

BEDSIDE RECOGNITION OF THE DISTURBANCES OF CARDIAC RHYTHM

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Present day knowledge of the disturbances of heart rhythm is based primarily upon earlier studies of the arrhythmias by means of the polygraph and the electrocardiograph. Correlation of the results of these studies with careful clinical observations has now made it possible to differentiate most of the arrhythmias at the bedside without instrumental aid. The electrocardiograph is, of course, an instrument of great value but its field of greatest usefulness no longer has to do with the disturbances of cardiac rhythm. Electrocardiograms should be made in every case of actual or suspected organic heart disease but, for the experienced observer, the recording of abnormal heart rhythms in this manner usually will serve only to corroborate an earlier clinical diagnosis. Even in these cases, however, the tracings may give additional information of importance, and for the detection of certain disturbances of rhythm, graphic registration remains indispensable.

SINUS ARRHYTHMIA

In any discussion of the irregularities of heart rhythm, mention should be made of sinus arrhythmia. In this condition there is a gradual increase in the heart rate during inspiration and a corresponding decrease during expiration. The disturbance is encountered commonly in children and is a usual finding in individuals who have neurocirculatory asthenia. It may also develop as a manifestation of digitalis overdosage, and this is the only situation in which it becomes of clinical importance. Sinus arrhythmia often can be accentuated by slow, deep breathing and abolished by exercise.

PREMATURE BEATS

Except for sinus arrhythmia, the most common disturbance of cardiac rhythm consists of premature beats or so-called extrasystoles. These may occur at frequent or infrequent intervals and may arise in the auricles, the ventricles, or the auriculoventricular node and the junctional tissue below it. They occur much more often in the entire absence of organic heart disease than in association with it but are far from rare in individuals who have valvular or heart muscle damage with or without evidence of impaired myocardial reserve. Regardless of their point of origin, premature beats are characterized by a disturbance of heart rhythm in which, at intervals, a heart beat occurs before its expected time. The degree of prematurity of the beat may be slight or marked,

BEDSIDE RECOGNITION OF DISTURBANCES OF CARDIAC RHYTHM

and those beats which follow the preceding normal beat at a very short interval often fail to cause a pulsation in the peripheral arteries. When each normal systole is followed by a premature beat (bigeminal rhythm), a high degree of prematurity may result in a radial pulse rate which is one-half of the ventricular rate.

The clinical recognition of premature beats is seldom difficult although without mechanical aid it is not always possible to distinguish between those of auricular and those of ventricular origin. Nodal premature beats can be identified as such only in the electrocardiogram. In all varieties which complicate an otherwise normal rhythm, it is possible to detect the basic regular rhythm of the heart by careful auscultation, and the differentiation of premature beats from other types of arrhythmias such as auricular fibrillation depends fundamentally upon the ability of the examiner to appreciate that a basic regular rhythm is present but is being disturbed, usually by a single beat, at regular or irregular, frequent or infrequent intervals. In the periods between the premature beats, the normal rhythm of the heart is present. Premature beats often can be diminished in numbers or even abolished by exercise sufficient to increase the heart rate, and this response is also of help at times in differential diagnosis.

With few exceptions, a premature beat is separated from the succeeding normal heart beat by an interval of abnormal length which is known as the compensatory pause. In the case of ventricular or nodal premature beats, this pause is completely compensatory so that the interval between the normal beat which preceded the extrasystole and the first normal ventricular contraction after the premature beat is exactly equal to the length of two normal cardiac cycles. In auricular premature beats, on the other hand, the compensatory pause is of shorter duration or incomplete. When the patient's heart rate is not elevated, it is often possible to estimate the completeness or incompleteness of the compensatory pause by careful auscultation and in this way form a reasonably accurate opinion as to whether the premature beats are of auricular or ventricular origin.

