the left lobe of the liver only by the diaphragm, sometimes imparts to that organ. The external jugular veins, on the other hand, have valves beyond which the pulsation cannot extend unless they also have become incompetent, the delicate walls yielding to the greatly increased pressure. A true pulsation of the jugulars is to be distinguished from false pulsation and undulation by compressing the vein by the finger; if the motion be caused by the contraction of the right ventricle, it will persist between the finger and heart after compression; it thus affords an important and almost conclusive sign of

tricuspid leakage. Respiratory distension and collapse of the cervical veins ceases when the breath is held. Occasionally the venous valves are seated nearly or quite an inch above the mouth of the vein, and pulsation, while the valve still holds, may then be seen just above the clavicle. Distension of the jugulars from tricuspid insufficiency can often be increased by firmly pressing tne liver upward and backward.

Tricuspid Stenosis is always combined with mitral stenosis, and often with regurgitation at one or both orifices; there is also at the same time aortic stenosis in 25 per cent. of the cases according to Fenwick. Under these circumstances the only physical sign directly pointing to the presence of the lesion is a presystolic murmur at the right apex differing in pitch and quality from that in the mitral area. It has once been the good fortune of the writer to diagnosticate this complex state of affairs with adherent pericardium, hepatic cirrhosis, and slight granular kidney, and to have the diagnosis confirmed in full by autopsy. The tricuspid presystolic murmur was heard only during a brief period some three years before death. During these years the woman supported herself much of the time, entering the hospital several times to be patched up, but the tricuspid and mitral presystolic murmurs could not again be differentiated.

Pulmonic Regurgitation is a condition of theoretical rather than clinical interest. Its diagnosis could

rest only on the presence of a diastolic murmur at and below the base of the heart, and loudest near the pulmonic area, with right sided enlargement, all proof of aortic leakage and of left sided enlargement being at the same time lacking.

Pulmonic Stenosis is less rare than insufficiency, and usually congenital. Its signs are a systolic murmur loudest in the pulmonic area and not transmitted into the great vessels; but associated with right sided enlargement, generally youth, and a history or the presence of cyanosis on, or independently of, exertion. If it were more common it might frequently lead to error, the seat and time of the most conspicuous anæmic murmur being the same.

Congenital Valvular Lesions may be due either to endocarditis during fœtal life, to malformation, or to both; inflammation being more prone to attack a malformed than a normal valve. The right heart is their favorite seat, and pulmonic stenosis is by far the commonest lesion. If this is more than slight, compensatory relief to the circulation must be had by means of a patent foramen ovale ductus arteriosus, or inter-ventricular septum, the passage of the blood through which may produce vibration and murmur precisely as through a constricted or incompetent valvular orifice. This fact introduces an element of doubt into the interpretation of congenital murmurs from which acquired lesions of the heart are free. The history of the case may hence assume greater importance than in those cases

with acquired lesions. In a young person of more or less stunted development, who from earliest childhood has been cyanotic and short of breath, with clubbed fingers and toes, and who is free from any history of rheumatism or rheumatic pains, the other physical signs of valvular disease point with almost absolute certainty to lesions of the right side of the heart dating from intra-uterine life. There are again other cases, widely differing in character from those just mentioned, which can safely be classed as congenital. A young person, namely, may present none of the rational signs of heart disease, and the heart may be but little if at all enlarged, and yet a persistent and very loud murmur may be heard at the base of the heart, over a large portion of the chest, nay on the top of the head and almost everywhere over the trunk, not attributable to aneurism or any other cause outside of the heart itself; it may also be heard at a distance from the chest and give rise to an extended thrill. In such case the defect is either a slight one —it is always to be remembered that the loudness of a murmur is no index of its gravity—or the compensation is so perfect that no inconvenience results, perhaps for a long period of time.

Having now concluded the physical signs and diagnosis of chronic valvular lesions we must next consider:

ACUTE ENDOCARDITIS, SIMPLE AND MALIGNANT.

