portance of not confusing them with true cardiac murmurs. They were formerly thought to indicate phthisis, but such is not the case.

Murmurs of Venous Origin.

I have already mentioned that the venous hum so often heard in the neck in cases of anæmia may be transmitted to the region of the base of the heart and heard there as a diastolic murmur owing to the acceleration of the venous current by the aspiration of the right ventricle during diastole. Such murmurs are very rare and may usually be obliterated by pressure upon the bulbus jugularis, or even by the compression brought to bear upon the veins of the neck when the head is sharply turned to one side. They are heard better in the upright position and during inspiration.

Arterial Murmurs.

(1) Roughening of the arch of the aorta, due to chronic endocarditis, is a frequent cause in elderly men of a systolic murmur, heard best at the base of the heart and transmitted into the vessels of the neck. Such a murmur is sometimes accompanied by a palpable thrill. From cardiac murmurs it is distinguished by the lack of any other evidence of cardiac disease and the presence of marked arterio-sclerosis in the peripheral vessels (see further discussion under Aortic Stenosis, p. 239, and under Aneurism, p. 282.

(2) A narrowing of the lumen of the subclavian artery, due to some abnormality in its course, may give rise to a systolic murmur heard close below the clavicle at its outer end. The murmur is greatly influenced by movements of the arm and especially by respiratory movements. During inspiration it is much louder, and at the end of a forced expiration it may disappear altogether. Occasionally such murmurs are transmitted through the clavicle so as to be audible above it.

(3) Pressure exerted upon any of the superficial arteries (carotid, femoral, etc.) produces a systolic murmur (see below, p. 237). Diastolic arterial murmurs are peculiar to aortic regurgitation.

(4) Over the anterior fontanelle in infants and over the gravid uterus systolic murmurs are to be heard which are probably arterial in origin.

CHAPTER X.

DISEASES OF THE HEART.

VALVULAR LESIONS.

Clinically it is convenient to divide the ills which befall the heart into three classes:

(1) Those which deform the cardiac valves (valvular lesions).
(2) Those which weaken the heart wall (parietal disease).
(3) Congenital malformations.

Lesions which affect the cardiac valves without deforming them are not often recognizable during life. The vegetations of acute endocarditis[1] do not usually produce any peculiar physical signs until they have so far deformed or obstructed the valves as to prevent their opening or closing properly.

The murmurs which are often heard over the heart in cases of acute articular rheumatism cannot be considered as evidence of vegetative endocarditis unless valvular deformities, and their results in valvular obstruction or incompetency, ensue. The chordæ tendineæ may be ruptured or shortened, thickened, and welded together into shapeless masses, but if these deformities do not affect the action of the valves we have no means of recognizing them during life. Congenital malformations are practically unrecognizable as such. If they do not affect the valves, we cannot with any certainty make out what is wrong.

For physical diagnosis, then, heart disease means either de-

[1] See Appendix.

formed valves of weakened walls. Whatever else may exist, we are none the wiser for it unless the autopsy enlightens us.

In this chapter I shall confine myself to the discussion of valvular lesions and their results.

Valvular lesions are of two types:

(*a*) Those which produce partial obstruction of a valve orifice or prevent its opening fully ("*stenosis*").

