Paroxysmal Supraventricular Tachycardia
Is the Atrium a Necessary Link?

MARK E. JOSEPHSON, M.D., AND JOHN A. KASTOR, M.D.

SUMMARY Whether or not the atrium plays an essential role in initiating and/or sustaining atrioventricular (A-V) nodal re-entrant tachycardia was evaluated in eight patients. In all eight patients, the atrium could be rendered refractory to retrograde atrial echoes during the tachycardia without interrupting the arrhythmia. This was accomplished by introducing atrial premature depolarizations prior to the time the atrium would normally be retrogradely depolarized by atrial echoes. In one patient, two atrial premature depolarizations could be introduced, producing A-V dissociation, without terminating the tachycardia. In another patient, the tachycardia could be initiated without an atrial echo.

Our data suggest that most, if not all of the atrium is unnecessary for the initiation and maintenance of A-V nodal re-entrant supraventricular tachycardia.

MOST CASES of paroxysmal supraventricular tachycardia are initiated and sustained through re-entry within the atrioventricular (A-V) node. Whether or not the atria form a portion of the re-entrant pathway remains unsettled. The present investigation of eight patients demonstrated that in each case no portion of the atrium recorded by our electrode catheters was required to sustain A-V nodal re-entrant supraventricular tachycardia and in one the atrium did not play an essential role in initiating the arrhythmia.

Methods, Materials, and Clinical Patient Information
Eight patients were studied in the nonsedated post-absorptive state after informed consent was obtained (table 1). All had symptomatic supraventricular tachycardia (SVT), and none demonstrated evidence of pre-excitation. No patient was taking antiarrhythmic drugs at the time of the study.

A quadripolar electrode catheter was introduced percutaneously into an antecubital and/or femoral vein and positioned under fluoroscopic control against the lateral wall of the high right atrium and/or the coronary sinus. The proximal pair of electrodes was used to record a high right atrial or coronary sinus electrogram, while the distal pair was used for atrial stimulation. A bipolar electrode catheter

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was similarly introduced into another antecubital vein and positioned in the right ventricular apex for stimulation. A His bundle electrogram was obtained using a tripolar electrode catheter introduced percutaneously into the right femoral vein. The intracardiac electrograms were filtered at 40 and 500 Hz and were simultaneously displayed with at least three surface ECG leads and timelines generated at 10 and 100 msec on a multichannel oscilloscope (Electronics for Medicine DR-16). Rectangular impulses of 1 msec duration and at approximately twice diastolic threshold were delivered by a specially designed programmed digital stimulator (Murray Bloom, Narberth, Pa.). All data were stored on magnetic tape (Honeywell 14 channel recorder) and later retrieved on photographic paper at speeds of 150 to 200 mm/sec.

In each patient, after initiation of A-V nodal re-entrant tachycardia, atrial premature depolarizations were introduced to activate the atria prior to the time they would have been retrogradely activated by atrial echoes during the tachycardia. In addition, refractory periods were determined by the extrastimulus method.

Results

All patients were in sinus rhythm at the beginning of the study and no abnormalities of conduction were present (table 1). In each patient, single atrial premature beats at coupling intervals ranging from 255-380 msec initiated the tachycardia. The echo zones1, 2 were 10 to 120 msec in duration. The critical A-H interval required for the initiation of the tachycardia ranged between 140 and 400 msec.3 The tachycardia could also be started in each patient during Wenckebach cycles produced by rapid atrial pacing,4 and in two patients (E.H. and H.H.), by one or more ventricular premature beats.

