gitation. I have recently reported (*Johns Hopkins Bull.*, May, 1903) three cases of intense anæmia associated with diastolic murmurs exactly like those of aortic regurgitation, but proved post mortem to be independent of any valvular lesion. The arterial phenomena were not marked, but the diagnosis of such cases is very hard. Luckily they are rare. The origin is obscure.

It must be remembered that aortic regurgitant murmurs are often exceedingly faint, and should be listened for with the greatest care and under the most favorable conditions.

Estimation of the Extent and Gravity of the Lesion.

The extent of the lesion is roughly proportional to—

(*a*) The amount of hypertrophy of the left ventricle.

(*b*) The degree to which the pulse collapses during diastole (provided the radial is not so much calcified as to make collapse impossible).

(*c*) The degree to which the murmur replaces the second sound as heard over the right carotid artery (Broadbent).

Irregularity of the pulse is a far more serious sign in this disease than in lesions of the mitral valve, and indicates the beginning of a serious failure of compensation.

Another grave sign is a diminution in the intensity of the murmur.

Complications.

(1) *Dilatation of the Aorta.*—Diffuse dilatation of the aortic arch is usually associated with aortic regurgitation and may produce a characteristic area of dulness to the right of the sternum (see Fig. 134). Not infrequently this dilatation is the cause of a systolic murmur to be heard over the region of the aortic arch and in the great vessels of the neck.

(2) *Roughening of the Aortic Valves.*—In almost all cases of aortic regurgitation the valves are sufficiently roughened to produce a systolic murmur as the blood flows over them. This murmur is heard at or near the conventional aortic area, and may be transmitted into the carotids. (The relation of these murmurs to the diagnosis of aortic stenosis will be considered with the latter lesion.)

(3) The return of arterial blood through the aortic valves into the left ventricle produces in time both hypertrophy and dilatation of this chamber, and results ultimately in a stretching of the mitral orifice which renders the mitral curtains incompetent. The result is a "*relative mitral insufficiency*," *i.e.*, one in which the mitral valve is intact but too short to reach across the orifice which it is intended to close. Such an insufficiency of the mitral occurs in most well-marked cases; it temporarily relieves the overdistention of the left ventricle and often the accompanying angina, although at the cost of engorging the lungs.[1]

(4) *The Austin Flint Murmur.*—The majority of cases of aortic regurgitation are accompanied by a presystolic murmur at the apex, which may be due to a genuine mitral stenosis or may be produced in the manner suggested by Austin Flint. (For a fuller discussion of this murmur see above, p. 227.)

(5) *Aortic stenosis* frequently supervenes in cases of aortic regurgitation, and results in a more or less temporary improvement in the patient's condition. It has the effect of increasing the intensity of the diastolic murmur, since the regurgitating stream has to pass through a smaller opening.

The general visible arterial pulsation becomes much less marked if stenosis supervenes on regurgitation.

AORTIC STENOSIS.

Uncomplicated aortic stenosis is by far the rarest of the valvular lesions of the left side of the heart, as well as the most difficult to recognize. Out of two hundred and fifty-two autopsies made at the Massachusetts General Hospital in cases of valvular disease there was not one of uncomplicated aortic stenosis. Twenty-nine cases occurred in combination with aortic regurgitation. During life the diagnosis of aortic stenosis is frequently made, but often on insufficient evidence—*i.e.*, upon the evidence of a systolic murmur heard with maximum intensity in the second right intercostal space

[1] This relative insufficiency of the mitral valve has been termed its "safety-valve" action, but the safety is but temporary and dearly bought.

and transmitted into the vessels of the neck. Such a murmur does indeed occur in aortic stenosis, but is by no means peculiar to this condition. Of the other diseases which produce a similar murmur more will be said under Differential Diagnosis.

For the diagnosis of aortic stenosis we need the following evidence:

(1) A systolic murmur heard best in the second right intercostal space and transmitted to the neck.

(2) The characteristic pulse (*vide infra*).

(3) A palpable thrill (usually).

(4) Absence or great enfeeblement of the aortic second sound.

Of these signs the characteristic *pulse* is probably the most important, and no diagnosis of aortic stenosis is possible without it. The heart may or may not be enlarged.

