Termination of Reciprocating Tachycardia by Atrial Stimulation

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SUMMARY

Recurrent tachycardia in a 10-year-old child with acute rheumatic carditis was interpreted as being produced by a reciprocating (circus) mechanism involving either the normal atrioventricular pathway or the anomalous pathway of the Wolff-Parkinson-White syndrome. The tachycardia could be stopped by appropriately timed, electrically induced, atrial premature systoles, which apparently interrupted the circus movement.

Additional Indexing Words:
A-V conduction
Intracardiac electrode
Circus movement
Acute rheumatic carditis
WPW syndrome

MOE AND ASSOCIATES\(^1\) produced tachycardia in a dog which they attributed to a reciprocating rhythm or a circus movement through the atrioventricular (A-V) transmission system. They could stop the tachycardia by electrically producing two atrial premature beats in quick succession at a critical time in the cardiac cycle. The present report pertains to recurrent tachycardia in a 10-year-old girl with acute rheumatic carditis. The arrhythmia was interpreted as a reciprocating tachycardia either through the normal A-V transmission system or involving the anomalous pathway of the Wolff-Parkinson-White syndrome. The tachycardia was stopped several times by an electrical stimulus which produced two successive atrial premature beats.

Observations

During the patient's first 25 days in the hospital, the tachycardia recurred frequently and lasted as long as 28 hours at a time. Although continuous records were not obtained, repeated observations were possible. A typical example of the many paroxysms of tachycardia and a detailed description are presented in figure 1. The beat just before the onset of the paroxysm consists of a normal sinus P conducted to the ventricles at a long P-R interval. The resulting QRS is in turn followed by a reciprocal P'. This P', however, differs from the previous two blocked P's in that it is self-perpetuating by virtue of reciprocating with itself (fig. 2).

The tachycardia at first was resistant to treatment. The patient was treated with corticosteroids and also received 0.2 g of whole leaf digitalis and 1.8 mg of digoxin in 9 hours followed by 0.25 mg of digoxin every 12 hours. This seemed to produce brief remissions, either spontaneous or enhanced by carotid sinus pressure or breath-holding. External direct current electrical shocks of 50 to 400 watt-sec strength stopped the tachycardia on a few but not all occasions. Several of the paroxysms failed to respond to carotid sinus pressure, breath-holding, phenobarbital, 30 mg every 6 hours, sodium phenobarbital, 75 mg given subcutaneously, diphenylhydantoin, 150 mg intravenously, lidocaine, 50 mg intravenously, or procainamide, 1 g intravenously. During the fourth week of observation and the fifth week of illness, a Wolff-Parkinson-
White abnormality of the electrocardiogram appeared for the first time (fig. 3).

During one of the protracted periods of tachycardia in the third week of observation when none of the measures tried was effective, a bipolar electrode was passed into the upper part of the right atrium through an arm vein; the contacts of the electrode were 1 cm apart. Stimuli were applied from a Medtronic 4837 pulse generator which produces stimuli 2 msec in duration, permits programming of the stimulus in relation to the R wave of the

Figure 1

X, Y, and Z leads (Frank) and a bipolar esophageal lead 30 cm from the nares (BE 30) recorded simultaneously. Tachycardia begins in the middle of the tracing. Before onset of the ectopic rhythm, the P-R intervals alternate between 0.13 (beats 1, 3, and 5) and 0.22 sec (beats 2, 4, and 6). Only beats possessing long P-R intervals are followed by retrograde atrial activation waves marked P' in the BE tracing. This is interpreted as reciprocal activation of the atria after the normal sinus P enters the A-V junctional tissue, turns about, and reenters the atrium in a retrograde fashion. The prolonged P-R interval is necessary for this phenomenon, for it enables the return pathway, the atrial myocardium, or both to recover completely before the arrival of the retrograde impulse. The first two retrograde P waves are blocked and are not followed by QRS. The third P', however, enters the ventricles, and, at the same time, initiates a tachycardia by perpetuating itself with 1:1 A-V relationship at a rate of 168/min. During tachycardia the P-R intervals are exactly 0.22 sec and therefore are identical with the long P-R before tachycardia; this suggests use of the same pathway.