AURICULAR FIBRILLATION

Auricular fibrillation may occur in young individuals with perfectly normal hearts but usually is due to rheumatic heart disease (particularly with mitral stenosis), coronary sclerosis, hypertensive heart disease or thyrotoxicosis. It may also occur as a complication of pneumonia and certain other febrile illnesses. In this arrhythmia there is no co-ordination in the contraction of the muscle fibres of the auricles so that a section of the auricular musculature at any given moment would

show fibers in all stages of contraction and relaxation. Auricular systole is abolished and the auriculoventricular node is showered with a rapid, irregular succession of impulses. Ventricular contractions occur in response to a variable number of these impulses, and a rapid, entirely irregular ventricular rhythm results. Ventricular contractions which follow the preceding response by a very brief interval often fail to cause a pulsation in the peripheral arteries, and a pulse deficit results. It is the absolute irregularity and the entire absence of an underlying basic rhythm that constitutes the pathognomonic feature of the disturbance. As long as the ventricular rate is elevated, auricular fibrillation can be recognized without difficulty, but when the rate has been reduced by means of digitalis, very careful auscultation may be required before one can be certain of the diagnosis.

At times one hears the statement that "slight fibrillation" is present. This is always erroneous, and the term should never be used. There are no degrees of auricular fibrillation; the arrhythmia either is present or it is absent. The ventricular rate may have been reduced by means of digitalis, and at the lower rate the irregularity of the ventricles may be less apparent. The auricles, however, are still fibrillating.

AURICULAR FLUTTER

Auricular flutter occurs much less commonly than auricular fibrillation. It is due to essentially the same type of auricular disturbance as auricular fibrillation but with the difference that regular auricular contractions of limited extent occur at a rate usually in the neighborhood of 300 per minute. Because a certain degree of auriculoventricular block is almost always present, the ventricular rate is slower than the auricular. In those cases in which the degree of block is constant, the ventricular rhythm is perfectly regular while in those in which the degree of block varies, an irregular rhythm is present.

When the auriculoventricular block is constant and of such a degree as to give a ventricular rate within the usual limits of normal, the presence of auricular flutter may easily be overlooked, and the arrhythmia may be discovered only when an electrocardiogram is taken because of associated organic heart disease. Occasionally, in cases of this kind, however, careful inspection of the venous pulsations in the neck may clearly reveal three or four or even more auricular pulsations to each ventricular wave. When auricular flutter is present with a regular ventricular rate of 120 to 180 beats per minute the rhythm must be differentiated from sinus tachycardia and from auricular paroxysmal tachycardia. In auricular flutter the ventricular rate remains constant within ~~very~~ narrow limits and is not affected appreciably by exercise. This is in contrast to the variability of the

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BEDSIDE RECOGNITION OF DISTURBANCES OF CARDIAC RHYTHM

rate in sinus tachycardia, but does not aid in distinguishing the condition from auricular paroxysmal tachycardia. Pressure upon the carotid sinus (~~at the bifurcation of the common carotid artery~~) may cause transient slowing of the rate in auricular flutter or may cause an abrupt standstill of the heart of variable duration followed by resumption of the original rate. The first of these responses may also be obtained in patients with sinus tachycardia but the second does not occur. In auricular paroxysmal tachycardia, pressure upon the carotid sinus either causes an abrupt reversion to normal sinus rhythm or has no effect at all.

~~Because of variations in the degree of auriculoventricular block the ventricular rhythm in auricular flutter may be very irregular and may at times strongly suggest auricular fibrillation.~~ Careful auscultation usually enables one to detect an underlying dominant rhythm, however, and this distinguishes the condition from auricular fibrillation. Furthermore, pressure upon the carotid sinus in auricular fibrillation has neither of the effects upon the rate of the ventricles which occur in auricular flutter.

AURICULAR PAROXYSMAL TACHYCARDIA

This is one of the most interesting of all the disturbances of heart rhythm. In it, an irritable focus in the auricles takes control of the cardiac mechanism and sends out contraction impulses at perfectly regular intervals, usually at a rate between 150 and 180 per minute. No auriculoventricular block is present, and the ventricular rate therefore is the same as the auricular. The condition is characterized clinically by paroxysms which develop suddenly, last for a few seconds to many days, and usually terminate abruptly. The heart rate during an attack seldom varies more than two or three beats per minute, and the disturbance occurs much more commonly in individuals who have normal hearts than in persons who have organic heart disease.