Acute Simple Endocarditis is ordinarily to be diagnosticated rather by the circumstances under which the physical signs appear, than by the physical signs themselves. If, for instance, during the progress of acute rheumatism an endocardial murmur develop at the mitral or aortic area, persist, and is followed by appropiate enlargement of the heart, it is clear that inflammation of the valvular endocardium was present. If, under the same circumstances, a mitral murmur comes, lasts for a variable time, but disappears without leaving traces behind, one cannot be sure whether the murmur was due to inflammation of the endocardium, of the myocardium, or simply to dynamic weakness. A later development of a valvular lesion without a recurrence of the rheumatic attack would settle the question in favor of endocarditis.

Malignant Endocarditis is an acute process, the pathological relations of which to the simple form are still unsettled. The physical signs in the heart itself are in no wise distinctive, and may be very slight or even entirely absent. For a description of the symptoms which often warrant a positive diagnosis this is not the place. The writer has seen two clear cases, one with post mortem, in which malignant endocarditis was the result of gonorrhœa.

D. CONDITIONS CHARACTERIZED SIMPLY BY ABNORMAL RATE OR RHYTHM, BY MURMURS, OR BY MODIFIED SOUNDS—FUNCTIONAL DISORDERS OF THE HEART.

The above peculiarities of cardiac action have been mentioned many times in connection with the various organic lesions of the organ. They are often, however, encountered in hearts which are free from serious or permanent changes; altered rate and rhythm chiefly as a result of faulty innervation, murmurs in connection with altered blood states and transitory weakness of the heart muscle.

The action may be abnormally slow—30 or even less; or abnormally rapid—160 to 200, under the influence of causes which are usually transitory. Less extreme degrees of slowness or rapidity may be of long duration, as in the habitually slow pulse of certain individuals, or in exophthalmic goitre where the heart may or may not be structurally sound, though its rate is greatly quickened. Irregularity and intermittency are often of little or no consequence, as, to take an extreme case, in a sleeping child.

In most cases of rate and rhythm change the stethoscope over the heart should control the evidence of the radial pulse, and it is advisable in cases which are not perfectly clear to suspend positive diagnosis until examination can be repeated. The habits, general condition of the patient, state of all his other

organs and tissues, etc., must receive careful consideration; and organic lesions of the component parts of the heart itself must be excluded. Thus only can we escape the serious blunder of mistaking myocarditis, fatty degeneration, or other grave structural change for functional disorder. A full discussion of this branch of our subject would lead us far beyond the scope of this little work.

The great Laennec recognized the fact that hearts over which murmurs are heard during life are sometimes found structurally intact after death, and much ingenuity and discussion have been expended on the elucidation of the mechanism of these murmurs without enabling us yet to arrive at perfectly clear knowledge. The tendency is to-day to attribute nearly all murmurs in the mitral area to regurgitation, the important practical question thus becoming whether in a given case the murmur is due to remediable or to irremediable causes, or, as Dr. George Balfour puts it, whether it is curable or incurable. A structural valvular lesion due to endocarditis which has passed the acute stage is, with few exceptions, permanent; an acute lesion may however, either undergo complete repair, or it may seem to do so but really lay the foundation of mischief which betrays itself long after. It will thus be seen that we are obliged to content ourselves with a classification of apex murmurs which is clinical rather than pathological.

The basic functional murmurs, so called, are the subject of less dispute, and are generally associated with anæmia, primary or secondary, and often with a venous hum in the neck which may be heard on one side only, or on both simultaneously. Their favorite seat is the pulmonic area; they are systolic, and rarely far transmitted. They may be heard less distinctly at the apex; sometimes anæmic murmurs are heard only there, and not at all at the base. In their diagnosis the same precautions must be taken as in the interpretation of changes in rate and rhythm.