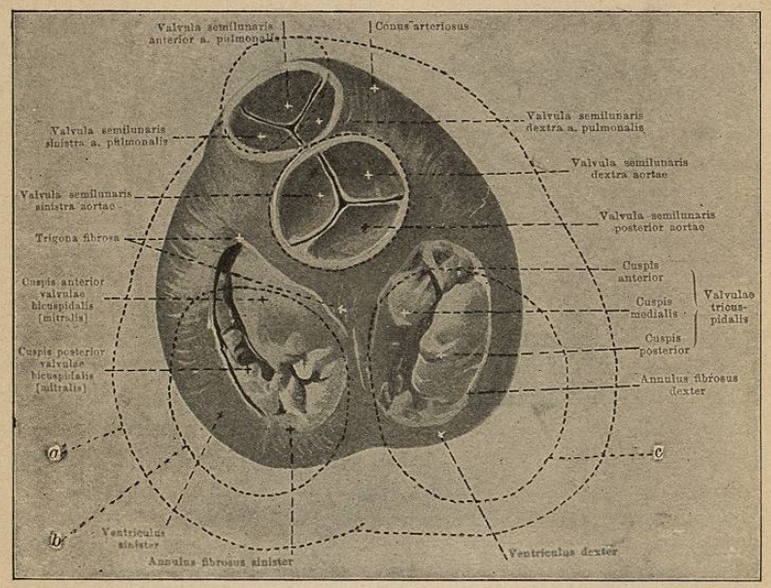

FIG. 117.—The Base of the Contracted Heart Showing Sphincteric Action of the Muscular Fibres Surrounding the Mitral and Tricuspid Valves. The outer dotted line is the outline of the relaxed heart. The inner dotted circles show the size of the mitral and tricuspid valves during diastole. *a*, Outline of the heart when relaxed; *b*, outline of the relaxed tricuspid valve; *c*, outline of the mitral orifice during diastole. (After Spalteholz.)

(*b*) Those which produce leakage through a valve orifice or prevent its closing effectively ("*regurgitation*," "*insufficiency*," "*incompetency*").

Stenosis results always from the stiffening, thickening, and contraction of a valve.

Regurgitation, on the other hand, may be the result either of—

(*a*) Deformity of a valve, or

(*b*) Weakening of the heart muscle.

The mitral and tricuspid orifices are closed not simply by the shutting of their valves, but also in part by the sphincter-like

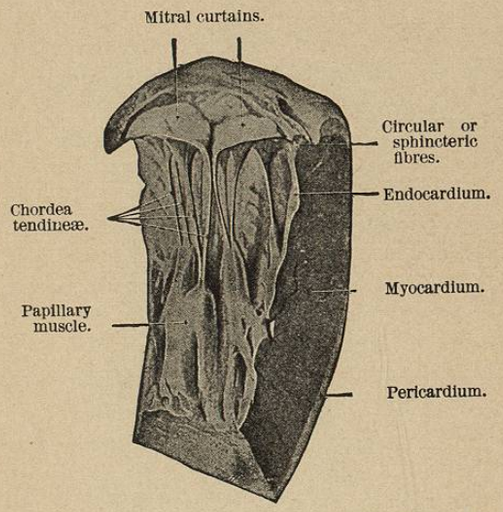

FIG. 118.—The Mitral Valve Closed, Showing the Action of the Papillary Muscles. (After Spalteholz.)

action of the circular fibres of the heart wall (see Fig. 117) and the contraction of the papillary muscles (Fig. 118).

In birds the tricuspid orifice has no valve and is closed wholly by the muscular sphincter of the heart wall.

In conditions of the acute cardiac failure, such as may occur after a hard run, the papillary muscles are in all probability relaxed, so that the valve-flaps swing back into the auricle and permit regurgitation of blood from the ventricle.

Valvular incompetence, then, differs from valvular *obstruction* in that the latter always involves deformity and stiffening of valves, while incompetence or leakage is often the result of deficient muscular action on the part of the heart wall. An osbtructed valve is almost always leaky as well, since the same deformities which prevent a valve from opening usually prevent its closure; *but this rule does not work backward.* A leaky valve is often *not* obstructed. It is leaky but not osbtructed if the valve curtain has been practically destroyed by endocarditis; or, again, it is leaky but not obstructed if the leak represents muscular weakening of the mitral sphincter or of the papillary muscles. Pure stenosis is very rare. Pure regurgitation is very common.

When valves are so deformed that their orifice is *both* leaky and obstructed, we have what is known as a "combined" or "double" valve lesion.

Since valvular lesions are recognized largely by their *results*, first upon the walls of the heart itself and then upon the other organs of the body, it seems best to give some account of these results before passing on to the description of the individual lesions in the heart itself.

