Pulsation in the liver must be distinguished from the "jogging" motion which may be transmitted to it from the abdominal aorta or from the right ventricle. To eliminate these transmitted impulses one must be able to grasp the liver bimanually, one hand in front and one resting on the lower ribs behind, and to feel it distinctly expand with every systole, or else to take its edge in the hand and to feel it enlarge in one's grasp with every beat of the heart.

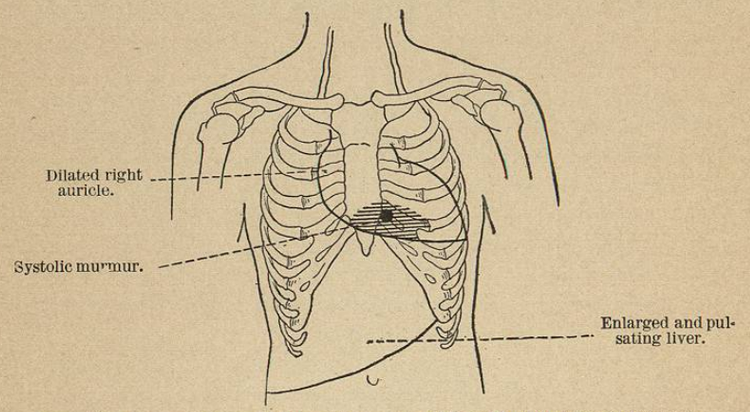

FIG. 142.—Tricuspid Regurgitation. The murmur is heard best over the shaded area.

Pressure upon the liver often causes increased distention and pulsation of the external jugulars if tricuspid regurgitation is present.

(3) Enlargement of the heart, both to the right and to the left, as well as downward, can usually be demonstrated. In rare cases a dilatation of the right auricle may be suggested by a percussion outline such as that shown in Fig. 142.

The pulmonic second sound is usually not accented. The importance of this in differential diagnosis will be mentioned presently. If a progressive diminution in the intensity of the sound occurs under observation, the prognosis is very grave.

(4) Cyanosis is usually very great, and dyspnœa and pulmonary œdema often make the patient's condition a desperate one.

Differential Diagnosis.

The statistics of the cases autopsied at the Massachusetts General Hospital show that tricuspid regurgitation is less often recognized during life than any other valvular lesion. The diagnosis was made ante mortem on only five out of twenty-nine cases. This is due to the following facts:

(*a*) Tricuspid regurgitation may be present and yet give rise to no physical signs which can be recognized during life.

(*b*) Tricuspid regurgitation occurs most frequently in connection with mitral regurgitation; hence its signs are frequently masked by those of the latter lesion. It is, therefore, a matter of great importance as well as of great difficulty to distinguish tricuspid regurgitation from

(1) *Mitral Regurgitation.*

The difficulties are obvious. The murmur of mitral regurgitation has its maximum intensity not more than an inch or two from the point at which the tricuspid murmur is best heard. Both are systolic in time. They are, therefore, to be distinguished only—

(*a*) In case we can demonstrate that there are two areas in which a systolic murmur is heard with relatively great intensity, with an intervening space over which the murmur is less clearly to be heard (see Fig. 143).

(*b*) Occasionally the two systolic murmurs are of different pitch or of different quality, and may be thus distinguished.

(*c*) Tricuspid murmurs are not transmitted into the left axilla and are rarely audible in the back, and this fact is of value in case we have to distinguish between uncomplicated tricuspid regurgitation and uncomplicated mitral regurgitation. Unfortunately these lesions are very apt to occur simultaneously, so that in practice our efforts are generally directed toward distinguishing between a pure mitral regurgitation and one complicated by tricuspid regurgitation.

(*d*) In cases of doubt the phenomena of venous pulsation in the jugulars and in the liver are decisive if present, but their absence proves nothing.

(*e*) Accentuation of the pulmonic second sound is almost invariably present in uncomplicated mitral disease and is apt to disappear in case the tricuspid begins to leak, since engorgement of the lungs is thereby for the time relieved, but in many cases the pulmonic second sound remains most unaccountably strong even when the tricuspid is obviously leaking.

