(3) *Hypertrophy and dilatation* of the right ventricle, which in turn becomes sooner or later overcrowded so that the tricuspid valve gives way and tricuspid leakage occurs.

(4) The capacity for hypertrophy possessed by the right auricle is soon exhausted, and we get then—

(5) *General venous stasis*, which shows itself first as venous pulsation in the jugulars and in the liver and later in the tissues drained by the portal and peripheral veins. This venous stasis increases the work of the left ventricle, and so we get—

(6) *Hypertrophy and dilatation of the left ventricle.* Hypertrophy of the left ventricle is also produced by the increased work necessary to maintain some vestige of sphincter action at the leaky mitral orifice, as well as by the labor of contracting upon the extra quantity of blood delivered to it by the enlarged left auricle.

At last the circle is complete. Every chamber in the heart is enlarged, overworked, and failure is imminent.

Returning now to the signs of mitral regurgitation, we shall find it most convenient to consider first the type of regurgitation produced by rheumatism and resulting in thickening, stiffening, and retraction of the valve.

Physical Signs.

(*a*) *First Stage—Prior to the Establishment of Compensation.*

We have but one characteristic physical sign:

A systolic murmur heard loudest at the apex of the heart, transmitted to the back (below or inside the left scapula) and to the left axilla. The murmur is not infrequently musical in character, and when this is the case diagnosis is much easier. Systolic musical murmurs so transmitted do not occur without valvular leakage. Rosenbach believes that adherent pericardium is capable of producing such a murmur, but only, if I understand him rightly, in case there is a genuine mitral leakage due to the embarrassing embrace of the pericardium which prevents the mitral orifice from closing.

"Functional" or "hæmic" murmurs are rarely heard in the back, and very rarely, if ever, have a musical quality.

Cases of mitral regurgitation are not very often seen at this stage, but in acute endocarditis after the fever and anæmia have subsided, or in chorea, such a murmur may exist for days or weeks before any accentuation of the pulmonic second sound or any enlargement of the heart appears. I have had the opportunity of verifying the diagnosis at autopsy in two such cases.

(*b*) *Second Stage—Compensation Established.*

As long as compensation remains perfect, the only evidence of regurgitation may be that obtained by auscultation, and I shall accordingly begin with this rather than in the traditional way with inspection, palpation, and percussion.

The distinguishing auscultatory phenomena in cases of well-compensated mitral insufficiency are:

(*a*) A systolic murmur whose maximum intensity is at or near the apex impulse of the heart, but which is also to be heard in the left axilla and in the back below or inside the angle of the left scapula (so far the signs are those of the first stage, above described).

(*b*) A pathological accentuation of the pulmonic second sound.

This is the minimum of evidence upon which it is justifiable to make the diagnosis of compensated mitral regurgitation. In the vast majority of cases, however, our diagnosis is confirmed by the following additional data:

(*c*) Enlargement of the heart as shown by inspection, palpation, and percussion.

The pulse in well-compensated cases shows no considerable abnormality. When compensation begins to fail, or sometimes before that time, the most characteristic thing about the pulse is its marked irregularity both in force and rhythm. Such irregularity is at once *more common and less serious in mitral disease than in that of any other valve;* it may continue for years and be compatible with very tolerable health.

Returning now to the details of the sketch just given, we will take up first —

(*a*) *The Murmur.*—In children the murmur of mitral regurgitation may be among the loudest of all murmurs to be heard in val-

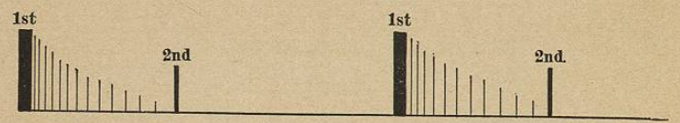

FIG. 122.—Diagram to Represent Systolic Mitral Murmur. The heavy lines represent the normal cardiac sounds and the light lines the murmur, which in this case does not replace the first sound and "tapers" off characteristically at the end.

vular disease, but this does not necessarily imply that the lesion is a very severe one. A murmur which *grows louder* under observation in a well-compensated valvular lesion may mean an advance of the disease, but if the case is first seen after compensation has failed a faint, variable whiff in the mitral area may mean the severest type of lesion. As the patient improves under the influence of rest and cardiac tonics, such a murmur may grow very much louder, or a murmur previously inaudible may appear.

