casionally the splashing of the fluid within may be felt as well as heard. It is essential, of course, to distinguish succussion due to the presence of air and fluid in the pleural cavity from similar sounds produced in the stomach, but this is not at all difficult in the majority of cases. It is a bare possibility that succussion sounds may be due to the presence of air and fluid in the pericardial cavity.

It is important to remember that succussion is never to be heard in simple pleuritic effusion or hydrothorax. The presence of air, as well as liquid, in the pleural cavity is absolutely essential to the production of succussion sounds.[1]

(2) *Metallic Tinkle or Falling-Drop Sound.*

When listening over a pleural cavity which contains both air and fluid, one occasionally hears a liquid, tinkling sound, due possibly to the impact of a drop of liquid falling from the relaxed lung above into the accumulated fluid at the bottom of the pleural cavity, and possibly to râles produced in the tissues around the cavity. It is stated that this physical sign may in rare cases be observed in large-sized phthisical cavities as well as in pneumohydrothorax and pneumopyothorax.

(3) *The Lung-Fistula Sound.*

When a perforation of the lung occurs below the level of the fluid accumulated in the pleural cavity, bubbles of air may be forced out from the lung and up through the fluid with a sound reminding one of that made by children when blowing soap-bubbles.

[1] It is well for the student to try for himself the following experiment, which I have found useful in impressing these facts upon the attention of classes in physical diagnosis: Fill an ordinary rubber hot-water bag to the brim with water. Invert it and squeeze out forcibly a certain amount (perhaps half) of the contents, by grasping the upper end of the bag and compressing it. While the water is thus being forced out, screw in the nozzle of the bag. Now shake the whole bag, and it will be found impossible to produce any splashing sounds owing to the fact that there is no air in the bag. Unscrew the nozzle, admit air, and then screw it in again. Now shake the bag again and loud splashing will be easily heard.

CHAPTER VIII.

AUSCULTATION OF THE HEART.

I. "Valve Areas."

In the routine examination of the heart, most observers listen in four places:

(1) At the apex of the heart in the fifth intercostal space near the nipple, the "*mitral area.*"

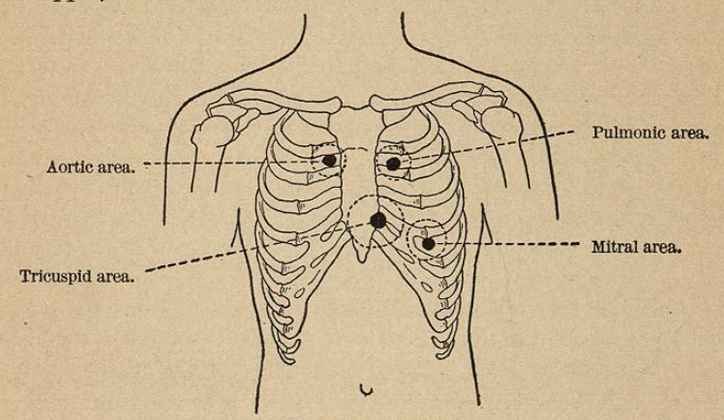

FIG. 108.—The Valve Areas.

(2) In the second left intercostal space near the sternum, the "*pulmonic area.*"

(3) In the second right intercostal space near the sternum, the "*aortic area.*"

(4) At the bottom of the sternum near the ensiform cartilage, the "*tricuspid area.*"

These points are represented in Fig. 108 and are known as

"*valve areas.*" They do *not* correspond to the anatomical position of any one of the four valves, but experience has shown that sounds heard best at the apex can be proved (by post-mortem examination or otherwise) to be produced at the mitral orifice. Similarly sounds heard best in the second left intercostal space are proved to be produced at the pulmonary orifice; those which are loudest at the second right intercostal space to be produced at the aortic orifice;[1] while those which are most distinct near the origin of the ensiform cartilage are produced at the tricuspid orifice.

