Second Degree Block During Reciprocal Atrioventricular Nodal Tachycardia

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SUMMARY Of 67 patients with reciprocal atrioventricular (A-V) nodal tachycardia consecutively studied by programmed electrical stimulation of the heart, nine patients showed second degree block toward the ventricle and one patient toward the atrium during tachycardia. In four patients the occurrence of block was critically related to the prematurity of the test stimulus initiating the tachycardia. In three patients block developed following increase in rate of tachycardia. In two patients block could be elicited by introducing premature ventricular stimuli during tachycardia. Our observations indicate that different mechanisms may be responsible for second degree block during reciprocal supraventricular tachycardia. The finding of second degree block during reciprocal supraventricular tachycardia excludes a tachycardia with A-V conduction over the A-V node – His pathway and V-A conduction over an accessory A-V pathway.

ONLY A FEW EXAMPLES of second degree block during reciprocal atrioventricular (A-V) nodal tachycardia have been reported in the literature. To determine the incidence and to study the mechanism of block, data were reviewed from 76 patients, in whom during programmed electrical stimulation of the heart a reproducible tachycardia could be initiated and terminated by appropriately timed premature beats. Signs of pre-excitation were not present in these patients during sinus rhythm and/or atrial pacing. All showed initiation of tachycardia following a critical delay in A-V nodal transmission time and a low-high sequence of atrial activation during tachycardia.

Application of the criteria given elsewhere for differentiation between a tachycardia circuit confined to the A-V node and a tachycardia circuit using an accessory A-V pathway suggested that in nine patients an accessory A-V pathway only conducting in the retrograde direction participated in the tachycardia circuit. During electrically-induced tachycardia episodes of second-degree block either toward the ventricle (7 patients) or toward the atrium (1 patient) were demonstrated in eight of the remaining 67 patients in whom the tachycardia circuit was thought to be confined to the A-V node. In two more patients persistence of tachycardia could be demonstrated in spite of block distal to the bundle of His, provoked by giving two closely spaced ventricular premature beats during tachycardia.

Material and Methods

Characteristics of the ten patients reported are given in table 1. None of the patients were receiving medication at the time of study. Following informed consent all 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 presented here show only those leads considered to be relevant.

Results

The reproducible initiation and termination of tachycardia by critically timed premature beats strongly suggested re-entry as the underlying mechanism for tachycardia in

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our patients. Application of criteria given elsewhere\(^a\) pointed to the A-V node as the site of the re-entry circuit.

**Initiation of Tachycardia and Development of Block During Tachycardia**

In nine of the ten patients tachycardia could be initiated only by a single atrial premature beat during atrial pacing, not by ventricular stimuli during ventricular pacing (table 1); in the remaining patient both an atrial and a ventricular premature beat produced the rhythm.

In one patient (patient C) atrial premature beats given in the interval range 290 to 260 msec (the refractory period of the atrium) resulted in tachycardia with always a 2 to 1 relation between atrial and ventricular activation. This patient, who has been described previously,\(^b\) showed a gap in A-V conduction in the premature beat interval range 390 to 300 msec. In four patients (patients A, D, F, and G) the development of block during tachycardia depended upon the timing of the tachycardia initiating atrial premature beat. A typical example is given in figures 1 and 2. During atrial pacing with a basic cycle length of 600 msec, atrial premature beats at a range of coupling intervals of 290 to 240 msec resulted in tachycardia with a 1 to 1 relation between atrial and ventricular activation (fig. 1, top). Atrial premature beats given

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**Table 1. Clinical Data and Findings Obtained during Programmed Electrical Stimulation**

