Atrial Tachycardia Secondary to Sino-Atrial Node Reentry

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In one patient reproducible episodes of atrial tachycardia could be elicited via programmed atrial premature depolarizations. Evaluation of the electrophysiologic studies suggested that the most likely mechanism was sino-atrial reciprocation. Intravenous atropine produced a pronounced shortening of sino-atrial conduction time (200 msec, control; 70 msec, atropine) and sustained atrial tachycardia no longer could be elicited. These studies support the concept that sino-atrial reciprocation can be a mechanism responsible for atrial tachyarrhythmias in man.

In recent years a number of in vivo and in vitro experimental studies have attempted to clarify the electrophysiologic characteristics of normal and abnormal sino-atrial node function. Additional studies have been directed towards possible involvement of the sino-atrial node in paroxysmal atrial tachyarrhythmias and the effects of pathologic changes on sino-atrial node function. Most recently studies have been undertaken utilizing some of these techniques to define more clearly the electrophysiologic properties of the sino-atrial node in man. Analysis of data obtained from some of these latter studies have suggested that some atrial tachyarrhythmias in man may be due to reentry between the sinus node and surrounding tissue.

This report describes an electrophysiologic study in man which supports the concept of sino-atrial reentry as a cause of sustained atrial tachycardia.

CASE REPORT

The patient is a 65-year-old man who was admitted to the hospital for evaluation of cardiac arrhythmias (bradycardia alternating with atrial tachycardia with AV block). There was no history of cardiac drug usage. The patient’s electrocardiogram showed left bundle branch block, superior axis deviation and a normal PR interval. As part of the diagnostic evaluation, electrophysiologic studies were performed (see methods).

METHODS

The patient was studied in the cardiac catheterization laboratory in the resting, nonsedated, postabsorptive state. Under fluoroscopy two bipolar 5 FR pacing catheters were inserted via an antecubital vein and positioned at the superior vena cava-right atrial junction for the purpose of both atrial pacing and high right atrial electrogram recordings. A 6 FR triplepolar catheter was introduced percutaneously via the right femoral vein and advanced to the septal leaflet of the tricuspid valve for the purpose of recording the His bundle electrogram. Three standard ECG leads were simultaneously recorded. All data were recorded on photographic paper at 100 mm paper speed with the use of a multichannel oscilloscope photographic recorder. Electrograms were filtered at 40 to 500 Hz.

Stimulus characteristics were as follows: (1) rectangular pulses, 10 times the diastolic threshold and 2 msec in duration were used for the basic driving stimuli; (2) extrastoles were initiated which utilized similar stimulation characteristics but with pulses of two times the diastolic threshold.

Records were obtained during sinus rhythm and following atrial pacing at increasing rates. Subsequently, AV refractory records and sinus recovery sequences were obtained by using the extra-stimulus technique. The procedure was then repeated after the administration of atropine 1.0 mg IV.

The following intervals were measured:

2. A2-A2: interval between the last stimulated beat and the induced atrial premature depolarization (APD)
3. A2-A3: interval between the APD and the next occurring sinus beat
4. H1-H2: interval between the His spike of last paced beat and the His spike of the APD
5. A2-H2: interval between the low right atrial depolarization of the APD and the resulting His bundle spike.

The A1-A2 and A2-A3 intervals were then expressed as a percent of the A1-A1 interval and plotted as shown in Figure 1. In addition, A1-A2 and H1-H2 intervals were then used to construct curves for the functional and effective refractory periods of the AV node as shown in Figure 2.

RESULTS

As the A1-A2 interval was progressively shortened in the range of 100 percent to 60 percent, the A2-A3 interval remained stable. However, once the A1-A2 interval reached less than 60 percent, the A2-A3 interval shortened significantly (Fig 1); with further decrements in the A1-A2 interval there was additional shortening of the A2-A3 interval. With an A1-A2 interval of ≤ 40 percent a sustained tachycardia was produced (Fig 1, 3) with the initial A2-
A CONTROL

