Modulation of Conduction and Refractoriness in Atrioventricular Junctional Reentrant Circuit

Effect on Reentry Initiated by Atrial Extrastimulus

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Abstract

The importance of activation sequence of an atrioventricular junctional reentrant (AVJRe) circuit, before delivery of an extrastimulus, has received little attention in studies concerned with clinical tachycardias. In this study a change in activation sequence was accomplished using bidirectional activation (V-A sequential pacing) during the basic drive (V_1A_1-V_1A_1). It was noted that, compared with an atrial extrastimulus (A_2) after an atrial drive (A_1-A_1), earlier activation (by V_1 impulse of the V_1A_1-V_1A_1 drive) consistently improved conduction, or decreased refractoriness, or both, in the anterograde as well as the retrograde pathway of the AVJRe circuit. In all patients, five with AV nodal reentry and six with Wolff-Parkinson-White syndrome, reentrant tachycardia could be prevented during V-A sequential pacing. In four of eleven patients, reentry was prevented despite achieving the so-called critical atrioventricular nodal delays that had previously caused reentry during control study. This finding suggested that conduction delay necessary for reentry was related to the site of block, which in turn was affected by V-A sequential pacing.

We concluded that changing the activation sequence during basic drive modulates conduction and refractoriness in AVJRe circuits, and allows the study of a wide range of electrophysical factors that prevent or permit reentry.

Introduction

Programmed electrical stimulation has proved to be a singularly useful method for study of reentrant circuits (1–18). Since the original description of this method by Schmitt and Erlanger (1); the conduction characteristics of the premature beat have remained the primary focus of studies concerning reentrant circuits. What has received very little attention is the influence of activation sequence, during basic drive, on behavior of the premature beat. In a previous study, we observed the effect of bidirectional activation of the His-Purkinje system on conduction, refractoriness, and occurrence of bundle branch reentry (19, 20). Since conduction delay and conduction block also play a pivotal role in atrioventricular junctional reentrant

(AVJRe)[1] tachycardias (8–12), we hypothesized that if conduction and refractoriness were affected by changing the activation sequence of the AVJRe circuit during basic drive, then a new set of relationships would emerge between conduction delay, conduction block, and coupling interval of the extrastimulus. These observations could potentially result in a clearer understanding of electrophysiological factors that facilitate or prevent clinical tachycardias.

In this study, we describe the electrophysiologic sequelae of changing the activation sequence during basic drive, on the reentrant process, in both small (atrioventricular [AV] nodal reentry) as well as large (Wolff-Parkinson-White syndrome) clinical circuits.

Methods

Study population. Eleven consecutive patients (six females and five males) with recurrent AVJRe tachycardias, in whom the clinical tachycardia could be reproduced by atrial premature stimulation, constituted the study group. Of the eleven patients, five had AV nodal reentry and six had Wolff-Parkinson-White syndrome. The mean age of the patients was 46±13 yr. There was no evidence of underlying structural heart disease in any of these patients. Two patients, however, had a history of hypertension.

All patients were in sinus rhythm. Right heart catheterization was done in the nonsedated, postabsorptive state. The nature of the electrophysiological procedure was explained, and an informed and signed consent was obtained.

Electrophysiology study. Three to four quadripolar 6F catheters were introduced percutaneously through peripheral veins and fluoroscopically positioned in the high right atrium, AV junction, coronary sinus, and right ventricular apex for local bipolar recordings (filtered at 30–500 Hz) and electrical stimulation. In addition to the bipolar electrograms, three surface electrocardiographic leads (I, II, and VI) and time lines were displayed simultaneously on a multichannel oscilloscope (VR-16; Electronics for Medicine Inc., Pleasantville, NY) and recorded on magnetic tape (model 5600 C; Honeywell Inc., Medical Electronics Div., Pleasantville, NY) for subsequent retrieval on photographic paper at 100 mm/s. Intracardiac stimulation was performed with a Bloom DTU 101 digital stimulator capable of delivering premature stimuli after a basic drive of A-V sequential as well as ventriculoatrial (V-A) sequential pacing with adjustable A-V (or V-A) intervals.

