Unusual Examples of Supraventricular Re-entrant Tachycardias

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SUMMARY
Programmed electrical stimulation of the heart uncovered unusual findings in four patients suffering from supraventricular tachycardias. They included 1) two supraventricular tachycardias of different location and frequency in the same patient, 2) increase in rate of a re-entrant atrioventricular (A-V) nodal tachycardia followed by the development of 2-to-1 A-V block, 3) a "gap" in A-V conduction and A-V nodal re-entrant tachycardia with 2-to-1 A-V block, and 4) the initiation of atrial re-entrant tachycardia by ventricular premature beats. The examples are given to help elucidate the understanding of mechanisms involved in re-entrant tachycardias and to localize the site of the re-entry circuit.

Additional Indexing Words:
Supraventricular tachycardia
Gap in atrioventricular conduction
Single test stimulus method
Re-entry
Atrioventricular block

The introduction of programmed electrical stimulation of the heart has enabled one to study the mechanisms of tachycardia directly in the human heart. A critically-timed premature beat can elicit tachycardia in hearts of patients with the pre-excitation syndrome; such a beat may also initiate tachycardia in the atrioventricular (A-V) node, the ventricle, and possibly also in the sinus node area. Although it has been shown recently by Cranefield and Aronson that, with perfusion of isolated Purkinje cardiac fiber preparations with sodium-free solution or ouabain, focal tachycardias can be initiated and terminated by premature beats. We believe that, in the intact heart, initiation and termination of tachycardias by a premature beat given within reproducible time intervals strongly suggests a re-entry mechanism. In reviewing our experience with stimulation studies in over 200 patients with symptomatic re-entrant tachycardias, examples of unusual tachycardias were noted. The four patients reported here all suffered from re-entrant supraventricular tachycardias. Although the mechanisms in these patients are uncommon, analysis of them is helpful in elucidating the underlying mechanisms operative in re-entrant tachycardias and in localizing their site of origin.

Material and Methods

Characteristics of the four patients studied are given in table 1. None of the patients were receiving medication at the time of study. Following informed consent, all patients were studied in the nonabsorptive state. Initiation of tachycardia was studied by means of the single test stimulus method during atrial and ventricular pacing and by regular pacing of atrium and ventricle at increasing frequencies. With the help of the synchronizing circuit of our stimulator, the effect of timed atrial and ventricular stimuli on the time relations during tachycardia and termination of tachycardia was subsequently studied. A description of the stimulator employed has been given previously. Simultaneous recordings were made of leads I, II, III, V1, and V6, an intracavitary high right atrial lead, and a His bundle electrogram. All data were stored on magnetic tape using an Ampex FR 1300 recorder. The figures presently demonstrated show only those leads considered to be relevant.

Results
Case 1. Two Supraventricular Re-entrant Tachycardias of Different Frequency in the Same Patient

As shown in the upper part of figure 1, a supraventricular tachycardia could be initiated during regular pacing in the atrium at a basic cycle length of 600 msec by giving an atrial premature beat in the interval range of 310 to 270 msec. The pattern of initiation of tachycardia — which revealed marked slowing of conduction through the A-V node with a sudden increase in the A-H interval of 50 msec on decreasing the premature beat interval from 320 to 310 msec — and the relation between atrial and ventricular activation during tachycardia were compatible with an A-V nodal re-entrant tachycardia. When the atrial premature beat coupling interval was shortened further to less than 270 msec, tachycardia could not be initiated until a premature beat interval of 240 msec was reached (lower part of fig. 1). This tachycardia had a different frequency and relation between atrial and ventricular activation as compared to the tachycardia initiated at longer premature beat intervals. As shown...
in the upper part of figure 2, an appropriately-timed atrial premature beat could change one type of tachycardia into the other. The lower part of figure 2 demonstrates reversion to the original tachycardia is effected by two premature beats given consecutively in the right ventricle. The tachycardia with the lowest frequency could be consistently terminated by a single atrial premature beat elicited 10 msec earlier than the one shown in the upper part of figure 2.

The other type tachycardia could only be terminated by two consecutively given ventricular premature beats. The second premature beat had to be given 20 msec earlier than the one shown in the lower part of figure 2.

### Table 1
Clinical Data of Patients Studied

<table>
<thead>
<tr>
<th>Case number</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Incidence of tachy</th>
<th>Duration (yr)</th>
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<tbody>
<tr>
<td>1</td>
<td>32</td>
<td>F</td>
<td>2/mo</td>
<td>6</td>
</tr>
<tr>
<td>2</td>
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<td>F</td>
<td>3/mo</td>
<td>4</td>
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<tr>
<td>3</td>
<td>42</td>
<td>M</td>
<td>3/yr</td>
<td>1</td>
</tr>
<tr>
<td>4</td>
<td>68</td>
<td>M</td>
<td>2/mo</td>
<td>11</td>
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</tbody>
</table>