<table>
<thead>
<tr>
<th>Pt/Age/Sex</th>
<th>Init of T.</th>
<th>R-R interval (msec)</th>
<th>Site of block</th>
<th>Term of T.</th>
</tr>
</thead>
<tbody>
<tr>
<td>A* 75/M</td>
<td>1 APB</td>
<td>300–310</td>
<td>A-V-node-His</td>
<td>2 APB, 2 VPB</td>
</tr>
<tr>
<td>B* 23/F</td>
<td>1 APB</td>
<td>260</td>
<td>His or distal</td>
<td>2 VPB</td>
</tr>
<tr>
<td>C* 42/M</td>
<td>1 APB</td>
<td>320</td>
<td>A-V-node-His</td>
<td>2 APB, 2 VPB</td>
</tr>
<tr>
<td>D* 28/M</td>
<td>1 APB</td>
<td>295</td>
<td>His or distal</td>
<td>2 VPB</td>
</tr>
<tr>
<td>E 27/F</td>
<td>1 APB</td>
<td>300</td>
<td>A-V-node-His</td>
<td>2 VPB</td>
</tr>
<tr>
<td>F* 20/M</td>
<td>1 APB</td>
<td>235–260</td>
<td>His or distal</td>
<td>3 APB, 2 VPB</td>
</tr>
<tr>
<td>G 34/M</td>
<td>1 APB</td>
<td>280</td>
<td>His or distal</td>
<td>2 APB, 2 VPB</td>
</tr>
<tr>
<td>H 48/M</td>
<td>1 APB</td>
<td>270</td>
<td>A-V-node-Atrial</td>
<td>3 APB, 2 VPB</td>
</tr>
<tr>
<td>I 41/F</td>
<td>1 APB</td>
<td>305</td>
<td>His or distal</td>
<td>2 VPB</td>
</tr>
<tr>
<td>J 36/M</td>
<td>1 APB, 1 VPB</td>
<td>290</td>
<td>His or distal</td>
<td>2 VPB</td>
</tr>
</tbody>
</table>

\(^a\) Patients A, B, C and D have been reported previously.

\(^b\) Abbreviations: Pt = patient; init = initiation; T = tachycardia; R-R interval = interval during T at time of block; Term = termination; APB = atrial premature beat; VPB = ventricular premature beat.

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**Figure 1.** Patient F. Top panel) Initiation of tachycardia by a single atrial premature beat given after 290 msec during atrial pacing with a basic cycle length of 600 msec. During tachycardia a 1 to 1 relation between atrial and ventricular activation is present. Lower panel) An atrial premature beat given after 200 msec is followed by tachycardia showing a 2 to 1 relation between atrial and ventricular activation. The His bundle recording shows that the site of 2 to 1 block was either in the bundle of His distal to the recording site, or more distally in the bundle branch system. Note that during tachycardia alternation in cycle duration between subsequent His bundle activation is present, and that the atrial rate during tachycardia in the lower panel is higher than in the top panel.
in the interval range 220 to 200 msec however initiated a tachycardia showing a 2 to 1 relation between atrial and ventricular activation (lower part of fig. 1). Atrial premature beats given in the interval range 240 to 220 initiated a tachycardia showing a Wenckebach second degree block in the His-Purkinje system distal to the site of His bundle recording (fig. 2).

In all four patients premature beats given up to a certain interval initiated a tachycardia with a 1 to 1 relation between atrial and ventricular activation, while premature beats given at shorter intervals resulted in tachycardia with block. As shown in figure 1 when second degree block occurred during tachycardia, the atrial rate during tachycardia was always higher than in the absence of block.

In three patients (B, E, and H) the tachycardia, which was initiated by a single atrial premature beat always showed at first a 1 to 1 relation between atrial and ventricular activation, to be followed, 15 to 60 beats later, by the sudden development of 2 to 1 block. An example is given in figure 3.

**Site of Block**

The table lists the most likely site of block in our patients. As shown, in four patients block was located distal to the site of the catheter recording from the His bundle, suggesting either block within the bundle of His or block distal to the bundle of His. In three patients block was located in the A-V node. In one patient (patient H) the location of 2 to 1 V-A block during tachycardia was either between the
bundle of His and the A-V node, in the A-V node, or between the A-V node and the atrium.

**Termination of Tachycardia**

As shown in the table in all patients the tachycardia could be terminated by appropriately timed atrial and/or ventricular premature beats. Not surprisingly, in view of the high rate during tachycardia at least two closely spaced premature beats were needed to terminate tachycardia.8

**Induction of Block during Tachycardia by Premature Stimuli**

In two of the 67 patients (I and J) two closely coupled ventricular premature beats given during tachycardia with a 1 to 1 relation between atrial and ventricular activation created refractoriness in the specific conduction system, distal to the bundle of His. As shown in figure 4 tachycardia persisted. Figure 4 illustrates the mechanism responsible for block distal to the bundle of His. The upper part of the figure suggests the possibility of block in the right bundle branch and the anterior fascicle of the left bundle branch following the two induced premature beats. The record shown in the lower part of the figure suggests additional block in the posterior fascicle of the left bundle branch. It cannot be excluded however that the beat following the two paced beats shown in the upper part of figure 4 is caused by local re-entry in the ventricle. It is of interest that the induced premature beats were not able to engage the His bundle retrogradely. The explanation for this phenomenon was given by studying V-A conduction during ventricular pacing. Following ventricular premature beats during ventricular pacing considerable delay in V-A conduction was found to occur in between the beginning of ventricular activation and retrograde activation of the bundle of His.

