

II.

THE RELATIONSHIP OF THE ABNORMAL HEART BEAT TO PROGNOSIS.

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THE heart beats abnormally when there is any abnormality of its rhythm, conduction, or contracting power. Sometimes all three and frequently two of these three cardiac functions are defective. During the past few years much attention has been directed to the determination and interpretation of signs and symptoms of abnormalities of the heart beat. The value of various phenomena in estimating cardiac damage and in forecasting the patient's future is being studied and this study should yield steadily more and more guides for our assistance. The field is still relatively recent, but enough years have now been spent by such men as James Mackenzie and Wenckebach to render profitable a summary of the subject as we know it at the present time. We may consider the abnormal heart beat most practicably by taking up in detail disorders of rhythm, conduction, and contraction.

I. DISTURBED RHYTHM.

The most common disorder of the rhythm of the heart is the *premature beat* (the extrasystole of old). It may arise in auricle, ventricle or junctional tissues. (Fig. 1.) Mackenzie² claims that the premature beat adds practically nothing to his opinion of a case whether favorable or unfavorable. In a young healthy person a premature beat should not be a source of concern, even though it does mean that there is an abnormal irritability somewhere in the heart. People have lived comfortably through lives of hard work till old age with the constant companionship of premature beats for fifty years or more. Sometimes this irregularity can be traced to tobacco or to exhaustion and in these cases it will stop

when the causes are stopped. The finding of ventricular premature beats in old people who are growing short of breath or in younger people with high blood pressure or syphilitic hearts may be important, for here one may discover the significant *pulsus alternans* which not infrequently follows the premature beat in such cases.

The frequent association of the much less common type of premature beat—that arising in the auricle (Fig. 1b)—with auricular fibrillation and paroxysmal auricular tachycardia (including flutter of the auricles) causes one to regard this type as more important prognostically than the ventricular premature beat as a possible forerunner of more serious conditions.

Finally, there is a third and uncommon type of premature beat, that which arises in the junctional tissues between auricles and ventricles. This premature beat is unimportant and may be dismissed with the observation that it shows an irritable condition of the node of Tawara or bundle of His and may be classed prognostically with the ventricular premature beat. The differentiation of the types of premature beats is usually possible only by graphic methods.

Paroxysmal tachycardia (Fig. 2) is the next disorder of the cardiac mechanism to be considered, following in direct sequence after premature beats. It consists of a rapid repetition of premature beats, all arising in the same abnormal focus in the heart muscle. It is almost always auricular in origin and sometimes occurs in patients who later show flutter or fibrillation of the auricles. There are people who have paroxysms of tachycardia at intervals for many years and, provided the paroxysms are brief, enjoy good health. Here again, however, the heart muscle is shown to be irritable and to a higher degree than in the case of premature beats. The irritability often expresses itself only under some mental or physical strain. Without other signs of cardiac trouble than the paroxysmal tachycardia the patient may usually be reassured but warned against excesses.

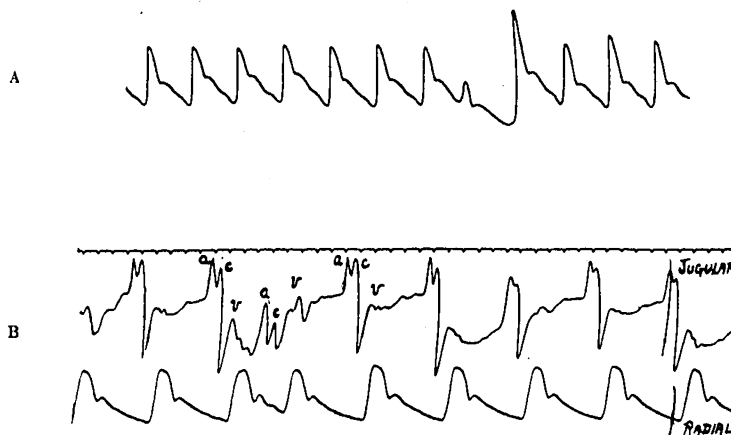


FIG. 1.

Radial pulse tracings showing premature beats.

A. Ventricular.

B. Auricular. Jugular pulse proves auricular origin of the premature beat.

Time interval = 0.2 sec.



FIG. 2.