II. The Normal Heart Sounds.

A glance at Fig. 109, which represents the anatomical positions of the four valves above referred to, illustrates what I said above; namely, that the traditional valve areas do not correspond at all with the anatomical position of the valves. If now we listen in the "*mitral area*," that is, in the region of the apex impulse of the heart, keeping at the same time one finger on some point at which the cardiac impulse is palpable, one hears with each outward thrust of the heart a low, dull sound, and in the period between the heart beats a second sound, shorter and sharper in quality.[2]

That which occurs with the cardiac impulse is known as the *first sound;* that which occurs between each two beats of the heart is known as the *second sound.* The second sound is generally admitted to be due to the closure of the semilunar valves. The cause of the first sound has been a most fruitful source of discussion, and no one explanation of it can be said to be generally received. Perhaps the most commonly accepted view attributes the first or systolic sound of the heart to a combination of two elements—

(*a*) The contraction of the heart muscle itself.

(*b*) The sudden tautening of the mitral curtains.

Following the second sound there is a pause corresponding to

[1] For exceptions to this rule, see below, page 235.

[2] The first sound of the heart, as heard at the apex, may be imitated by holding a linen handkerchief by the corners and suddenly tautening one of the borders. To imitate the second sound, use one-half the length of the border instead of the whole.

the diastole of the heart. Normally this pause occupies a little more time than the first and second sounds of the heart taken together. In disease it may be much shortened.

The first sound of the heart is not only longer and duller than the second (it is often spoken of as "booming" in contrast with the "snapping" quality of the second sound) but is also considerably more intense, so that it gives us the impression of being accented like the first syllable of a trochaic rhythm. After a little practice one grows so accustomed to this rhythm that one is apt to rely upon

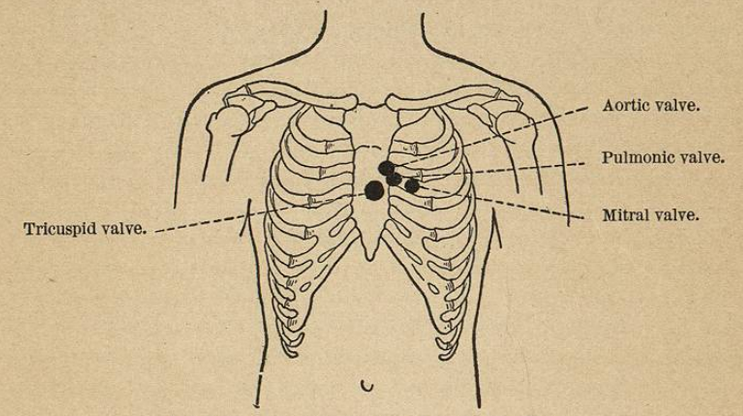

Fig. 109.—Anatomical Position of the Cardiac Valves.

his appreciation of the rhythm alone for the identification of the systolic sound. This is, however, an unsafe practice and leads to many errors. Our impression as to which of the two sounds of each cardiac cycle corresponds to systole should always be verified either by sight or touch. We must either see or feel the cardiac impulse and assure ourselves that it is synchronous with the heart sound which we take to be systolic.[1] This point is of especial importance in the recognition and identification of cardiac murmurs, as will be seen presently.

[1] When the cardiac impulse can be neither seen nor felt, the pulsation of the carotid will generally guide us. The radial pulse is not a safe guide.

So far, I have been describing the normal heart sounds heard in the "mitral area," that is, at the apex of the heart. If now we listen over the pulmonary area (in the second left intercostal space), we find that the rhythm of the heart sounds has changed and that here the stress seems to fall upon the "second sound," *i.e.*, that corresponding to the beginning of diastole; in other words, the first sound of the heart is here heard more feebly and the second sound more distinctly. The sharp, snapping quality of the latter is here even more marked than at the apex, and despite the feebleness of the first sound in this area we can usually recognize its relatively dull and prolonged quality.

Over the *aortic area* (*i.e.*, in the second right interspace) the rhythm is the same as in the pulmonary area, although the second sound may be either stronger or weaker than the corresponding sound on the other side of the sternum (see below, p. 176).