The results of valvular lesions are first conservative and later destructive. The conservative results are known as:

The establishment of compensation through hypertrophy.

The destructive or degenerative results are known as:

The failure of compensation through (or without) dilatation.

I shall consider, then,

(*a*) The establishment and the failure of compensation.
(*b*) Cardiac hypertrophy.
(*c*) Cardiac dilatation.

ESTABLISHMENT AND FAILURE OF COMPENSATION IN VALVULAR DISEASE OF THE HEART.

We may discriminate three periods in the progress of a case of valvular heart disease:

(1) The period before the establishment of compensation.
(2) The period of compensation.
(3) The period of failing or ruptured compensation.

(1) *Compensation Not Yet Established.*

In most cases of acute valvular endocarditis, whether of the relatively benign or of the malignant type, there is a time when the lesion is perfectly recognizable despite the fact that compensatory hypertrophy has not yet occurred. In some cases this period may last for months; the heart is not enlarged, there is no accentuation of either second sound at the base, there is no venous stasis, and our diagnosis must rest solely upon the presence and characteristics of the murmur. For example, in early cases of mitral regurgitation due to chorea or rheumatism, the disease may be recognized by the presence of a loud musical murmur heard in the back as well as at the apex and in the axilla. In the earlier stages of aortic regurgitation occurring in young people as a complication of rheumatic fever, there may be absolutely no evidence of the valve lesion except the characteristic diastolic murmur. In most textbooks of physical diagnosis I think too little attention is given to this stage of the disease.

(2) *The Period of Compensation.*

Valvular disease would, however, soon prove fatal were it not for the occurrence of compensatory hypertrophy of the heart walls. To a certain extent the heart contracts as a single muscle, and increases the size of all its walls in response to the demand for increased work; but as a rule the hypertrophy affects especially one ventricle—that ventricle, namely, upon which especially demand is made for increased power in order to overcome an increased resistance in the vascular circuit which it supplies with blood. Whatever increases the resistance in the lungs brings increased work upon the right ventricle; whatever increases the resistance in the aorta or peripheral arteries increases the amount of work which the left ventricle must do.

Now, any disease of the mitral valve, whether obstruction or leakage, results in engorgement of the lungs with blood, and hence demands an increased amount of work on the part of the right ventricle in order to force the blood through the overcrowded pulmonary vessels; hence it is in mitral disease that we find the greatest compensatory hypertrophy of the right ventricle.

On the other hand, it is obvious that obstruction at the aortic valves or in the peripheral arteries (arterio-sclerosis) demands an increase in power in the left ventricle, in order that the requisite amount of blood may be forced through arteries of reduced calibre, while if the aortic valve is so diseased that a part of the blood thrown into the aorta by the left ventricle returns into that ventricle, its work is thereby greatly increased, since it has to contract upon a larger volume of blood.

In response to these demands for increased work, the muscular wall of the left ventricle increases in thickness, and compensation is thus established at the cost of an increased amount of work on the part of the heart.[1]

(3) *Failure of Compensation.*

Sooner or later in the vast majority of cases the heart, handicapped as it is by a leakage or obstruction of one or more valves, becomes unable to meet the demands made upon it by the needs of the circulation. Failure of compensation is sometimes associated with dilatation of the heart and weakening of its walls, but in many cases no such change can be found to account for its failure, and we have to fall back upon changes in the nutrition of the heart wall or upon some hypothetical derangement of the nervous mechanism of the organ as an explanation. Whatever the cause may be, the result of ruptured compensation is *venous stasis;* that is, œdema or dropsy of various organs appears. If the left ventricle is especially weakened, dropsy appears first in the legs, on account of the influence of gravity, soon after in the genitals, lungs, liver, and the serous cavities. Engorgement of the lungs is especially marked in cases of mitral disease with weakening of the right ventricle, and is manifested by dyspnœa, cyanosis, cough, and hæmoptysis. In many cases, however, dropsy is very irregularly and unaccountably distributed, and does not follow the rules just given. In pure aortic disease, uncomplicated by leakage of the mitral valve, dropsy is a relatively late symptom, and dyspnœa and precordial pain (angina pectoris) are more prominent.