The *length* of the murmur varies a great deal in different cases and is not of any great practical importance. It rarely ends abruptly, but usually "tails off" at the end of systole (see Fig. 122). *Musical* murmurs are heard more often in mitral regurgitation than in any other valve lesion, but the musical quality rarely lasts throughout the whole duration of the murmur, contrasting in this respect with musical murmurs produced at the aortic valve. The

FIG. 123.—Systolic Mitral Murmur Replacing the First Sound of the Heart.

first sound of the heart may or may not be replaced by the murmur (see Fig. 123). When the sound persists and is heard either with or before the murmur, one can infer that the lesion is relatively slight in comparison with cases in which the first sound is wholly

obliterated. *Post-systolic* or *late systolic* murmurs, which are occasionally heard in mitral regurgitation, are said to point to a relatively slight amount of disease in the valve (see Fig. 122). Rosen-

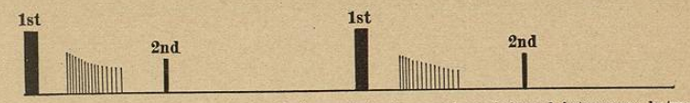

FIG. 124.—Late Systolic Murmur. The first sound is clear and an interval intervenes between it and the murmur.

bach claims that the late systolic murmur is always due to organic disease of the valves and never occurs as a functional murmur.

When compensation fails, the murmur may altogether disappear for a time, and if the patient is then seen for the first time and dies without rallying under treatment, it may be impossible to

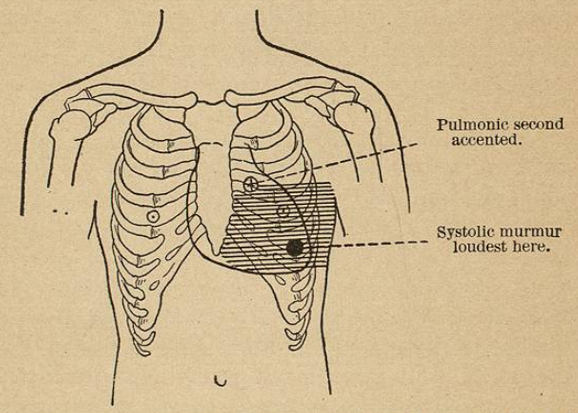

FIG. 125.—Mitral Regurgitation. The murmur is heard over the shaded area as well as in the back.

make the diagnosis. The very worst cases, then, are those in which there is no murmur at all.

The murmur of mitral regurgitation is conducted in all directions, but especially toward the axilla and to the back (*not* around the chest, but directly). In the latter situation it is usually louder

than it is in mid-axilla, and occasionally it is heard as loudly in the back as anywhere else. This is no doubt owing to the position of the left auricle (see Figs. 125 and 126).

(*b*) After compensation is established and as long as it lasts an *accentuation of the pulmonic second sound* is almost invariably to be made out, and may be so marked that we can feel and see it, as well as hear it. Not infrequently one can also see and feel the pulsation of the conus arteriosus—*not* the left auricle—in the second and third left intercostal space. (It may be well to mention again

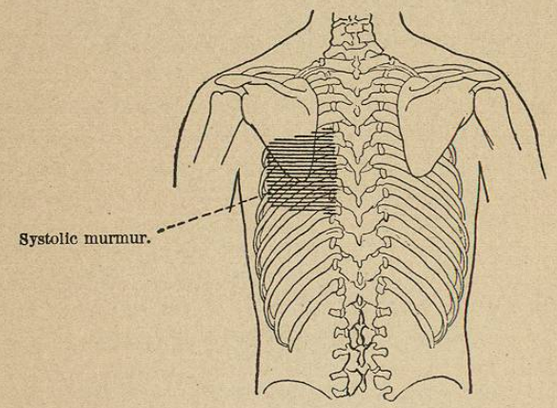

FIG. 126.—Mitral Regurgitation. Murmur heard over the shaded area.

here that by accentuation of the pulmonic second sound one does not mean merely that it is louder or sharper in quality than the aortic second sound, since this is true in the vast majority of cases in healthy individuals under thirty years of age. Pathological accentuation of the pulmonic second sound means *a greater intensity of the sound than we have a right to expect at the age of the individual in question.*) Occasionally the pulmonic second sound is reduplicated, but as a rule this points to an accompanying stenosis of the mitral valve. At the apex the second sound (*i.e.*, the transmitted aortic second) is not infrequently wanting altogether, owing

to the relatively small amount of blood which recoils upon the aortic valves.

