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.

ent of the existence of any valvular disease. Attention has been especially called to this point by Lees and Boynton (*British Med. Jour.*, July 2, 1898) and by S. West.

Influenza is also complicated not infrequently by acute cardiac dilatation.

Chronic Myocarditis ("Weakened Heart").

Fatty or fibroid changes in the heart wall occurring in *chronic* disease are usually the result of sclerosis of the coronary arteries and imperfect nutrition of the myocardium, but chronic toxæmias, like pernicious anæmia, may also produce a very high grade of fatty degeneration of the heart and especially of the papillary muscles.

Whether fatty or fibroid changes predominate, the physical signs are the same.

Physical Signs of Chronic Myocarditis.

For the recognition of these changes in the myocardium our present methods of physical examination are always unsatisfactory and often wholly inadequate. Extensive degenerations of the heart wall are not infrequently found at autopsy when there has been no reason to suspect them during life. On the other hand, the autopsy often fails to substantiate a diagnosis of degeneration of the heart muscle, although all the physical signs traditionally associated with this condition were present during life.[1] To a considerable extent, therefore, our diagnosis of myocarditis must depend upon the history and symptoms of the case; physical examination can sometimes supplement these, sometimes not. Symptoms of *cardiac weakness* developing in a man past middle life, especially in a patient who shows evidences of arterio-sclerosis or high arterial tension, or who has suffered from the effects of alcohol and syphilis, suggest parietal disease of the heart, fatty or fibroid. The probability is increased if there have been attacks of angina pectoris, Cheyne-Stokes breathing, or of syncope.

Inspection and *palpation* may reveal nothing abnormal, or there

[1] A well-known Boston pathologist recently told me that he had never known a case of myocarditis correctly diagnosed during life.

may be an unusually diffuse, slapping cardiac impulse associated perhaps with a displacement of the apex beat to the left and downward. Marked irregularity of the heart beat, both in force and in rhythm, is sometimes demonstrable by these methods, and an increase in the area of cardiac dulness may be demonstrable in case dilatation has followed the weakening of the heart wall. *Auscultation* may reveal nothing abnormal except that the aortic second sound is unusually sharp; in some cases *feeble and irregular heart sounds* are heard, although the first sound at the apex is not infrequently sharp. Reduplication of one or both sounds and disturbance of rhythm, especially the "gallop rhythm," are not infrequent. If the mitral sphincter is dilated, or the papillary muscles are weakened, as not infrequently happens, we may have evidences of mitral regurgitation, a systolic murmur at the apex heard in the left axilla and back with accentuation of the pulmonic second sound.

Summary.

1. The history and symptoms of the case or the condition of other organs are often of more diagnostic value than is the physical examination of the heart itself, which may show nothing abnormal.

2. Among the rather unreliable physical signs, those most often mentioned are:

(*a*) Weakness and irregularity of the heart sounds.

(*b*) Accentuation of the aortic second sound.

(*c*) A diffuse slapping cardiac impulse.

(*d*) Reduplication of some of the cardiac sounds (gallop rhythm).

(*e*) Evidences of cardiac dilatation.

(*f*) Murmurs—especially the murmur of mitral insufficiency which often occurs as a result of dilatation of the valve orifices and weakening of the cardiac muscle.

Differential Diagnosis.

We have to distinguish myocarditis from—

(*a*) Uncomplicated valvular lesions.

(*b*) Cardiac neuroses.

(*a*) It has been already pointed out that valvular lesions do not necessarily give rise to any murmurs when compensation has failed. Under such circumstances one hears only irregular and weak heart sounds, as in myocarditis. The history of a long-standing valvular trouble, a knowledge of the previous existence of murmurs, the age, method of onset, and symptoms of the case may assist us in the diagnosis. Cases of myocarditis are less likely to be associated with extensive dropsy than are cases of valvular disease whose compensation has been ruptured.

