II.

In the *second stage* the murmur and thrill are usually longer and may occupy th whole of diastole, beginning with considerable intensity just after the reduplicated second sound, quickly diminish-

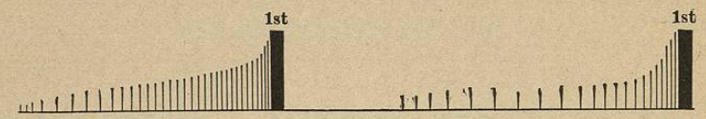

FIG. 131.—Type of Presystolic Murmur Often Heard in the Second Stage of Mitral Stenosis. Here the murmur fills the whole of diastole, with a gradual increase of intensity as it approaches the first sound. No second sound is audible at the apex.

ing until it is barely audible, and then again increasing with a steady crescendo up to the first sound of the next cycle.[1] These changes may be graphically represented as in Figs. 130 and 131. Diastole is now still more prolonged, so that the characteristic rhythm of this lesion is even more marked than in the earlier stages of the disease. In many cases at this stage no second sound is to be heard at all at the apex, although at the pulmonic orifice it is loud and almost invariably double. (This is one of the reasons for believing that the second sound which we usually hear at the apex is the transmitted aortic second sound. In mitral disease the aortic valves

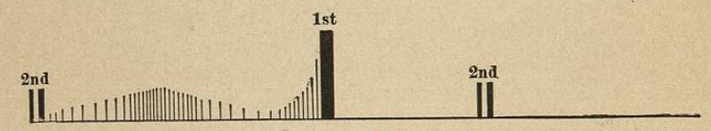

FIG. 132.—Type of Presystolic Murmur Sometimes Heard in the Second Stage of Mitral Stenosis. There is a double crescendo. The second sound seems reduplicated.

shut feebly owing to the relatively small amount of blood that is thrown into the aorta.)

At this stage of the disease enlargement of the heart begins to make itself manifest. The apex impulse is displaced to the left—

[1] Rarely one finds a crescendo in the middle of a long presystolic roll with a diminuendo as it approaches the first sound.

sometimes as far as the mid-axillary line, and often descends to the sixth interspace. Occasionally the cardiac dulness is increased to the right of the sternum.

The instability and fleeting character of the murmur in the earlier stages of the disease are much less marked in this, the second stage. The first sound at the apex still retains its sharp, thumping quality, and is often audible *without the murmur* in the back.

The irregularity of the heart is generally greater at this stage than in the earlier one.

III.

The third stage of the affection is marked by the disappearance of the characteristic murmur, and is generally synchronous with the development of tricuspid regurgitation. The right ventricle becomes dilated sometimes very markedly. Indeed, it may produce a visible pulsating tumor below the left costal border and be mistaken for cardiac aneurism (Osler). The snapping first sound and the "double-shock" sound usually remain audible, but the latter may be absent altogether. Diagnosis in this stage rests largely upon the peculiar snapping character of the first sound, together with the prolongation of diastole and the very great irregularity of the heart, both in force and rhythm. At times a presystolic thrill may be felt even when no murmur is to be heard.

The pulse shows nothing characteristic in many cases except that early and persistent irregularity which has been already alluded to. In other cases the wave is low, long, easily compressed, but quite perceptible between beats; but for the lack of sufficient power in the cardiac contractions the pulse would be one of high tension.

As the disease advances the irregularity of the pulse becomes more and more marked, and sometimes presents an amazing contrast with the relatively good general condition of the circulation. Even when not more than a third of the beats reach the wrist, the patient may be able to attend to light work and feel very well. Such cases make us feel as if a pulse were a luxury rather than a necessity.

Under the influence of digitalis the pulse is especially apt to

assume the *bigeminal* type in mitral stenosis. Every other beat is then so abortive that it fails to send a wave to the wrist, and the weak beat is succeeded by a pause. According to Broadbent the weak beat corresponds to an abortive contraction of the left ventricle accompanied by a normal contraction of the right ventricle, so that for each *two* strong beats of the right side of the heart we have one strong and one weak beat of the left side of the heart.

Mitral stenosis is in the great majority of cases combined with mitral regurgitation, and it often happens that the signs of regurgitation are so much more prominent than those of stenosis that the latter escape observation altogether, especially in the third stage of the disease, when the typical presystolic roll has disappeared. In such cases combined stenosis and regurgitation is to be distinguished from pure regurgitation by the sharpness of the first sound, which would be very unusual at this stage of a case of pure mitral regurgitation. The presence of reduplicated second sound, a "double-shock sound" at the outset of the prolonged diastolic pause, and of great irregularity in force and rhythm, is further suggestive of mitral stenosis.

