Intraventricular Conduction Delay as a Determinant of Atrial Echo Beats

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SUMMARY

Re-entry within the atrioventricular (A-V) node is a common cause of atrial echo beats and paroxysmal atrial tachycardia. This report deals with observations made in four patients in whom the determining factor for atrial echo beats appeared to be a critical conduction delay occurring within the His-Purkinje system and not within the A-V node. The evidence in support of this conclusion is as follows: 1) at a constant A-V nodal delay, echo beats only occurred when a critical His-Purkinje delay was reached; 2) in the presence of different intraventricular conduction times, atrial echoes were dependent on a requisite His-Purkinje conduction delay; 3) at variable A-V nodal conduction times, echo beats appeared only when a requisite delay in His-Purkinje conduction developed; 4) at a constant premature coupling interval atrial echo beats could be abolished when refractoriness within the His-Purkinje system was decreased by decreasing the cycle length of the basic drive; and 5) at constant A-V nodal conduction times, a reciprocal relationship existed between the magnitude of the His-Purkinje delay and the "echo interval" defined as the interval between the onset of ventricular depolarization and the echo beat.

Additional Indexing Words:

Re-entry Conduction times His electrograms Atrial stimulation Reciprocation His-Purkinje system

RE-ENTRY WITHIN THE ATRIOVENTRICULAR (A-V) NODE has been demonstrated to be a common mechanism of atrial echo beats and paroxysmal supraventricular tachycardia.1 A critical delay in A-V nodal conduction appears to be the major factor in the genesis of both atrial echo beats and re-entrant supraventricular tachycardias.2,3 This report deals with observations made in four patients in whom the determining factor for atrial echo beats was a critical conduction delay occurring within the His-Purkinje system and not within the A-V node.

Materials and Methods

The first patient, a 64-year-old man, and the second patient, a 36-year-old man, were referred to our laboratory for cardiac catheterization procedures designed to evaluate chest pains not diagnostic of angina pectoris. The ECG of the first patient showed a right bundle branch block pattern and the ECG of the second patient was normal. Physical examination and chest X-rays were normal in both patients.

The third patient, a 47-year-old asymptomatic man, was referred because of an ECG demonstrating variable degrees of left bundle branch block pattern. Physical examination and chest X-ray were within normal limits. Cardiac catheterization including coronary arteriograms performed at another hospital were normal. The fourth patient was a 34-year-old female who was admitted to the hospital because of frequent attacks of palpitations. During these attacks she developed lightheadedness with occasional syncope, precordial pain radiating to the left arm, and fatigue. She had been tried on various antiarrhythmic drugs without success. At the time of study the frequency of the attacks was increasing. Physical examination, chest X-ray and the ECG between attacks were normal.

Electrophysiological Studies

All patients were studied in the postabsorptive, nonsedated state. The procedure was explained and an informed consent was obtained. The patients were not on any cardioactive drugs at the time of the study.

A quadripolar catheter was percutaneously inserted into a right antecubital vein and under fluoroscopic control positioned against the lateral wall of the right atrium near its junction with the superior vena cava. The proximal pair of electrodes was used to record a high right atrial electrogram and the two distal electrodes were used to stimulate the right atrium. A tripolar catheter was advanced via the right femoral vein to the tricuspid region and His bundle electrograms obtained as previously described.4 The right atrium was paced using stimuli of 1.5 msec duration at twice diastolic threshold. The atrial extrastimulus method was used to determine the refractory periods of the various components of the A-V conducting system both during sinus rhythm and at various paced atrial rates.5,6 The stimulator

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used in this study has digital circuitry and permitted stimuli to be introduced at an accuracy of 1 msec.

Electrocardiographic leads I, II, III, V1, a high right atrial bipolar electrogram, His bundle electrogram, and time marks generated at 10 and 100 msec were displayed on a switched-beam multichannel oscilloscope and recorded on magnetic tape. Analog tracings were subsequently transferred to photographic paper at paper speeds of 150 mm/sec. Electrograms were recorded at a frequency setting of 40-500 Hz and all equipment was carefully grounded.

Definition of Terms

A1 A2 — is the interval between atrial depolarization of either sinus or paced beat.

A1 A2 — is the interval between atrial depolarizations of either sinus or paced origin and a stimulated premature atrial beat.

A-H — the interval measured from the onset of the low atrial depolarization to the onset of the His deflection from the His bundle electrographic (HBE) recordings. This interval was used as a measure of A-V nodal conduction time.

H-V — the interval between the onset of the His deflection to the earliest onset of ventricular depolarization, as determined from either the surface electrocardiographic leads or the ventricular electrograms.

