decreased during systole and the pressure of the air causes a sinking in of the intercostal spaces. Pulsation of the vessels of the neck may be either venous or arterial in origin; in the former case the result of an incompetent tricuspid valve, and in the latter due to an insufficient aortic valve.

Pulsation in the epigastric region is generally due to the pulsation of the right ventricle transmitted through the liver to the surface. Should the right ventricle be hypertrophied, the epigastric pulsation is much more marked. This is noted in emphysema, and in those cases of valvular disease of the left heart which cause the right side to hypertrophy in the attempt to compensate for the lesion. Epigastric pulsation may also be caused by the normal pulsation of the abdominal aorta, by aneurism of the abdominal aorta, or by a pulsating liver.

Palpation.—Many of the observations made by inspection may be confirmed by palpation, and may often be more easily appreciated. The location of the apex beat, often invisible, can usually be felt, and if not palpable when the patient stands erect, may become so if the patient is made to lean forward. In addition to the site of the apex beat, its regularity and force can be observed, and the presence of thrills determined. Changes in the location of the apex are suggestive of certain abnormal conditions. Hypertrophy and dilatation of the left ventricle and fluid or air in the right pleural cavity displace the apex downward and to the left.

Congenital malformations, retraction of the right lung from old pleurisy or tuberculosis, and fluid or air in the left pleural sac dislocate the apex to the right.

Retraction of the left lung, or pressure from below, as in ascites, tympanites, enlargement of the abdominal organs or tumors, displace the apex upward. Downward displacement is usually the result of aneurism of the aorta or of emphysema.

The regularity or rhythm may be changed by disease. The heart may be irregular in the force and frequency of its beats; and it may intermit or drop out a beat from time to time.

The force of the heart beat may be increased or diminished. It is diminished by any of the exhausting diseases: by myocarditis, by lesions of the valves causing dilatation of the cavities and weakening of the cardiac muscle, by the presence of fluid in the pericardium, by the presence of lung tissue between the chest wall and the apex, as in emphysema, or by very thick chest walls.

Hypertrophy of the ventricles, over-exertion, or thin chest walls increase the force of the apex beat.

Where the weakness or force is due to changes in the thickness of the thoracic wall or to the interposition of lung tissue between the costal wall and the apex, the weakness or strength is only apparent, and the real strength or weakness must be determined by the examination of the pulse.

Thrills may be detected by the palpating hand. When noted at the base of the heart and systolic in time they are suggestive of aneurism of the aorta or of aortic stenosis. At the apex a systolic thrill may occur in mitral insufficiency; and a presystolic thrill is observed in mitral stenosis.

Pericarditis occasionally causes a rubbing, which may be appreciated on palpation—the so-called pericardial friction fremitus. It is usually to be felt at the base of the heart in cases of dry pericarditis, and occurs with systole and diastole.

Percussion.—Percussion of the præcordium to determine the size of the heart may be practised in the ordinary manner or auscultatory percussion may be used. Both of these methods have their advantages, and may be used to supplement each other.

By this means of examination not only the fact that the heart is enlarged can be made out, but the part of the heart which is the site of hypertrophy may be determined.

The superficial and deep areas of cardiac dulness should be marked out (Plate XXII., Fig. 1). The superficial area of cardiac dulness is that portion of the heart which is not covered by lung substance in full inspiration. It is somewhat triangular in shape and is bounded internally by the left edge of the sternum, externally by an oblique line drawn from the fourth costal cartilage at its sternal junction to the fifth space just within the apex, and below by a line drawn from this point to the end of the sternum. In marking out this area the examiner is finding in reality the edges of the lungs, and, as the layer of lung tissue overlying the heart is thin, percussion must be light; for this examination finger percussion is most satisfactory. If the volume of the lungs is increased as in emphysema, the superficial area of cardiac dulness will be much diminished; unless the heart is also hypertrophied, in which case the borders of the lungs may be pushed back, and an apparently normal area of superficial cardiac dulness will be found on percussion. The deep area of cardiac dulness is the dulness detected by percussing over the edges of the heart; as there is, however, a thick layer of lung between the right and left borders of the heart and the chest wall, the area, as made out by digital percussion, does not accurately correspond with the outline of the heart, and, to determine with more exactness the right and left edges of the heart, auscultatory percussion may be employed. This method of examination was introduced by Dr. Cammann, who also devised the binaural stethoscope, and depends upon the fact that every organ has a percussion note peculiar to itself. When ordinary digital percussion is practised, the note produced is conveyed to the ear through the air, and the sound is thus more or less diffused and changed in character; when, however, the sound of the percussion note is brought to the ear direct by the use of the stethoscope, differences in quality and intensity are readily recognized.