Pacing protocol (Figs. 1–3). Initial refractory period studies were done according to previously described pacing methods (21). In general, after a basic atrial drive (A_1-A_1) (range, 400 to 700 ms), the diastolic interval was scanned with premature atrial beat (A_2) at progressively shorter A_1-A_2 intervals until the A_2 encountered atrial muscle refractoriness or resulted in repetitive atrial responses (control study, Fig. 1 *A*). After the zone of AV junctional reentry had been defined with the control method described above, the pacing protocol was repeated with the basic atrial drive (A_1-A_1) substituted by a V-A

Figure 1. Pacing protocol. *A* shows the control method, which consists essentially of scanning the diastole with A_2 after a basic drive (A_1-A_1). The retrograde preexcitation protocols are depicted in *B* and *C*. The ventricle and atrium are paced sequentially (V-A sequential pacing, V_1A_1-V_1A_1) and again the diastole is scanned with A_2. A_1-A_2 intervals and cycle lengths of V-A sequential drive V_1A_1-V_1A_1 are identical to that during control. In *C* the V-A interval is longer (compared with *B*), which results in greater retrograde preexcitation (see text, Figs. 2 and 3).
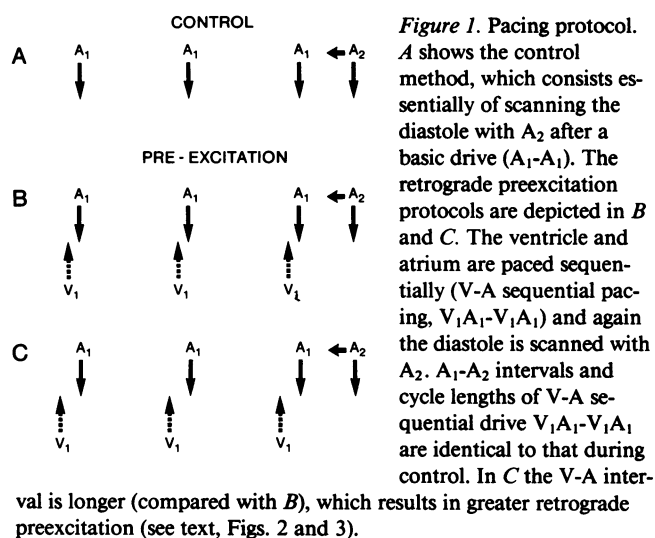
sequential drive (V_1A_1-V_1A_1) (Figs. 1, *B* and *C*) at the same cycle length (V-A sequential method). The initial V-A interval was arbitrarily selected as 0 ms. This was done to cause collision of the paced atrial and ventricular impulses in the AV node as well as in the accessory pathways, when present. Thus, when compared with the control method, the paced ventricular impulse during VA sequential drive caused earlier or preexcitation of segments of the reentrant circuits (i.e., AV node or accessory pathways) (see following paragraph, titled Definition of terms, and Figs. 2 and 3). After the basic V-A sequential drive (V_1A_1-V_1A_1), A_2 was introduced at progressively shorter coupling intervals, identical to those during control method, and the zone of AV junctional reentry was again determined (Figs. 1–3). The pacing protocol was then repeated with progressively longer (by 10–20 ms) V-A intervals programmed to analyze the effect of greater preexcitation of the AVJRe circuit (Fig. 1 *C*) until the zone of reentry was completely abolished.

Definition of terms

Anterograde refractory periods. A-H interval was measured from the initial low frequency deflection of the low atrial electrogram to the onset of His-bundle deflection.

CONTROL

Figure 2. Schematic representation of pacing protocol in patients with AV nodal reentry. The top shows conduction through the AV node (AVN) both during basic drive (A_1) and premature atrial stimulus (A_2). Note the schematic depiction of conduction through slow and fast AV nodal pathway along with unidirectional AV nodal block with A_2. During V-A sequential pacing, the paced ventricular impulse causes earlier excitation of a segment of AV node as compared with control method. The effect of such preexcitation on the AV node is discussed in the text. While not shown here, increasing the V-A interval during basic drive will increase the fraction of AV node that is preexcited. His, His bundle.
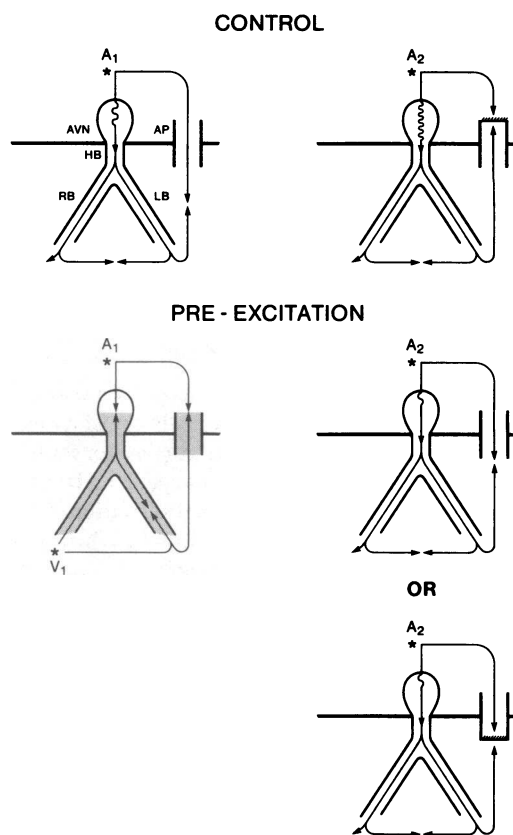
Figure 3. Schematic illustration of pacing protocol in patients with AV junctional reentry incorporating an accessory pathway. The upper panel shows conduction through reentrant circuit consisting of AV node (AVN), His-bundle (HB), right and left bundle branches (RB and LB), and accessory pathway (AP) during basic drive A_1. Note slow conduction through AV node and unidirectional block in accessory pathway with premature stimulation (A_2). With V-A sequential pacing, the paced ventricular impulse (V_1) preexcites the reentrant circuit (compared with control method). The effect of such physiologically altered circuit on conduction of A_2 is discussed in the text.