Over the tricuspid area one hears sounds practically indistinguishable in quality and in rhythm from those heard at the apex.

When the chest walls are thick and the cardiac sounds feeble, it may be difficult to hear them at all. In such cases the heart sounds may be heard much more distinctly if the patient leans forward and toward his own left so as to bring the heart closer to the front of the chest. Such a position of the body also renders it easier to map out the outlines of the cardiac dulness by percussion.

In cardiac neuroses and during conditions of excitement or emotional strain, the first sound at the apex is not only very loud but has often a curious *metallic reverberation* ("*cliquetis metallique*") corresponding to the trembling, jarring cardiac impulse (often mistaken for a thrill) which palpation reveals.

III. Modifications in the Intensity of the Heart Sounds.

It has already been mentioned that in young persons with thin, elastic chests, the heart sounds are heard with greater intensity than in older persons whose chest walls are thicker and stiffer. In obese, indolent adults it is sometimes difficult to hear any heart sounds at all, while in young persons of excitable temperament the sounds may have a very intense and ringing quality. Under diseased conditions either of the heart sounds may be increased or diminished in intensity. I shall consider

(1) *The First Sound at the Apex (sometimes Called the Mitral First Sound).*

(*a*) Increase in the length or intensity of the first sound at the apex of the heart occurs in any condition which causes the heart to act with unusual degree of force, such as bodily or mental exertion, or excitement. In the earlier stages of infectious fevers a similar increase in the intensity of this sound may sometimes be noted. Hypertrophy of the left ventricle sometimes has a similar effect upon the sound, but less often than one would suppose, while dilatation of the left ventricle, contrary to what one would suppose, is not infrequently associated with a loud, forcible first sound at the apex. In mitral stenosis the first sound is usually very intense and is often spoken of as a "thumping first sound" or as a "sharp slap."

(*b*) Shortening and weakening of the first sound at the apex.

In the course of continued fevers and especially in typhoid fever the granular degeneration which takes place in the heart muscle is manifested by a shortening and weakening of the first sound at the apex, so that the two heart sounds come to seem much more alike than usual. In the later stages of typhoid, the first sound may become almost inaudible. The sharp "valvular" quality, which one notices in the first apex sound under these conditions, has been attributed to the fact that weakening of the myocardium has caused a suppression of one of the two elements which go to make up the first sound, namely, the muscular element, so that we hear only the short, sharp sound due to the tautening of the mitral curtains. Chronic myocarditis, or any other change in the heart wall which tends to enfeeble it, produces a weakening and shortening of the first sound similar to that just described. Simple weakness in the mitral first sound without any change in its duration or pitch may be due to fatty overgrowth of the heart, to emphysema or pericardial effusion in case the heart is covered by the distended lung or by the accumulated fluid. Among valvular diseases of the heart

the one most likely to be associated with a diminution in intensity of the first apex sound is mitral regurgitation.

(*c*) Doubling of the first sound at the apex.

It is not uncommon in healthy hearts to hear in the region of the apex impulse a doubling of the first sound so that it may be suggested by pronouncing the syllables "turrupp" or "trupp." In health this is especially apt to occur at the end of expiration. In disease it is associated with many different conditions involving an increase in the work of one or the other side of the heart. It seems, however, to be unusually frequent in myocarditis.

(2) *Modifications in the Second Sounds as Heard at the Base of the Heart.*

Physiological Variations.—The relative intensity of the pulmonic second sound, when compared with the second sound heard in the conventional aortic area, varies a great deal at different periods of life. Attention was first called to this by Vierordt,[1] and it has of late years been recognized by the best authorities on diseases of the heart, though the majority of current text-books still repeat the mistaken statement that the aortic second sound is always louder than the pulmonic second in health.