To practise this method of percussion, the bell of the stethoscope, held by an assistant or the patient, should be placed over the organ, the dimensions of which are to be determined, at the point where the organ comes in contact with the thoracic or abdominal walls, and moderate percussion be made beginning at some distance from the point where the stethoscope is placed and gradually approaching it. When the pleximeter or finger reaches the point on the surface of the chest or abdomen below which the border of the organ lies, the percussion note changes abruptly, becomes much more marked in intensity, and usually higher in pitch. In mapping out the outline of the heart, it is best to find a number of points on the border of the heart at the extremities of its diameters, the vertical, transverse, and right and left oblique; these diameters intersect at a point near the centre of the heart. The eight points found, on being joined, give a fair outline of the heart. The vertical diameter is drawn parallel to the left border of the sternum; the transverse at right angles to the vertical diameter through its centre; the right oblique diameter is drawn, on the line from the right shoulder to the apex, from the right auricle to the apex; and the left oblique at a right angle to the latter and through its centre.

It is claimed that a skilful examiner can so accurately mark out the cardiac outlines on the cadaver that sharp-pointed steel needles driven through the chest wall at the given points will pass between the cardiac muscle and the pericardium.

On the living subject the outline cannot be so exactly determined, as the heart is constantly changing its size in systole and diastole. It is found, however, that the sum of the diameters in a large number of cases of adult males averaged sixteen and five-sixth inches, and this for the purpose of comparison is of use, though the length of the diameters considered singly is worthless.

In addition to determining the size of the heart and the location of its borders, this method of examination may be used to locate the edges of the liver in the normal subject, or when there is fluid in the pleural sac or abdominal cavity; also when a consolidated right lobe is in contact with the liver. The size of the spleen can be made out in ascites, or when in contact with an enlarged liver, provided that at some point the spleen is in contact

EXPLANATION OF
PLATE XXII.

EXPLANATION OF PLATE XXII.

(In all the figures the blue color indicates the superficial, and the red color the deep, cardiac dulness. The liver dulness is also indicated by the blue color.)

FIG. 1.—Area of Superficial and Deep Cardiac Dulness in Normal Conditions.

FIG. 2.—Area of Heart Dulness in Dilatation of the Right Ventricle. The increase in area of dulness is almost entirely to the right of sternum.

FIG. 3.—Area of Heart Dulness in Dilatation of the Left Ventricle. The increase in dulness is limited to the left side of the sternum.

FIG. 4.—Area of Heart Dulness in Dilatation of Left Auricle and Ventricle, with Uncovering of the Pulmonary Artery. (*a*) Deep cardiac dulness, right and left ventricles; (*b*) superficial dulness, the left auricle; (*c*) dulness due to pulmonary artery.

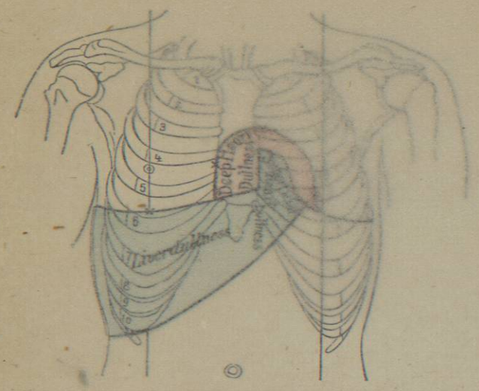

FIG. 1.