Effective refractory period (ERP) of the AV node was defined as the longest A_1-A_2 interval that failed to propagate to the His-bundle. Unless otherwise indicated, this was also the definition of ERP of the anterograde pathway of the AVJRe circuit, both in patients with Wolff-Parkinson-White syndrome as well as in patients with AV nodal reentry without demonstrable dual pathways.

In patients with dual AV nodal pathways, ERP of the fast AV nodal pathway was defined as the longest A_1-A_2 interval that resulted in an abrupt marked increase in A_2-H_2 interval, usually associated with reentry. Consequently, in the patients with discontinuous refractory period curves, ERP of the slow pathway was evident as the longest A_1-A_2 interval, which failed to propagate anterogradely to the His-bundle.

ERP of the accessory pathway was the longest A_1-A_2 interval that failed to conduct over the accessory pathway.

To evaluate the effect of V-A sequential pacing on AV nodal conduction time, A_2-H_2 intervals were compared with those during control, at the same A_1-A_2 intervals. To more precisely evaluate the effect on conduction time in the anterograde pathway of the AVJRe circuit, such comparisons were made only when A_2-H_2 delays were associated with AV junctional reentry.

Determination of V-A intervals. Generally, the absence of anterograde His-bundle deflection during the basic V-A sequential drive indicated that the paced ventricular impulse had penetrated and collided with the paced atrial impulse in the AV node (i.e., proximal to His-

bundle). As determination of the level of retrograde impulse penetration was not the purpose of the study, in all patients the V-A sequential pacing protocol was started with a V-A interval equaling 0 ms. Thereafter, the study design called for progressive increase in the V-A sequential interval to observe the effect of preexcitation (see below) of progressively greater segments of the reentrant circuit.

Retrograde preexcitation. The term preexcitation is used as defined by Ohnell (22), and denotes depolarization of tissue by an additional excitatory spread in advance of that during normal or, as is the case in this study, the control method (Figs. 1–3). The term retrograde is used here to qualify the generic physiological term, and to indicate that preexcitation is being used in a context different from the so-called clinical preexcitation syndrome. Thus, the retrograde preexcitation of the reentrant circuits (composed of either the AV node exclusively or the AV node and an accessory pathway) resulted from the ventricular impulse during the V-A sequential method. The use of progressively longer (by 10–20 ms) V-A intervals resulted in a greater portion of the reentrant circuit depolarized by the ventricular impulse and thus greater preexcitation (Figs. 2 and 3).

Zone of reentry. Zone of reentry was defined as the range of A_1-A_2 interval, where A_2 produced the atrial echo phenomenon (due to AV junctional reentry) (10–12). The outer and inner limits of the zone of reentry were the longest and the shortest A_1-A_2 intervals, respectively, in a given patient, which resulted in the atrial echo phenomenon.

Results

Effect of retrograde preexcitation on conduction in anterograde (AV nodal) pathway. All five patients with AV nodal reentry
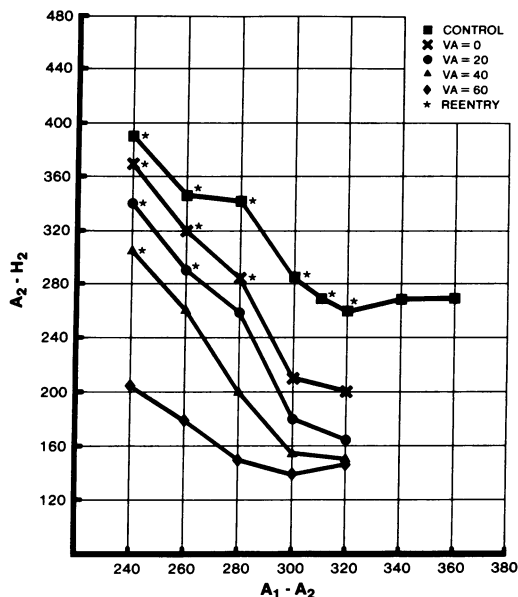