One other class of murmurs, the cardio-respiratory, deserves mention as possibly leading to error. They are associated with some phase of the respiratory act, are systolic in time, disappear or are greatly modified when the breath is held, and are usually loudest at the end either of inspiration or expiration. They are not necessarily connected with appreciable changes in the lungs, and are analogous to the subclavian systolic whiff heard in some cases of apex phthisis and pleurisy, as well as in the absence of those diseases.

The following table may aid in the diagnosis of this whole class of murmurs:

THE SERIOUS MURMURS.	THE NON-SERIOUS MURMURS.
Occur at any time in the cardiac revolution, either at the base or apex.	Are systolic in time, and more common at the base.

THE SERIOUS MURMURS.	THE NON-SERIOUS MURMURS.
Are apt to be associated with rheumatism or its history, or with degenerative changes in other parts or organs.	Are usually associated with anæmia, fevers, nervous excitement, or respiration.
Have definite lines of propagation.	Have no definite lines of propagation.
Are persistent.	Are usually transitory.
Involve more or less well-marked enlargement of the heart.	Involve no marked enlargement of the heart.

CHAPTER III.

THE PERICARDIUM.

Pericarditis, inflammation of the pericardium, is quite analogous in itself and in its results to inflammation of other serous membranes, and especially of the pleura, though the physical signs differ, of course, in some important respects. Just as in pleurisy, so here, we must distinguish between local, dry, and adhesive pericarditis, and that which is attended by appreciable effusion. A division into the acute and chronic forms is less important from our point of view.

In the *dry form* the pericardial surfaces no longer glide freely upon one another as the heart contracts, dilates, and rotates. Inspection shows nothing more than, perhaps, quickened respiration with a rapid or irregular action of the heart; palpation may show tenderness of the præcordia, and friction fremitus; percussion is negative. The distinctive physical signs are auscultatory, and the chief of these is friction, most commonly heard over the front of the right ventricle where it nearest approaches the chest wall, double,—to and fro—systolic and diastolic, superficial, intensified by pressure with the stethoscope, not always perfectly synchronous with the heart sounds, sometimes single. The friction may obscure the heart sounds, or, if the action is rapid, these are apt to be

short and weak; disordered rhythm is common. Pericardial friction should persist when the breath is held, but even this test does not distinguish with certainty a pericardial rub from one arising in the pleura overlying the heart, and excited by the movements of that organ. Pericardial inflammation is often secondary to left pleurisy, and the co-existence of the two enhances the difficulty of the diagnosis. But acute pericarditis may be entirely latent as far as direct physical signs and symptoms go. The writer has examined the cardiac area with the greatest care twice daily for more than a week, having good reason to believe that dry pericarditis was present, without hearing friction; yet the autopsy showed general recent adhesions.

Adherent pericardium involves, of course, obliteration of the cavity either in whole or in part, is apt to derange the function of the organ, and may lead later to advanced myocarditic changes. If the adhesions are limited to the visceral and parietal layers of the sac their presence cannot be diagnosticated during life. They may give rise to no symptoms at all, or any symtoms to which they do give rise are in no way distinctive. If, however, the external surface of the pericardium is densely adherent to the chest wall in front, suggestive signs may be present. Such are systolic retraction of the apex region with diastolic rebound; recession of the epigastrium on deep inspiration, the area of cardiac flatness and the position of

the apex being unchanged thereby; immobility of the heart on change of position of the patient; and diastolic collapse of distended veins in the neck. The first of these signs, systolic retraction of the chest wall, cannot be looked for if the costal cartilages are ossified; and may be ill-marked or absent if the heart's action is feeble.

Pericardial Effusion. — The pericardium is a closed sac which extends up a certain distance on the great vessels, and is thence reflected. As fluid is thrown out it will naturally, unless limited by adhesions, occupy the lowest portion of the sac containing it, and, as it increases, will tend to lift the heart upward and forward and to tilt it to the left. Previous enlargement or fixation of the heart, or emphysematous, fixed, or consolidated edges of the lung in the cardiac region, will modify this tendency in ways which are readily understood. It will also be seen that the physical signs must vary according to the amount of the effusion; and that, if this is absorbed, they will during absorption follow an inverse order of sequence to that which characterized the stage of increasing effusion.