(2) From "functional" systolic murmurs tricuspid insufficiency may generally be distinguished by the fact that its murmur is best

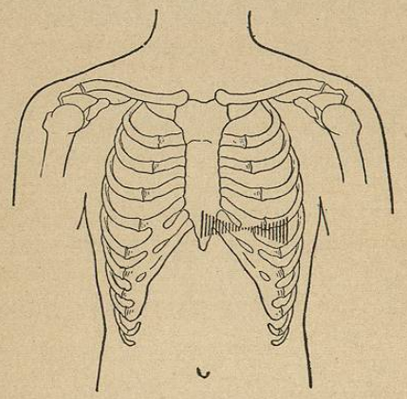

FIG. 143.—Two Systolic Murmurs (Mitral and Tricuspid) with a "Vanishing Point" between.

heard in the neighborhood of the ensiform cartilage, and not in the second right intercostal space where most functional murmurs have their seat of maximum intensity. Functional murmurs are unaccompanied by venous pulsation, cardiac dilatation, or cyanosis.

(3) Occasionally a pericardial friction rub simulates the murmur of tricuspid insufficiency, but, as a rule, pericardial friction is much more irregular in the time of its occurrence and is not regularly synchronous with any definite portion of the cardiac cycle.

TRICUSPID STENOSIS.

One of the rarest of valve lesions is narrowing of the tricuspid valve. No case has come under my observation, and in 1898, Herrick was able to collect but 154 cases from the world's literature. Out of these 154 cases, 138, or 90 per cent, were combined with mitral stenosis, and only 12 times has tricuspid stenosis been known to occur alone.[1] These observations account for the fact that tricuspid stenosis has hardly ever been recognized during life, since the murmur to which it gives rise is identical in time and quality and nearly identical in position with that of mitral stenosis. Narrowing of the tricuspid valve is to be diagnosed, therefore, only by the recognition of a presystolic murmur best heard in the tricuspid area and distinguished either by its pitch, quality, or position from the other presystolic murmur due to the mitral stenosis which is almost certain to accompany it.

The heart is usually enlarged, especially in its transverse direction, but the enlargement is just such as mitral stenosis produces, and does not aid our diagnosis at all.

The diagnosis is still further complicated in many cases by the presence of an aortic stenosis in addition to a similar lesion at the tricuspid and mitral valves, so that it seems likely that in the future as in the past the lesion will be discovered first at autopsy.

PULMONARY REGURGITATION.

Organic disease of the pulmonary valve is excessively rare in post-fœtal life, but may occur as part of an acute ulcerative or septic endocarditis. A *temporary* functional regurgitation through the pulmonary valve may be brought about by any cause producing very *high pressure in the pulmonary artery*. I have known two medical students with perfectly healthy hearts who were able, by prolonged holding of the breath, to produce a short, high-pitched diastolic murmur best heard in the second and third left intercostal spaces and ceasing as soon as the breath was let out. Of the occurrence of a murmur similarly produced under pathological conditions, especially in mitral stenosis, much has been written by Graham Steell.

[1] Out of 87 cases collected from the post-mortem records of Guy's Hospital, 85, or 97 per cent, were associated with still more extensive mitral stenosis.

From the diastolic murmur of aortic regurgitation we may distinguish the diastolic murmur of pulmonary incompetency by the fact that the latter is best heard over the pulmonary valve, is never transmitted to the apex of the heart nor to the great vessels, and is never associated with a Corrigan pulse nor with capillary pulsation.[1] The right ventricle is hypertrophied, the pulmonic second sound is sharply accented and followed immediately by the murmur. Evidences of septic embolism of the lungs are frequently present and assist us in diagnosis. The regurgitation which may take place through the rigid cone of congenital pulmonary stenosis is not recognizable during life.

Pulmonary Stenosis.

Among the rare congenital lesions of the heart valves this is probably the commonest. The heart, and particularly the right ventricle, is usually much enlarged. There is a history of cyanosis and dyspnœa since birth. Pulmonary tuberculosis complicates from one-fourth to one-third of all cases. A systolic thrill is usually to be felt in the second left intercostal space, and a loud systolic murmur is heard in the same area. The pulmonic second sound is weak.

The region in which this murmur is best heard has been happily termed the "*region of romance*" on account of the multiplicity of mysterious murmurs which have been heard there. The systolic murmur of pulmonary stenosis must be distinguished from

(*a*) Functional murmurs due to anæmia and debility or to severe muscular exertion, and possibly associated with a dilatation of the conus arteriosus.