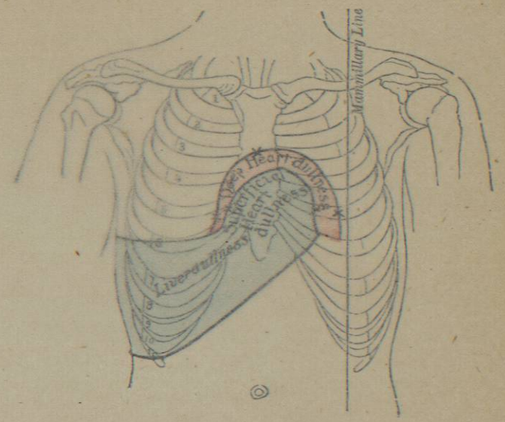

FIG. 2.

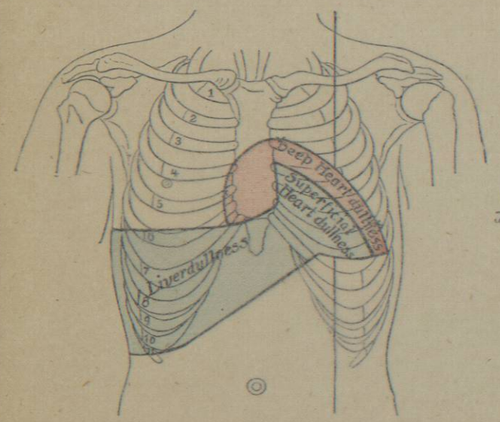

FIG. 3.

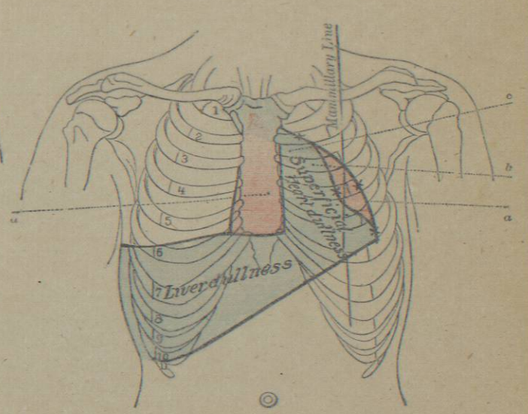

FIG. 4.

GRAPHIC REPRESENTATION OF SUPERFICIAL AND DEEP CARDIAC DULNESS IN THE NORMAL HEART AND IN HYPERTROPHY AND DILATATION

From Sahli's "Lehrbuch der klinischen Untersuchungs-Methoden," etc.

EXPLANATION OF PLATE XXII.

(In all the figures the [illegible] cardiac dulness. The liver dulness [illegible])

FIG. 1.—Area of [illegible]

FIG. 2.—Area of [illegible] The [illegible] in area of dulness is almost [illegible]

FIG. 3.—Area of [illegible] The increase in dulness is limited to the left side of the [illegible]

FIG. 4.—Area of [illegible] with Uncovering of the [illegible] superficial dulness, [illegible]

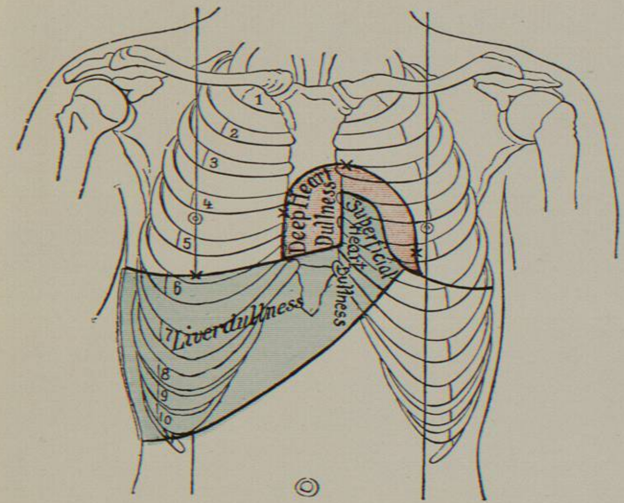

FIG. 1.