In all patients, atrial premature depolarizations could be introduced at intervals resulting in atrial depolarizations 10 to 130 msec prior to the time of spontaneous retrograde atrial activation without interrupting the tachycardia or altering its cycle length. In three patients, both right and left atrial (coronary sinus) sites were prematurely depolarized without influencing the tachycardia. Examples are shown in figures 1-3. Note in particular that the low right atrium is depolarized: a) 30 and 20 msec early in beats 2 and 4 of figure 1; b) 25 msec early in beat 3 of figure 2; c) 70 msec early in beat 3 of figure 3A and 100 msec early in beat 3 of figure 3B. Furthermore, the coronary sinus is depolarized 120 msec early in figure 3A and 55 msec early in figure 3B. The configuration of the high and low atrial electrograms were different in the atrial premature depolarizations (Ap) when compared to the atrial echoes (Ae). In addition, the atrial electrograms of the premature depolarizations introduced from the right or left atrium were recorded before the onset of the His bundle deflection in the patient illustrated in figure 3. Thus in each of these three cases, that part of the atria giving rise to the high right atrial electrogram, low right atrial electrogram (in the His bundle recording), and in one case the coronary sinus electrogram, could be prematurely depolarized without interrupting the tachycardia. Since the atrial refractory periods of our patients were 210-300 msec, the atria would have been refractory for at least 200 msec after Ap, during which time Ae would have appeared (10 to 130 msec after the atrium was prematurely depolarized).

In patient K.M., two premature atrial depolarizations (Ap) could be introduced which depolarized both the high and low right atrium without terminating the tachycardia (fig. 4). In addition, after the two stimulated atrial

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**Table 1. Clinical Data**

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<th>Pt/Age/Sex</th>
<th>Dx</th>
<th>PR*</th>
<th>QRS*</th>
<th>AH*</th>
<th>HV*</th>
<th>A-H required to initiate SVT*</th>
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<td>K.M./27/F</td>
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<td>70</td>
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<td>140</td>
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<td>90</td>
<td>92</td>
<td>45</td>
<td>290</td>
</tr>
</tbody>
</table>

*All values are given in msec.

Abbreviations: ASHD = atherosclerotic heart disease; MVP = mitral valve prolapse; IMI = inferior myocardial infarction.

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**Figure 1. Atrial stimulation during supraventricular tachycardia in patient K.M.** This figure and all subsequent intracardiac recordings are similarly organized. From top to bottom are ECG leads I, II, III, V1; high right atrial electrogram (HRA), His bundle electrogram (HBE), and timelines (T) generated at 10 and 100 msec. During supraventricular tachycardia (SVT), which has a stable cycle length of 340 msec, each QRS is associated with atrial echoes (Ae) reflecting retrograde depolarization of the atria. Note that the atrial electrogram of the Ae in the HBE precedes that in the HRA. Atrial premature beats (Ap) are introduced at a coupling interval of 225 msec after the first and third QRS complexes. In the Ap, the HRA precedes the atrial electrogram in the HBE and both occur earlier than they would during the tachycardia. Despite the fact that the atrium is depolarized prior to the time the Ae would normally occur, the tachycardia continues its cycle length unaffected by the stimulated atrial beats. S represents the stimulus artifact.
premature depolarizations, there is a pause in atrial activity followed by what appears to be a sinus P wave (A<sub>a</sub>). A<sub>a</sub> does not conduct because the A<sub>a</sub>H interval of 40 msec is too short (AH during sinus rhythm was 70 msec). Thus a period of A-V dissociation resulted without terminating the tachycardia. That A<sub>a</sub> arises in the area of the sinus is supported by: a) the sequence of atrial activation (high right atrium activated prior to low right atrium), and b) the normal appearance of the P wave seen in the surface ECG (heavy arrow, fig. 4). The pause and transient change in ventricular cycle length developed because the first Ap penetrated the A-V node and depolarized the ventricles while the second Ap did not penetrate the A-V node. The tachycardia continued even though no atrial echoes (Ae) were seen following the fifth and sixth QRS complexes.