(*b*) Weakness and irregularity of the cardiac sounds, when due to nervous affection of the heart and unassociated with parietal or valvular changes, is usually less marked after slight exertion. The heart "rises to the occasion" if the weakness is a functional one. On the other hand, if fatty or fibroid changes are present, the signs and symptoms are much aggravated by any exertion.

In some cases of myocarditis the pulse is excessively slow and shows no signs of weakness. This point will be referred to again in the chapter on Bradycardia.

Fatty Overgrowth.

An abnormally large accumulation of fat about the heart may be suspected if, in a very obese person, signs of cardiac embarrassment (dyspnœa, palpitation) are present, and if on examination we find that the heart sounds are feeble and distant but preserve the normal difference from each other. When the heart wall is seriously weakened (as in the later weeks of typhoid), the heart sounds become more alike owing to the shortening of the first sound.

In fatty overgrowth this is not the case.

The diagnosis, however, cannot be positively made. We suspect it under the conditions above described, but no greater certainty can be attained.

Fatty Degeneration.

There are no physical signs by which fatty degeneration of the heart can be distinguished from other pathological changes which result in weakening the heart walls. An extensive degree of fatty

degeneration is often seen post mortem in cases of pernicious anæmia, although the heart sounds have been clear, regular, and in all respects normal during life. The little we know of the physical signs common to fatty degeneration and to other forms of parietal disease of the heart has been included in the section on Myocarditis (see p. 257).

Cardiac Neuroses.

Tachycardia (Rapid Heart).

Simple quickening of the pulse rate, or tachycardia, which may pass altogether unnoticed by the patient himself, is to be distinguished from palpitation, in which the heart beats, whether rapid or not, force themselves upon the patient's attention.

The pulse rate may vary a great deal in health. A classmate of mine at the Harvard Medical School had a pulse never slower than 120, yet his heart and other organs were entirely sound. Such cases are not very uncommon, especially in women. Temporarily the pulse rate may be greatly increased, not only by exercise and emotion, but by the influence of fever, of gastric disturbances, or of the menopause. Such a tachycardia is not always of brief duration. The effects of a great mental shock may produce an acceleration of the pulse which persists for days or even weeks after the shock.

Among organic diseases associated with weakening of the pulse the commonest are those of the heart itself. Next to them, exophthalmic goitre, tumors or hemorrhage in the medulla, and obscure diseases of the female organs of generation, are the most frequent causes of tachycardia.

The only form of tachycardia which is worthy to be considered as a more or less independent malady is

Paroxysmal Tachycardia.

As indicated in the name, the attacks of this disease are apt to begin and to cease *suddenly*. They may last a few hours or several days. The pulse becomes frightfully rapid, often 200 per minute or more. Bristowe records a case with a pulse of 308 per minute.

In the radial artery the pulse beat may be impalpable. The heart sounds are regular and clear, but the diastolic pause is shortened and the first sound becomes short and "valvular," resembling the second ("*tic-tac heart*"). The paroxysm may be associated with aphasia and abnormal sensations in the left arm. Occasionally the heart becomes dilated, and œdema of the lungs, albuminuria, and other manifestations of stasis appear. As a rule, however, paroxysmal tachycardia can be distinguished from the rapid heart-beat associated with cardiac dilatation by the fact that the heart remains perfectly regular. This same fact also assists us in excluding the cardiac neuroses due to tobacco, tea, and other poisons. From the tachycardia of Graves' disease the affection now in consideration differs by its paroxysmal and intermittent character.

Bradycardia (Slow Heart).

In many healthy adults the heart seldom beats over 50 times a minute.

I. Among the causes which may produce for a short time an abnormally slow heart-beat are:

(*a*) *Exhaustion;* for example, after fevers, after parturition, or severe muscular exertion.

(*b*) *Toxæmia;* for example, jaundice, uræmia, auto-intoxications in dyspepsia.

(*c*) In certain *hysterical and melancholic* states and in *neurotic children*, the pulse may be exceedingly slow. *Pain* has also a tendency to retard the pulse.