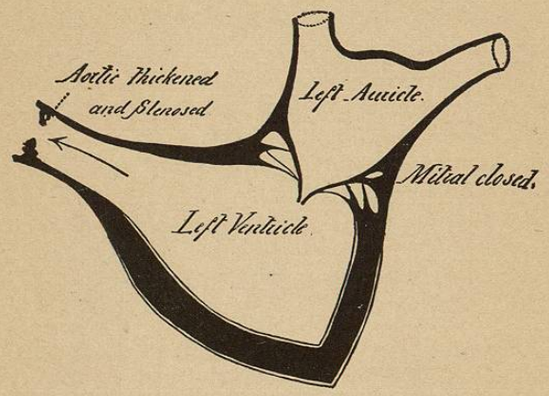

FIG. 139.—Aortic Stenosis. The heart is in systole and the blood column is obstructed by the narrowed aortic ring. The mitral is closed (as it should be).

Each of these points will now be described more in detail.

(1) *The Murmur.*

(*a*) The *maximum intensity* of the murmur, as has already been said, is usually in the second right intercostal space near the sternum or a little above that point near the sterno-clavicular articulation, but it is by no means uncommon to find it lower down, *i.e.*,

in the third, fourth, or fifth right interspace, and occasionally it is best heard to the left of the sternum in the second or third intercostal space. (*b*) *The time* of the murmur is *late systolic;* that is, it follows the apex impulse at an appreciable interval, contrasting in this respect with the systolic murmur usually to be heard in mitral regurgitation. (*c*) The murmur is usually *widely transmitted*, often being audible over the whole chest and occasionally over the skull and the arterial trunks of the extremities (see Fig. 140). It is usually heard less well over that portion of the precordia occupied by the right ventricle, while, on the other hand, it is relatively loud in the region of the apex impulse, whither it is transmitted through the left ventricle. The same line of transmission

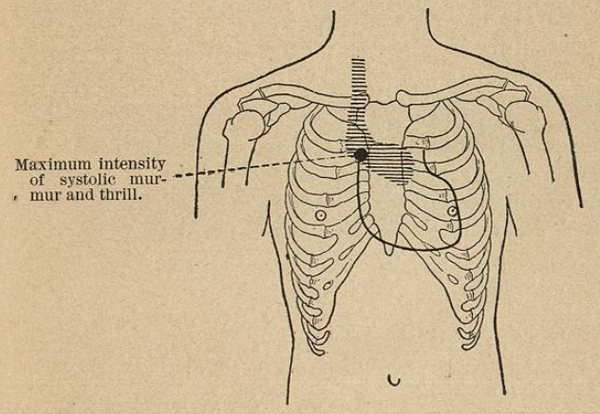

FIG. 140.—Aortic Stenosis. The murmur is audible over the shaded area and sometimes over the whole chest.

was mentioned above as characteristic of the murmur of aortic regurgitation in many cases. The murmur is also to be heard over the carotids and subclavians, and can often be traced over the thoracic aorta along the spine and down the arms.

Until compensation fails the murmur is apt to be a very loud one, especially in the recumbent position; it is occasionally audible at some distance from the chest, and is often rough and vibrating, sometimes musical or croaking. Its length is unusually

great, extending throughout the whole of systole, but to this rule there are occasional exceptions. The first sound in the aortic region is altogether obliterated, as a rule, and the second sound is either absent or very feeble.[1]

(2) *The Pulse.*

Owing to the opposition encountered by the left ventricle in its attempt to force blood into the aorta, its contraction is apt to be prolonged; hence the pulse wave *rises* gradually and late, *and falls away slowly.* This is shown very well in sphygmographic tracings (see Fig. 141). But further, the blood thrown into the aorta by the left ventricle is prevented, by the narrowing of the aortic valves, from striking upon and expanding the arteries with its ordinary force; hence the pulse wave is not only slow to rise but *small in*

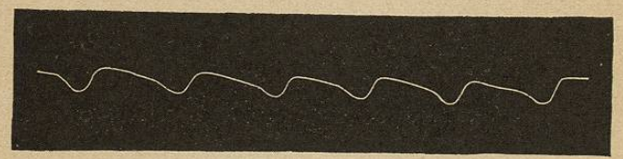

FIG. 141.—Sphygmographic Tracing of the Pulse in Uncomplicated Aortic Stenosis. Compare with the normal pulse wave and with that of aortic regurgitation (page 174).

height, contrasting strongly with the powerful apex beat (*"pulsus parvus"*). Again, the delay in the emptying of the left ventricle, brought about by the obstruction at the aortic valves, renders the contractions of the heart relatively *infrequent*, and hence the pulse is *infrequent* (*pulsus rarus*) as well as small and slow to rise. The *"pulsus rarus, parvus, tardus"* is, therefore, a most constant and important point in diagnosis, but unfortunately it is to be felt in perfection only in the *very* rare cases in which aortic stenosis occurs uncomplicated. When stenosis is combined with regurgitation, as is almost always the case, the above-described qualities of the pulse are greatly modified as a result of the regurgitation. It

[1] "Occasionally, as noted by W. H. Dickinson, there is a musical murmur of great intensity in the region of the apex, probably due to a slight regurgitation at high pressure through the mitral valve."—OSLER.

is also to be remembered that the pulse of aortic stenosis is by no means unalterable and does not exhibit its typical plateau at all times.