The work of Dr. Sarah R. Creighton, done in my clinic during the summer of 1899, showed that in 90 per cent of healthy children under ten years of age, the pulmonic second sound is louder than the aortic. In the next decade (from the tenth to the twentieth year) the pulmonic second sound is louder in two-thirds of the cases. About half of 207 cases, between the ages of twenty and twenty-nine, showed an accentuation of the pulmonic second, while after the thirtieth year the number of cases showing such accentuation became smaller with each decade, until after the sixtieth year we found an accentuation of the *aortic second in sixty-six out of sixty-eight cases* examined. These facts are exhibited in tabular form in

[1] Vierordt: "Die Messung der Intensität der Herztöne" (Tübingen, 1885). See also Hochsinger, "Die Auscultation des kindlichen Herzens"; Gibson, "Diseases of the Heart" (1898); Rosenbach, "Diseases of the Heart" (1900); Allbutt, "System of Medicine."

Figs. 110 and 111 and appear to show that the relative intensity of the two sounds in the aortic and pulmonic arteries depends primarily upon the age of the individual, the pulmonic sound predominating in youth and the aortic in old age, while in the period of middle life there is relatively little discrepancy between the two.

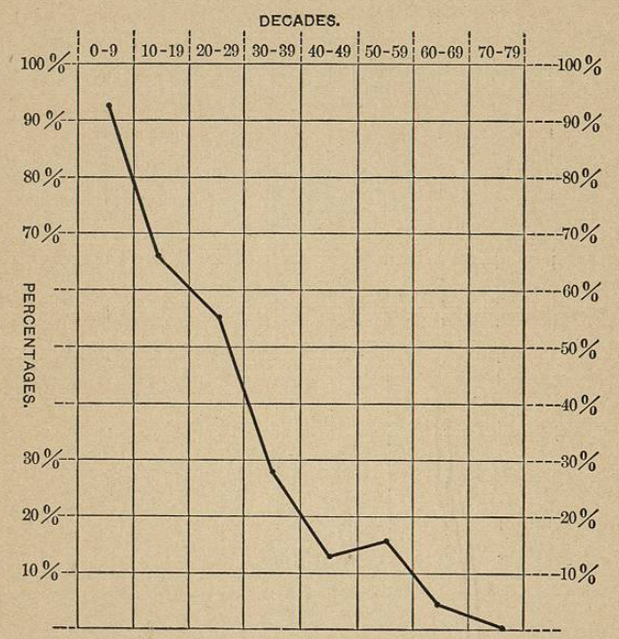

FIG. 110.—Showing the Per Cent of Accentuated Pulmonic Second Sound in Each Decade. Based on 1,000 cases.

It is, therefore, far from true to suppose that we can obtain evidence of a pathological increase in the intensity of either of the second sounds at the base of the heart simply by comparing it with the other. Pathological accentuation of the pulmonic second sound must mean a greater loudness of this sound *than should be expected at the age of the patient in question*, and not simply a greater intensity than that of the aortic second sound. The same

observation obviously applies to accentuation of the aortic second sound.

Both the aortic and the pulmonic second sounds are sometimes

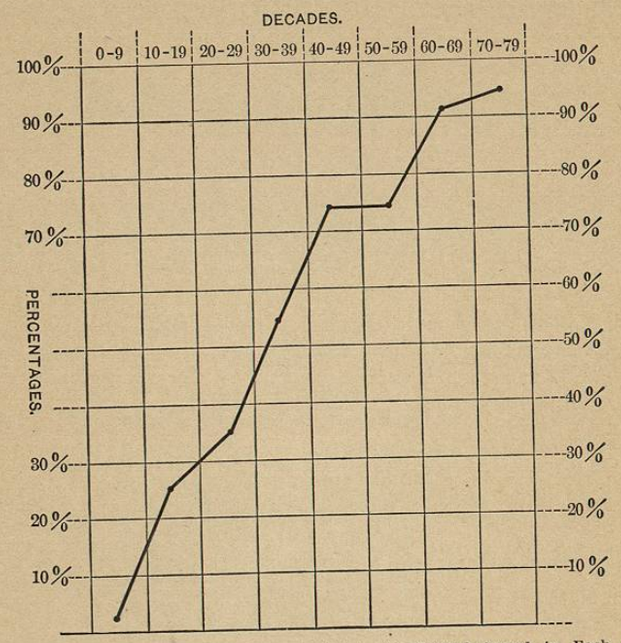

FIG. 111.—Showing the Per Cent of Accentuated Aortic Second Sound in Each Decade. Based on 1,000 cases.

very intense during great emotional excitement or after muscular exertion, and sometimes without any obvious cause.