The sudden onset of a perfectly regular tachycardia in which the heart rate is not affected by rest or exercise is always suggestive of auricular paroxysmal tachycardia. Paroxysmal auricular flutter may give rise to tachycardia of similar grade and great regularity but the two conditions usually can be distinguished without difficulty. Mention already has been made of the effect of pressure on the carotid sinus in auricular flutter. In auricular paroxysmal tachycardia, pressure on the carotid sinus either has no effect at all on the heart rate or causes an abrupt resumption of the normal heart rhythm. There are a number of other procedures that often terminate the paroxysm of auricular tachycardia with the same promptness. One of the most

effective of these is the simple act of having the patient hold a deep breath as long as he is able. Firm pressure on the eyeballs, abrupt flexion of the body at the hips and the induction of nausea or vomiting are also effective in many patients. Sudden termination of an attack of regular tachycardia by any of these measures enables one to make an unqualified diagnosis of auricular paroxysmal tachycardia.

VENTRICULAR PAROXYSMAL TACHYCARDIA

Unlike auricular paroxysmal tachycardia, tachycardia of ventricular origin is almost always the result of serious organic heart disease. As in auricular tachycardia, the paroxysms usually are sudden in onset and termination, and the ventricular rate generally is in the neighborhood of 160 to 180 beats per minute. Levine¹ has pointed out a number of characteristics of ventricular tachycardia which are of value in diagnosis. In the first place, the rhythm is seldom perfectly regular although the variation may occur only at infrequent intervals and usually can be detected only by careful auscultation. In the second place, auscultation at the apex for a considerable length of time reveals an occasional accentuation of the first heart sound which apparently is due to simultaneous contraction of the auricles and ventricles. Finally, pressure upon the carotid sinus and the other measures which may terminate an attack of auricular tachycardia have no influence on paroxysmal tachycardia of ventricular origin.

AURICULOVENTRICULAR BLOCK

Auriculoventricular block may be of any degree from slight prolongation of auriculoventricular conduction to complete dissociation of the auricles and ventricles. Simple prolongation of the conduction time between the auricles and ventricles can seldom be detected without the aid of instruments but the various grades of heart block above this often can be recognized by careful auscultation alone. Partial heart block of that degree in which a ventricular beat fails to occur at occasional intervals is characterized by a period of silence over the precordium only slightly shorter than the time of two complete cardiac cycles. This condition can be differentiated from sino-auricular block or so-called sinus arrest only by means of phlebograms or electrocardiograms, and on auscultation one must also make certain that a faint premature beat is not being overlooked in the early part of the period of silence. In 2:1 heart block a ventricular response fails to occur after every other auricular beat. The ventricular rate usually is less than 50 per minute, and it may be possible to hear the sound, usually quite faint, which results from the auricular contractions that are not followed by a ventricular systole. In complete heart block also one can often detect the

BEDSIDE RECOGNITION OF DISTURBANCES OF CARDIAC RHYTHM

sound of the auricular beats. Here the ventricular rate is usually less than 40 beats per minute. There is one other auscultatory finding of diagnostic importance, namely, that on listening over the apex of the heart one will note an occasional accentuation or reduplication of the first heart sound due to the chance occurrence of practically simultaneous contraction in the dissociated auricles and ventricles.

Complete heart block and partial heart block with a slow ventricular rate must be distinguished from sinus bradycardia. Unless one of the above auscultatory signs of heart block are present or unless one can detect extra auricular waves on inspection of the jugular pulse, the differentiation may not be possible by physical examination alone. It is important to remember, however, that exercise and such drugs as atropine cause little or no change in the ventricular rate in complete heart block while in sinus bradycardia a distinct increase in rate usually results.

SUMMARY

The characteristics of the various disturbances of cardiac rhythm have been so well established that it is possible to recognize all but a few of the arrhythmias without electrocardiographic aid. Premature beats, auricular fibrillation, auricular flutter, auricular paroxysmal tachycardia, ventricular paroxysmal tachycardia and complete heart block usually can be detected by careful physical examination alone, and the various grades of partial heart block often can be diagnosed or their presence at least be surmised. Electrocardiographic corroboration of the clinical diagnosis is always desirable but the electrocardiogram today is of much greater value in the detection of heart muscle damage and disturbances of intraventricular conduction than in the differentiation of the cardiac arrhythmias.

REFERENCE

1. Levine, S. A.: Treatment of acute coronary thrombosis, J. A. M. A., 99:1737-1740, November 19, 1932.