Inspection shows marked prominence of the præcordia only in patients with elastic chest-walls, notably in young subjects and in very large effusions; in the latter case there may be distinct bulging at the epigastrium, with dislocation of the liver and of other organs lying immediately below the diaphragm. The

apex beat is raised and moved somewhat outwards, being seen sometimes in the fourth, or even in the third, space; it may be indistinct or absent, according to the quantity of the fluid and the presence or absence of complications in the heart itself or the adjacent portion of lung.

Palpation confirms the results of inspection, and may detect friction over the superficial portion of the sac above the level of the fluid. The seat and intensity of the friction may undergo change with change in the position of the patient. Percussion shows increase in the flat and dull areas which, in large and uncomplicated effusions, presents a characteristic shape. The lowest is the most distensible part of the pericardial sac; flatness begins, therefore, to increase here, and, with a mounting effusion, grows laterally and rises upward till it assumes a more or less well-marked pear shape, the smaller part of the pear corresponding to the reflection of the sac from the great vessels. The experiments of Rotch indicate that flatness in the fifth right interspace extending as much as three centimetres beyond the sternal border occurs in pericardial effusion, but practically never in enlargement of the heart. Dislocation may produce it, but this possibility is really excluded by the absence of any cause for such dislocation. Auscultation shows a gradual ascent and disappearance of the friction if the case be under observation and correctly diagnosticated from the start. The heart-sounds become

muffled, distant, and indistinct, the first being short and valvular owing to the embarrassment and weakening of the ventricular contractions resulting from the disadvantage at which the heart works, and perhaps coexistent endo- and myo-carditis. Endocardial murmurs may or may not be present; if they, and left dry pleurisy, are both associated with pericardial friction, the auscultatory phenomena may be of a highly complicated character.

Orthopnœa, cyanosis, quick, and rhythmically disordered action of the heart may be present according to the severity of the inflammation, the rapidity and the amount of the effusion, and the co-existence of complications. The paradoxical pulse, absence of the pulse-wave during inspiration, is not strictly confined to pericarditis. Large pericardial, like large pleural effusions, may be remarkably latent as far as symptoms are concerned; but the latency has, of course, no influence on the direct physical signs.

The chief source of error in diagnosis is the dilated and weak heart.

Hydropericardium, or dropsy of the pericardium, gives rise to the same physical signs as inflammatory effusion except that friction is less common while dropsical transudation is elsewhere more constant in cases of the former.

Rheumatism, left pleurisy, and other causes of pericarditis are to be taken into account in establishing the diagnosis.

Pneumo-hydro- or pneumo-pyo-pericardium is a rare condition the nature of which is sufficiently indicated by its name. The liquid seeks the lower, the air the upper portion of the sac, and the two change place with change in the position of the patient unless adhesions prevent. Splashing and churning sounds are heard on auscultation over the cardiac area, in more or less regular synchronism with the heart beat.

CHAPTER IV.

THORACIC ANEURISM.

Aneurismal dilatation of the aorta may occupy any portion of the vessel, but is most common in the ascending, and thence onward progressively diminishes in frequency; it may involve only part of the circumference —saccular, the whole circumference of a limited segment —fusiform, or the dilatation of the aortic arch may be pretty general as the result of atheroma and aortic regurgitation; the latter should not, strictly speaking, be classed as aneurismal. The affection befalls by preference the male sex and early middle life, men during this period being more exposed to the strain of muscular exertion acting in conjunction with weakening of the vascular walls due to degenerative changes set up by syphilis and the abuse of alcohol. A more or less local yielding of the arterial coats to the blood pressure tends to increase in size and to form a tumor, pulsating synchronously with the heart, displacing, pressing on, or destroying, the tissues and parts in its immediate vicinity. The symptoms and signs must, therefore, vary with and depend on the portion of the aorta affected, the size of the aneurism, and the aspect of the vessel from which it springs. It is customary to divide the signs