Pathological Variations.

A. Accentuation of the Pulmonic Second Sound.

Pathological accentuation of the second sound occurs especially in conditions involving a backing up of blood in the lungs, such as occurs in stenosis or insufficiency of the mitral valve, or in obstructive disease of the lungs (emphysema, bronchitis, phthisis, chronic interstitial pneumonia). Indirectly accentuation of the pulmonic second sound points to hypertrophy of the right ventricle, since without such hypertrophy the work of driving the blood through the obstructed lung could not long be performed. If the right ventricle becomes weakened, the accentuation of the pulmonic second sound is no longer heard.

B. Weakening of the Pulmonic Second Sound.

Weakening of the pulmonic second sound is a very serious symptom, sometimes to be observed in cases of pneumonia or cardiac disease near the fatal termination. It is thus a very important indication for prognosis, and is to be watched for with the greatest attention in such cases.

C. Accentuation of the Aortic Second Sound.

I have already shown that the aortic second sound is louder than the corresponding sound in the pulmonary area in almost every individual over sixty years of age and in most of those over forty. A still greater intensity of the aortic second sound occurs—

(*a*) In interstitial nephritis or any other condition which increases arterial tension and so throws an increased amount of work upon the left ventricle. Indirectly, therefore, a pathologically loud aortic sound points directly to increased tension in the peripheral arteries and indirectly to hypertrophy of the left ventricle.

(*b*) A similar increase in the intensity of the aortic second sound occurs in aneurism or diffuse dilatation of the aortic arch.

D. Diminution in the Intensity of the Aortic Second Sound.

Whenever the amount of blood thrown into the aorta by the contraction of the left ventricle is diminished, as is the case especially in mitral stenosis and to a lesser degree in mitral regurgitation, the aortic second sound is weakened so that at the apex it may be inaudible. A similar effect is produced by any disease which weakens the walls of the left ventricle, such as fibrous myo-

carditis, fatty degeneration, and cloudy swelling. Relaxation of the peripheral arteries has the same effect. In conditions of collapse the aortic second sound may be almost or quite inaudible.

In persons past middle life the second sounds are often louder in the third or fourth interspace than in the second, so that if we listen only in the second space we may gain the false impression that the second sounds are feeble.

Accentuation of both the second sounds at the base of the heart may occur in health from nervous causes or when the lungs are retracted by disease so as to uncover the conus arteriosus and the aortic arch. Under these conditions the second sound may be seen and felt as well as heard. In a similar way, an apparent increase in the intensity of either one of the second sounds at the base of the heart may be produced by a retraction of one or the other lung.

Summary.—(1) The *mitral first sound* is increased by hypertrophy or dilatation of the left ventricle, and among valvular diseases especially by mitral stenosis. It is weakened or reduplicated by parietal disease of the heart. Any of these changes may occur temporarily from physiological causes.

(2) The *pulmonic second sound* is usually more intense than the aortic in children and up to early adult life. Later the aortic second sound predominates. Pathological accentuation of the second pulmonic sound usually points to obstruction in the pulmonary circulation (mitral disease, emphysema, etc.). Weakening of the pulmonic second means failure of the right ventricle and is serious.

(3) The *aortic second sound* is increased pathologically by any cause which increases the work of the left ventricle (arteriosclerosis, chronic nephritis). It is diminished when the blood stream, thrown into the aorta by the left ventricle, is abnormally small (mitral disease, cardiac failure).

(4) Changes in the tricuspid sounds are rarely recognizable, while changes in the first aortic and pulmonic sounds have little practical significance.

Modifications in the Rhythm of the Cardiac Sounds.