A2 H2 — is the A-H interval of the stimulated atrial premature depolarization.

H2 V2 — is the H-V interval of the stimulated atrial premature beat.

Results

All four patients demonstrated atrial echo beats following premature stimulation of the atrium. These echo beats appeared to be related to significant delays in His-Purkinje conduction and unrelated to delays in A-V nodal conduction. Figures 1 and 2 are representative tracings from patients 1 and 4, respectively. In both examples, atrial echo beats only occurred when the delay in His-Purkinje conduction (H2 V2 interval) was in excess of 200 msec (panel B of both figures). It is apparent that the atrial echo beats were unrelated to A-V nodal delay since the A2 H2 interval remained the same irrespective of whether an echo was present or not. In patients 1, 2, and 3, the critical His-Purkinje delay which resulted in an atrial echo beat always produced an aberrant LBBB pattern while in patient no. 4 atrial echo beats occurred with either a RBBB or LBBB aberrant pattern (see figure 4 below).

The finding that appearance of atrial echo beats was dependent on a requisite delay within the His-Purkinje system was further evaluated by studying the effect of changes in preceding cycle length (A1 A2 interval) on the echo phenomenon. Figure 3 is representative of these studies. In panel A, an atrial echo beat occurs when the H2 V2 interval is 250 msec. In panel B, the preceding cycle length was decreased to 750 msec which in turn decreased refactoriness within the His-Purkinje system as reflected in the fact that a premature atrial beat (A2) introduced at the same coupling interval (300 msec) as in panel A results in a less prolonged H2 V2 interval (160 msec). An atrial echo beat does not occur at the shorter H2 V2 interval. In panel C, the preceding cycle length was held constant at 750 msec and an atrial echo beat reappeared when the requisite His-Purkinje delay was achieved by decreasing the coupling interval of the premature atrial beat. In panel D the echo beat was again prevented from occurring by further decreasing the preceding cycle length to 650 msec.

In all cases a reciprocal relationship existed between the magnitude of the His-Purkinje delay and the "echo interval" defined as the interval between the onset of ventricular depolarization and the atrial echo beat. This relationship is illustrated in figure 4. In panel A, the "echo interval" is shorter (120 msec) and

![Figure 1](http://circ.ahajournals.org/)

The tracings from top to bottom in both panels are: electrocardiographic leads 1, 2, 3 and V1, a high right atrial electrogram (HRA). His bundle electrogram (HBE) and time lines (T) at 10 and 100 msec. The basic atrial cycle (A, A1) in both panels is the same at 850 msec and the sequence of atrial activation is high to low. The A-H and H-V intervals of the basic drive beats are 80 and 50 msec respectively. In panel A, a premature atrial beat (A2) introduced at an A2 H2 interval of 330 msec is aberrantly conducted to the ventricles (LBBB) with a prolonged H2 V2 interval of 160 msec. No echo beat occurs. In panel B, the A2 H2 interval is decreased to 300 msec. aberrant conduction to the ventricles occurs with an H2 V2 interval of 220 msec following which an atrial echo beat (A') occurs. Note the inverted P waves in leads 2 and 3 and the low to high sequence of atrial activation. Also, to be noted is the constant A, H2 interval of 115 msec in both panels. Similar abbreviations will be used for subsequent figures.

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atrial echo beats follow a and in instant within the preceding cycle A, B, shorter the right conduction patterns. In independent these panels occurred. to 280 rate dependent became and C, many episodes in incomplete A-V nodal cycle lengths (panels A to C), had arising in addition to one bundle branch for antegrade and the other for retrograde conduction because if this were the case one would expect the bundle branch block pattern to have persisted throughout the tachycardia.

Discussion

Re-entry can occur in any tissue in which there are areas of different conduction velocity and nonuniform recovery. In order for re-entry to occur, conduction through certain areas of tissue needs to be sufficiently delayed so that other parts of the tissue could recover and be re-excited by the delayed wavefront. This phenomenon has been demonstrated in in vitro studies of the sinus node, A-V node and Purkinje strands. Re-entry within the region of the sinus node and in the A-V node have been demonstrated in both experimental animals and man. Intraventricular re-entry has been postulated to be a mechanism for coupled extrasystoles, parasystolic rhythms, and certain types of ventricular tachycardia in man.

In most patients with paroxysmal supraventricular tachycardia (SVT), the site of re-entry can be localized to the A-V node by demonstrating that single echo beats and SVT are dependent on a critical A-V nodal delay. In previous studies of A-V nodal re-entry no other factor was found to be essential for the initiation or maintenance of the tachycardia in these patients. In contrast, in the present study, echo beats occurred only at a critical His-Purkinje conduction delay.