[1] Rosenbach brings forward evidence to show that the arteries, the lungs, and other organs actively assist in maintaining compensation.

HYPERTROPHY AND DILATATION.

Since cardiac hypertrophy or dilatation are not in themselves diseases, but may occur in any disease of the heart (valvular or parietal), it seems best to give some account of them and of the methods by which they may be recognized, before taking up separately the different lesions with which they are associated.

1. *Cardiac Hypertrophy.*

Hypertrophy of the heart is usually due to the following causes:

First (and most frequent): Valvular disease of the heart itself. Second: Obstruction of the flow of blood through the arteries owing to increase of arterial resistance, such as occurs in chronic nephritis and arterio-sclerosis. Third: Obstruction to the circulation of the blood through the lungs (emphysema, cirrhosis of the lung, fibroid phthisis). Fourth: Severe and prolonged muscular exertion (athlete's heart).

In valvular disease the greatest degree of hypertrophy is to be seen usually in relatively young persons, and especially when the advance of the lesion is not very rapid.

Hypertrophy of the heart in valvular disease is also influenced by the amount of muscular work done by the patient, by the degree of vascular tension, and by the treatment. In the great majority of cases of hypertrophy, from whatever cause, both sides of the heart are affected, but we may distinguish cases in which one or the other ventricle is *predominantly* affected.

(1) *Cardiac hypertrophy affecting especially the left ventricle.*

(*a*) The apex impulse is usually lower than normal, often in the sixth space, occasionally in the seventh or eighth.[1] It is also farther to the left than normal, but far less so than in cases in which the hypertrophy affects especially the right ventricle. The area of visible pulsation is usually increased, and a considerable portion of the chest wall may be seen to move with each systole of the heart, while frequently there is a systolic *retraction* of the interspaces in place of a systolic impulse.

(*b*) Palpation confirms the results of inspection and shows us also that the apex impulse is unusually powerful. Percussion shows in many cases that the cardiac dulness is more intense and its area increased downward and to a lesser extent toward the left.[2]

(*c*) If we listen in the region of the maximum cardiac impulse, we generally hear an unusually long and low-pitched first sound, which may or may not be of a greater intensity than normal. A very loud first sound is much more characteristic of a cardiac neurosis than of pure hypertrophy of the left ventricle.

The second sound at the apex (the aortic second sound transmitted) is usually much louder and sharper than usual. Auscultation in the aortic area shows that the second sound at that point is loud and ringing in character. Not infrequently the peripheral arteries (the subclavians, brachials, carotids, radials, and femorals) may be seen to pulsate with each systole of the heart. This sign is most frequently observed in cases of hypertrophy of the left ventricle, which are due to aortic regurgitation, but is by no means peculiar to this disease and may be repeatedly observed when the cardiac hypertrophy is due to nephritis or muscular work. I have frequently observed it in athletes, blacksmiths, and others whose muscular work is severe.

The radial pulse wave has no constant characteristics, but depends rather upon the cause which has produced the hypertrophy than upon the hypertrophy itself.

[1] This is due partly to a stretching of the aorta, produced by the increased weight of the heart.

[2] Post mortem hypertrophy of the left ventricle is often found despite the absence of the above signs in life.

(2) *Cardiac Hypertrophy Affecting Especially the Right Ventricle.*

It is much more difficult to be certain of the existence of enlargement of the right ventricle than of the left. Practically we have but two reliable physical signs:

(*a*) Increase in the transverse diameter of the heart, as shown by the position of the apex impulse and by percussion of the right and left borders of the heart; and

(*b*) Accentuation of the pulmonic second sound, which is often palpable as well as audible.