As shown in figure 3, a supraventricular tachycardia could be initiated by an atrial premature beat given after 290 msec during regular pacing of the atrium with a basic cycle length of 500 msec. The pattern of initiation of tachycardia, and the relation between atrial and ventricular activation during tachycardia, were suggestive of a re-entrant tachycardia situated in the A-V node. Following initiation of tachycardia, there was an increase in rate of tachycardia. As shown in figure 4, the ventricular rate during tachycardia suddenly fell to one-half the previous rate when the interval between two consecutive His bundle electrograms became 260 msec. The His bundle lead illustrates that there was persistence of the 1-to-1 relation between His bundle and atrial activation. In this patient one could reproducibly record the development of 2-to-1 A-V block distal to the bundle of His as soon as the tachycardia interval measured 260 msec. Unfortunately the effective refractory period of the distal Purkinje system could not be determined during atrial pacing using the single test stimulus method because, at a basic cycle length of 500 msec, the atrium became refractory at 265 msec. At this premature beat interval there was still conduction to the ventricle and initiation of tachycardia. Termination of...
SUPRAVENTRICULAR RE-ENTRANT TACHYCARDIAS

Figure 3
Case 2. Initiation of tachycardia by an atrial premature beat given after 290 msec during pacing of the atrium with a basic cycle length of 500 msec. As indicated in the diagram, the pattern of initiation of tachycardia suggested an A-V nodal re-entrant tachycardia. Note the increase in rate of tachycardia following its onset. α and β designate postulated dual A-V nodal pathways.

tachycardia was only possible when two ventricular premature beats were elicited in close succession.

Case 3. Gap in A-V Conduction and A-V Nodal Re-entrant Tachycardia with 2-to-1 A-V Block

During pacing of the atrium at a basic cycle length of 700 msec, an atrial premature beat elicited in intervals ranging from 390 to 300 msec was not conducted to the ventricle (fig. 5, panels B and C). Because the blocked atrial premature beat was not followed by a His bundle electrogram, one has to assume that block occurred in the atrium or in the A-V node. An atrial premature beat elicited in the interval range 290 to 260 msec (see fig. 5, panel D) was conducted to the ventricle and was followed by a tachycardia having a 2-to-1 relation between atrial and ventricular activation. The interval between two consecutive atrial complexes during tachycardia measured 320 msec. As indicated by the His bundle recording, the A-V block (2:1) during tachycardia was situated above the level of the His bundle electrogram recording site and was presumably in the A-V node. The tachycardia could be terminated by two consecutive premature beats elicited in either the atria or ventricles.

Case 4. Atrial Re-entrant Tachycardia Initiated by Ventricular Premature Beats

In this patient tachycardia could be initiated by a ventricular premature beat during ventricular pacing. There were two distinct ranges of premature beat coupling intervals during which a tachycardia could be initiated. As shown in figure 6, the ranges were from 390 msec to 350 msec and from 300 to 290 msec. As demonstrated in figures 6 and 7, initiation of tachycardia was not related to critical slowing in the A-V node. Instead, it related to the interval between atrial activation following the last beat of the basic ventricular rhythm (A1) and atrial activation following the premature beat (A2). If this A1–A2 interval fell to 405 msec or less, tachycardia resulted. The increase in ventriculo-atrial conduction following ventricular premature beats given with increasing prematurity, together with emergence of a His bundle potential following ventricular premature beats given after a premature beat interval of 350 msec or less, indicates that ventriculo-atrial conduction occurred through the

Same patient as figure 3. Nine tachycardia complexes were present between the end of figure 3 and the beginning of the registration shown in figure 4. There has been a further increase in rate during tachycardia. On reaching an interval between two consecutive His bundle complexes of 290 msec, the QRS complexes show the development of 2-to-1 block. As shown in the His bundle lead and illustrated in the diagram, this 2-to-1 block was situated below the site of the catheter recording from the His bundle. Note the persistence of left bundle branch block following the development of 2-to-1 block. Either concealed retrograde invasion into the left bundle or the “fatigue” phenomenon could explain this phenomenon.

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Figure 6

Case 4. Five different ventricular premature beat intervals are shown during ventricular pacing with a basic cycle length of 600 msec. Note that tachycardias are only initiated (middle panel, left and right) when the interval between atrial activation following the last ventricular complex of the basic rhythm (A1) and atrial activation following the ventricular premature beat (A2) reaches a value of 405 msec or less. As shown in both panels on the right, ventriculo-atrial conduction occurs via the A-V node. Because there is no relation between delay in conduction through the node and initiation of tachycardia, the sequence of events suggests the tachycardia circuit to be located in the atrium.

His-A-V node pathway and excludes the possibility of V-A conduction over an accessory pathway. The finding of the critical relation between A1-A2 interval and initiation of tachycardia, with no relation to retrograde transmission time through the A-V node, favors an atrial location for the re-entry circuit. No tachycardias could be initiated during atrial pacing with a basic cycle length of 600 msec by test stimuli given in the range of 450 to 260 msec. The tachycardia could reproducibly be terminated by a single atrial premature beat.