Figure 4. Effect of retrograde preexcitation on AV nodal conduction and zone of reentry (patient 2). A family of refractory period curves is depicted (A_1-A_2 intervals versus A_2-H_2 delays). During control study, a relatively linear curve is generated. A_2-H_2 delays associated with AV nodal reentry are indicated by an asterisk. During retrograde preexcitation (V-A interval, 0) less A_2-H_2 delays are encountered at a given coupling interval; this is associated with a decrease in the zone of reentry. Increasing the V-A intervals show progressively greater effect of retrograde preexcitation until (at V-A interval, 60 ms) the resulting A_2-H_2 delays are no longer associated with AV nodal reentry. Note that during V-A sequential method, AV nodal reentry occurs with A_2-H_2 delays that are less than that during control (see zone of A_1-A_2 intervals from 240–280 ms).

Table I. Effect of Retrograde Preexcitation on Anterograde Pathway

| | | A_2-H_2 interval associated with reentry | |
Patient number	A_1-A_2 interval	Control method	V-A sequential method
AV nodal reentry			
1	300	470	390
2	240	390	305
3	280	430	380
4	240	480	290
5	300	440	380
Wolff-Parkinson-White			
6	240	190	160
7	360	360	310
8	310	275	190
9	260	310	280
10	300	270	185
11	220	270	270

All intervals in milliseconds. $P < 0.001$.

demonstrated a decrease in anterograde AV nodal conduction time (A_2-H_2) after retrograde preexcitation by V_1. An example is shown in Fig. 4, which depicts refractory period curves, both during control study as well as retrograde preexcitation, in a patient with AV nodal reentry. Note that the A_2-H_2 interval is shorter when a given A_1-A_2 interval during the retrograde preexcitation protocol is compared with the control method. With progressively greater preexcitation (i.e., V-A interval of 20, 40, and 60 ms), there was progressively greater decrease in anterograde AV nodal conduction time at a given A_1-A_2 interval. Of particular note was the fact that at certain A_1-A_2 intervals, AV nodal reentry occurred despite shorter A_2-H_2 intervals during VA sequential pacing (Table I, Fig. 4). This observation, suggesting improvement in conduction through the anterograde pathway of the AV nodal reentrant circuit, was noted in all five patients with AV nodal reentry. Another example is shown in Fig. 5 taken from the one patient with typical discontinuous AV nodal refractory period curves. In Fig. 5, conduction through the so-called slow pathway is heralded by a "sudden jump" and associated with AV nodal reentry, both during control as well as with retrograde preexcitation protocols. The improvement in anterograde conduction through the slow pathway, after retrograde preexcitation, is seen in the zone of A_1-A_2 intervals ranging from 340 to 280 ms (Fig. 5).

In five of the six patients with Wolff-Parkinson-White syndrome, A-V junctional reentry associated with shorter A_2-H_2 intervals could also be demonstrated at certain A_1-A_2 intervals, after retrograde preexcitation (Table I). In the remaining patient with concealed accessory pathway there was no leftward shift of the AV nodal refractory period curves, even with progressively greater preexcitation (Fig. 6). This was the sole example in which improvement in anterograde pathway conduction was not seen after retrograde preexcitation of the AV node.

Effect of retrograde preexcitation on ERP of anterograde pathway (AV node). In the five patients with AV nodal reentry,
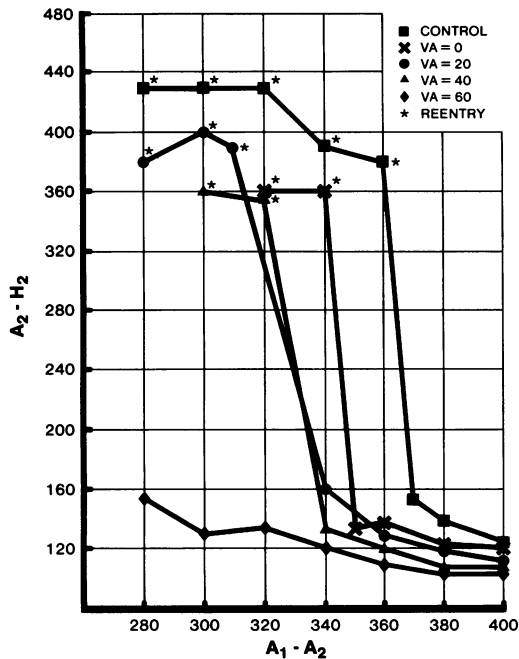
Figure 5. Effect of preexcitation in a patient with dual pathways (patient 3). This figure depicts another family of refractory period curves. During control method, a sudden jump in A_2-H_2 interval, characteristic of so-called dual pathways, is seen. Note how (with increasing preexcitation) the ERP of the fast pathway is progressively shortened and that AV nodal reentry (*) is always associated with conduction through slow pathway. With V-A interval of 60 ms the ERP of fast pathway was less than ERP of the atrium, thus effectively abolishing AV nodal reentry at all coupling intervals.