(*d*) An *increase of intracranial pressure*, as in meningitis, cerebral hemorrhage, depressed fracture of the skull. Possibly in this category belong the cases of bradycardia sometimes seen in epileptiform or during syncopal attacks. Bradycardia from any one of these causes is apt to be of comparatively short duration.

II. *Permanent bradycardia* is most often associated with coronary sclerosis and myocarditis. In this disease the pulse may remain below 40 for months or years, though strong and regular, yet the patient may be free from disagreeable symptoms of any kind. The rate of the heart-beat cannot always be estimated by counting the radial pulse. Not infrequently many pulsations of the heart are not of sufficient force to transmit a wave to the radial artery, and the mistake should never be made of diagnosing bradycardia simply by counting the radial pulse.

Arrhythmia.

1. *Physiological Arrhythmia.*—Arrhythmia, or irregularity in the force or rhythm of the heart-beat, is to a certain extent physiological. The heart normally beats a little faster and a little more strongly during inspiration than during expiration. Any psychical disturbance or muscular exertion may produce irregularity as well as a quickening of the heart-beat. Rarely the pulse may be irregular throughout life in perfectly healthy persons. This irregularity is usually of rhythm alone; every second or third beat may be regularly omitted without the individual knowing anything about it or feeling any disagreeable symptoms connected with it. More rarely the heart's beats may be permanently irregular in force as well as rhythm despite the absence of any discoverable disease.

In children the pulse is especially apt to be irregular, and during sleep some children show that modification of rhythm known as the "*paradoxical pulse*," which consists in a quickening of the pulse with diminution in volume during inspiration.

(2) If we leave on one side diseases of the heart itself, *pathological arrhythmia* is most frequently seen in persons who have used tobacco or tea to excess, or in dyspepsia. In these conditions it is often combined with palpitation and becomes thereby very distressing to the patient. In connection with cardiac disease the following types of arrhythmia may be distinguished:

(*a*) *Paradoxical Pulse.*—Any cause which leads to weakening of the heart's action may occasionally be associated with paradoxical pulse. Fibrous pericarditis has been supposed to be frequently associated with this type of arrhythmia, but if so it is by no means its only cause.

(*b*) The *bigeminal pulse* is seen most frequently in cases of uncompensated heart disease (particularly mitral stenosis) after the administration of digitalis. Every other beat is weak or abortive

and is succeeded by an unusually long pause. Sometimes every third beat is of the abortive type, or an unusually long interval may divide the heart-beats into groups of three (*"triçeminal pulse"*).

(*c*) *Embryocardia*, or the "tic-tac heart," represents a shortening of the diastolic pause and of the first sound of the heart so that it resembles the second sound, as in the fœtal heart. Any case of uncompensated heart disease, whether valvular or parietal, may be associated with this disturbance of rhythm.

(*d*) The *gallop rhythm.*

Owing to a reduplication of one of the heart sounds (usually the second), we may have three sounds instead of two with each beat of the heart, the sounds possessing a rhythm which reminds us of the hoof-beats of a galloping horse (see p. 181). This rhythm is heard especially in the failing heart of interstitial nephritis or coronary sclerosis.

(*e*) *Delirium cordis* is a term used to express any great irregularity and rapidity of the heart-beats which cannot be reduced to a single type or rhythm. It is seen in the gravest stages of uncompensated heart disease.

Palpitation.

Defined by Osler as "irregular or forcible heart action *perceptible to the individual.*" The essential point is that the individual becomes conscious of each beat of his heart, whether or no the heart action is in any way abnormal.

(*a*) In irritable conditions of the nervous system, such as occur at puberty, at climacteric, or in neurasthenic persons, palpitation may be very distressing. Temporary disturbances, such as fright, may produce a similar and more or less lasting effect.

(*b*) The effect of high altitudes, or of even a moderate elevation (1,500 feet) is sufficient to produce in many healthy persons a quickening and strengthening of the heart's action, so that sleep may be prevented. After a few nights this condition usually passes off, provided the heart is sound.