(*b*) Uncovering of the conus arteriosus through lack of expansion of the lung.

(*c*) Aortic stenosis.

(*d*) Mitral regurgitation.

(*e*) Aneurism.

(*f*) Roughening of the intima of the aortic arch.

[1] By registering the variations of pressure in the tracheal column of air Gerhardt has shown graphically that a systolic pulsation of the pulmonary capillaries may occur in pulmonary regurgitation. With the stethoscope a systolic whiff may be heard all over the lungs.

(*a* and *b*) Functional murmurs, and those produced in the conus arteriosus, are rarely if ever accompanied by a thrill, are rarely so loud as the murmur of pulmonary stenosis, and are not associated with dyspnœa, cyanosis, and enlargement of the right ventricle.

(*c*) The murmur of aortic stenosis is usually upon the right side of the sternum and is transmitted to the neck, whereas the murmur of pulmonary stenosis is never so transmitted and is not associated with characteristic changes in the pulse (see above, p. 242).

(*d*) The murmur of *mitral regurgitation* is occasionally loudest in the region of the pulmonary valve, but differs from the murmur of pulmonary stenosis in being, as a rule, transmitted to the back and axilla and associated with an accentuation of the pulmonary second sound.

(*e*) *Aneurism* may present a systolic murmur and thrill similar to those found in pulmonary stenosis, but may usually be distinguished from the latter by the presence of the positive signs of aneurism, viz.—pulsation, and dulness in the region of the murmur, and signs of pressure on the trachea or on other structures in the mediastinum.

(*f*) Roughening of the aortic arch occurs after middle life, while pulmonary stenosis is usually congenital. The murmur due to roughening may be transmitted into the carotids; that of pulmonary stenosis never. Enlargement of the right ventricle is characteristic of pulmonary stenosis, but not of aortic roughening.

COMBINED VALVULAR LESIONS.

It is essential that the student should understand from the first that the number of murmurs audible in the precordia is no gauge for the number of valve lesions. We may have four distinct murmurs, yet every valve sound except one. This is often the case in aortic regurgitation—systolic and diastolic murmurs at the base of the heart, systolic and presystolic at the apex, yet no valve injured except the aortic. In such a case the systolic aortic murmur is due to roughening of the aortic valve. The systolic apex murmur results from relative mitral leakage (with a sound valve). The presystolic apex murmur is of the "Flint" type. Hence in this

case the *diastolic* murmur alone of the four audible murmurs is due to a valvular lesion.

It is a good rule not to multiply causes unnecessarily, and to explain as many signs as possible under a single hypothesis In the above example the mitral leak might be due to an old endocarditis, and there *might* be mitral stenosis and aortic stenosis as well, but since we can explain all the signs as results—direct and indirect—of one lesion (aortic regurgitation) it is better to do so, and post-mortem experience shows that our diagnosis is more likely to be right when it is made according to this principle.

The most frequent combinations are:

(1) Mitral regurgitation with mitral stenosis.

(2) Aortic regurgitation with mitral regurgitation (with or without stenosis).

(3) Aortic regurgitation with aortic stenosis, with or without mitral disease.

(1) *Double Mitral Disease.*

(*a*) It very frequently happens that the mitral valve is found to be both narrowed and incompetent at autopsy when only one of these lesions had been diagnosed during life. In fact mitral steno-

FIG. 144.—Mitral Stenosis and Regurgitation, showing relation of murmur to first heart sound.

sis is almost never found at autopsy without an associated regurgitation, so that it is fairly safe to assume, whenever one makes the diagnosis of mitral stenosis, that mitral regurgitation is present as well, whether it is possible to hear any regurgitant murmur or not (see Fig. 144).

(*b*) On the other hand, with a double mitral lesion one may have only the regurgitant murmur at the mitral valve and nothing to suggest stenosis unless it be a surprising sharpness of the first mitral sound. In chronic cases the changeableness of the murmurs both in type and position is extraordinary. One often finds at one

visit evidences of mitral stenosis and at another evidences of mitral regurgitation alone. Either murmur may disappear altogether for a time and reappear subsequently. This is peculiarly true of the presystolic murmur, which is notoriously one of the most fleeting and uncertain of all physical signs.