Figure 6. Refractory period curves in a patient with accessory pathway without retrograde AV nodal conduction (patient 11). In this figure, A_1-A_2 intervals are plotted against resulting AV delays (A_2-V_2). Note absence of significant and consistent improvement in AV conduction with V-A sequential pacing. At some coupling intervals, His-Purkinje system delay seen during control study was abolished with V-A sequential pacing, resulting in some shortening of AV delay. Overall, despite little change in A_2-V_2 delay, there was progressive shortening of the zone of reentry with greater preexcitation (see bar graph). A postulated mechanism was a distal shift in site of block in the accessory pathway (see Fig. 3). As the site of block occurred more distally with increasing V-A intervals, progressively greater AV nodal delays were required to initiate reentry. Thus, A_1-A_2 intervals, at which the zone of reentry started, became progressively shorter with greater preexcitation. Fig. 9 shows selected panels from this patient. (*) Denotes the only two A_1-A_2 intervals where AV reentry occurred.

AV nodal ERP was not attained during control study because of the following reasons: (a) it was less than the ERP of atrium (three patients), or (b) induction of unsustained atrial arrhythmias (two patients) and further testing at shorter A_1-A_2 intervals was not attempted. With retrograde preexcitation, there was no change either in the ERP of the atrium or in induction of unsustained atrial arrhythmias. In four of six patients with Wolff-Parkinson-White syndrome, ERP of the AV node was attained during the control study. With retrograde preexcitation, ERP of the AV node was achieved at shorter A_1-A_2 intervals. In other words, retrograde preexcitation resulted in a decrease in the ERP of anterograde pathway, when this parameter could be determined during the control method.

Effect of retrograde preexcitation on ERP of "fast" AV nodal pathway or accessory pathway. Of the three patients with manifest anterograde conduction over the accessory pathway, in two the ERP of the accessory pathway decreased with V-A sequential pacing (Figs. 3 and 7), and in one patient there was no change. The decrease in ERP of the fast pathway in the sole patient with discontinuous AV nodal pathways is evident in Fig. 5. In the remaining four patients with AV nodal reentry, the effect on ERP of the fast pathway could not be determined, as there was no clear distinction between the so-called fast and slow pathways at the cycle lengths tested. For the same reason, improvement in anterograde conduction time in the fast AV nodal pathway with retrograde preexcitation could only be clearly demonstrated in the patient with discontinuous AV nodal curves (Fig. 5).
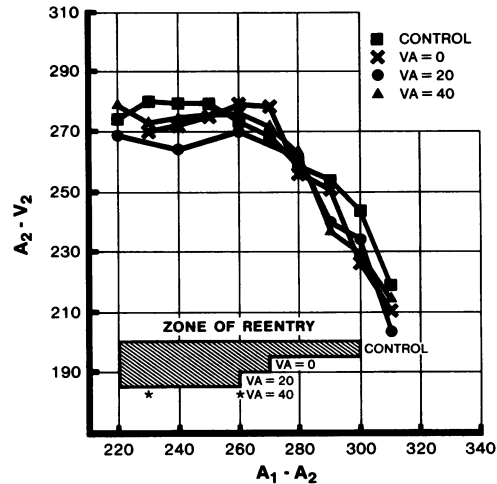
Effect of retrograde preexcitation on initiation of reentry by atrial extrastimulus. Several of the functional changes described above acted alone or in concert to prevent initiation of reentry by A_2. In all patients the zone of reentry was decreased by V-A sequential pacing. Both AV nodal reentry as well as AV junctional reentry incorporating an accessory pathway was abolished at all coupling intervals by increasing the V-A interval during basic drive. In 10 of 11 patients there appeared to be a relationship between the improvement in anterograde conduction and the decrease in the zone of reentry (Figs. 4 and 5). In the remaining one patient, the zone of reentry decreased progressively with greater retrograde preexcitation, even though there was no improvement in anterograde AV nodal conduction (Figs. 6 and 8). In three patients an additional mechanism for prevention of reentry was a decrease in ERP of accessory or fast AV nodal pathway (Fig. 7). In three other patients AV junctional reentry failed to occur, even though the A_2-H_2 delay during retrograde preexcitation was greater than the A_2-H_2 delay associated with reentry during control method (Fig. 9).

