Paroxysmal Supraventricular Tachycardia

Is the Atrium a Necessary Link?

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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.1-3 Whether or not the atrium 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 reentrant 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 postabsorptive 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 quadrupolar 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
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.\textsuperscript{4} 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.

\section*{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 zones\textsuperscript{1, 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.\textsuperscript{4} The tachycardia could also be started in each patient during Wenckebach cycles produced by rapid atrial pacing,\textsuperscript{3} 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 I–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

\begin{table}[h]
\centering
\caption{Clinical Data}
\begin{tabular}{|l|c|c|c|c|c|}
\hline
Pt/Age/Sex & Dx  & PR* & QRS* & AH* & HV* \\
\hline
K.M./27/F  & MVP & 125 & 75 & 70 & 50 & 400 \\
H.H./66/M  & ASHD & 140 & 90 & 95 & 35 & 170 \\
M.B./17/F  & MVP & 165 & 65 & 90 & 40 & 145 \\
W.P./56/M  & ASHD & 160 & 82 & 78 & 45 & 140 \\
E.H./37/F  & MVP & 135 & 86 & 50 & 50 & 295 \\
M.U./53/M  & ASHD & 155 & 88 & 85 & 48 & 195 \\
J.R./18/F  & None & 148 & 90 & 80 & 45 & 250 \\
C.N./72/F  & ASHD & 160 & 90 & 92 & 45 & 290 \\
\hline
\end{tabular}
\end{table}

\*All values are given in msec.
Abbreviations: ASHD = atherosclerotic heart disease; MVP = mitral valve prolapse; IMI = inferior myocardial infarction.
premature depolarizations, there is a pause in atrial activity followed by what appears to be a sinus P wave (Aa). Aa does not conduct because the AaH 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 Aa 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 (Aa) if the resulting AaH 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.”

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. 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-
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.

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.6-8 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, 16

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 Mendez5 included the atrium and a longitudinally dissociated A-V node to form the re-entrant circuit. More recently, Wit et al.23 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,24 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 subatral, 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 Wallace25 found that ventricular echoes resulted from re-entry within the A-V node and did not require the atrium. Mendez and Moe6 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."26 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 extranodal 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.

References

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