(*c*) Abuse of tobacco and tea have a similar effect.

Auscultation of a palpitating heart shows nothing more than unusually loud and ringing heart sounds, but since palpitation is often associated with arrhythmia of one or another type we must be careful to exclude the palpitation symptomatic of acute dilatation of the heart, such as may occur in debilitated persons after violent or unusual exertion. In this condition the area of cardiac dulness is increased and dyspnœa upon slight exertion becomes marked. It goes without saying that in almost any case of organic disease of the heart palpitation may be a very marked and distressing symptom.

CONGENITAL HEART DISEASE.

From the time of birth it is noticed that the child is markedly and permanently cyanosed, hence the term "blue baby." Dyspnœa is often, though not always, present, and may interfere with sucking. The cyanosis is practically sufficient in itself for the diagnosis.

Among congenital diseases of the heart the commonest and the most important (because it is less likely than any of the others to prove immediately fatal) is:

1. *Pulmonary Stenosis.*

This lesion is usually the result of fœtal endocarditis, and is often associated with malformations and defects, such as patency of the foramen ovale and persistence of the ductus arteriosus. The physical signs of pulmonary stenosis are:

(*a*) A palpable systolic thrill most distinct in the pulmonary area.

(*b*) A loud murmur (often rough or musical) heard best in the same region, but usually transmitted to all parts of the chest.

(*c*) A weak or absent pulmonic second sound.

(*d*) An increased area of cardiac dulness corresponding to the right ventricle.

Unlike most other varieties of congenital heart disease, pulmonary stenosis is compatible with life for many years, and "blue babies" with this lesion may grow up and enjoy good health, al-

though usually subject to pulmonary disorders (pneumonia or tuberculosis). For a discussion of the differential diagnosis of this lesion, see above, p. 252.

2. *Defects in the Ventricular Septum.*

The loud *systolic murmur* produced by the rush of blood through an opening between the ventricles is heard, as a rule, over the whole precordia. Its point of maximum intensity differs in different cases, but is hardly ever near the apex of the heart. The most important diagnostic point is the *absence* of a palpable thrill. With almost every other form of congenital heart disease in which a loud murmur is audible, there is a thrill as well. Hypertrophy of both ventricles may be present, but is seldom marked in uncomplicated cases.

(*Patency of the foramen ovale*, if unassociated with other defects, does not usually produce any murmur or other signs by which it can be recognized during life, and causes no symptoms of any kind.)

3. *Persistence of the Ductus Arteriosus.*

The most characteristic sign is a loud, vibratory systolic murmur with its intensity at the base of the heart and *unassociated with hypertrophy of either ventricle.* If complicated with stenosis at or close above the pulmonary valves, persistence of the ductus arteriosus cannot be diagnosed, as the murmur produced by it cannot with certainty be distinguished from that of the pulmonary stenosis, and the presence of hypertrophy of the right ventricle deprives us of the one relatively characteristic mark of a patent arterial duct.

It has been claimed that a murmur persisting through systole and into diastole is diagnostic of an open arterial duct, but this supposition is not borne out by post-mortem evidence.

The signs produced by the other varieties of congenital heart disease, such as aortic stenosis and tricuspid or mitral lesions, do not differ materially from those characterizing those lesions in

adults. Excluding these, we may summarize the signs of the other lesions as follows:

(*a*) Practically all cases of congenital heart disease, which produce any physical signs beyond cyanosis and dyspnœa, manifest themselves by a loud systolic murmur heard all over the precordia and often throughout the chest. Its maximum intensity is usually at or near the base of the heart.

(*b*) If there is *no thrill* and *no hypertrophy*, the lesion is probably a defect in the ventricular septum.

(*c*) If there is a *thrill but no hypertrophy*, the lesion is probably a patent ductus arteriosus.

(*d*) If there is a *thrill and hypertrophy* of the right ventricle, the lesion is probably pulmonic stenosis, especially if the pulmonic second sound is feeble.