The apex beat is displaced both to the left and downward, *but especially to the left.* In cases of long-standing mitral disease, the cardiac impulse may be felt in mid-axilla, several inches outside the nipple, and yet not lower down than the sixth intercostal space. In a small percentage of cases (*i.e.*, when the right auricle is engorged), an increased area of dulness to the right of the sternum may be demonstrated. Accentuation of the pulmonic second sound is almost invariably present in hypertrophy of the right ventricle, though it is not peculiar to that condition. It may be heard, for example, in cases of pneumonia when no such hypertrophy is present, but in the vast majority of cases of cardiac disease we may infer the presence and to some extent the amount of hypertrophy of the right ventricle from the presence of a greater or lesser accentuation of the pulmonic second sound. The radial pulse shows nothing characteristic of this type of hypertrophy.

Epigastric pulsation gives us no evidence of the existence of hypertrophy of the right ventricle, despite contrary statements in many text-books. Such pulsation is frequently to be seen in persons with normal hearts, and is frequently absent when the right ventricle is obviously hypertrophied. It is perhaps most often due to an unusually low position of the whole heart.

Dilatation of the Heart.

(1) *Acute Dilatation.*—Immediately after severe muscular exertion, as, for example, at the finish of a boat race, or of a two-mile run (especially in persons not properly trained), an acute dilatation of the heart may occur, and in debilitated or poorly nourished subjects such an acute dilatation may be serious or even fatal in its results.

(2) Chronic dilatation comes on gradually as a result of valvu-

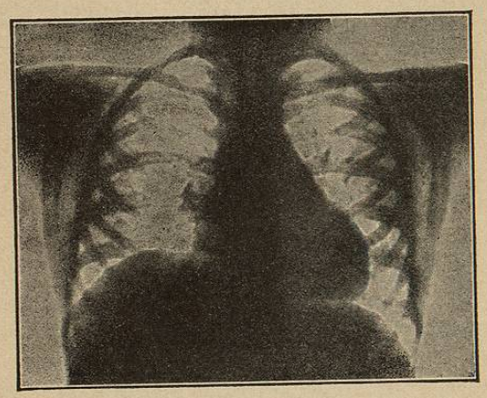

Fig. 119.—Dilated Heart. From v. Ziemssen's Atlas.

lar disease or other cause, and gives rise to practically the same physical signs as those of acute dilatation, from which it differs chiefly as regards the accompanying physical phenomena and the prognosis. Briefly stated, the signs of dilatation of the heart, whether acute or chronic, are:

(*a*) *Feebleness and irregularity* of the apex impulse and of the radial impulse, (*b*) *enlargement* of the heart, as indicated by inspection, palpation, and percussion, and (sometimes) (*c*) *murmurs* indicative of stretching of one or another of the valvular orifices.

Dilatation of the Left Ventricle.

Inspection shows little that is not better brought out by palpation. Palpation reveals a "flapping" cardiac impulse, or a vague shock displaced both downward and to the left and diffused over an abnormally large area of the chest wall. Percussion verifies the position of the cardiac impulse and sometimes shows an unusually blunt or rounded outline at the apex of the heart.

On auscultation, the first sound is usually *very short and sharp*, but *not feeble* unless it is accompanied by a murmur. In case the mitral orifice is so stretched as to render the valve incompetent, or in case the muscles of the heart are so fatigued and weakened that they do not assist in closing the mitral orifice, a systolic murmur is to be heard at the apex of the heart. This murmur is transmitted to the axilla and back, but does not usually replace the first sound of the heart. The aortic second sound, as heard in the aortic area and at the apex, is feeble.

Dilatation of the right ventricle of the heart is manifested by an increase in the area of cardiac dulness to the right of the sternum (corresponding to the position of the right auricle), by feebleness of the pulmonic second sound together with signs of congestion and engorgement of the lungs, and often by a systolic murmur at the tricuspid valve; *i.e.*, at or near the root of the ensiform cartilage. When this latter event occurs, one may have also systolic pulsation in the jugular veins and in the liver (see below, p. 248).

In cases of acute dilatation, such as occur in infectious fevers or at the end of well-contested races, there is often to be heard a systolic murmur loudest in the pulmonary area and due very possibly to a dilatation of the conus arteriosus.