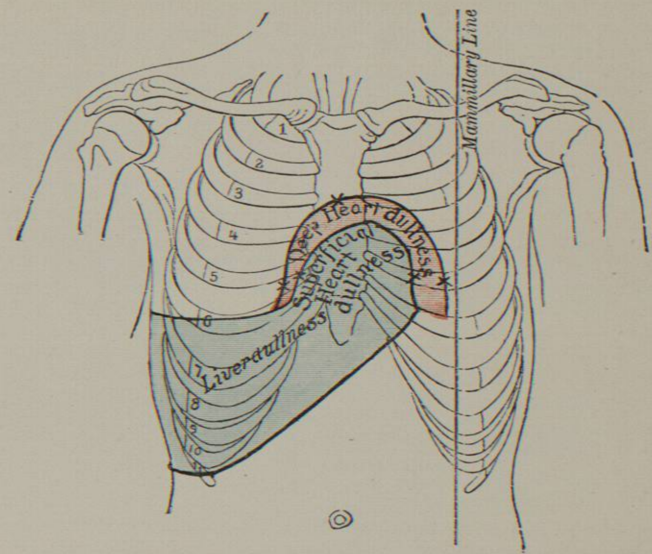

FIG. 2.

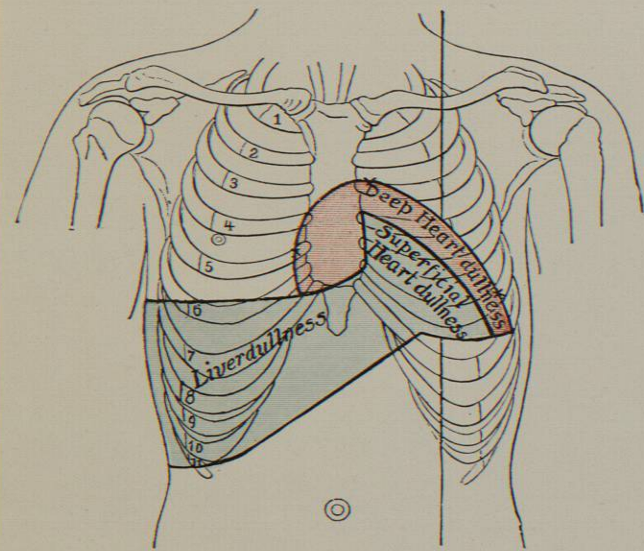

FIG. 3.

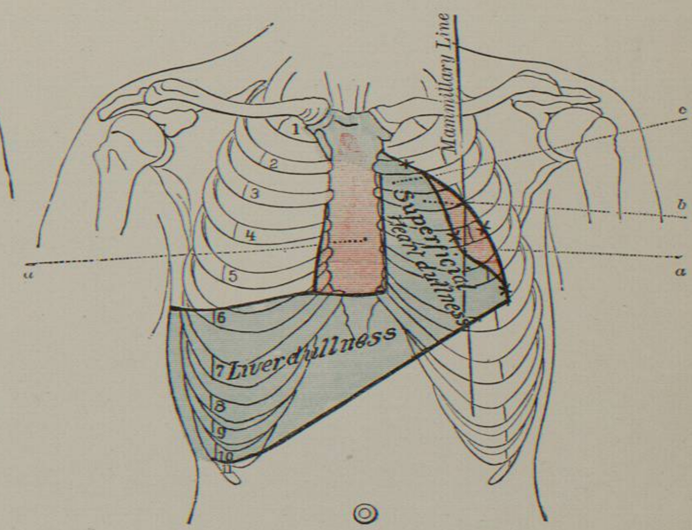

FIG. 4.

GRAPHIC REPRESENTATION OF SUPERFICIAL AND DEEP CARDIAC DULNESS IN THE NORMAL HEART AND IN HYPERTROPHY AND DILATATION

From Sahli's "Lehrbuch der klinischen Untersuchungs-Methoden," etc.

with the parietal wall. The kidneys may be mapped out with the exception of the inner border.

Another form of auscultatory percussion has come into use with the invention of the phonendoscope. Here instead of percussing, the surface of the chest in the neighborhood of the organ under examination is gently scratched with the finger. This produces a sound the quality of which, as well as the intensity and pitch, instantly changes when the density of the organs beneath the surface changes. It is merely a modification of auscultatory percussion, but is perhaps capable, in skilful hands, of giving even more definite information in regard to the size of the solid organs of the body, of tumors, the level of fluid exudates or transudates, and the line of contact between tumors and solid organs, or between solid organs and fluid.

As in percussing the lungs, so in the examination of the heart by percussion, much information may be gained by noting the sense of resistance beneath the finger in contact with the chest wall.