(1) Whenever the walls of the heart are greatly weakened by disease, for example, in the later weeks of a case of typhoid fever, the diastolic pause of the heart is shortened so that the cardiac sounds follow each other almost as regularly as the ticking of a clock; hence the term "*tick-tack* heart." As this rhythm is not unlike that heard in the fœtal heart, the name of "*embryocardia*" is sometimes applied to it. The "tick-tack" rhythm may be heard in any form of cardiac disease after compensation has failed, or in any condition leading to collapse.

(2) A less common change of rhythm is that produced by a shortening of the interval between the two heart sounds owing to an incompleteness of the contraction of the ventricle. This change may occur in any disease of the heart when compensation fails.

(3) *The "Gallop Rhythm."*—Shortening of the diastolic pause together with doubling of one or another of the cardiac sounds results in our hearing at the apex of the heart three sounds instead of two, which follow each other in a rhythm suggesting the hoof beats of a galloping horse. Such a rhythm may occur temporarily in any heart which is excited or overworked from any cause, but when permanent is usually a sign of *grave cardiac weakness*. The rhythms so produced are usually anapæstic, ⏑ ⏑ — ′, ⏑ ⏑ — ′, ⏑ ⏑ — ′, or of this type: ⏑ — ′ ⏑, ⏑ — ′ ⏑, ⏑ — ′ ⏑.

Doubling of the Second Sounds at the Base of the Heart.—At the end of a long inspiration this change may be observed in almost any healthy person if one listens at the base of the heart. It is still better brought out after muscular exertion or by holding the breath. In such cases it probably expresses the non-synchronous closure of the aortic and pulmonic valves, owing to increased pressure in the pulmonary circulation. Similarly in diseased conditions, anything which increases the pressure either in the peripheral arteries or in the pulmonary circulation, and thus throws increased work upon one or the other ventricle, will cause a doubling of the second sound as heard at the base of the heart.

In mitral stenosis a double diastolic sound is usually to be

heard at the apex, and in the diagnosis of this disease this "double shock sound" during diastole may be an important piece of evidence, and may sometimes be felt as well as heard. The "double shock sound" of mitral stenosis is not generally believed to represent a doubling of the ordinary second sound, although it corresponds with diastole. Just what its mechanism is, is disputed.

I have said nothing about modifications in the second sound at the apex, since this sound is now generally agreed to represent the aortic second sound transmitted by the left ventricle to the apex. The first sounds at the base of the heart have also not been dwelt upon, since they have no special importance in diagnosis.

Metallic Heart Sounds.

The presence of air in the immediate vicinity of the heart, as, for example, in pneumothorax or in gaseous distention of the stomach or intestine, may impart to the heart sounds a curious metallic quality such as is not heard under any other conditions.

"Muffling," "Prolongation," or "Unclearness" of the Heart Sounds.

These terms are not infrequently met with in literature, but their use should, I think, be discontinued. The facts to which they refer should be explained either as faintness of the heart sounds, due to the causes above assigned, or as faint, short murmurs. In their present usage such terms as "muffled" or "unclear" heart sounds represent chiefly an unclearness in the mind of the observer as to just what it is that he hears, and not any one recognized pathological condition in the heart.

IV. Sounds Audible Over the Peripheral Vessels.

(1) The normal heart sounds are in adults audible over the carotids and over the subclavian arteries. In childhood and youth only the second heart sound is thus audible.

(2) In about 7 per cent of normal persons a systolic sound can be heard over the femoral artery. This sound is obviously not

transmitted from the heart, and is usually explained as a result of the sudden systolic tautening of the arterial wall.

In aortic regurgitation this arterial sound is almost always audible not only in the femoral but in the brachial and even in the radial, and its intensity over the femoral becomes so great that the term "pistol-shot" sound has been applied to it. In fevers, exophthalmic goitre, lead poisoning, and other diseases, a similar arterial sound is to be heard much more frequently than in health.

Venous Sounds.

The violent closure of the venous valves in the jugular is sometimes audible in cases of insufficiency of the tricuspid valve. The sound has no clinical importance, and is difficult to distinguish owing to the presence of the carotid first sound mentioned above.