**Discussion**

As pointed out by Pick et al.4 in the absence of digitalis excess, myocarditis, or coronary heart disease, clinical examples of second degree block toward atrium or ventricle during reciprocal A-V junctional tachycardia have rarely been published.3,5

It is of interest that two-to-one block to the ventricle was documented during a spontaneous attack of tachycardia in only one of our patients (patient C), in spite of the fact that several recordings of spontaneous attacks of tachycardia were available from all patients. This suggests that although block during reciprocal A-V nodal tachycardia can be seen during the stimulation study in approximately 15% of patients (10 out of 67), their true occurrence during spontaneous attacks of tachycardia is probably rare.

**Site of Block**

In nine of the ten patients block developed between the intranodal re-entry circuit and the ventricle. In three patients block was situated in the A-V node distal to the re-entry circuit or between the A-V node and the bundle of His. In the remaining six patients block occurred either within the bundle of His or distal to it. In two of these six patients it was produced by giving two ventricular premature beats during tachycardia. In one patient block was located between the re-entry circuit in the A-V node and the atrium.

**Mechanism of Block**

It is of interest that in four patients the development of block during tachycardia was related to the prematurity of the tachycardia initiating premature beat. One can only speculate upon possible mechanisms responsible for this
phenomenon. One might suppose that part of the delay following the very early premature beats occurred proximal to the re-entry circuit, resulting in later arrival at the circuit and faster transmission time through the circuit. The shorter re-entry cycle would lead to a faster rate during tachycardia. Or, in view of the observations by Moore and Touboul et al., one could postulate that the site of slow conduction and the re-entry circuit might vary depending upon the prematurity of the test pulse. These differences in site and length of the re-entry circuit could result in differences in frequency during tachycardia. Under both circumstances block during tachycardia must occur outside the re-entry circuit, in an area with a refractory period longer than the transmission time through the re-entry circuit. Block would tend to be perpetuated because the length of the refractory period in the area of block and distal to it will be related to the frequency present there. Three patients showed the development of block following a “spontaneous” increase in rate during tachycardia. In two patients block occurred between the re-entry circuit and the ventricle, in the other between the re-entry circuit and the atrium. Again apparently structures outside the re-entry circuit must have had a longer refractory period than those components of the circuit itself. In two patients, refractoriness distal to the re-entry circuit could be created by giving two closely coupled ventricular premature beats during tachycardia.

Significance of Second Degree Block During Reciprocal Supraventricular Tachycardia

Evidence has recently been published that in patients suffering from reciprocal supraventricular tachycardia, who never showed signs of pre-excitation, an accessory pathway may be operative in ventriculo-atrial direction only. Since both the functional properties of the constituents of the tachycardia pathway and its location are of importance as far as treatment of the tachycardia is concerned, a correct decision as to whether an accessory A-V pathway is incorporated in the tachycardia circuit is of more than theoretical interest. The finding of persistence of tachycardia in spite of block in the A-V node, between the A-V node and the bundle of His, in the bundle of His, or block in both main bundle branches excludes a tachycardia circuit consisting of atrium → A-V node → bundle of His → bundle branches → ventricle → accessory pathway → atrium. Theoretically it does not exclude the possibility of a tachycardia circuit composed of atrium → A-V node → atrial-nodal fiber (James fiber) → atrium. In the absence of a short P-R interval and findings suggesting A-V conduction over an atrio-nodal fiber during atrial stimulation, such a fiber should then only be operative in the retrograde direction.

Persistence of tachycardia following the development of ventriculo-atrial block (in the A-V node, or between the A-V node and the atrium, as in patient H) also excludes a tachycardia circuit incorporating an accessory pathway connecting atrium and ventricle. Theoretically the possibility remains that in such a patient a tachycardia pathway is present composed of bundle of His → bundle branches → ventricle → fasciculo-ventricular fiber (Mahaim fiber) → bundle of His. It is of interest that the finding of persistence of tachycardia following the development of V-A block probably excludes in this patient the atrium as being a necessary link in the A-V nodal re-entry circuit.

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