The P waves with short P-R intervals are not followed by reciprocal P' either because they entered the ventricles by way of a separate route far from the return pathway or because they used the same route as that of the P waves with long P-R intervals but found refractory tissue on their way back to the atria.
Figure 2

Schematic drawing depicting the suggested mechanisms for the behavior of arrhythmia and its electrical termination in the present case. The speckled areas represent the sino-atrial node and the atrioventricular junction including the node and paranodal fibers. The dashed lines indicate fast, and thick dotted lines, slow A-V conduction.

Before onset of tachycardia, conduction proceeds alternately across two pathways. One is depicted to the left and, utilizing the fast pathways within the A-V junction, reaches the ventricles by a short P-R interval (see beats nos. 1, 3, and 5 of fig. 1). The other type of beat with long P-R interval results from passage through the slow pathway within the A-V node (beats 2, 4, and 6 of fig. 1). The latter beats are followed by reciprocal P' because the impulse, after traversing the slow A-V path, gains contact with the accessory bundle drawn just to the right of the A-V junction. Finding the accessory bundle and the atrial tissue, this retrograde impulse invades the atrium and gives rise to the retrograde P'.

During tachycardia, conduction through the slow A-V path proceeds as normal and returns toward the atrium (reciprocating) as depicted above. However, the returning limb in turn establishes connection with the orthograde slow pathway and, finding it recovered, follows its original path in quick sequences (circus). Termination of tachycardia by one electrical stimulus resulting in 2 successive P waves is depicted to the right of the illustration. So long as the electrical impulse, spreading both cephalad and caudal meets refractory SA node tissue on its retrograde passage, it succeeds only in producing a premature atrial beat (pathway I). On its way toward the A-V junction, this impulse encounters the returning limb of the reciprocal P' and is locally extinguished. On the other hand, when the ascending portion of the electrical stimulus finds the SA node responsive, it passes through it and reenters the atrium in orthograde direction (pathway II). It is this impulse which is responsible for the second PX in figure 4. By the time this impulse reaches the A-V junction, the circus pathway has already recovered and is thus penetrated and extinguished by the impulse. The pathway responsible for the reciprocal activation of the atrium in this case is probably the same pathway used in orthograde fashion during the WPW abnormality.
The first beat in each lead is normally conducted and the second beat shows features of Wolff-Parkinson-White syndrome. The precordial leads were recorded at half standardization, (1 mV = 5 mm) except V₅ and V₆ of the WPW syndrome which were recorded at normal standardization. The normal tracing and the one showing WPW were taken 2 days apart during the same hospital admission at a time when rheumatic activity was declining.
RECIPROCATING TACHYCARDIA

Termination of the reciprocating tachycardia by electrically produced atrial premature beats. Conventions as in figure 1. Arrows point to stimulus artifacts. They can be recognized as notches in the R waves of leads Y and Z and as brief breaks in the base line of BE just before atrial responses marked PX. P = sinus P; P' = retrograde atrial activation; PX = atrial premature beats produced by electrical stimulation. The first three stimuli produce single atrial premature beats and do not stop the tachycardia. The fourth stimulus differs from the preceding three stimuli in that it produces two atrial premature beats in quick succession and stops the tachycardia. The second PX, traveling in a direction opposite to the P' waves, is responsible for the tachycardia. (See figure 2 for explanation.)

Figure 4

During a large part of the cycle the stimulus elicited no responses as it met refractory atrial myocardium. As the stimulus approached R, that is, moved away from the preceding P, atrial premature beats were produced. Single electrically induced atrial premature beats were ineffective. However, the electrical impulses followed by two successive atrial premature beats separated from one another by 0.22 sec were regularly capable of terminating the tachycardia. (See legends of figures 2 and 4 for explanation.)

On three occasions during tachycardia, application of atrial stimuli in the manner described above failed to produce two successive P waves and, therefore, did not terminate the rapid rhythm. Instead, they converted it to atrial flutter at rates of 320 to 370/min lasting 6 to 17 sec. These stimuli were found subse-

electrocardiogram, and delivers currents at amplitudes up to 10 ma. By careful exploration with slight changes in the timing of the stimulus, a location was found near R and 190 msec after onset of the preceding P' as measured from the esophageal tracing where a stimulus stopped the tachycardia (fig. 4). This was successful during a number of recurrences.