Mitral stenosis is apt to be associated with hæmoptysis, with engorgement of the liver and ascites, and especially with arterial embolism. No other valve lesion is so frequently found associated with embolism. The lungs are generally very voluminous, and may therefore mask an increase in area or intensity of the cardiac dulness.

Differential Diagnosis.

I have already discussed the difficulty of distinguishing a double lesion at the mitral valve from a simple mitral regurgitation (see above, p. 215).

Other murmurs which may be mistaken for the murmur of mitral stenosis are:

(*a*) The Austin Flint murmur.

(*b*) The murmur of tricuspid stenosis.

(*c*) A rumbling murmur sometimes heard in children, after an attack of pericarditis.

(*a*) The Austin Flint murmur.

In 1862 Austin Flint studied two cases in which during life a typical presystolic roll was audible at the apex of the heart, yet in which post mortem the mitral valve proved to be perfectly normal, and the only lesion present was aortic insufficiency. This observation has since been verified by Osler, Bramwell, Gairdner, and other competent observers. At the Massachusetts General Hospital we have had seven such cases with autopsy. Yet, despite repeated confirmation, Flint's observation has remained for nearly forty years unknown to physicians at large. Its importance is this: Given a case of aortic regurgitation—a presystolic murmur at the apex does not necessarily mean stenosis of the mitral valve even though the murmur has the typical rolling quality and is accompanied by a palpable thrill. It may be only one of the by-effects of the aortic incompetency. How it is that a presystolic murmur can be produced at the apex in cases of aortic regurgitation has been much debated. Some believe it is due to the impact of the aortic regurgitant stream upon the ventricular side of the mitral valve, floating it out from the wall of the ventricle so as to bring it into contact with the stream of blood descending from the left auricle. Others suppose that the mingling of the two currents of blood, that from the mitral and that from the aortic orifice, is sufficient to produce the murmur.

Between the "Austin Flint murmur" thus defined and the murmur of true mitral stenosis, complicating aortic regurgitation, diagnosis may be impossible. If there are no dilatation of the mitral orifice and no regurgitation, either from this cause or from deformities of the mitral valve itself, any evidence of engorgement of the pulmonary circuit (accentuation of the pulmonic second sound, œdema of the lungs, hæmoptysis, and cough) speaks in favor of an actual narrowing of the mitral valve, while the absence of such signs and the presence of a predominating hypertrophy of the left ventricle tend to convince us that the murmur is of the type described by Austin Flint, *i.e.*, that it does not point to any stenosis of the mitral valve. The sharp, snapping first sound and systolic shock so characteristic of mitral stenosis are said to be

modified or absent in connection with murmurs of the Austin Flint type.

(*b*) Tricuspid obstruction.

Luckily for us as diagnosticians, stenosis of the tricuspid valve is a very rare lesion. Like mitral stenosis it is manifested by a presystolic rolling murmur whose point of maximum intensity is sometimes over the traditional tricuspid area, but may be at a point so near the mitral area as to be easily confused with stenosis of the latter valve.

The difficulty of distinguishing tricuspid stenosis from mitral stenosis is further increased by the fact that the two lesions almost invariably occur in conjunction. Hence we have two presystolic murmurs, perhaps with slightly different points of maximum intensity and possibly with a difference in quality, but often quite undistinguishable from each other. In the vast majority of cases, therefore, tricuspid stenosis is first recognized at the autopsy, and the diagnosis is at best a very difficult one.

(*c*) Broadbent, Rosenbach, and others have noticed in children who have just passed through an attack of pericarditis a rumbling murmur near the apex of the heart, which suggests the murmur of mitral stenosis. It is distinguished from the latter, however, by the absence of any accentuation of the first sound at the apex, as well as by the conditions of its occurrence and by its transiency. Such cases are important, since their prognosis is much more favorable than that of mitral stenosis.

Phear (*Lancet*, September 21, 1895) investigated 46 cases in which a presystolic murmur was observed during life and no mitral lesion found at autopsy. In 17 of these there was aortic regurgitation at autopsy; in 20 of these there was adherent pericardium at autopsy; in 9 nothing more than dilatation of the left ventricle was found. In none of these cases was the snapping first sound, so common in mitral stenosis, recorded during life.

It should be remembered that patients suffering from mitral stenosis are very frequently unaware of any cardiac trouble, and seek advice for anæmia, wasting, debility, gastric or pulmonary complaints. This is less often true in other forms of valvular dis-

ease. We should be especially on our guard in cases of supposed "nervous arrhythmia" or "tobacco heart," if there has been an attack of rheumatism or chorea previously. Such cases may present *no* signs of disease except the irregularity—yet may turn out to be mitral stenosis.