Radial pulse tracing showing a brief paroxysm of tachycardia. Preceding the paroxysm appears a ventricular premature beat. Time interval = 0.2 sec.

Closely associated with paroxysmal tachycardia is *auricular flutter*, which is an auricular tachycardia of high degree, almost always with heart block associated with it. The prognosis is less favorable than in the case of simple paroxysmal tachycardia, the evidence pointing as a rule to more serious myocardial damage. In some instances, however, patients showing this condition may live comfortably for years after their return to normal cardiac rhythm. Of seven cases of auricular flutter seen at the Massachusetts General Hospital during the past ten months (August, 1914, to June, 1915) two have died (29%), one had auricular fibrillation when last heard from, and the other four have had recurrences of the flutter. Of the last mentioned four, one has now an atrioventricular rhythm (that is, a pacemaker situated in the junctional tissues between auricle and ventricle), another has frequent ventricular premature beats and alternation of the pulse, a third has so many paroxysms of flutter that she is incapacitated, and the fourth, although in fair health, has shown auricular premature beats between attacks. In distinguishing flutter from paroxysmal tachycardia graphic records are practically essential.

Now we come to the most important disturbance of rhythm—the absolute irregularity of the heart in *auricular fibrillation*. (Fig. 3.) The prognosis in this condition is extremely variable, being dependent on the condition of the ventricular muscle, which must bear the brunt of the irregular passage of the impulse from the auricles, and also dependent on the response of the patient to digitalis therapy. From the moment of discovery of this irregularity until death any length of time, from a few hours to many years, may supervene. In 1881 Potain, by tracings of the radial pulse, found the presence of absolute arrhythmia in a young *externe* of the Paris hospitals; in 1913, thirty-two years later, Heitz¹ by polygram, found

the fibrillation still existing in this man after an active professional life during which the irregularity had never ceased. Of 86 patients with auricular fibrillation seen at the Massachusetts General Hospital in the past ten months (August, 1914 to June, 1915) 14 (16%) are known to have died. Five are now living comfortably who have suffered in the past from the rather unusual paroxysmal type of fibrillation.

Sometimes a marked arrhythmia is produced through nervous influences acting directly on the pacemaker at the sino-auricular node. This so-called sinus arrhythmia usually is respiratory in origin, but may be due to factors such as increased intracranial pressure and to other causes not well defined. Although at times distinctly abnormal, this disturbance of rhythm as a rule has an extracardiac origin, and therefore should not impress one unfavorably with respect to the condition of the heart. Finally, disorder of the cardiac rhythm may be produced by heart-block, which should be considered under the second heading.

II. DISTURBANCES OF CONDUCTION.

All degrees of *heart-block* may be found from the simple prolongation of the interval between auricular and ventricular contractions, through the stage of dropped beats and 2 and 3 to 1 block, to the rare condition of complete heart-block. (Fig. 4.) Toxic block, usually transient, may be caused by digitalis and allied drugs, acute infection and asphyxia (shown experimentally). This type of block has little value prognostically, except in some cases to evidence a damaged bundle which has been easily depressed by the toxin. If the block is not traceable to toxic cause, but seems to be due entirely to disease or degenerative changes in the bundle, the prognosis must be guarded, for the block in such a case is but an



FIG. 3.

Radial pulse tracing showing the absolute irregularity of auricular fibrillation. Time interval = 0.2 sec.

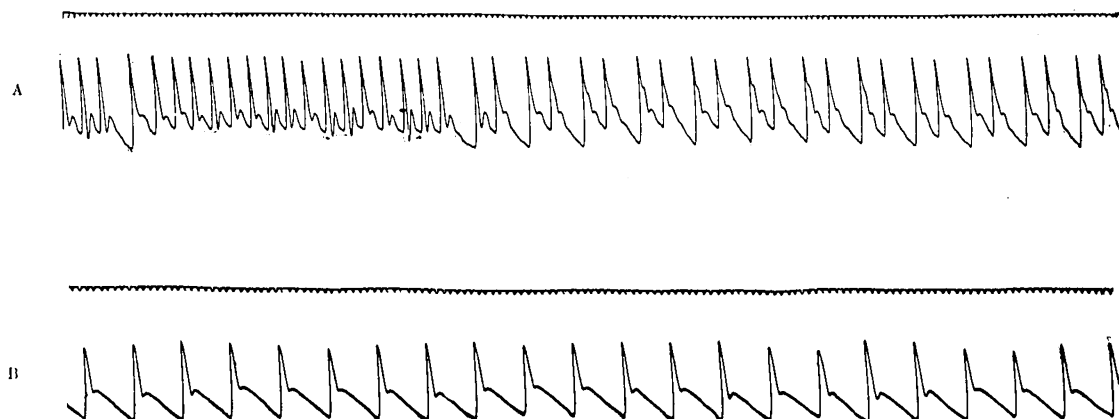


Fig. 4.