The diagnosis of dilatation of the heart seldom rests entirely upon physical signs referable to the heart itself. In acute cases our diagnosis is materially aided by a knowledge of the cause, which is often tolerably obvious. In chronic cases the best evidence of dilatation is often that furnished by the venous stasis which results from it.

(4) CHRONIC VALVULAR DISEASE.

I. Mitral Regurgitation.

The commonest and on the whole the least serious of valvular lesions is incompetency of the mitral. It results in most cases from the shortening, stiffening, and thickening of the valve produced by rheumatic endocarditis in early life. It is the lesion present in most cases of chorea (see Figs. 120 and 121).

Temporary and curable mitral regurgitation may result from weakening of the heart muscle, which normally assists in closing the mitral orifice through the sphincter-like contraction of its circular fibres.

Great muscular fatigue, such as is produced by a hard boat race, may result in a temporary relaxation of the mitral sphincter or of the papillary muscles sufficient to allow of genuine but temporary and curable regurgitation through the mitral orifice. In conditions of profound nervous debility, excitement, or exhaustion, similar weakening of the cardiac muscles may allow of a leakage through the mitral, which ceases with the removal of its cause. Stress has been laid upon these points by Prince, and recently by Arnold.

Mitral insufficiency due to stretching of the ring into which the valve is inserted occurs not unfrequently as a result of dilatation of the left ventricle, and is commonly known as *relative insufficiency* of the mitral valve. The valve orifice can enlarge, the valve cannot, and hence its curtains are insufficient to fill up the dilated orifice. This type of mitral insufficiency frequently results from aortic regurgitation with the dilatation of the left ventricle which that lesion produces, or from myocarditis, which weakens the heart wall until it dilates and widens the mitral orifice.

The *results* of any form of mitral leakage occur in this order:

1. *Dilatation or hypertrophy of the left auricle*, which has to receive blood both from the lungs and through the leaky mitral from the left ventricle.

2. The overfilled left auricle cannot receive the blood from the lungs as readily as it should; hence the blood "backs up" in the lungs and thereby increases the work which the right ventricle must do in order to force the blood through them. Thus result œdema of the lungs, and—

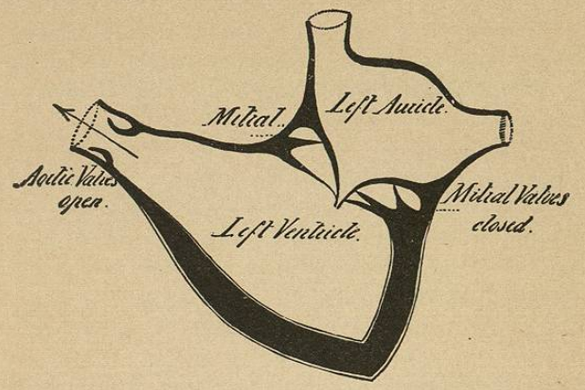

FIG. 120.

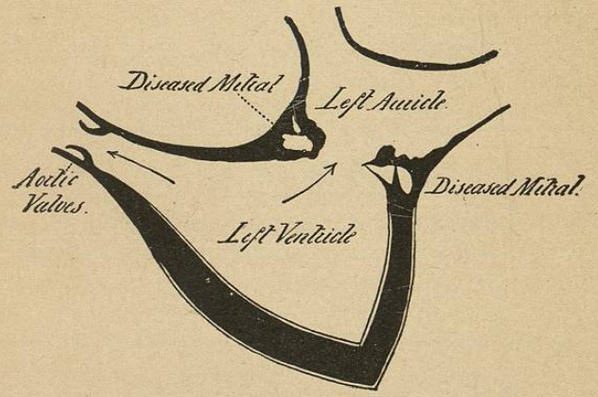

FIG. 121.

FIG. 120.—Normal Heart during Systole. Mitral valve closed; blood flowing through the open aortic valves into the aorta.

FIG. 121.—Mitral Regurgitation. The heart is in systole and the arrows show the current flowing back in the left auricle as well as forward into the aorta.