If by either of the methods of percussion described the area of the heart dulness is found increased, it is suggestive of hypertrophy of the heart muscle, and the locality of the dulness indicates the portion of the heart which is enlarged; of fluid in the pericardium; of aneurism, or of a consolidated lung or tumor displacing the heart.

Should the area of cardiac dulness be diminished, the usual cause will be found in an emphysematous condition of the lungs.

Auscultation.—By auscultation the character, rhythm, and point of maximum intensity of the normal heart sounds are observed, any changes in the intensity or rhythm are noted, and the presence of any abnormal sounds or murmurs is detected. In this examination the immediate or mediate method may be employed. The mediate is, however, the more common, and the use of the stethoscope enables the examiner to localize the area of the maximum intensity of the sounds more exactly; it also intensifies the sounds, so that sounds inaudible to the ear may by its aid be clearly heard.

Two sounds are heard on auscultation: the first, or systolic sound, occurring with the systole of the heart, somewhat dull and booming in character, and resembling the syllable "tubb"; the second, or diastolic sound, occurring with the diastole of the heart, short, quick, and snappy in character, like the syllable "dupp."

The first and second sounds are separated by a short pause, and a longer pause occurs between the second sound and the succeeding first sound. This rhythmical recurrence of the first and second sounds with the interposed pauses makes up the cardiac cycle.

The first sound is produced by the closure and tension of the auriculo-ventricular valves and the tension of the walls of the ventricles; it is, therefore, a composite sound made up of several elements. The second sound is caused by the sudden closing of the aortic and pulmonary valves.

Normally these sounds recur in rhythmical sequence, and may be heard all over the præcordium. There are, however, areas where the individual sounds may be heard with greater distinctness. For example, the first sound may be heard most clearly at the apex, and not immediately over the point of production, the location of the mitral and tricuspid valves. This area at the apex is called the mitral area.

The aortic element of the second sound is most clearly heard in the aortic area, the second right intercostal space near the sternum; and the pulmonary element in the second left intercostal space, the pulmonary area. At the apex the quality of the systolic sound is soft and booming, the pitch is low, the intensity marked, and the duration long. The quality of the second sound is clicking or sharp, the pitch is higher than that of the first sound, the intensity less, and the duration shorter.

At the base the intensity of the first sound is diminished, and that of the second sound increased. As a rule, the second sound is heard more distinctly in the aortic area than in the pulmonary; in other words, the aortic element of the second sound is normally the louder.

Disease may cause changes in the normal heart sounds, which modifications are noted in the quality, intensity, duration, and rhythm.

The quality of the first sound may be impure or rough, and there may be reduplication of the first sound, which is caused by the non-synchronous contraction of the ventricles and closure of the auriculo-ventricular valves.

The intensity of the first sound is much increased in cases in which there is hypertrophy of the left ventricle, in acute febrile diseases, and in patients with very thin chest walls. Diminution in the intensity is noted in emphysema, in patients with very thick chests, in cases of pericarditis with effusion, in all exhausting diseases, and in dilated heart.

In hypertrophy the duration of the first sound may be prolonged, even to the point of sounding like a murmur; and in dilatation this sound is so shortened that it may be mistaken for the second sound. The same shortening of the first sound is often observed in cases of mitral stenosis.

The rhythm is often irregular or intermittent; this is particularly marked in cases of mitral stenosis and in dilated heart. The aortic element of the second sound, or the aortic second sound as it is called, is accentuated in any disease which causes an increase in the blood pressure in the aorta and systemic circulation; in other words, when the arterial tension is high, or when there is an increased amount of blood in the aorta, as in aneurism or dilatation of the aorta. When the action of the heart is weak, as in dilated heart, in fevers, and in myocarditis, the amount of blood and the tension in the aorta are diminished, and the intensity of the aortic second sound is lessened. The pulmonic second sound is accentuated when the pulmonary circulation is under increased tension; in mitral disease, either stenosis or insufficiency of the valve, and in pneumonia or emphysema. When the tension in the pulmonary circulation is low, the second sound in the pulmonary area is diminished in intensity.

There is not uncommonly a reduplication of the second sound, which is due to the fact that the aortic and pulmonary valves do not close at the same moment. This occurs when the tension of the blood, or blood pressure, is unequal in the systemic and pulmonary circulation. It is frequently observed in mitral stenosis.