(*c*) *Enlargement of the heart*, and more especially of the right ventricle, is generally to be made out, and in the majority of cases this enlargement is manifested by displacement of the apex impulse both downward and toward the left, but more especially to the left. Percussion confirms the results of inspection and palpation regarding the position of the cardiac impulse. The normal substernal dulness is increased in intensity, and we can sometimes demonstrate an enlargement of the heart toward the right (see Fig. 123).

In children (in whom adhesive pericarditis often complicates the disease) a systolic THRILL may not infrequently be felt at the apex, and the precordia may be bulged, and even in adults such a systolic thrill is not so rare as some writers would have us suppose.

(*d*) *The pulse*, as said above, shows nothing characteristic at any stage of the disease. While compensation lasts, there is usually nothing abnormal about the pulse, although it may be somewhat irregular in force and rhythm, and may be weak when compared to the powerful beat at the apex in case the regurgitant stream is a very large one. Irregularity at this period is less common in pure mitral regurgitation than in cases complicated by stenosis.

(*c*) *Third Stage—Failing Compensation.*

When compensation begins to fail, the pulse becomes weak and irregular, and many heart beats fail to reach the wrist, but there is still nothing characteristic about the pulse, which differs in no respect from that of any case of cardiac weakness of whatever nature.

(*e*) Evidence of *venous stasis*, first in the lungs and later in the liver, lower extremities, and serous cavities, does not show itself so long as compensation is sufficient, but when the heart begins to fail the patient begins to complain not only of palpitation and cardiac distress, but of dyspnœa, orthopnœa, and cough, and examination reveals a greater or lesser degree of cyanosis with pulmo-

nary œdema manifested by crackling râles at the base of the lungs posteriorly, and possibly also by hæmoptysis or by evidences of hydrothorax (see below, p. 330). If compensation is not re-established, the right ventricle dilates, the tricuspid becomes incompetent, the liver becomes enlarged and tender, dropsy becomes general, the heart and pulse become more and more rapid and irregular, the heart murmur disappears and is replaced by a confusion of short valvular sounds, "*gallop rhythm*" or "*delirium cordis*," often considerably obscured by the noisy, labored breathing with numerous moist râles. In a patient seen for the first time in such a condition diagnosis may be impossible, yet mitral disease of some type may usually be suspected, since murmurs produced at the aortic valve are not so apt to disappear when compensation fails. The relative tricuspid insufficiency which often occurs is likely to manifest itself by an enlargement of the right auricle, sometimes demonstrable by percussion and later by venous pulsation in the neck and in the liver.

(*d*) *Differential Diagnosis.*

The murmur of mitral regurgitation may be confused with

(1) Tricuspid regurgitation.

(2) Functional murmurs.

(3) Stenosis or roughening of the aortic valves.

(1) The post-mortem records of the Massachusetts General Hospital show that in the presence of a murmur due to mitral regurgitation it is very easy to fail altogether to recognize a tricuspid regurgitant murmur. Only 5 out of 29 cases of tricuspid regurgitation found at autopsy were recognized during life. Allbutt's figures from Guy's Hospital are similar. In the majority of these cases, mitral regurgitation was the lesion on which attention was concentrated during the patient's life. This is all the more excusable because the tricuspid area is so wide and uncertain. Murmurs produced at the tricuspid orifice are sometimes heard with maximum intensity just inside the apex impulse, and if we have *also* a mitral regurgitant murmur, it may be impossible under such circumstances to distinguish it from the tricuspid murmur. Sometimes the two are of different pitch, but more often tricuspid regurgitation must be recognized *indirectly* if at all, *i.e.*, through the evidence given by venous pulsation in the jugular veins and in the liver. Tricuspid murmurs are not transmitted to the left axilla and do not cause accentuation of the pulmonic second sound, although they are compatible with such accentuation. They are to be distinguished from the murmurs of mitral regurgitation by their different seat of maximum intensity, possibly by a difference in pitch, but most clearly by the concomitant phenomena of venous pulsation above mentioned.

(2) "Functional" murmurs are usually systolic and may have their maximum intensity at the apex of the heart, but in the great majority of cases they are heard best over the pulmonic valve or just inside or outside the apex beat (Potain). They are faint or inaudible at the end of expiration, and are more influenced by position than organic murmurs are. In the upright position they are often very faint. They are rarely transmitted beyond the precordia and are unaccompanied by any evidences of enlargement of the heart, by any pathological accentuation of the pulmonic second sound,[1] or any evidences of engorgement of the lungs or general venous system.