As a rule the same inflammatory changes which produce mitral regurgitation in early life result as they extend in narrowing the mitral valve, so that the signs of stenosis come to predominate in later years. Coincidently with this narrowing of the diseased valve a certain amount of improvement in the patient's symptoms may take place, and Rosenbach regards the advent of stenosis in such a case as an attempt at a regenerative or compensatory change. In many cases, however, no such amelioration of the symptoms follows.

(2) *Aortic Regurgitation with Mitral Disease.*

The signs of mitral disease occurring in combination with aortic regurgitation do not differ essentially from those of pure

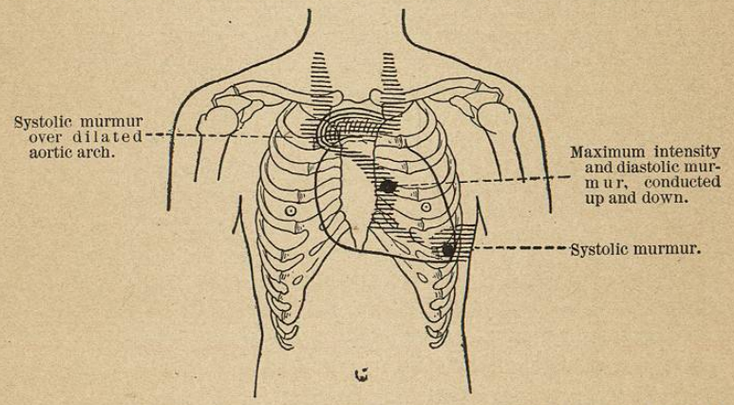

FIG. 145.—Aortic and Mitral Regurgitation. The shaded areas are those in which the murmurs are loudest.

mitral disease except that the enlargement of the heart is apt to be more general and correspond less exclusively to the right ventricle (see Figs. 145 and 146). The manifestations of the aortic le-

sion, on the other hand, are considerably modified by their association with the mitral disease. The Corrigan pulse is distinctly less sharp at the summit and rises and falls less abruptly. Capillary

FIG. 146.—Showing Relation of Murmurs to Heart Sound in Regurgitation at the Aortic and Mitral Valves.

pulse is less likely to be present, and the throbbing of the peripheral arteries is less often visible.

(3) *Aortic Regurgitation with Aortic Stenosis.*

If the aortic valves are narrowed as well as incompetent, we find very much the same modification of the physical signs characteristic of aortic regurgitation as is produced by the advent of a mitral lesion; that is to say, the throbbing in the peripheral arteries is less violent, the characteristics of the radial pulse are less marked, and the capillary pulsation is not always to be obtained at all. Indeed, this blunting of all the typical manifestations of aortic regurgitation may give us material aid in the diagnosis of aortic stenosis, provided always that the mitral valve is still performing its function.

(4) The association of mitral disease with tricuspid insufficiency has been already described on p. 218.

CHAPTER XI.

PARIETAL DISEASE.—CARDIAC NEUROSES.—CONGENITAL MALFORMATIONS OF THE HEART.

Parietal Disease of the Heart.

Acute Myocarditis.

THE myocardium is seriously, though not incurably, affected in all continued fevers, owing less to the fever itself than to the toxæmia associated with it. "Cloudy swelling," or granular degeneration of the muscle fibres, is produced by relatively mild infections, while a general septicæmia due to pyogenic organisms may produce extensive *fatty* degeneration of the heart within a few days.

The *physical signs* are those of *cardiac weakness*. The most significant change is in the quality of the first sound at the apex of the heart, which becomes gradually shorter until its quality is like that of the second sounds, while in some cases its feebleness makes the second sounds seem accented by comparison. Soft blowing systolic murmurs may develop at the pulmonary orifice, less often at the apex or over the aortic valve.

The apex impulse becomes progressively feebler and more like a tap than a push. *Irregularity* and *increasing rapidity* are ominous signs which may be appreciated in the radial pulse, but still better by auscultation of the heart itself. In most of the acute infections evidence of dilatation of the weakened cardiac chambers is rarely to be obtained during life (although at autopsy it is not infrequently found),[1] but in *acute articular rheumatism* an acute dilatation of the heart appears to be a frequent complication, independ-

[1] Henchen's recent monograph on this subject, "Ueber die acute Herzdilatation bei acuten Infectionskrankheiten," Jena, 1899, does not seem to me convincing.