A real-time display of the A1-A2 interval (horizontal axis) and the A2-A3 interval (vertical axis). Both intervals are expressed as a percentage of the basic sinus cycle length. The left-hand panel (A) shows the values obtained in the control state. Coupling intervals of 100 percent to 65 percent produced a relatively constant A2-A3 response. At A1-A2 coupling intervals of 80 percent to 40 percent a substantial reduction in the A2-A3 response was observed; the shortest A1-A2 interval resulted in sustained tachycardia. The right hand panel (B) shows the values obtained following intravenous atropine administration. At A1-A2 coupling intervals of 90 percent to 45 percent a relatively stable A2-A3 response was observed. Shorter coupling intervals resulted in marked reduction in the resultant A2-A3 intervals but without sustained tachycardia. (See text)

A3 interval of 42 percent. The P wave morphology was similar to that observed in sinus rhythm and the high-low atrial activation sequence preserved; there was no evidence of AV nodal reentry (Fig 2). Repeated stimulation sequences produced identical results with similar coupling intervals.

Following administration of atropine (Fig 1) the range through which the A1-A3 was diminished and for which a relatively stable A2-A3 interval was maintained was 100 percent to 50 percent. When the A1-A2 interval was further shortened (≤ 45 percent) a marked decrease in A2-A3 was again noted. With an A1-A2 interval of 37 percent and 35 percent similar though not sustained episodes of tachycardia (nine and four beats, respectively) resulted (Fig 1). Moreover, atropine administration, as com-

![Graphs relating the A1-A2 interval (horizontal axis) and the A2-A3 interval (vertical axis). Both intervals are expressed as a percentage of the basic sinus cycle length. The left-hand panel (A) shows the values obtained in the control state. Coupling intervals of 100 percent to 65 percent produced a relatively constant A2-A3 response. At A1-A2 coupling intervals of 80 percent to 40 percent a substantial reduction in the A2-A3 response was observed; the shortest A1-A2 interval resulted in sustained tachycardia. The right hand panel (B) shows the values obtained following intravenous atropine administration. At A1-A2 coupling intervals of 90 percent to 45 percent a relatively stable A2-A3 response was observed. Shorter coupling intervals resulted in marked reduction in the resultant A2-A3 intervals but without sustained tachycardia.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/20958/)

![Effects of atrial premature depolarizations. The three panels show records obtained at progressively shorter coupling intervals (580, 455 and 255 msec, respectively). The tracings for each panel are, from above downward: a high right atrial electrogram (AEG), the His bundle electrogram (HBE) and standard leads 1, 2, and 3. The coupling interval, in milliseconds, is shown above the AEG. S1 and S2 signify the basic and test stimuli. The basic His bundle responses are labeled A1, H1, and V1 and the test responses are labeled A2, H2 and V2. The resultant return sinus beat is labeled A3. Note the AV nodal delay seen as the coupling interval shortened (panel B). In panel C, at a coupling interval of 255 msec, block was observed at the level of the AV node. A very short A3 response was seen with a resultant sustained tachycardia with variable AV block. The high-low atrial activation sequence, as observed during sinus rhythm, was preserved and P wave morphology was similar to the sinus beats.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/20958/)
Figur 3. A graph showing the relationship between the basic (A1) and test (A2) atrial coupling intervals (horizontal axis) as compared to, on the vertical axis, the resultant His bundle responses (H1, basic response; H2, test response). Responses plotted in the control state (filled circles) are compared to responses following atropine administration (open circles). Note the shift of the responses down and to the left following atropine administration. Sustained tachycardia was seen only in the control state. The shortest atrial coupling interval resulted in block in the AV node but is shown for timing purposes only.

pared to control, produced a shortened A2-A3 response for any given A1-A2 interval (Fig 1).

Data were plotted summarizing the results of programmed atrial premature stimuli before and after atropine. The AV refractory period plots shown in Figure 3 demonstrate an abbreviation of AV nodal refractoriness following atropine administration.

Discussion

Childers and co-workers, in their studies on sinus nodal echoes, describe potential responses to an atrial premature depolarization which include: sinus echo, complete or incomplete interpolation. A sinus echo is defined as an impulse which occurs earlier than the expected sinus (or sinus escape) beat in response to an atrial premature depolarization. Theoretic explanations for this phenomenon have been offered by Han and associates in studies with isolated right atrial preparations utilizing microelectrode techniques. They suggest that sinus node echoes or reentry result from atrial premature depolarizations which, arriving at one site of the SA node and finding this site refractory, enter the node via another site, traverse the node slowly and exit as an echo beat. Paulay and co-workers, studying sinus node reentry in vivo in the dog were able, in one dog, to produce multiple sinus echo beats. In addition, they demonstrated that there is a critical coupling interval of the atrial premature depolarization necessary to produce sinus node echoes. They could distinguish sinus node reentry from local, atrial or AV node reentry in that echo beats failed to occur when the sinus node was destroyed.