A less characteristic, but decidedly frequent, variation in the pulse wave of aortic stenosis is the *anacrotic* curve. The slow, long pulse with a long plateau at the summit is seen also in some cases of mitral stenosis and renal disease, and is not peculiar to aortic stenosis, but taken in connection with the other signs of the disease it has great value in diagnosis.

(3) *The Thrill.*

In the majority of cases an intense purring vibration may be felt if the hand is laid over the upper portion of the sternum, especially over the second right intercostal space. This thrill is continued into the carotids, can occasionally be felt at the apex, and rarely over a considerable area of the chest. It is a very important aid in the diagnosis of aortic stenosis, but is by no means pathognomonic, since aneurism may produce a precisely similar vibration of the chest wall.

The heart is slightly enlarged to the left and downward as a rule, but the apex impulse is unusually indistinct, "a well-defined and deliberate push of no great violence" (Broadbent). Corresponding to the protracted sustained systole the first sound at the apex is dull and long, but not very loud.

Differential Diagnosis.

A systolic murmur heard loudest in the second right intercostal space is by no means peculiar to aortic stenosis, but may be due to any of the following conditions:

(*a*) Roughening, stiffness, fenestration, or slight congenital malformation of the aortic valves.

(*b*) Roughening or diffuse dilatation of the arch of the aorta.

(*c*) Aneurism of the aorta or innominate artery.

(*d*) Functional murmurs.

(*e*) Pulmonary stenosis.

(*f*) Open ductus arteriosus.

(*g*) Mitral regurgitation.

(*a* and *b*) The great majority of such systolic murmurs at the base of the heart, first appearing after middle life, are due to the causes mentioned above under *a*, *b*, and *c*. In such cases it is usually combined with accentuation and ringing quality of the aortic second sound owing to the arterio-sclerosis and high arterial tension associated with the changes which produce the murmur. This *accentuation of the aortic second sound* enables us, except in extraordinarily rare cases, to exclude aortic stenosis, in which the intensity of the aortic second sound is almost always much reduced.

Diffuse dilatation of the aorta, such as often accompanies aortic regurgitation, is a frequent cause of a systolic murmur loudest in the second right interspace. This may be recognized in certain cases by the characteristic area of dulness on percussion and by its association with aortic regurgitation of long standing (see Fig. 134).

Roughening of the intima of the aorta (*endaortitis*) is always to be suspected in elderly patients with calcified and tortuous peripheral arteries, and such a condition of the aorta doubtless favors the occurrence of a murmur, especially when accompanied by a slight degree of dilatation. The absence of a thrill and a long, slow pulse with a low maximum serves to distinguish such murmurs from those of aortic stenosis.

(*c*) Aneurism of the ascending arch of the aorta or of the innominate artery may give rise to every sign of aortic stenosis except the characteristic pulse and the diminution of the aortic second sound. In aneurism we may have a well-marked tactile thrill and a loud systolic murmur transmitted into the neck, but there is usually some pulsation to be felt in the second right intercostal space and often some difference in the pulses or in the pupils, as well as a history of pain and symptoms of pressure upon the trachea and bronchi or recurrent laryngeal nerve. In aneurism the aortic second sound is usually loud and accompanied by a shock, and the pulse shows none of the characteristics of aortic stenosis.

(*d*) Functional murmurs, sometimes known as "hæmic," are occasionally best heard in the aortic area instead of in their usual situation (second left intercostal space). They occur especially in young, anæmic persons, are not accompanied by any cardiac enlargement, by any palpable thrill, any diminution in the aortic second sound, or any distinctive abnormalities in the pulse.

(*e*) Pulmonary stenosis, a rare lesion, is manifested by a systolic murmur and by a thrill whose maximum intensity is usually on the left side of the sternum. In the rare cases in which this murmur is best heard in the aortic area it may be distinguished from the murmur of aortic stenosis by the fact that it is not transmitted into the vessels of the neck, has no effect upon the aortic second sound, and is not accompanied by the characteristic changes in the pulse.

(*f*) The murmur due to persistence of the ductus arteriosus may last through systole and into diastole; it may be accompanied by a thrill, but does not affect the aortic second sound nor the pulse.