In patient W.P., supraventricular tachycardia was initiated in the absence of an atrial echo (Ae) (fig. 5). On other occasions, an atrial echo was seen following the stimulated atrial premature depolarization (A<sub>a</sub>) if the resulting A<sub>a</sub>H interval exceeded 200 msec. This was the only patient we observed in whom no atrial echo followed the atrial premature depolarizations initiating the tachycardia. In two of the eight patients, A-V nodal refractory curves were consistent with "dual pathways."16

**Discussion**

The purpose of these studies was to determine whether the atria form part of the re-entrant circuit in patients with A-V nodal re-entrant tachycardia.1-9 In an attempt to answer this question, premature atrial depolarizations were introduced during the tachycardia prior to the time the atria would have been retrogradely activated during the tachycardia. Per-

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**Figure 2.** Atrial stimulation during supraventricular tachycardia in patient M.B. The panel is organized as in figure 1. Supraventricular tachycardia is present at a cycle length of 470 msec. After the third QRS an atrial premature beat (Ap) is introduced at a coupling interval of 400 msec. In the Ap, the HRA precedes the atrial depolarization in the His bundle electrogram (HBE), a reversal of the sequence of the atrial echo (Ae). Despite this, the tachycardia is unaffected. Also note the change in the atrial electrograms produced by Ap.

**Figure 3.** Atrial stimulation during supraventricular tachycardia in patient E.H. Panels A and B are organized from top to bottom leads I, aV<sub>1</sub>, V<sub>1</sub>, high right atrial electrogram (HRA), coronary sinus electrogram (CS), His bundle electrogram (HBE) and timelines (T). Supraventricular tachycardia (SVT) is present at a cycle length of 370 msec in each panel. Note that during supraventricular tachycardia, the atrial electrograms in the HBE, HRA and CS follow the onset of the His deflection. In panel A, an atrial premature beat (Ap) is stimulated from a catheter in the distal coronary sinus 250 msec after the prior CS electrogram. This results in depolarization of both the high and low atrium, as well as that part of the left atrium recorded by the CS electrogram prior to the inscription of the His bundle, without altering the H-H interval or cycle length of the supraventricular tachycardia. Thus both atria were depolarized 70-120 msec prior to the time Ae would have occurred without altering the SVT. In panel B, an Ap from the high right atrium is introduced 240 msec after the prior high right atrial electrogram. The high and low right atrial and coronary sinus electrograms appear 130, 100, and 55 msec premature respectively without altering the SVT.
Figure 4. Atrial premature beats causing A-V dissociation during supraventricular tachycardia in patient K.M. During SVT, two Ap's are introduced depolarizing both the high and low atrium prematurely without terminating the tachycardia. The next spontaneous atrial depolarization (As) has the appearance of a sinus beat both by its surface ECG appearance (heavy arrow) and the sequence of atrial activation. Clearly the HRA precedes the atrial electrogram in the HBE as it does in sinus beats. A period of A-V dissociation is present since As occurs almost simultaneously with the His deflection and could therefore not be responsible for it. Thus, no Ae is seen after the fifth and sixth QRS complexes, and in spite of transient A-V dissociation, the SVT continues.

The Route of A-V Nodal Re-entry

The physiologic basis of A-V nodal re-entry depends on the presence of longitudinal dissociation of the A-V node into two pathways: one with fast conduction and a long refractory period (beta pathway), and the other with slower conduction but shorter refractoriness (alpha pathway). Sustained tachycardias develop when atrial premature depolarizations are blocked in the beta pathway but conduct sufficiently slowly down the alpha pathway so that the impulse can retrogradely activate the previously refractory beta pathway returning through atrial tissue back to the alpha pathway to complete the circuit. This concept proposed by Moe and Mendez included the atrium as a necessary link in the re-entrant circuit.

