Atrial Reciprocal Rhythm and Reciprocating Tachycardia in Wolff-Parkinson-White Syndrome

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SUMMARY
In an infant with type A Wolff-Parkinson-White syndrome, atrial reciprocal beats and attacks of reciprocating tachycardia were repeatedly recorded. Their dependence on a prolongation of the P-R interval could be well demonstrated during Wenckebach periods. Because of the normal aspect of the QRS complex during arrhythmia, the short ventriculo-atrial conduction time (0.08 sec), and the vectorial orientation of the secondary P wave, it was concluded that retrograde reactivation of the atria probably took place via the anomalous bundle. The versatility of the conduction through the accessory bundle is shown by the fact that its direction may change from antegrade in one beat to retrograde in the next. The importance of a circus movement in the genesis of some types of tachycardia in the WPW syndrome is discussed.

Additional Indexing Words:
A-V conduction Reentry A-V dissociation Retrograde conduction
Circus movement Wenckebach period

A N IMPULSE originating in the sinus node or in the atria may, on its way across the atrioventricular junction, turn back and reactivate the atria. The resulting electrocardiographic sequence is a P-QRS-P' complex in which P', owing to the retrograde activation, has a contour different from that of P. Various terms have been applied to this variety of reentry, for example, atrial return extrasystole, atrial echo beat, and reversed reciprocal rhythm. For reasons of clarity and historical priority, recently reviewed by Kistin,1 it seems preferable to use the term "atrial reciprocal rhythm." When the phenomenon is repeated and tachycardia ensues, the term "reciprocating rhythm" is applied.

Pick and Katz2 stressed the importance of a reentry mechanism in various types of arrhythmia observed in the Wolff-Parkinson-White syndrome. They formulated the hypothesis, already conjectured by Wolferth and Wood,3 that supraventricular tachycardia in the pre-excitation syndrome might start when an impulse conducted to the ventricles through the normal atrioventricular junction turns back toward the atria via the accessory pathway and thus initiated a circus movement. Their hypothesis was mainly based on the finding that the last beat preceding an attack of tachycardia was not of the pre-excitation type. Direct electrocardiographic evidence of atrial reciprocal or reciprocating rhythm in the Wolff-Parkinson-White syndrome has since been presented in a few studies.4-8 Apart from the theoretical importance of these observations, a better understanding of the genesis of repetitive tachycardia in the Wolff-Parkinson-White syndrome is essential in the therapeutic approach. If a circus movement is responsible for the rapid heart action and if its anatomic pathways can be localized, surgical section of the anomalous muscle...
bridge might, in selected patients, be the treatment of choice.\(^8\)

The present observation in an infant with Wolff-Parkinson-White conduction is reported because of the unusually frequent alternation of slow and rapid heart rate and hence the opportunity to analyze the spontaneous onset and termination of paroxysms of tachycardia on numerous tracings. The intimate relationship between the Wolff-Parkinson-White syndrome, atrial reciprocal beats, and reciprocating tachycardia is demonstrated by the fact that all three phenomena were often recorded within seconds. To our knowledge identical spontaneous sequences have not been published before. Recently, however, Durrer and associates,\(^9\) were able to produce atrial reciprocal beats and reciprocating tachycardia in a patient with Wolff-Parkinson-White syndrome by intracardiac electrical stimulation and the induction of atrial premature beats.

**Report of Case**

This girl, who had never been ill before, was admitted to another hospital at the age of 8 months, because of tachypnea which was attributed to an upper respiratory infection. On admission she was pale, restless, and dyspneic. The liver was felt 6 cm below the costal margin. She had a regular heart rate at 200/min, and the ECG showed supraventricular tachycardia. The clinical response to digitalization was satisfactory, and intermittently the heart rate slowed down to approximately 100/min. However, steady sinus rhythm could not be restored. After an unsuccessful trial of quinidine (three times 75 mg a day), the baby was transferred to our department. Aside from tachycardia, the heart was normal on auscultation. There were no longer signs of heart failure. The chest roentgenograms revealed no abnormality. The most striking observation was the variability of the cardiac rate. While the patient was asleep or at rest, the heart rate was usually irregular and slow (between 70 and 100/ min), sometimes giving the impression of bigeminy or trigeminy. On awaking or on the slightest excitement it briskly reversed to a regular tachycardia of 180 to 240/min. During these episodes of tachycardia, which sometimes lasted for a whole day, the baby was pale, anxious, and transpired profusely. These signs were so closely linked with the presence of tachycardia that observers who knew the infant well could predict the type of arrhythmia from the patient's general aspect. During the 2 months' hospital course, neither pulmonary nor hepatic congestion was observed, not even after prolonged periods of tachycardia. The administration of digoxin was continued throughout.