In addition, the studies of Strauss and co-workers and of Goldreyer and Damato have demonstrated that the coupling interval is of prime importance in determining whether or not an atrial premature depolarization can penetrate the SA node (SA entrance block, reset or not reset the sinus cycle) and result in complete or incomplete interpolation. Goldreyer and Damato showed that as an A1-A2 interval progressively shortened over a range of 100 percent to 70 percent the A2-A3 interval progressively increased (A1-A2 and A2-A3 expressed as percentages of A1-A1). With coupling intervals of 70 percent to 40 percent, a plateau in the A2-A3 response was observed and interpreted as maximum depression of the sinus node pacemaker. A further decrease in the A1-A2 interval (below 40 percent) produced an abrupt decrease in the A2-A3 interval and was considered to represent SA node entrance block. Strauss and co-workers have obtained similar data but have interpreted it in a somewhat different manner. These authors feel that atrial depolarizations with a 70 percent to 100 percent coupling interval (zone 1) do not enter the SA node, analogous to a compensatory pause seen with a premature ventricular systole. Shorter coupling intervals result in a plateau effect (zone 2) considered to represent premature activation with reset.

Furthermore, Strauss and co-workers have suggested that one can utilize data so plotted to calculate an approximate SAN-atrial conduction time; the range of calculated conduction time in their four patients was 68 to 156 msec. It is interesting that the calculated control conduction time in our patient during control conditions was 200 msec. Moreover, following atropine administration estimated conduction time diminished significantly (77 msec) and no sustained episodes of tachycardia could be elicited. This latter observation offers further credence to the concept of SA node reentry as the cause of the arrhythmia in our patient.

One must, however, consider the effect of atrial pacing on the calculated sinus to atrial conduction time. Although the basic drive rate was slow (90 per minute), it is conceivable that some overdrive effect could alter the calculated conduction time.

In the case presented here we have demonstrated a sudden decrease in the A2-A3 interval when the A1-A2 interval is sufficiently short. However, it appears that rather than being blocked at the SA node the atrial premature depolarization is, in fact, able to enter the node and produce a sustained atrial tachycardia presumably via sinus node atrial reentry. It has become apparent that there are certain necessary criteria for the establishment of an SA node reentry phenomena. These include: (1) that the P wave vector is directed inferiorly and the
morphology is similar to the P waves seen during sinus rhythm; (2) that there is a critical coupling interval between the premature depolarization which results in a short returning cycle; and (4) that there is no evidence of AV nodal reentry. AV nodal reentry was clearly not the mechanism in our patient. P wave morphology was similar to sinus rhythm and no initial retrograde atrial activation sequence was seen. Moreover, the A2 precipitating the tachycardia was apparently blocked proximal to the His bundle. It is conceivable that AV nodal reentry could occur without conduction to the bundle of His. Nevertheless, retrograde atrial activation should occur in a low-to-high atrial activation sequence. The tachycardias terminated spontaneously after periods ranging from 30 seconds to 2 minutes. Because of the short-lived nature of the rhythm disturbances no attempts were made to terminate the rhythm with atrial stimuli. It is possible that the A2 responsible for the start of this tachycardia entered the atrial relative refractory period resulting in repetitive firing. However, the stimulus to A2 interval was not significantly altered and the subsequent rhythm was regular (Fig 2c). Nevertheless, because of the rapid atrial rate and the prolonged calculated sinus-to-atrial conduction time one cannot completely exclude a reentrant arrhythmia occurring within the atria but outside the confines of the SA node.

In all of the previous studies only single reentry beats or at the most cyles of 3 to 5 systoles were produced in response to an atrial premature depolarization. In our report we have presented a case of sustained tachycardia which we believe is an example of SA node reentry in that all of the criteria for sino-atrial echo responses have been met.

In the past, SA node reentry has been considered as a mechanism for paroxysmal atrial tachyarrhythmias based on the production in experimental preparations of one or several echo beats following premature atrial depolarizations with short coupling intervals. This present report would appear to establish more clearly SA nodal reentry as a mechanism for paroxysmal atrial tachyarrhythmias in man.

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