Radial pulse tracings showing heart block.

A. Partial block. Early in the tracing there is a "dropped beat." Later every third beat is dropped.

B. Complete heart block. Rate = 42.

Time interval = 0.2 sec.

index of widespread damage in the myocardium. The progressive nature of the destructive process may be followed easily in some cases by the observation of the evolution of the block itself from mild to more extensive grades. The Stokes-Adams syndrome is most frequent in partial heart block, with marked depression of the bundle at intervals. When the block becomes complete in such a case the patient may improve considerably and live comfortably for years. The prognosis of any case of permanent block, especially of the higher grades, should be regarded as grave, although in some instances years may elapse between the discovery of the block and death.

Not only may the main stem of the bundle of His be involved to produce a blocking of the cardiac impulse from the auricles, but also its branches, large and small, may be damaged. At present it is impossible to detect lesions of the smaller twigs of the conduction system in the ventricle, but it is possible by electrocardiograms to recognize gross defects of the two main branches, right and left, which go to right and left ventricles respectively. The left branch is involved much less frequently than the right. At the Massachusetts General Hospital, in the past eight months, of twelve cases of bundle branch defect only one has been of the left branch. The main stem of the bundle of His in such cases may not be damaged sufficiently to cause delay between auricular and ventricular contractions, and the pulse may be perfectly regular at a rate close to 70 per minute. (Fig. 5.) The hidden damage is, however, revealed by the electro-cardiogram and is of considerable value prognostically. Any beats in which there is defective conduction in the branches of the bundle of His are called aberrant, thus indicating the abnormal distribution of the cardiac impulse in the ventricles. Reduplications of the heart sounds may accompany these branch lesions, in which one ventricle receives the impulse appreciably before the other. As in the case of lesions of the main stem, so lesions of the branches of

the bundle evidence widespread myocardial damage and are found usually in sclerotic hearts. Two of the twelve patients with branch lesions at the Massachusetts General Hospital have died within eight months of the time when the lesions were discovered; several of the survivors are in poor condition at the present time.

III. DISTURBANCES OF CONTRACTING POWER.

Finally we come to abnormalities of the contracting power of the heart. Weakness in the contraction of the heart muscle is often associated with defects in rhythm and conduction. The coexistence of considerable abnormalities of two or of three of these properties of the heart increases the gravity of the prognosis (for example, heart block or arrhythmia combined with myocardial weakness). Defect in the contracting power of the heart may itself be enhanced by disturbance of the rhythm, as in the rapid irregular action of auricular fibrillation. Help may come on the other hand if the conductivity of the heart is defective—such defect may be artificially produced through the administration of digitalis. In the estimation of myocardial exhaustion the symptoms and general physical signs of the patient taken collectively are of much value and are generally used as the criterion of the cardiac condition: dyspnea on exertion, weak heart sounds, edema of extremities and lungs, and cyanosis. But the history of the frequency of "cardiac breaks" and the way in which the patient reacts or has reacted in the past to rest and digitalis must control one's prognosis.

It is in the doubtful cases showing little else than arrhythmia, palpitation or slight dyspnea on exertion that oftentimes one is most interested in the prognosis. Swan⁴ in a recent investigation, in a series of cases, of several methods which have been proposed as guides in the determination of incompetence of the myocardium in doubtful cases, concludes that two of the methods have more value than the others, namely