In view of the unsatisfactory control of the tachycardia, a trial was made with propranolol. A dose of 2.5 mg was given by mouth. Three hours later a regular heart rate at 120/min was noted. The ECG showed sinus rhythm and type A Wolff-Parkinson-White conduction. The rhythm remained regular for about 24 hours; thereafter it went back to regular tachycardia at 240/min. Subsequent trials with small doses of propranolol remained unsuccessful. Therefore, and also because of inexperties of the chronic use of this drug in infants, it was decided to discontinue its administration. All laboratory data, including hematological examination and the determination of protein-bound iodine, were normal.

The child was discharged home to continue treatment with digoxin, 0.15 mg a day. She has been examined twice at the outpatients clinic, at intervals of 3 months. According to the parents and the family doctor she had been doing well, except for a few episodes of tachycardia. One of them lasted as long as 2 weeks, and then the heart reverted spontaneously to a slow regular rate. The last electrocardiogram, recorded approximately 6 months after the first attack of tachycardia, showed regular sinus rhythm and Wolff-Parkinson-White conduction.

**Analysis of the Electrocardiograms**

As far as frequent auscultation and monitoring could reveal, the dominant rhythm during the hospital course was paroxysmal supraventricular tachycardia (fig. 1). The QRS complex was preceded by a P wave at a P-R interval of 0.24 sec. The P wave was negative in leads I, II, III, aVF, and the left precordial leads, positive in aVR, and biphasic in V\(_{6R}\) and V\(_1\). This might be interpreted as indicating supraventricular tachycardia with an impulse originating somewhere in the left atrium and conducted to the ventricles with a first degree A-V block. The alternative explanation would be that the P wave was conducted in a retrograde direction with an unusually short R-P interval. This interpretation seems correct in view of the following observation. When a bout of tachycardia ends (fig. 1, leads V\(_2\) and V\(_3\)), the last QRS complex is followed by a P wave which has the
Representative electrocardiogram during attack of tachycardia with rate of 200/min. A short interruption is seen in V₂ and V₃. The P wave which follows the fourth QRS complex in these leads has the same configuration as the P waves seen during tachycardia. Its contour and fixed time relationship to the preceding QRS complex suggest that it is due to retrograde reactivation of the atria. After a pause a normal antegrade P wave emerges which is identical with the P waves during tachycardia. In the precordial leads the lower channel lags 0.08 sec behind the upper one. Time intervals in milliseconds.

The same contour and R-P interval as those during tachycardia. In the same strip it will be noted that following a long pause (0.50 sec) a P wave emerges which has a different configuration and which is identical with the P waves recorded during sinus rhythm (see fig. 4). This sinus P wave (preceding the fifth QRS complex in V₂ and V₃, fig. 1) is conducted to the ventricles with a P-R interval of 0.12 sec. The QRS complex is not followed by a negative P wave. The next sinus P wave, preceding the last QRS complex shown in this strip, is conducted at a longer P-R interval (0.24 sec). The ensuing QRS complex is this time followed by a negative P wave, indistinguishable from the ones recorded during tachycardia. The resulting sequence may be described as a P-QRS-P’ “sandwich.”

A detailed analysis of the termination and the beginning of a paroxysm of tachycardia is illustrated in figure 2. The last QRS complex of the bout of tachycardia (upper strip) is followed by a retrograde P wave. As indicated in the diagram underneath the record, one may assume that this retrograde atrial impulse inactivates the sinus pacemaker, as the following pause is not fully compensatory. Indeed this pause (0.80 sec) is shorter than twice the sinus interval (0.42 sec). Similar noncompensatory pauses are shown in the lower strip of figure 2.

The interpretation that the negative P waves (P’) following some QRS complexes are actually atrial reciprocal beats is supported by (1) their fixed time relationship (0.08 sec) to the preceding QRS complex, (2) their configuration and their vectorial direction roughly opposite to that of the sinus P waves, (3) the characteristic sequences in which a QRS complex is “sandwiched” between two P waves.

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Continuous tracing demonstrating the termination and beginning of an attack of tachycardia. The last QRS complex of the bout of tachycardia (upper strip) is followed by a retrograde P wave which is not conducted to the ventricles but probably discharges the sinus node as the following pause is not fully compensatory. The next sinus beat has a normal P-R interval (0.12 sec) and is followed by a QRS complex only. The last QRS complex, shown in the upper strip, is preceded by a long P-R interval (0.22 sec) and followed by a retrograde P' wave (P-QRS-P' sequence). In the lower strip three pairs of QRS complexes are seen in which the first QRS is preceded by a normal P-R (0.12 sec), and the second by a prolonged P-R interval (0.22 sec). This suggests Wenckebach periodicity. Only the QRS complexes preceded by a long P-R interval are followed by a retrograde P' wave. In the last Wenckebach period this retrograde P' initiates a circus movement and thus an attack of supraventricular tachycardia (reciprocating rhythm). S-A = sinus-atrial; A = atrial; A-V = atrioventricular; V = ventricular.
of different configuration, and (4) their dependence on the preceding A-V conduction time. The last point is illustrated by several sequences recorded during relative bradycardia.