IV. Aortic Regurgitation.

Rheumatic endocarditis usually occurs in early life and most often attacks the mitral valve. The commonest cause of aortic disease on the other hand—arterio-sclerosis—is a disease of late mid-

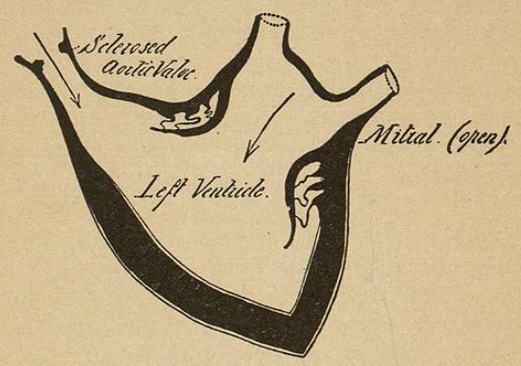

Fig. 133.—Diastole in Aortic Regurgitation. The blood is flowing back through the stumpy and incompetent aortic valves.

dle life, and attacks men much more often than women. When we think of aortic regurgitation, the picture that rises before us is usually that of a man past middle life and most often from the classes who live by manual labor. Nevertheless cases occur at all ages and in both sexes, and rheumatic endocarditis does not spare the aortic cusps altogether by any means.

Whether produced by arterio-sclerosis extending down from the aorta, or by rheumatic or septic endocarditis, the lesion which results in aortic regurgitation is usually a *thickening and shortening* of the cusps (see Fig. 133). In rare cases an aortic cusp may be ruptured as a result of violent muscular effort, and the signs and

symptoms of regurgitation then appear suddenly. But as a rule the lesion comes on slowly and insidiously, and unless discovered accidentally or in the course of routine physical examination it may exist unnoticed for years. Dropsy and cyanosis are relatively late and rare, and the symptoms which first appear are usually those of dyspnœa and precordial distress.

It is a disputed point whether relative and temporary aortic insufficiency due to stretching of the aortic orifice ever occurs. If it does occur, it is certainly exceedingly rare, as the aortic ring is very tough and inelastic.

Dilatation of the aortic arch—practically diffuse aneurism—*occurs in almost every case* of aortic regurgitation, and produces several important physical signs. This complication is a very well-known one, but has not, I think, been sufficiently insisted on in text-books of physical diagnosis. It forms part of that general enlargement of the arterial tree which is so characteristic of the disease.

Physical Signs.

Inspection reveals more that is important in this disease than in any other valvular lesion. In extreme cases the patient's face or hand may blush visibly with every systole. Not infrequently one can make the diagnosis across the room or in the street by noting the violent throbbing of the carotids, which may be such as to shake the person's whole head and trunk, and even the bed on which he lies. No other lesion is so apt to cause a heaving of the whole chest and a bobbing of the head, and no other lesion so often causes a bulging of the precordia, for in no other lesion is the enlargement of the heart so great (*cor bovinum* or ox-heart). The throbbing of the dilated aorta can often be felt and sometimes seen in the suprasternal notch or in the second right interspace. Not only the carotids but the subclavians, the brachials and radials, the femoral and anterior tibial, and even the digital and dorsalis pedis arteries may visibly pulsate, and the characteristic jerking quality of the pulse may be seen as well as felt. This visible pulsation in the peripheral arteries, while very characteristic of aortic

regurgitation, is occasionally seen in cases of simple hypertrophy of the heart from hard muscular work (*e.g.*, in athletes). If the arteries are extensively calcified, their pulsation become much less marked.

The peculiar conditions of the circulation whereby it is "changed into a series of discontinuous discharges as if from a catapult" (Allbutt) throws a great tensile strain upon all the arteries, and results, in almost every long-standing case, in increasing both their length

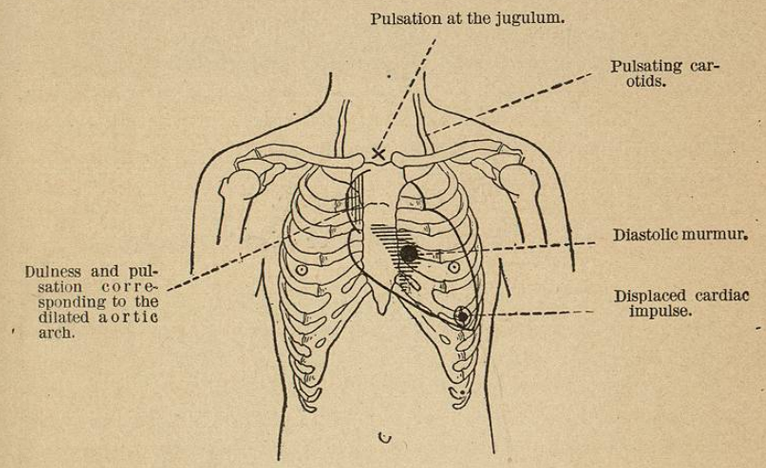

FIG. 134.—Aortic Regurgitation, Showing Position of the Diastolic Murmur and Areas of Visible Pulsation.