In contrast, facilitation of reentry by retrograde preexcitation was also seen in three patients with Wolff-Parkinson-White syndrome. In all three the zone of reentry was limited by ERP of the AV node during control study. Preexcitation resulted in abolition of AV nodal conduction block with decrease in the ERP of the AV node, thus permitting reentry to
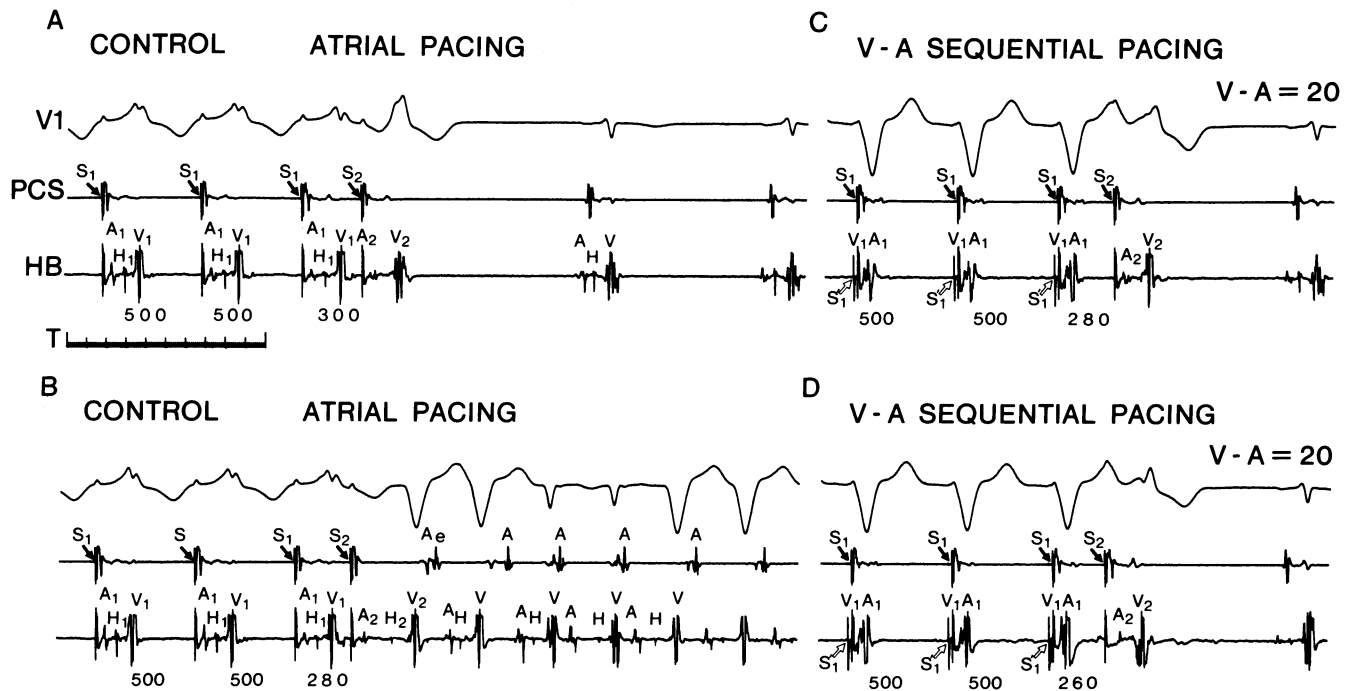
A
CONTROL ATRIAL PACING

V1

PCS

HB

500 500 300

T

B
CONTROL ATRIAL PACING

500 500 280

C
V - A SEQUENTIAL PACING

V - A = 20

500 500 280

D
V - A SEQUENTIAL PACING

V - A = 20

500 500 260

Figure 7. Effect of V-A sequential pacing on ERP of accessory pathway (patient 6). Electrograms from top to bottom show surface ECG (V₁), proximal coronary sinus (PCS), and His-bundle (HB) electrograms. T, Timing markers. *A* shows conduction of paced atrial impulse over the accessory pathway both during basic drive as well as premature stimulus (A₂). At S₁-S₂ interval of 280 ms (*B*), AV junctional tachycardia is initiated as the ERP of the accessory pathway is achieved. Note occurrence of intermittent functional bundle branch block. *C* and *D* show continued conduction over accessory pathway at shorter S₁-S₂ intervals during V-A sequential pacing.