In addition to the normal heart sounds, other sounds may be heard which are the result of diseased conditions of the heart or vessels. These abnormal sounds may be produced either within or without the heart or in the great vessels, and are therefore called endocardial, exocardial, or vascular murmurs. In listening to a murmur, in order to decide upon its nature and the lesion upon which its production depends, it is necessary to note the time of the cardiac cycle at which the murmur is heard, its point of maximum intensity, the quality of the sound, and the direction in which it is transmitted.

Exocardial Murmurs.—These sounds are produced by the rubbing together of the surfaces of the pericardium made rough by inflammation, or by the movement of the pulmonary pleura against the pericardial sac; hence these sounds are called pericardial friction sounds or murmurs or pleuro-pericardial sounds.

Pericardial murmurs occur in pericarditis, and they are heard with both the systole and the diastole of the heart. From this fact they are often spoken of as "see-saw" or "to-and-fro" murmurs. The murmur is usually loudest at the base of the heart and along the line indicating the auriculo-ventricular groove. The quality is rubbing and creaking, not of marked intensity and often very faint; the intensity may frequently be increased by pressure on the chest wall with the stethoscope. The sound is near the ear, and is not transmitted.

Pleuro-pericardial frictions or murmurs occur in cases of dry pleurisy involving the pleura of the pericardial sac and the adjacent lung. The sounds are not synchronous with the heart action, but occur with the respiratory movements, and may disappear when the patient

holds his breath; they are usually heard loudest over the borders of the ventricles, when respiration is deep. The quality is rubbing, the intensity not marked, and it may be increased by pressure with the stethoscope.

Endocardial Murmurs.—Endocardial murmurs may be due to gross changes in the valves or lining membrane of the heart, or to changes in the quality of the blood. In the first case they are called organic, and in the second functional, hæmic, or inorganic murmurs respectively.

Organic Endocardial Murmurs.—For the production of a murmur two factors are necessary: force and rapidity of the blood current, and narrowing or change in calibre of the passages through which the blood flows.

The blood normally flows with force and rapidity through a passage with smooth walls, and of even or gradually diminishing calibre. When, as the result of disease, the cusps of the valves do not open properly to allow the blood to flow through, or when the curtains of the valves do not close perfectly and so allow the blood to flow back through the opening, a sudden diminution in the calibre of the passage occurs. Thus the two factors for the production of a sound or murmur, the blood current passing with force and rapidity through a narrow opening into a wider space, are present within the diseased heart.

Should the velocity and force of the blood current be diminished, as the result of a weak heart, when a diminution in the calibre exists, either there will be no murmur or a murmur of slight intensity will be heard. The valves on the left side of the heart are those most commonly the seat of organic disease in adults. In early infant life and in intra-uterine life the valves of the right heart are usually those affected.

Inorganic, Functional, and Hæmic Murmurs.—In addition to the organic valvular lesions which render the valves unfit for the work they are destined to perform, the valves may be rendered incompetent on account of a dilated condition of the muscular walls of the heart and a consequent widening of the orifices, which the cusps of the valves are unable to fill in and close. This inability to close the dilated auriculo-ventricular orifice, for example, is due to the fact that the cusps of the mitral valve are too small, and also to the fact that the papillary muscles are displaced by the dilatation of the heart and so prevent the cusps from closing perfectly.

An increased blood pressure in the aorta or pulmonary artery, or a loss of elasticity of the arterial coats, which causes a dilated condition of the vessels, may allow a regurgitation of blood at the aortic or pulmonary orifices. Murmurs due to these causes are spoken of as inorganic, or the result of relative insufficiency of the valves.

Changes in the character of the blood may also produce sounds, to which the name "hæmic" or "functional" murmurs is given.

Organic Murmurs.—Sounds due to organic lesions are heard during either the systole or the diastole of the heart, and are therefore spoken of as systolic or diastolic murmurs. Those murmurs which occur during the diastole, but which end at the beginning of the systole, are called "presystolic" murmurs.