and their diameter. The visible arterial trunks become tortuous and distended, while the arch of the aorta is diffusely dilated and becomes practically an aneurism (see Fig. 134). With each heart beat the snaky arteries are often jerked to one side as well as made to throb.

Inspection of the region of the cardiac impulse almost always shows a very marked displacement of the apex beat both downward and outward (but especially the former), corresponding to the hypertrophy and still more to the dilatation of the left ventricle,

which is usually very great, and to the downward sagging of the enlarged aorta. Dilatation is in this disease an essentially helpful and compensatory process. In a small proportion of the cases no enlargement of the heart is to be demonstrated. This was true of 5 out of the last 67 cases which I have notes of, and generally denotes an early and slight lesion. Not at all infrequently one finds a systolic *retraction* of the interspaces near the apex beat instead of a systolic *impulse*. This is probably due to the negative pressure produced within the chest by the powerful contraction of an hypertrophied heart. In the suprasternal notch one often feels as well as sees a marked systolic pulsation transmitted from the arch of the dilated aorta, and sometimes mistaken for saccular aneurism.

Arterial pulsation of the liver and spleen are rarely demonstrable by a combination of sight and touch.

Capillary Pulsation.

If one passes the end of a pencil or other hard substance once or twice across the patient's forehead, and then watches the red mark so produced, one can often see a systolic flushing of the hyperæmic area with each beat of the heart. This is by far the best method of eliciting this phenomenon. It may also be seen if a glass slide is pressed against the mucous membrane of the lip so as partially to blanch it, or if one presses upon the finger-nail so as partially to drive the blood from under it; but in both these manœuvres error may result from inequality in the pressure made by the observer upon the glass slide or upon the nail. Very slight movements of the observer's fingers, even such as are caused by his own pulse, may give rise to changes simulating capillary pulsation. Capillary pulsation of normal tissues is not often seen in any condition other than aortic[1] regurgitation, yet occasionally one meets with it in diseases which produce very low tension of the pulse, such as phthisis or typhoid, anæmic and neurasthenic conditions, and I have twice seen it in perfectly healthy persons. In such cases the pulsation is usually less marked than in aortic regurgitation. Rarely pulsation may be detected in the peripheral veins.

[1] Jumping toothache and throbbing felon are common examples of capillary pulsation in inflamed areas.

Palpation.

Palpation verifies the position of the cardiac impulse and the heaving of the whole chest wall suggested by inspection. The shock of the heart is very powerful and deliberate unless dilatation

FIG. 135.—Sphygmographic Tracing from Normal Pulse.

is extreme, when it becomes wavy and diffuse. In the supraclavicular notch a systolic thrill is often to be felt. A diastolic thrill in the precordia is very rare.

The pulse is important, usually characteristic. The wave rises

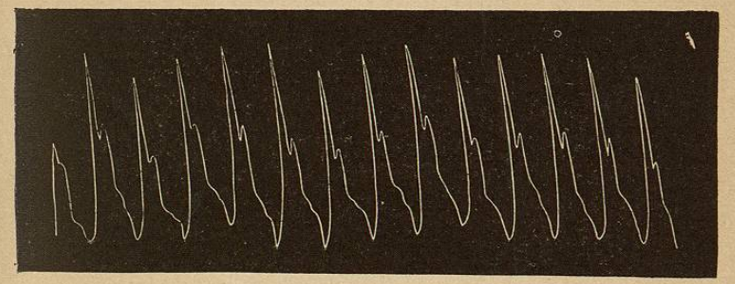

FIG. 136.—Sphygmographic Tracing of the "*Pulsus Celer*" in Aortic Regurgitation. Its collapsing character is well shown.

very suddenly and to an unusual height, then collapses completely and with great rapidity (pulsus celer) (see Figs. 135, 136).

This type of pulse, which is known as the "Corrigan pulse" or "water-hammer pulse," is exaggerated if one raises the patient's arm above the head so as to make the force of gravity aid in emptying the artery. The quality of the pulse in aortic regurgitation