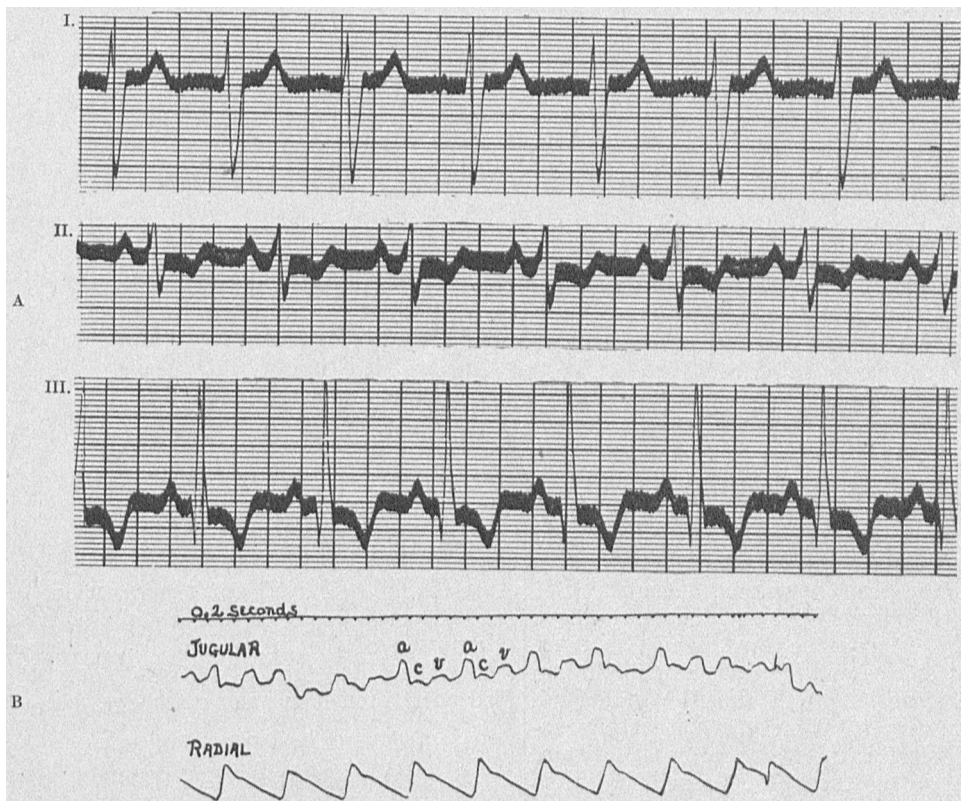


Fig. 5.

Graphic records from patient showing defective conduction in the left branch of the bundle of His.

A. The three electrocardiographic leads.

B. Radial and jugular tracings, showing an absolutely regular pulse with slightly prolonged a-c interval.

Time interval = 0.2 sec.

Tigerstedt's efficiency factor, which is found by dividing the systolic pressure multiplied by the pulse rate into the pulse pressure multiplied by the pulse rate, the other method being the determination of the percentage of pulse pressure formed by the second auscultatory phase. He considers as abnormal figures of 40% or more in the former and 30% or less in the latter test. It is very doubtful, however, whether these methods will prove of real worth; in some cases at least they are contradictory and in others far from convincing. Continued study of the blood flow in the determination of the time volume of the heart beat may yield in the future indications of value in the estimation of the efficiency of the heart.

Moritz³ has recently summarized the present status of clinical signs of beginning heart weakness, and concludes that nearly all signs or symptoms taken alone have little value, and that one must study a case from all angles in order to make a fair estimation of the heart's strength. He does not consider the size of the pulse or the blood pressure as reliable signs which can be interpreted at present. He is inclined to lay more stress on venous pressure as evidence of stasis, on the persistence (especially during sleep) of increased pulse rate following exercise, on nocturnal dyspnea, on the gallop rhythm of the heart, and on abnormal sensations in chest and arm. He pays little attention to the character of

the pulse because of its considerable modification by local conditions of the artery. On the other hand there is one condition of the pulse which is an important evidence of exhaustion of the myocardium—that is the variation in force of the pulse beats found either with absolute irregularity in some cases of auricular fibrillation or alternating regularly in some cases with normal rhythm. In the first instance the arrhythmia itself points to an abnormal heart, but in the second the pulse may be perfectly regular or interrupted only occasionally by ventricular premature beats. The alternating character of the force of pulse waves is best seen on radial tracings and gives the condition its name of *pulsus alternans* (Fig. 6). Alternation of the pulse is one of the most important signs which we possess of defective power of contraction of the heart and has been shown by Mackenzie and others to be of very great prognostic significance in cases not showing paroxysmal rapid heart action at the time (in such cases there may be temporary exhaustion from the rapid rate). The alternation may exist in individuals obviously mortally ill but also in cases in which one sometimes would doubt the presence of myocardial weakness. Patients who show *pulsus alternans* usually survive but a few years at the most, the longest case on record being one of Tabora's⁵, living six years after the alternation was first discovered. Of 83 patients with *pulsus alternans*