A
CONTROL ATRIAL PACING

1

HRA

LRA

HB

T

600 600 300

B
CONTROL ATRIAL PACING

600 600 220

C
V - A SEQUENTIAL PACING V - A = 40

600 600 300

D
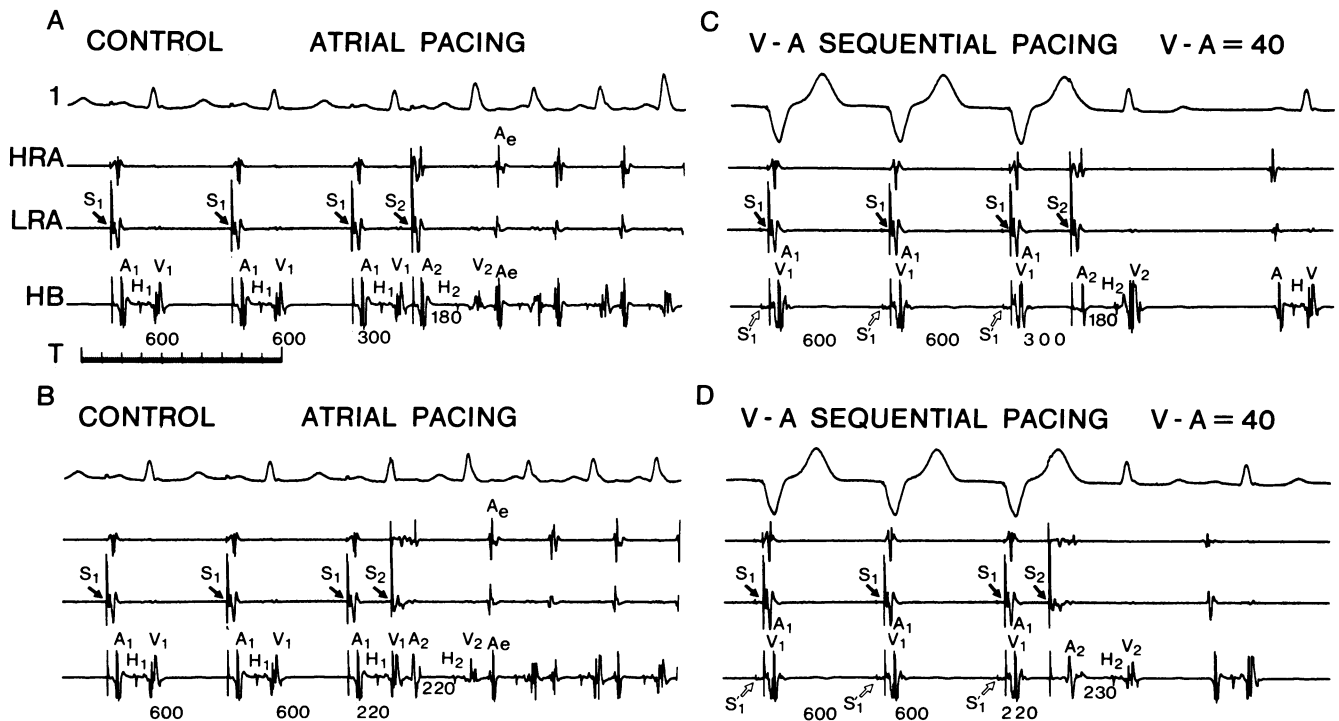V - A SEQUENTIAL PACING V - A = 40

600 600 230

Figure 8. Effect of V-A sequential pacing on reentry involving accessory pathway (patient 11). Electrograms from top to bottom show surface ECG lead (1), high right atrial (HRA), low right atrial (LRA), and His-bundle (HB) electrograms. T, Timing markers. *A* and *B* show the outer (S₁-S₂, 300 ms) and inner (S₁-S₂, 220 ms) limits of the zone of reentry in a patient with concealed accessory pathway. Note the AV nodal delays associated with reentry. *C* and *D* show V-A sequential pacing with S₁-S₂ intervals identical to *A* and *B*, respectively. Of interest is the fact that the AV nodal delays are similar (despite preexcitation), and yet no AV reentry takes place. The absence of improvement in AV nodal conduction may be explained by absence of retrograde penetration by the preexciting ventricular impulse. However, the site of block (accessory pathway) is preexcited, and possibly the resultant distal shift in site of block prevents reentry despite achievement of AV delays similar to control method (see text and Figs. 3 and 7).
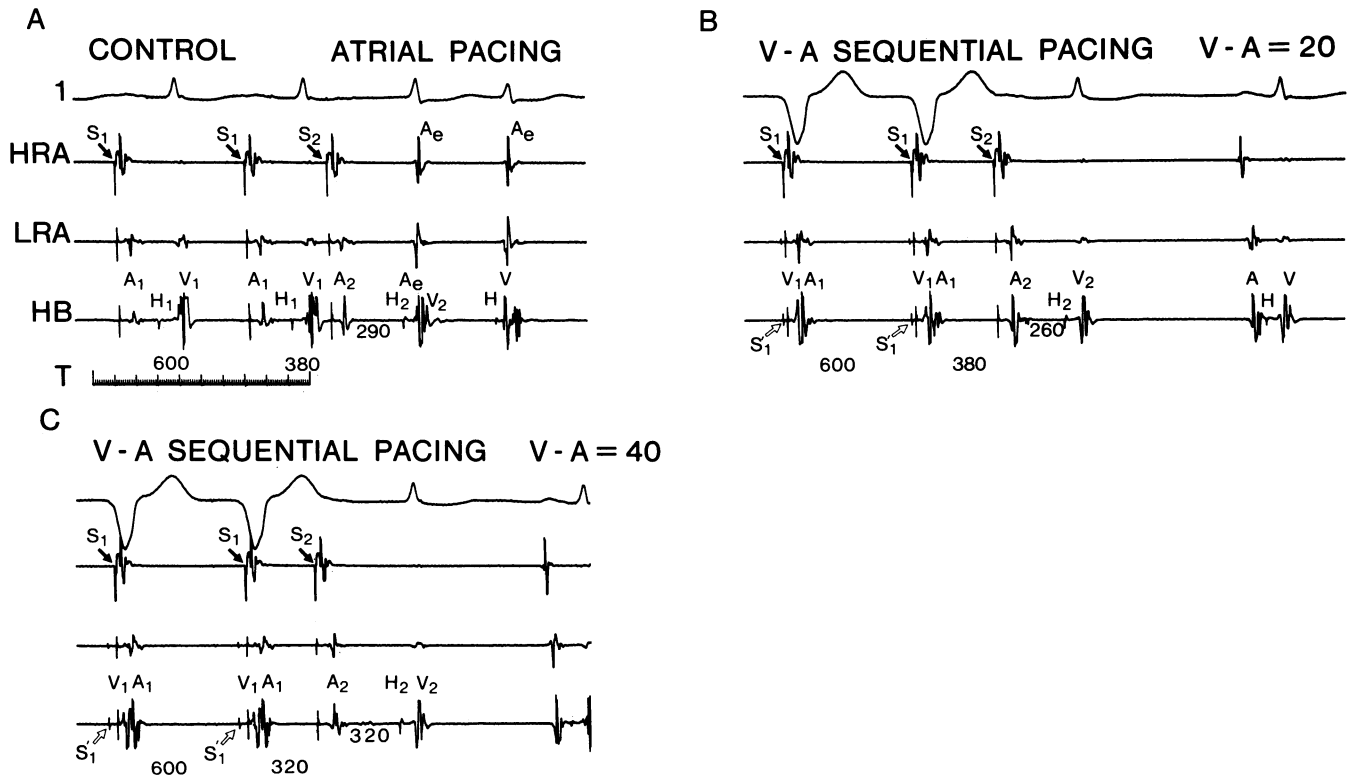
A

B

C

Figure 9. Electrophysiologic mechanisms of prevention of AV nodal reentry (patient 1). Electrograms from top to bottom show surface ECG lead 1, high right atrial (HRA), low right atrial (LRA), and His-bundle (HB) electrograms. T, Timing markers. *A* shows induction of AV nodal reentry by atrial extrastimulus A_2 at coupling intervals of 380 ms. Note AV nodal delay (A_2-H_2, 290 ms) sufficient for onset of AV nodal reentry. With V-A sequential pacing (V-A interval, 20 ms), A_2 at same coupling interval now results in decrease in AV nodal