In order to investigate the mechanism by which atrial stimulation stopped the arrhythmia, tracings from several episodes were carefully scrutinized and stimuli were delivered to the right atrium during various parts of the heart cycle. The critical location near R which had previously been found to stop the tachycardia was avoided, and the remainder of the cycle was explored, changing the timing of the stimulus by less than 10 msec at a time.
ently to have been delivered to the atrium during the atrial T wave, that is, the vulnerable period for this chamber.

Discussion

Reciprocal rhythms are those in which there is circus movement in or near the A-V junction. A sinus, atrial or ventricular impulse on its way across the A-V junction finds a bypass and returns to reactivate the chamber of origin. Or, an impulse originating in the A-V junction and spreading to the atria or ventricles finds a bypass, turns back, and enters the other chamber in the opposite direction. The term "reciprocating"3 has been used when the circus movement continues for a number of cycles. Reciprocal rhythm with single cycles probably occurs frequently in man.4-7 The frequency of reciprocating tachycardia is not known, because its recognition requires observation at the onset of the tachycardia and because the standard electrocardiographic leads usually provide no evidence for suspecting the mechanism. Since reciprocal rhythm is but one form of reentry, its incidence has an important bearing on the role of reentry in the genesis of human arrhythmias in general.

The tachycardia reported here resembles others previously described.5 The interpretation that the arrhythmia in this case was produced by a reciprocating mechanism, while based on inference, seems strongly supported first by the retrograde configuration of the P waves labeled P′ in figures 1 and 4 and second by the association of the onset of the tachycardia with partial A-V block.

The P′ waves were opposite in direction to the sinus P waves in the esophageal lead as would be expected with retrograde atrial activation. Similar P waves occur with retrograde atrial activation associated with ventricular premature beats produced by irritation of a ventricle by a cardiac catheter.8, 9

In the present case both types of retrograde P′, that is, the isolated P′ and those initiating the tachycardia were always preceded by prolonged P-R intervals. The partial block presumably allows time for some fibers in or around the A-V junction to recover in time to serve as a return pathway or that the long P-R interval allows sufficient time for full recovery of the atrial myocardium so that it can now respond to the returning, reciprocal impulse. An alternative explanation is that the sinus P waves with short P-R intervals and those with long P-R intervals utilize two different pathways with different characteristics of conduction velocity and duration of refractory period.1-3, 5, 10 The pathway passing through or more closely associated with the A-V node would then be responsible for the prolonged P-R intervals, and therefore, more conducive to the establishment of a reciprocal rhythm; while the pathway lying further from the A-V node would conduct faster, at a P-R interval which is too short to allow reciprocal activation of the atrium.

The presence of the Wolff-Parkinson-White abnormality suggests the possibility that the reciprocal mechanism in this case was caused by antegrade conduction by way of the usual A-V path and retrograde conduction by way of the anomalous path.2, 10 The acute rheumatic carditis which was probably responsible for the partial A-V block in this case could conceivably have contributed to the occurrence of the reciprocal rhythm by allowing the anomalous path sufficient recovery time for retrograde conduction. It was not possible in this case to determine which of these mechanisms was responsible.

The technique used by Moe and his associates1 may have practical applications in man and may provide a new approach to the treatment and further study of some types of supraventricular tachycardia. These authors stopped the tachycardia with two premature beats in rapid succession, but they could not stop it with one. They discussed in detail the critical requirements of timing for an impulse to reach and enter the circus path at a time when it may interrupt the propagating impulse. In the present case also the tachycardia was stopped by a pair of atrial beats occurring after a single electrical stimulus delivered to the atrium. While termination of the tachycardia in the manner described fits the concept of the interruption of a circus movement,
as Moe and his associates\(^1\) pointed out, it is also conceivable that premature beats may under suitable conditions inactivate a single focus producing tachycardia by repeated discharges.

When supraventricular tachycardia occurs during cardiac catheterization, it is sometimes possible to stop it by advancing the catheter into the ventricle and deliberately producing ventricular premature beats. One of the authors (R.A.M.) has observed this twice, and it has been noted repeatedly by others (personal communication from Lewis Scott, Children's Hospital, Washington, D.C.). One may speculate that the impulse from the ventricular premature beat is conducted to the A-V junction where it interrupts a circus movement, or that it is conducted to the A-V junction or the atria to inactivate a repetitive focus.

References
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