into two main classes; the direct, those due immediately to the tumor itself; and the indirect, those due to the pressure which it may exert on neighboring structures or parts. The first class is encountered especially when the aneurism springs from the anterior or lateral portions of the ascending or transverse aorta and grows more or less forward, the latter when it extends inward and backward toward the root of the lungs or rises from the inner or posterior aspect of the vessel. One class only of signs, or both simultaneously, may be represented.

1. *Direct Signs.*—Inspection reveals pulsation or a pulsating tumor under, to the right, or under and to the right of the sternum, near to or above the base of the heart. The pulsation is synchronous with the cardiac impulse, which occupies its normal seat unless the heart be enlarged or displaced from some other cause. It is important to remember that an aneurism does not usually in itself throw extra work on the heart and thus lead directly to enlargement of that organ, though arterial degeneration may be an underlying cause of both. The pulsation may be limited in extent, or more diffuse and prominent, according to the size of the aneurism and the amount of absorption of the sternum and ribs which it has caused.

Palpation may detect a pulsation which is not visible, or confirms and enlarges the results of inspection. The pulsation is expansile, equal alike at the summit and sides of the tumor, which is to a degree

compressible, especially if it is not lined with a thick layer of laminated clot. A thrill, usually systolic, is not uncommon.

Percussion gives dullness or flatness over the pulsation or pulsating tumor according to its size and the relation which it bears to the lung tissue; or there may be localized dullness although inspection and palpation are negative; or percussion also may be negative. The reasons for these differences are obvious.

Auscultation also yields variable or negative re-results. In the first place, nothing abnormal may be heard; secondly, normal heart sounds of unusual intensity for that place may be found over the seat of the aneurism, the second sound being especially distinct; thirdly, there may be a murmur or murmurs, and these may be generated either in the sac itself or at one or more of the cardiac orifices. A murmur originating in the sac is apt to be systolic, but may be double; one arising at a cardiac orifice is generally associated with enlargment of the heart unless it is due simply to a little roughening of the aortic valves. The results of auscultation alone are, therefore, not to be much depended on.

2. *Indirect Signs.*—These vary greatly with the direction in which pressure is exerted, and may consequently afford testimony of extreme value as to the presence and seat of a thoracic tumor while most of them throw no special light on its nature. Re-

tardation of the systole in the peripheral arteries as compared with the apex beat may occur in some cases of aneurism of the ascending aorta; or retardation, weakening, or obliteration, may be noted in the right or left radial as compared with its fellow if the aneurism involve the innominate, or is seated between the innominate and the left subclavian, or involve the latter, respectively. Such delay or weakening of the pulse, can generally be appreciated by the finger.

Pressure on the left innominate vein or the inferior cava may be caused if the transverse or ascending portions are involved, and is manifested by distention of the branches emptying into these trunks, turgescence of the face, œdema, etc.

Pressure on the left recurrent laryngeal nerve which winds round the transverse portion of the arch is common in aneurisms of that part. The corresponding vocal cord may be either paralyzed or spastic according as the pressure irritates the nerve or destroys its conducting power, and aphonia, hoarseness, and stridor may result. The laryngoscope is then an important aid to diagnosis.

Pressure on a primary bronchus may also cause stridor, but without laryngoscopic change; also unilateral dullness on percussion, enfeeblement of the respiratory murmur, and whistling respiration.

Pressure on the œsophagus is liable to give rise to dysphagia, and the seat of obstruction may sometimes be localized by auscultation while the patient

swallows. The passage of a bougie may, in such a case, result in rupture of the sac.

Pressure on the sympathetic may cause inequality of the pupils and vaso-motor changes on one side of the face and neck, both together being much more suggestive than either one alone.