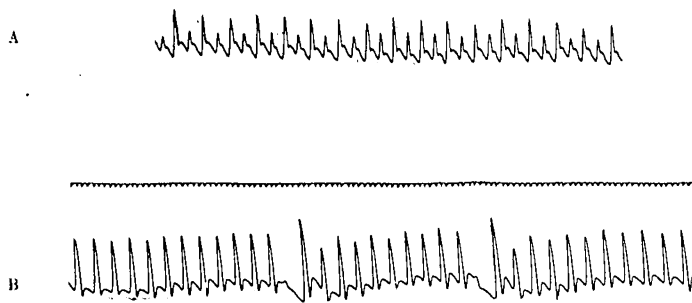


Fig. 6.
Radial tracings showing alternation of the pulse.
A. Constant *pulsus alternans*.
B. Post-premature beat alternation.
Time interval = 0.2 sec.

and heart-rate below 125 and as a rule below 100 per minute, seen at the Massachusetts General Hospital in the past ten months (August, 1914 to June, 1915) twenty-six are known to have died—that is nearly one-third, which is twice the death rate of cases with auricular fibrillation during the same length of time. Whether we accept the theory of Wenckebach, that alternation of the heart is due to an alternating total hyposystole, or the theory of Gaskell and Hering, that it is due to an alternating partial asystole resulting from the prolongation of the refractory phase of the muscle fibres, at any rate we can feel certain that the *pulsus alternans* is evidence of defect in the contracting power of the heart.

SUMMARY.

Of disturbances of the rhythm of the heart beat, auricular fibrillation (absolute irregularity) and auricular flutter have the gravest prognosis, for not only are these conditions indices of myocardial damage, but they themselves increase the difficulty of the circulation by their rapid driving of the heart. Sometimes, however, many years may elapse before the death of these patients. Of 86 cases of auricular fibrillation seen at the Massachusetts General Hospital 16% are known to have died within ten months of discovery. Premature beats and paroxysmal tachycardia are compatible with a long and active life, but their frequent association with cardiac damage should lead one to be a little cautious in prognosis.

Permanent damage to the atrioventricular bundle of His or its branches is evidence of widespread damage to the myocardium. Hence the prognosis of a patient showing such a condition should be guarded.

Defective contraction of the heart as shown by familiar symptoms and signs, has a prognosis dependent on the number and degree of these symptoms and signs, plus a consideration of similar trouble in the past and of the way in which

the heart responds to rest and digitalis. Of all the individual signs of abnormal contracting power of the heart *pulsus alternans* is one of the most important and consistent. Of 83 cases of alternation of the pulse seen at the Massachusetts General Hospital 31.3% are known to have died within ten months of discovery.

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- ⁴ Swan, J. M.: Arch. Int. Med., 1915, Vol. xv, p. 269.
- ⁵ Tabora (von): Conversation reported by Gravier, L., "L'Altérence du Cœur. Étude Critique et Clinique," p. 282.

III.

THE TREATMENT OF HEART DISEASE.

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THE treatment of heart disease, the subject assigned to me, is no small one. Respecting, therefore, time and place, it will be my effort this afternoon to deal with principles rather than details. Congenital heart disease I shall not discuss.

The treatment of heart disease.

In the first place, we rarely treat heart disease. In rheumatic endo-pericarditis we use the salicylates, though, at least as far as my experience goes, they have no such control here as they have over rheumatic arthritis. If I am right, a possible explanation may lie in the fact that the heart cannot rest as the joints can. Some syphilitic hearts are greatly benefited by potassium iodide. Which these are can be rarely known. We must try. With salvarsan in luetic heart disease I have no experience, my hospital life having closed three years ago. Dr. Sears tells me he has had brilliant results from its use in some cases, notably in aortitis, and in endocarditis of combined luetic and rheumatic origin. I trust we may hear more on this most important point. The discovery of the pale-faced spirocheta has greatly enlarged and clarified our knowledge of cardiac syphilis.