The slow heart action is characterized by groupings of two or three beats arranged in short Wenckebach periods. In figure 2 three pairs of beats are shown in which the P-R interval increases from 0.12 to 0.22 sec. In

Figure 3

Similar sequences as seen in figure 2. However, the sinus interval (0.50 to 0.52 sec) and the pause following reciprocal beats are slightly longer, which gives the A-V node the opportunity to escape. The third and sixth complexes are typical A-V dissociation beats. At the end of the tracing the commencement of an attack of tachycardia is seen.

Figure 4

Regular sinus rhythm and Wolff-Parkinson-White conduction (type A).
each of these pairs only the second QRS is followed by a retrograde P wave. It is postulated (see diagram) that the retrograde atrial activation discharges the sinus pacemaker, thus interrupting the Wenckebach period.

The commencement of an attack of tachycardia is closely related to this Wenckebach periodicity. As illustrated in the lower strip of figure 2, the second QRS complex of the third Wenckebach period is preceded by a sinus P wave with a P-R interval of 0.22 sec, and followed by a negative P wave, produced by rapid retrograde conduction to the atria (0.08 sec). In its turn this atrial impulse is conducted in an antegrade direction to the ventricles, initiating a circus movement and thus producing an attack of tachycardia. The P-R intervals during the attack are slightly longer (0.22 to 0.26 sec) than the P-R intervals recorded during isolated P-QRS-P' complexes (0.20 to 0.22 sec). An identical sequence, initiating a run of tachycardia, is seen in figure 3. To these repeated cycles of atrial reciprocal rhythm, the term “reciprocating rhythm” may be applied.

In figure 3 the sinus interval (0.50 to 0.52 sec) is slightly longer than that in figure 2 where it is 0.42 to 0.44 sec. Consequently, the pause following a reciprocal atrial beat is equally longer. This permits the A-V node to escape and to interfere with the sinus P wave. In some cycles the P wave coincides with the QRS complex (the third beat in figure 3), while in others it projects immediately after QRS (the sixth beat).

Figure 4 was recorded after the oral administration of 2.5 mg of propranolol. It shows a regular sinus rhythm with upright P waves in leads I, II, III, aVF, and the pre-cordial leads. The pre-excitation syndrome is revealed by the combination of a short P-R interval, a distinct delta wave, a widening of the QRS to 0.14 sec, and secondary ST-T changes. Because of the positivity of the delta wave in both the right and the left pre-cordial leads, the tracing is characteristic of a type A pre-excitation syndrome.

The three related phenomena (WPW conduction, atrial reciprocal rhythm, and reciprocating tachycardia) are demonstrated in this short strip. A progressive shortening of the sinus interval (0.46 sec, 0.44 sec, and 0.40 sec) is seen in the first three pairs of QRS complexes. The shortest P-P interval is followed by a WPW complex (compare its configuration with lead I and lead II in figure 4). This may indicate that the His bundle has not yet recovered or that the impulse occurs in a supernormal phase of conduction of the anomalous bypass. Note the versatility of the conduction through the anomalous bundle and the reversal of its polarity from beat to beat: the conduction is antegrade in the WPW complex and retrograde in the next sequence in which an atrial reciprocal beat heralds a run of reciprocating rhythms. The first QRS complex is distorted by a standardization mark.
Figure 5 recapitulates in a short strip the three related disturbances (pre-excitation syndrome, atrial reciprocal rhythm, and reciprocating tachycardia) which were often recorded in rapid succession. The first two complexes form a Wenckebach group of which the second QRS is followed by an atrial reciprocal beat, producing a P-QRS-P' sequence. The sixth QRS is a typical WPW complex. Its presence among normally conducted QRS complexes may be explained by the fact that the sinus interval is slightly shorter than it is in the preceding normally conducted complexes. The P-P interval is 0.46 sec in the first group, 0.44 sec in the second, and only 0.40 sec in the sequence in which the WPW complex occurs. It is conceivable that the ordinary A-V pathway has not yet completely recovered at such an early time and that the atrial impulse is transmitted preferentially through the anomalous bundle. The hypothesis of a supernormal phase of conduction in the bypass tissue, following within critical time limits after QRS, might equally well explain the observation. Such WPW complexes, interpolated between normally conducted beats, were repeatedly recorded. As in the example shown, they usually occurred shortly before the beginning of an attack of reciprocating tachycardia.