Pressure on the bodies of the vertebræ, the sternum, and the ribs, tends to cause erosion and absorption of the bone and cartilage; vertebral pressure is especially apt to be accompanied by severe and boring pain, which, in the appropriate part of the dorsal spine, should always awaken suspicion.

In cases of more or less general dilatation of the aorta without the formation of a true aneurismal sac, the pulsation of the transverse portion may be felt by pressing the finger behind the sternal notch, and percussion is usually dull over the upper piece of the sternum.

Aneurism of the Innominate or Subclavians is seated higher and farther to the right or left than one springing from the aorta itself.

If a tumor is present the equal expansion in all directions with the cardiac systole is the most important evidence of its aneurismal nature. All the other signs, direct or indirect, may be produced by any solid intrathoracic tumor which may border or encroach on any portion of the course of the aorta. But aneurism far surpasses in frequency all other varieties of tumor in this situation; and, in doubtful

cases, the predisposing causes of aneurism are to be taken into account. Aneurism, moreover, does not impair the general health and nutrition unless it wears the patient out by pain or prevents sufficient ingestion of food through pressure on the œsophagus. Aneurism, again, is more liable to undergo temporary variation in size—from variations in the blood-pressure behind it—than any other solid tumor, with a corresponding and more or less rapid change in its indirect signs and symptoms.

Pulsating pleurisy, almost invariably left-sided; malposition of the aorta from rickets; malformation of the chest; a dilated auricle; unusual retraction of the lung normally overlying the heart; and lung consolidation, especially near the pulmonary artery, are all possible sources of error.

Aneurism of the Abdominal Aorta gives rise to much the same direct signs as that of the thoracic; but the anatomical relations of the sub-diaphragmatic portion of the vessel are such that the chief indirect symptom is pain from vertebral pressure and erosion. A solid tumor—gastric cancer, for instance—may overlie the vessel and transmit its pulsation; but lateral expansion is in such a case lacking, and the pulsation may disappear or become less distinct when the patient is put on his hands and knees. In very thin persons the accessibility of the aorta may occasion serious error, which is also not infrequently caused by unusually marked pulsation of neurotic origin in the

healthy vessel or antero-posterior spinal curvature. Every physician has seen great unhappiness caused by the lack of sufficient care in excluding the above innocuous conditions.

CHAPTER V.

PHYSICAL EXPLORATION OF THE LIVER, SPLEEN, STOMACH, AND PANCREAS.

Changes in these organs, which lie in more or less close juxtaposition to those of the thorax are liable to modify the physical signs of the latter and thus lead to error. A brief consideration of such changes seems, therefore, to be in order. Here, again, accurate knowledge of the normal anatomical relations as well as of the limits of normal variation is of prime importance.

THE LIVER.

The liver occupies chiefly the right upper abdomen, lying immediately beneath the arch of the diaphragm, and, speaking broadly, filling the whole space between the diaphragmatic arch and the right costal border, though in the back and side it extends only to the eleventh rib. The left lobe extends into and across the epigastrium, covered in its upper portion by the heart, in its lower to a varying degree by the stomach.

Percussion and Palpation in Health.—The upper border can be outlined with considerable accuracy anteriorly and laterally. In the front dullness begins at the lower edge of the fifth rib, flatness on the sixth,

these lines running parallel and nearly horizontal outward and backward and intersecting the ribs successively as these run downwards and forwards. These lines are naturally somewhat modified by the ascent and descent of the diaphragm in forced expiration and inspiration. The lower border can generally be distinguished throughout nearly its whole extent from the resonant digestive canal by very gentle percussion.

In the perfectly normal condition the organ cannot be felt. The gall bladder is not accessible to percussion or palpation.

Percussion in Disease.—Modifications in the size of the liver may be apparent or real, general or local. The modes in which disease above the diaphragm may alter the relations of the organ have been already indicated, but the whole subject of change in hepatic percussion is well epitomized by Weil as follows:

1. The lower border is normal.

(*a*). The upper border is high; enlargement of the liver upward; moderate pleural effusion, the dullness of which joins that of the liver; enlargement of the liver with coincident displacement upward, as in hyperæmia or amyloid disease with ascites.