The evidence that in these four patients echo beats were dependent on a critical His-Purkinje delay and not dependent on a critical A-V nodal delay is as follows: 1) at a constant A-V nodal delay echo beats occurred only when a critical His-Purkinje delay was present (figs. 1, 2, and 3); 2) in the presence of different H-V values, atrial echoes were dependent on a requisite His-Purkinje conduction delay (fig. 3); 3) at variable A-V nodal conduction times, echo beats appeared only when a requisite delay in His-Purkinje conduction developed (fig. 4); 4) at a constant premature coupling interval, atrial echo beats could...
Figure 3
The effect of decreasing preceding cycle length on the echo phenomenon. In panel A, the basic cycle length \( (A_1, A_1) \) was 850 msec. \( A_1, A_2 \) was 300 msec and an atrial echo beat \( (A^*) \) followed an \( H_2 V_2 \) interval of 250 msec. In panel B, the \( A_1, A_2 \) coupling interval was the same (300 msec). As a result of decreasing the basic cycle to 750 msec, \( A_2 \) was conducted with a less prolonged \( H_2 V_2 \) interval (160 msec) and the echo beat was prevented from occurring. In panel C, the \( A_1, A_2 \) cycle length was held constant at 750 msec and \( A_1, A_2 \) was decreased to 270 msec. \( A_2 \) was conducted at an \( H_2 V_2 \) interval of 210 msec and an atrial echo occurred. In panel D, the \( A_1, A_2 \) interval was decreased to 650 msec and despite a shorter \( A_1, A_2 \) coupling interval, no echo occurred at an \( H_2 V_2 \) interval of 150 msec.

Figure 4
Panels demonstrate the reciprocal relationship between His-Purkinje delay and the "echo interval."
Figure 5
Tracings from patient 3 with rate dependent bundle branch block patterns. Panels A to C demonstrate that at basic cycle lengths of 600 to 800 msec a LBBB pattern was present and at a cycle length of 840 msec (panel D) an incomplete LBBB pattern occurred. A-V nodal delay (A2 H1 interval) up to 280 msec (panels A to C) was produced without the occurrence of an atrial echo beat. In panel D an atrial echo beat occurs following His-Purkinje delay of 200 msec.

Panels C and D of figure 6 illustrate how, subsequent to delay in the His-Purkinje system, the re-turning retrograde impulse, if delayed sufficiently, may reciprocate within the bundle of His (panel C) or A-V node (panel D) and thereby initiate a sustained supraventricular tachycardia. The QRS complexes of the supraventricular tachycardia may be either normal or aberrant. Since retrograde reciprocation within the A-V node is probably more common than reciprocation within the bundle of His, the mechanism depicted in panel C is favored as the more plausible explanation for the tachycardias noted in patient 4. However, recent studies using microelec-trode techniques have demonstrated that the bundle of His can also be a site of re-entry.

Reciprocation within the His-Purkinje system resulting in atrial re-excitation as proposed above requires that the bundle of His also be retrogradely re-excited. Failure to demonstrate the expected retrograde His potential in our four patients suggests that retrograde His activation may be occurring during the inscription of the ventricular electrogram and is obscured by it. Although in these four patients, atrial echo beats occurred in association with H-V intervals of 200 msec or greater, it is possible that atrial re-excitation can occur with normal H-V intervals provided that sufficient time has elapsed for the A-V node and atrium to recover. For example, if an atrial
premature beat conducts normally within one bundle branch (e.g., left bundle) and is antegradely blocked within the contralateral bundle (e.g., right bundle), the H-V interval will be within normal limits and aberrant ventricular activation will occur. If the contralateral bundle (the right bundle in this example) manifests unidirectional block, then atrial re-excitation can occur.

Understanding and localizing the site(s) of re-entry is not only of electrophysiological interest but may also have important therapeutic implications. Drugs such as procaine amide and quinidine could conceivably precipitate or increase the frequency of echo beats because of their ability to delay His-Purkinje conduction. It is also conceivable that these drugs, which at times are effective in abolishing or preventing A-V nodal re-entry, may shift the site of re-entry from the A-V node to the His-Purkinje system. Atrial pacing at rapid rates increases A-V nodal conduction time and may precipitate A-V nodal re-entrant tachycardia. On the other hand, rapid rates decrease refractoriness within the His-Purkinje system and this could abolish or prevent echo beats or re-entrant tachycardias which were dependent on a requisite His-Purkinje conduction delay.

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