Discussion

By means of programmed electrical stimulation of the heart, it has been possible to reproducibly initiate and terminate tachycardias with appropriately timed premature beats. When this can be demonstrated, re-entry has been identified as the causal mechanism. The first patient presently described demonstrates that two types of supraventricular re-entrant tachycardias can exist in the same patient. The initiation of the two varieties of tachycardia was critically related to the timing of the premature beat. It is impossible to identify the exact site of origin of the tachycardias. The pattern of initiation of the tachycardia with the highest frequency is compatible with a location in the A-V node.9, 10 If one believes that P wave polarity indicates the origin of the atrial impulse, the tachycardia with the lowest frequency could have originated high in the atrium, possibly in the sinus node.9 If one does not accept this hypothesis, the possibility that both types of tachycardias had their site of origin in the A-V node, or one in the A-V node, and the other somewhere in the atrium, cannot be excluded. The interesting feature of this patient was the ease with which one type of tachycardia could be changed into the other by use of critically timed atrial and ventricular premature beats.

Patient 2 showed a phenomenon which in our experience is unusual in patients with A-V nodal
SUPRAVENTRICULAR RE-ENTRANT TACHYCARDIAS

Graph showing the relation between the interval from atrial activation \( A_1 \) to atrial activation \( A_2 \) following basic rhythm to atrial activation \( A_2 \) following the premature ventricular beat (the \( A_1-A_2 \) interval) and the prematurity of the ventricular test pulse. All points falling upon or below the horizontal line were followed by tachycardia.

reciprocal tachycardia. Following initiation of tachycardia, there was an increase in tachycardia frequency from approximately 180 beats/min to approximately 230 beats/min. The increase in frequency following onset of tachycardia, called “warming-up” phenomenon, might suggest a focal origin for the tachycardia.\(^{12-14}\) However, the pattern of initiation and the reproducible termination of tachycardia by two closely coupled ventricular premature beats in the patient presently described point more to a re-entry mechanism. At a tachycardia frequency of approximately 230 beats/min, the patient developed 2-to-1 A-V block which, based on the His bundle recording, had to be located distal to the bundle of His or in the His bundle itself. Atrioventricular block during reciprocal A-V nodal tachycardia is not rare.\(^{9, 18}\) Six examples were encountered in 58 consecutive patients with re-entrant A-V nodal tachycardia. In five of these patients, there was a 2-to-1 relation between atrial and ventricular activation. The other patient showed a 3-to-2 Wenckebach type of second degree block between the A-V node and the ventricle. Three patients showed a 1-to-1 relation between His bundle and atrial activation, suggesting that the block was situated in or below the bundle of His. In the other three patients the A-V block was located in the A-V node (see also patient 3).

In our patient 2 the increase in rate during tachycardia was based upon shortening in A-H interval, the H-A interval remaining constant. One does not know what was responsible for this shortening in antegrade A-V conduction time. Changes in antegrade conduction without changes in retrograde conduction have also been observed in our laboratory in patients with A-V nodal tachycardias following the administration of drugs like digitalis. This suggests that the electrophysiologic properties of the antegrade and retrograde pathways might be different.

Patient 3 showed a “gap” in A-V conduction.\(^{16-19}\) In order to have resumption of atrioventricular conduction following the gap, it is essential to have slowing of conduction in the area proximal to the area in which block occurs. Slowing of conduction is not only required for overcoming the gap but is also a necessity for initiation of A-V nodal re-entry.\(^9\) The combination of the “gap” phenomenon and the initiation of a re-entrant tachycardia in the A-V node is not therefore surprising. Because of our ability to record the electrical activity of the bundle of His, it is possible to localize, by catheter techniques, gaps in and below the bundle of His. Unfortunately, since we cannot register the electrical activity of the A-V node, exact localization of the site of gap in our patient is not possible. Although we cannot exclude the possibility that the area responsible for the “gap” was situated in the atrium, the observation that 2-to-1 block was present proximal to the bundle of His during tachycardia makes it tempting to speculate that the area responsible for the “gap” was situated in the A-V node distal to the re-entry circuit.

It is of interest that Janse et al.\(^{20}\) studying the isolated rabbit A-V nodal preparation with the microelectrode technique, could demonstrate the occurrence of a “gap” in the A-V node, followed by re-entrant tachycardia. It is of interest that they also observed, as in our patient, 2:1 block occurring between the node and the bundle of His during the tachycardia.

Patient 4 demonstrated that atrial tachycardia can be initiated by critically timed ventricular premature beats. While the pattern of V-A conduction following premature beats given with increasing prematurity showed retrograde conduction through the A-V node, thus excluding conduction along an accessory pathway, it could also be clearly demonstrated that initiation of tachycardia was not related to critical slowing of conduction through the node. This observation and the finding that initiation of tachycardia was dependent upon critical time relations at the atrial level seems to exclude an A-V nodal tachycardia and to argue for a tachycardia of atrial origin. It is important to realize that ventricular tachycardias can be initiated by an atrial premature beat\(^4\) and atrial tachycardias by a ventricular premature beat in patients in whom the A-V node is the only connection between atrium and ventricle. All that is necessary for either of these phenomena to occur is for conduction.
to go through the node at a speed which permits the impulse to arrive in the other chamber within an interval permitting initiation of re-entry.

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