(*b*). The upper border is depressed; moderate emphysema. In such a case the height of the dull hepatic zone above the pneumono-hepatic line is normal or increased.

2. The lower border is depressed.

(*a*). The upper border is raised; great enlargement of the liver; large pleural effusion.

(*b*). The upper border is normal; enlargement of the liver; anomalous position of the organ, as in fatty or corset liver.

(*c*) The upper border is depressed; great emphysema; pneumothorax; in either case the relative dullness above is wanting or diminished.

3. The lower border is too high.

(*a*) The upper border is high; displacement of the liver upward.

(*b*). The upper border is normal; atrophy of the liver; obliquity of position without marked displacement.

4. The hepatic flatness is absent.

Oblique position of the liver with meteorism and ascites; intervention of intestinal coils containing gas, or of free gas in the peritoneum, between the convex surface of the liver and the abdominal wall.

5. Change of the hepatic flatness to the other side of the body in cases of visceral transposition.

Great enlargement may give rise to dullness and feeble respiration in the right, or even in both backs. This the writer has seen exemplified in a case of hydatids of the liver as well as in other affections.

The lower border of the healthy organ may be depressed to a greater or less degree by thoracic disease, or by sub-diaphragmatic abscess, for instance; or it may become accessible to touch as a result of

enlargement, either general or local. If the abdominal wall is lax the whole contour of the lower border anteriorly and laterally, the shape and density of its edge, and the character of a varying portion of the surface as regards smoothness or roughness, can be well made out. The descent of the edge against the fingers on deep inspiration is often wonderfully distinct, and peritoneal friction can sometimes be felt over the liver when the portion of the membrane investing that organ is not too acutely inflamed. The friction may also be heard with the stethoscope. In some cases of ecchinococcus disease the hydatid thrill may be felt. In many cases the hyperæsthetic rigidity of the abdominal muscles, tension of the hepatic capsule, or actual inflammation, render it either difficult or impossible to obtain positive results from palpation, which should always be used as a control method to percussion in cases of suspected hepatic enlargement or displacement. Repeated examination may remove doubts engendered at first. In cases of moderate ascites the fingers, by a quick but gentle thrust, can often be made to penetrate the fluid and reach the surface of the enlarged liver; if the ascites is great and the abdominal wall tense an examination immediately after paracentesis may be necessary in order to enable us to determine the size and surface of the liver.

The determination of the cause of either atrophy or enlargement involves a careful study of the special

features of the change, of the history of the case, and of the rest of the organism. It therefore does not lie within our province here.

A distended *gall bladder* may become accessible to sight, percussion, and touch; forming a rounded tumor, dull on percussion, elastic or fluctuant, in the angle between the lower edge of the liver and the outer edge of the right rectus abdominis muscle. In some cases the tumor can be diminished in size by pressure, the contents thus being forced out.

THE SPLEEN.

This organ lies deep in the left hypochondrium, its long diameter being directed obliquely from above and behind, downwards and forwards. Its upper end lies under the diaphragm near the body of the tenth dorsal vertebra and is covered by the edge of the left lung; the lower end lies beneath the tip of the eleventh rib just behind the mid-axillary line; the transverse diameter extends from the ninth to the eleventh ribs.

Percussion and Palpation in Health.—Examination can be made either in the dorsal, right lateral, or erect position, the two latter being the more convenient. Palpation is negative in health. The upper third of the organ lies so deep that it is not accessible to percussion, and the relations of the rest of the spleen to air-containing parts are such that its limits are ordinarily determined by dullness rather than flatness. Moreover, the gaseous or solid contents

of the stomach or intestines may either mask or apparently increase the splenic dullness. It is, consequently, unsafe to rely implicitly on the results of a single examination. The area of splenic dullness is, then, in the erect posture, bounded by the scapular and mid-axillary lines between the ninth and eleventh ribs, and measures 7–8 centimeters in its long by 5–6 centimeters in its short diameter. In the right lateral position the boundaries of the dullness are slightly different, and it may be useful to contrast the results of the two. But, of course, the erect position is often inadmissible. The organ descends somewhat with forced inspiration which diminishes its area of dullness by distending the portion of the left lung which overlies its upper and posterior border.