(3) Roughening or narrowing of the aortic valves may produce a systolic murmur with maximum intensity in the second right intercostal space, but this murmur is not infrequently heard all over the precordia and quite plainly at the apex, so that it may simulate the murmur of mitral regurgitation. The aortic murmur may indeed be heard more plainly at the apex than at any other point *except* the second right intercostal space, owing to the fact that the right ventricle, which occupies most of the precordial region between the aortic and mitral areas, does not lend itself well to the propagation of certain types of cardiac murmurs. Under these circumstances "a loud, rough aortic murmur may be heard at the

[1] It must be remembered that in chlorosis, a disease in which functional murmurs are especially prone to occur, the pulmonic second sound is often surprisingly loud, owing to a retraction of the left lung, which uncovers the root of the pulmonic artery.

apex as a smooth murmur of a different tone" (Broadbent). Such a murmur is not, however, likely to be conducted to the axilla or heard beneath the left scapula, nor to be accompanied by accentuation of the pulmonic second sound nor evidences of engorgement of the lungs and general venous system.

II. Mitral Stenosis.

Narrowing or obstruction of the mitral orifice is almost invariably the result of a chronic endocarditis which gradually glues together the two flaps of the valve until only a funnel-shaped opening or a slit like a buttonhole is left see Figs. (127 and 128). As we examine post mortem the tiny slit which may be all that is left of the mitral orifice in a case of long standing, it is difficult to conceive how sufficient blood to carry on the needs of the circulation could be forced through such an insignificant opening.

Usually a slow and gradually developed lesion, mitral stenosis often represents the later stages of a process which in its earlier phases produced pure mitral regurgitation. By some observers the advent of stenosis is regarded as representing an attempt at compensation for a reduction of the previous mitral leakage. Others consider that the stenosis simply increases the damage which the valve has suffered.

A remarkable fact never satisfactorily explained is the predilection of mitral stenosis for the female sex.[1] A large proportion of the cases—seventy-six per cent in my series—occur in women.

It is also curious that so many cases are associated with pulmonary tuberculosis.

Physical Signs.

Mitral stenosis may exist for many years without giving rise to any physical signs by which it may be recognized, and even after signs have begun to show themselves they are more fleeting and inconstant than in any other valvular lesion of the heart. In the early stages of the disease the heart may appear to be entirely nor-

[1] Fenwick's explanation, viz., that the sedentary life of women favors the slow adhesive inflammation of the valve and its curtains, resulting in stenosis, does not seem to me to be satisfactory.

mal if the patient is at rest, and especially if examined in the recumbent position, characteristic signs being elicited only by exertion; or again a murmur which is easily audible with the patient in the upright position may disappear in the recumbent position; or a murmur may be heard at one visit, at the next it may be im-

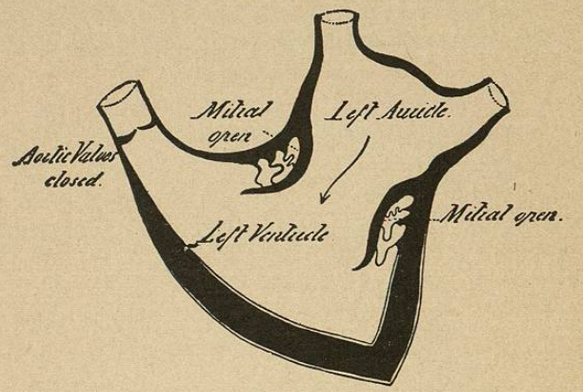

FIG. 127.

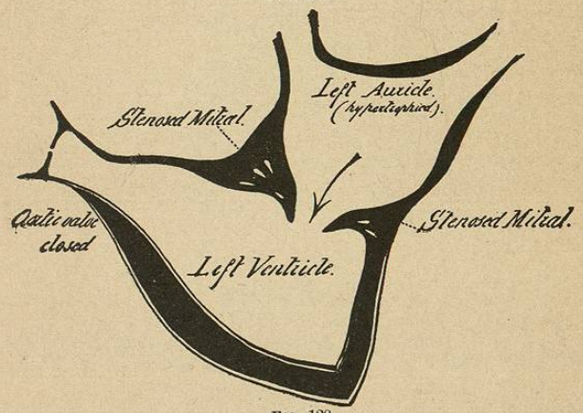

FIG. 128.