The data obtained in this study suggest that most, if not all, of the atria are uninolved in the re-entrant circuit. In each case, both the high and low right atrium could be depolarized without altering the tachycardia. In addition, the left atrium could be “captured” without influencing the tachycardia in three cases, suggesting that the demonstration of atrial “capture” without altering the tachycardia was unrelated to the direction of the wavefront of the premature atrial depolarization. Furthermore, A-V dissociation during the tachycardia was produced in one patient (fig. 4), and an atrial echo was not required to initiate the tachycardia in another (fig. 5). These findings suggest that none of the recorded atrial tissue was required to sustain the arrhythmia. If any atrial tissue is necessary, it may be a very small rim immediately adjacent to the A-V node that was not recorded by our catheters. Resolution of this question must come from appropriate intra-atrial mapping and stimulation during open heart surgery in patients with A-V nodal re-entrant tachycardia.

Alternative Explanations

One could postulate that each premature atrial depolarization was conducted down the antegrade limb of the re-entrant circuit and fortuitously failed to alter the cycle length of the tachycardia due to an exact “compensatory” delay of conduction down that limb. However, premature atrial depolarizations could be delivered over a range of 20–50 msec without changing the tachycardia. Some irregularity of cycle length should have been noted during the scanning interval. Furthermore, in the presence of premature capture of the low right atrium by as much as 100 msec (fig. 3B), it seems very unlikely that the impulse would not have altered the cycle length of the tachycardia.

Fortuitous atrial participation certainly could not explain the findings noted in figure 4 in which two successive premature depolarizations capture the atrium without interrupting the tachycardia. Although the first Ap could have and probably did capture the ventricle, the second could not possibly have penetrated the re-entrant circuit since it had just been depolarized by the first Ap and would have been refractory. The persistence of the tachycardia after two Ap’s, despite the altered cycle length, the absence of echoes, and subsequent period of A-V dissociation, argues strongly against fortuitous atrial participation.

In one patient, W.P., the tachycardia was initiated in the absence of an atrial echo following the premature beat (fig. 5). This phenomenon, not previously described, strongly implies that the atrium was not necessary to initiate the tachycardia. Superficial examination of the record might lead one to suggest that retrograde atrial depolarization did...
occur but that the atrial electrogram was "buried" in the ventricular electrogram. This conclusion is unlikely for three reasons. First, during the tachycardia the atrial electrogram clearly appears before each QRS, and would therefore certainly be present in that position for the initiation. Second, if atrial depolarization did occur and was buried within the ventricular electrogram, one would have to assume simultaneous intra-atrial block preventing it from propagating to the high right atrium since there is no evidence of activity at that site. No intra-atrial block or latency was seen at similar coupling intervals during premature stimulation and these phenomena are less likely to occur in this situation since the introduction of $A_2$ would shorten atrial refractoriness for the subsequent beat. Third, with a greater increase in the $A_2H$ interval, the atria had time to recover and an $Ae$ was demonstrated at the onset of the tachycardia. Thus evidence for "hidden" atrial depolarization after $A_2$ is lacking.

**Figure 5. Initiation of supraventricular tachycardia without a critical atrial echo.** The panels are organized as in figure 1, but in addition, the top panel has a ladder diagram underneath depicting the mechanism of the arrhythmia. In the ladder diagram and top panel, $A_1$ is eighth of a series of paced atrial beats at a cycle length of 700 msec. $A_2$ represents the premature beat. In the ladder diagram, $A$ is atrium, AVN is A-V node, HPS is His-Purkinje, and $V$ is ventricle. $A_2$ is introduced at a coupling interval of 300 msec and SVT is initiated. However, no atrial echo is seen during or after the stimulated beat, but is present in subsequent beats of the SVT. As depicted in the ladder diagram, re-entry occurs within the A-V node; the atrium is not depolarized initially because it is still refractory. The lack of an initial $Ae$ strongly suggests that the $Ae$ is not necessary to initiate SVT. In panel opposite, $A_2$ is delivered at a coupling interval of 300 msec after the eighth paced beat at a cycle length of 600 msec. The resulting $A_2H$ of 220 msec is long enough to allow atrial refractoriness to recover and atrial echo to resume.