Systolic murmurs, heard loudest in the aortic and pulmonary areas, are due to a stenosis or obstructed condition of the aortic or pulmonary orifices. Systolic murmurs heard at the mitral or tricuspid areas are due to incompetent mitral or tricuspid valves, which allow a regurgitation of blood through them. Diastolic murmurs in the aortic and pulmonary areas are produced by blood regurgitating through an incompetent aortic or pulmonary valve. Murmurs diastolic in time and heard in the mitral and tricuspid areas are caused by an obstructed or stenotic mitral or tricuspid valve.

The relative frequency of these murmurs is in the following order (Broadbent): mitral regurgitation, mitral stenosis, aortic regurgitation, aortic stenosis, tricuspid regurgitation, tricuspid stenosis, pulmonary stenosis, pulmonary regurgitation. To take up the consideration of the murmurs in the order of their frequency:

Mitral Systolic Murmur.—Mitral valve incompetent, blood regurgitates into left auricle during systole of left ventricle. The most common cause of incompetence of the mitral valve is rheumatic endocarditis. Myocarditis, dilated left ventricle from any cause, or the infectious diseases may also produce this lesion.

The murmur occurs with the systole of the heart; the quality is soft and blowing like a whispered "who," pitch is low and intensity not loud; the duration is long. Area of maximum intensity at the apex; the murmur may be localized or may be transmitted toward the left into the axilla; it may also be heard in the left interscapular region along the internal border of the scapular.

Mitral Presystolic Murmur.—The mitral valve is stenosed or obstructed, and the blood is therefore hindered in its passage from the left auricle to the left ventricle during the auricular systole. The cause in nearly all cases is rheumatic endocarditis.

The murmur occurs during the auricular systole; toward the end of the long pause it becomes gradually louder and louder, and ends with the first sound of the heart. The quality is harsh, rough, and rolling, intensity marked, the area of maximum intensity usually small and just within the apex. This murmur is not, as a rule, transmitted.

Aortic Diastolic Murmur.—Aortic valve incompetent, the blood therefore regurgitates into the left ventricle during the period of diastole. Rheumatic endocarditis is a common cause of this lesion; also atheromatous changes in the aorta and the cusps of the valve, as well as dilatation of the orifice.

The murmur occurs with and replaces the second sound of the heart; it is soft and blowing in quality, often almost musical; generally of rather high pitch, of variable intensity and short duration. The area of maximum intensity varies, it may be most marked in the aortic area; sometimes to the left of the sternum in the second, third, or fourth intercostal space; and at times at the tip of the sternum. It is transmitted across and down the sternum to the ensiform cartilage, and to the apex.

Aortic Systolic Murmur.—Aortic valve stenosed or obstructed, the blood hindered in its passage during the systole of the left ventricle from the ventricle into the aorta. This murmur may be due to true stenosis of the orifice resulting from rheumatic endocarditis or atheromatous changes. It may be caused by atheroma of the first part of the aorta, by thickening and roughening of the aortic cusps, and sometimes as the result of aneurism and rupture of the valves.

The murmur occurs with or replaces the first sound. Its quality is harsh and rough; it may, however, at times be soft, the intensity is usually marked and the duration long. Heard with maximum intensity in the aortic area, and transmitted up the vessels of the neck. It may sometimes be heard all over the præcordium, and in the second, third, and fourth intercostal spaces on the left side near the vertebral column.

Hypertrophy and Dilatation of the Heart.—Hypertrophy of the heart is a thickening of the walls of the heart which may involve the walls of all the cavities or be limited to those of one. Dilatation is an increase in the capacity of the cavities of the heart; as in hypertrophy one or more of the cavities may be involved at the same time.

Hypertrophy and dilatation may occur together or separately. With hypertrophy there is usually a certain amount of dilatation. Hypertrophy results from the effort of the heart to do more work than normal. This demand for extra work may be due to excessive exercise, to functional causes, for example exophthalmic goitre, to mechanical defects in the circulatory system, to valvular disease, or to increased resistance in the peripheral or in the pulmonary circulation, depending on Bright's disease, chronic bronchitis, or emphysema. The endeavor of the heart to overcome any of the valvular lesions by hypertrophy is an effort of the organism to supply extra force where extra work is needed. If the hypertrophy is sufficient to overcome the symptoms arising from the valvular lesion, the lesion is said to be

EXPLANATION OF
PLATE XXIII