FIG. 127.—Diagram to Represent the Position of the Valves in the Normal Heart during Diastole, the Open Mitral Allowing the Blood to Flow Down from the Left Auricle, the Aortic Closed.

FIG. 128.—Mitral Stenosis—Period of Diastole. The blood flowing from the left auricle is obstructed by the thickened and adherent mitral curtains.

possible to elicit it by any manœuvre, while at the third visit it may be easily heard again. These characteristics explain to a certain extent the fact that differences of opinion so often arise regarding the diagnosis of mitral stenosis, and that out of forty-eight cases in which this lesion was found at autopsy at the Massachusetts General Hospital, only twenty-three were recognized during life. No common lesion (with the exception of tricuspid regurgitation) has been so frequently overlooked in our records.

I shall follow Broadbent in dividing the symptoms into three stages, according to the extent to which the lesion has progressed.

I.

In the first stage inspection and palpation show that the apex beat is little if at all displaced, and percussion reveals *no increase*

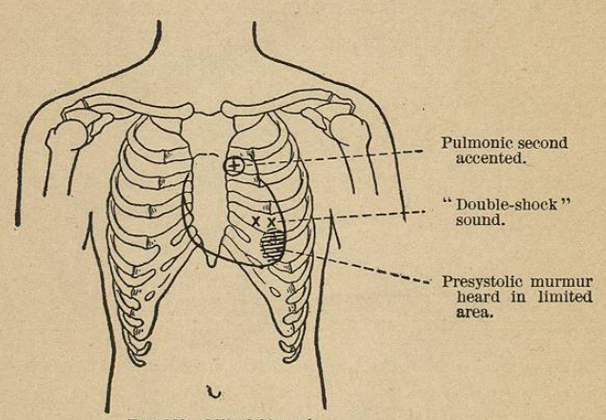

FIG. 129.—Mitral Stenosis.

in the area of cardiac dulness; indeed, in rare cases the heart may be smaller than usual. If one lays the hand lightly over the origin of the apex beat, one can generally feel the *purring presystolic thrill* which is so characteristic of this disease, more common indeed than in any other. This thrill is more marked in the second stage of the disease, but can generally be appreciated even in the first. It runs up to and ceases abruptly with the very sharp first sound,

the sudden *shock* of which may be appreciated even by palpation. The thrill is sometimes palpable even when no murmur can be heard, and often the thrill is transmitted to the axilla when the murmur is confined to the apex region. On auscultation one hears, especially after the patient has been exerting himself, and particularly if he leans forward and to the left, a *short low-pitched rumble or roll immediately preceding the systole* and increasing in intensity as it approaches the first sound. At this stage of the disease the second sound can still be heard at the apex. The first sound is very sharply accented or snapping, and communicates a very decided shock to the ear when a rigid stethoscope is used. As a rule, the murmur is closely confined to the region of the apex beat and not transmitted any considerable distance in any direction. I have seen cases in which it was to be heard only

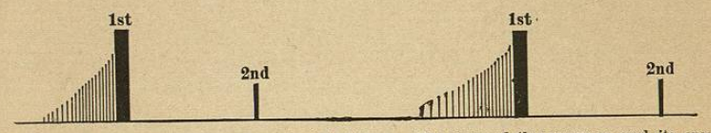

FIG. 130.—The Murmur of Mitral Stenosis—First Stage. The place of the murmur and its crescendo character are indicated by the position of the light lines just before the first sound and by their increasing length.

over an area the size of a half-dollar.[1] Very characteristic of mitral stenosis is a prolongation of the diastolic pause so that the interval beween the second sound of one cycle and the first sound of the next is unduly long. Occasionally the diastolic sound is reduplicated ("double-shock sound"—Sansom) at this stage of the disease, but this is much more frequent in the later phases of the lesion.[2]

Irregularity of the heart beat both in force and rhythm is very frequently present even in the early stages of the affection. The heart may be regular while the patient is at rest, but slight exertion is often sufficient to produce marked irregularity.

[1] It may, however, be widely transmitted to the left axilla and audible in the back or even over the whole of the left chest, especially when the stenosis is combined with regurgitation.

[2] This is the opinion of most observers. Sansom states that the "double-shock sound" may precede *all* other evidences of mitral stenosis.