**Therapeutic Implications**

Clinical practice has established that drugs and maneuvers which prolong A-V nodal conduction can interrupt and prevent episodes of supraventricular tachycardia. Electrophysiologic studies have demonstrated that digitalis, propranolol, Valsalva maneuver, and carotid sinus pressure prolong A-V nodal conduction and refractoriness, thereby altering the temporal relationships within the A-V node that must be established to initiate and sustain supraventricular tachycardia. Quinidine and procainamide, which are less useful in the treatment of supraventricular tachycardia, have minimal effects on A-V nodal conduction and tend to shorten A-V nodal refractoriness by their vagolytic properties. Similarly, because of its lack of effect on the A-V node, lidocaine is of little value in the management of supraventricular tachycardia. However, quinidine and procainamide are useful in preventing supraventricular tachycardia by suppressing the atrial or ventric-
ular premature beats that initiate the arrhythmia.

It has been recently demonstrated that extra-nodal bypass tracts exist and can function as part of the retrograde limb of the re-entrant circuit, even in the absence of their utilization in an antegrade fashion.16-18 Demonstration that the atrium is not required to sustain supraventricular tachycardia argues against the presence of functional bypass tracts during the tachycardia. From a pharmacologic standpoint, if such retrograde bypass tracts and atrial tissue formed part of the re-entrant pathway, drugs affecting atrial and bypass conduction and refractoriness, i.e., quinidine and procainamide, would be useful.14, 15

Occasionally, permanent atrial or ventricular pacing is employed to treat patients whose supraventricular tachycardia is refractory to drug therapy.21, 22 The success of pacemaker therapy in supraventricular tachycardia depends on the ability of the induced atrial or ventricular beat to penetrate the re-entrant circuit and interrupt the arrhythmia. Infrequently, the use of single atrial beats is ineffective; the atrium may be depolarized without terminating the arrhythmia. In such cases, lack of atrial requirement during supraventricular tachycardia may explain the lack of success.

Evidence from Animal Experiments

Whether or not the atrium forms a requisite link in sustaining atrial echoes, supraventricular tachycardia, or ventricular echoes has not been resolved in the animal laboratory. The original mechanism of supraventricular tachycardia described by Moe and Mendez was included the atrium and a longitudinally dissociated A-V node to form the re-entrant circuit. More recently, Wit et al showed that in the isolated rabbit heart, a small but significant portion of the right atrium was required to sustain supraventricular tachycardia. However, Janse and co-workers, using a similar preparation with a plaque electrode over the A-V node, came to the opposite conclusion, i.e., that re-entry was entirely subaortic, located entirely in the A-V node. Both these latter studies demonstrated that infranodal structures were unnecessary to sustain supraventricular tachycardia. Findings in studies attempting to evaluate the role of the atrium in the production of ventricular echoes have contributed to the controversy. Using the technique of "atrial pre-excitation," Mignone and Wallace found that ventricular echoes resulted from re-entry within the A-V node and did not require the atrium. Mendez and Moe came to the opposite conclusion in the isolated rabbit heart. Thus, several carefully designed animal studies have failed to resolve the question of atrial requirement in these re-entrant arrhythmias.

Conclusion

Our data strongly suggest that in certain patients with A-V nodal re-entrant supraventricular tachycardia, no part of the recorded atrium is required to sustain or initiate A-V nodal re-entrant tachycardia. The demonstration of lack of the atrial requirement could be shown in the absence of A-V nodal refractory curves demonstrating "dual pathways."23 It therefore seems likely that, in these cases, the basic re-entrant circuit is limited to the A-V node and that the atrial and ventricular responses during supraventricular tachycardia are "secondary" phenomena. The finding of lack of atrial requirement in a patient with paroxysmal supraventricular tachycardia suggests the absence of a functional extra-nodal bypass tract and directs pharmacologic therapy toward those drugs which have a major action on A-V nodal conduction and/or refractoriness. Since the atria can be captured without terminating the tachycardia, an explanation is now available for the occasional inability to terminate supraventricular tachycardia with induced atrial premature beats.

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