Percussion and Palpation in Disease.—Downward dislocation may be produced by intrathoracic disease which depresses the diaphragm, such as left pleural effusion or pneumothorax, and advanced emphysema; upward dislocation by great tympanites, ascites, or any cause which unduly elevates the diaphragm. In the condition known as "floating spleen," the organ is generally, though not necessarily, enlarged, and its normal dullness disappears inasmuch as it sinks more or less into the abdominal cavity, and can there be felt. Apparent increase in the size has been already alluded to. In actual enlargement the shape of the normal dullness is closely preserved, but its size is increased in all directions. If the enlargement is mod-

erate, the organ may be felt under, or in front of the rib margin only at the end of deep inspiration, provided that palpation is not rendered nugatory by hyperæsthesia, tenderness, or abdominal distension. In great enlargement distinct fullness of the left side of the abdomen may be evident to the eye, while the non-resonant organ may be outlined by palpation, its notches felt, and its inspiratory descent determined. To distinguish positively between splenic tumor and enlargement, or tumor of the left kidney, it may be necessary to inflate the colon with air; if the tumor is of renal origin, it is then traversed by a belt of resonance.

THE STOMACH.

Scarcely any organ in the body varies so widely physiologically, as regards size and the character of its contents, as the stomach—a fact which renders the determination of its pathological conditions by the ordinary methods of physical exploration a matter of the greatest difficulty. Its proximity to the transverse colon is another pitfall. When the organ is moderately distended, and the person lies on the back, a small part of the anterior surface and the larger part of the greater curvature are in contact with the abdominal wall, the rest of it being covered by the left lobe of the liver and by the left lung. The pyloric portion lies in the right half of the epigastrium, barely reaching the right costal arch. The lower border crosses the epigastrium in a curved line nearly mid-

way between the ensiform cartilage and the navel. Percussion over this portion of the organ, which is more or less accessible to direct examination, is usually tympanitic, of varying intensity and pitch. But the truth is that gastric percussion, even auscultatory percussion, is liable to so many sources of error as to be practically valueless for fine diagnostic purposes unless the stomach is artificially inflated. This is best done by passing the sound, and then pumping in air with a Davidson syringe. The outlines of the organ in the natural and distended conditions may then be contrasted, and the lower limit is often evident to the eye, in cases of great dilatation nearly as low as the pubes. If the abdominal wall is lax and the stomach dilated, gastric peristalsis may be distinctly seen. Swashing over the stomach is common enough during gastric digestion; to acquire any pathological significance it must be persistent, and present at a time when the organ should be empty or nearly so. Tumors seated in the anterior wall or the pyloric region may often be felt.

THE PANCREAS.

This organ lies so deep in the abdomen that it is practically never accessible to physical examination in health, and rarely in disease It extends transversely from the hilus of the spleen to the concavity of the duodenum, at the level of the first lumbar vertebra, overlies the aorta, and is covered by the stomach

and the left lobe of the liver. It therefore corresponds to a zone on the external surface, the lower margin of which is about an hand's-breadth above the umbilicus. The only affections of the organ which can well give rise to changes sufficiently gross to produce local physical signs, are cancer and cysts. The former may, if it form a tumor over or bordering on the aorta, simulate aneurism; the latter may cause a more or less dull tympanitic bulging at the epigastrium, somewhat similar to that which is seen in some cases of double pleural, or pericardial fluid accumulation. In an extreme case a pancreatic cyst may extend below the navel.