Discussion

The importance of a reentry mechanism in the origin of some types of tachycardia was first recognized by physiologists. Mines stimulated excised portions of the heart of various species, cut in such a way as to include atrial, ventricular, and junctional tissue, and demonstrated alternating contractions of the atria and ventricles, due to the circus movement of a single impulse. He assumed that slight differences in the duration of the refractory period of certain bundles of the atroioventricular junction might be responsible for the phenomenon. If critically timed, an atrial impulse might thus find part of the junctional tissue refractory and would be conducted along the responsive fibers. During this conduction the refractory fibers might recover and serve as a pathway for retrograde reactivation of the atria from the ventricles. If this process is repeated, an attack of supraventricular tachycardia will ensue. This hypothesis of a functional dissociation of the atioventricular junction into two (or multiple) pathways has since been amplified and the physiological conditions under which it may occur have been well established. The electrocardiographic characteristics and the diagnostic criteria of atrial reciprocal rhythm have been repeatedly discussed. All authors emphasize the importance of partial atroioventricular block or Wenckebach periodicity in the genesis of the arrhythmia: Normal or short P-R intervals are followed by a QRS complex, and prolonged P-R intervals, by a QRS-P' sequence. In the latter case the slow A-V conduction through some fibers might allow recovery of other parts of the bundle which might then become accessible for retrograde conduction (P').

Because of the existence of two anatomically separated atroioventricular pathways, patients with the WPW syndrome are expected to be particularly prone to ventricular-atrial reentry. This mechanism has in fact been invoked to explain the high incidence of various types of arrhythmia in the pre-excitation syndrome. It is surprising therefore that only a few well-documented cases of atrial reciprocal rhythm or reciprocating tachycardia in association with the WPW syndrome have been published. Only Massumi and associates recorded both types of rhythm disturbance in the same patient and in rapid succession. The arrhythmia reported by Harnischfeger is slightly different as the site of origin of the impulse was not the sinus node or the atrium, but the atroioventricular node.

In all these cases, as in the present one, the retrograde activation time (R-P') was short and comparable with the antegrade activation time through the anomalous bundle. As stressed by Wolff this observation gives considerable support to the theory that retrograde activation occurs through the accessory bundle. The case of reciprocating rhythm reported by Burchell, though occurring in a
patient in whom WPW conduction had been observed several years previously, is probably not an example of retrograde activation through the anomalous bundle. The long R-P' interval (0.20 sec) in isolated P-QRS-P' sequences and during attacks of tachycardia suggests reactivation through the His bundle.

Besides the short R-P' interval, the vectorial orientation of P' lends support to the concept that retrograde conduction takes place via the accessory pathway. It is generally accepted that the anomalous bundle in type A WPW syndrome connects the left atrium with the posterobasal areas of the left ventricle. If a retrograde impulse invades the atrium from this location, one expects a P'-wave vector as seen in the present case. Epicardial exploration indicates that the accessory muscle bridge in the WPW syndrome of type B is located near the base of the right ventricle and close to the atrioventricular groove, as the earliest ventricular activation is recorded in this region. A retrograde P' originating from this area would in most leads be oriented in a direction comparable to that of the normal P wave and not too different from the ST-T vector. This may be one of the reasons why the retrograde P', at least in type B of the WPW syndrome, is not conspicuous and why esophageal leads are often needed.

A final argument in favor of retrograde activation, presumably through the anomalous bundle, is the dependence of reciprocal or reciprocating rhythm on prolongation of the atrioventricular conduction. This could be repeatedly demonstrated in our patient because of the frequent occurrence of Wenkebach periods. The observation is in complete accordance with previous studies in the case reported by Massumi and associates in partial A-V block, due to rheumatic carditis, was considered to be the immediate cause of atrial reciprocal rhythm.

Comparable tracings have recently been obtained by Durrer and associates. When they critically timed electrically induced atrial premature contractions in a patient with WPW syndrome and a history of palpitations, they were able to elicit atrial reciprocal beats and sometimes bouts of supraventricular tachycardia. In view of the short R-P interval (P' beginning 0.02 to 0.04 sec after the end of the QRS complex) and the polarity of P' the authors concluded that retrograde conduction took place through the Kent bundle. Furthermore the attacks, if they did not end spontaneously, could be stopped by a single atrial premature beat which presumably made the atrium refractory and blocked retrograde Kent conduction from the ventricle. These clinical experiments as well as the analysis of spontaneous attacks of tachycardia, as presented here, fit the hypothesis of a circus movement of the impulse in certain forms of tachycardia in the pre-excitation syndrome.

References


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