(*g*) The systolic murmur of aortic stenosis may be heard loudly at the apex, and hence the lesion may be mistaken for mitral regurgitation. But the maximum intensity of the murmur of aortic stenosis is almost invariably in the aortic area, and its association with a thrill and a long, slow pulse should enable us easily to differentiate the two lesions.

By the foregoing differentiæ aortic stenosis may be distinguished from the other conditions which resemble it, *provided it occurs uncomplicated*, but unfortunately this is very rare. As a rule, it occurs in connection with aortic regurgitation, and its characteristic signs are therefore obscured or greatly modified by the signs of the latter disease. We may suspect it in such cases (provided the mitral valve is sufficient) when we have, in addition to the signs of aortic regurgitation, a systolic murmur and palpable thrill in the aortic area transmitted into the great vessels, a modification of the Corrigan pulse in the direction of the "*pulsus tardus, rarus, parvus,*" and less visible arterial pulsation than is to be expected in pure aortic regurgitation.

Occasionally one can watch the development of an aortic stenosis out of what was formerly a pure regurgitant lesion, the stenosis gradually modifying the characteristics of the previous condition. One must be careful, however, to exclude a relative mitral insuffi-

ciency which, as has been already mentioned above, is very apt to supervene in cases of aortic disease, owing to dilatation of the mitral orifice, and which may modify the characteristic signs of aortic regurgitation very much as aortic stenosis does.

TRICUSPID REGURGITATION.

Endocarditis affecting the tricuspid valve is rare in post-fœtal life; in the fœtus it is not so uncommon. In cases of ulcerative or malignant endocarditis occuring in adult life, the tricuspid valve is occasionally involved, but the majority of cases of tricuspid disease occur as a result of disease of the mitral valve and in the following manner: Hypertrophy of the right ventricle occurs as a result of the mitral disease, is followed in time by dilatation, and with this dilatation comes a stretching of the ring of insertion of the tricuspid valve, and hence a regurgitation through that valve. Tricuspid regurgitation, then, occurs in the latest stages of almost every case of mitral disease and sometimes during the severer attacks of failing compensation.

Out of 405 autopsies at Guy's Hospital in which evidence of tricuspid regurgitation was found, 271, or two-thirds, resulted from mitral disease, 68 from myocardial degeneration, 55 from pulmonary disease (bronchitis, emphysema, cirrhosis of the lung). Very few of these cases had been diagnosed during life, and in all of them the valve was itself healthy but insufficient to close the dilated orifice.

Gibson and some other writers believe that temporary tricuspid regurgitation is the commonest of all valve lesions, and results from weakening of the right ventricle in connection with states of anæmia, gastric atony, fever, and many other conditions. It is very difficult to prove or disprove such an assertion.

Tricuspid regurgitation is often referred to as serving like the opening of a "*safety valve*" to relieve a temporary pulmonary engorgement. This "safety-valve" action, however, may be most disastrous in its consequences to the organism as a whole, despite the temporary relief which it affords to the overfilled lungs. The engorgement is simply transferred to the liver and thence to the

abdominal organs and the lower extremities, so that as a rule the advent of tricuspid regurgitation is recognized not as a relief but as a serious and probably fatal disaster.

Physical Signs.

(1) A systolic murmur is heard loudest at or near the fifth left costal cartilage.

(2) Systolic venous pulsation in the jugulars and in the liver.

(3) Engorgement of the right auricle producing an area of dulness beyond the right sternal margin.

(4) Intense cyanosis.

(1) *The Murmur.*—The maximum intensity of the systolic murmur of tricuspid regurgitation is usually near the junction of the fifth or sixth left costal cartilages with the sternum. Leube finds the murmur a rib higher up, but it is generally agreed that the tricuspid area is a large one, so that the murmur may be heard anywhere over the lower part of the sternum or even to the right of it. On the other hand, there are some tricuspid murmurs which are best heard at a point midway between the apex impulse and the ensiform cartilage. The murmur is not widely transmitted and is usually inaudible in the back; at the end of expiration its intensity is increased.

In some cases we have no evidence of tricuspid regurgitation other than the murmur just described, but—

(2) Of more importance in diagnosis is the presence of a systolic pulsation in the external jugular veins and of the liver, which unfortunately is not always present, but which when present is pathognomonic. I have already explained (see p. 88) the distinction between true *systolic* jugular pulsation, which is practically pathognomonic of tricuspid regurgitation, and simple presystolic undulation or distention of the same veins, which has no necessary relation to this disease. The decisive test is the effort permanently to empty the vein by stroking it upward from below. If it instantly refills from below and continues to pulsate, tricuspid regurgitation is almost certainly present. If, on the other hand, it does not refill from below, the cause must be sought elsewhere.