delay (*B*) with prevention of AV nodal reentry. This mechanism is graphically illustrated in Fig. 2. *C* depicts the effect of preexcitation with V-A interval of 40 ms. Note that AV nodal delay is now significantly greater than in *A*, yet AV nodal reentry is again prevented. A possible explanation may be that preexcitation of fast pathway causes A_2 to block more distally in the fast pathway, in turn requiring greater delay in the slow pathway for reentry. (See text for details.)

occur at shorter coupling intervals and extending the zone of reentry. However, further increases in V-A intervals were followed by progressive decrease and then abolition of the zone of reentry.

Discussion

The extrastimulus method has proved to be a potent and useful technique for reproducing clinical arrhythmias in the electrophysiology laboratory. The wake of refractoriness of the basic drive determines the coupling interval of the extrastimulus which results in the necessary conduction delay and conduction block required for reentry (10, 11, 16). In the AV node, increase in the rate of basic drive results in greater delay at a given coupling interval (23–25). In the His-Purkinje system, an abrupt change in cycle length influences refractoriness, and thereby reentry (26). Ventricular tachycardia is more likely to be induced by a premature ventricular beat after a ventricular drive as compared with that programmed during sinus rhythm (18). This suggests that both direction as well as sequence of activation of the reentrant circuit may be important.

In this study, varying fractions of both large as well as small AV junctional reentrant circuits were excited earlier from retrograde direction during the basic drive. This resulted in improvement in conduction and decrease in refractoriness in

both the anterograde as well as retrograde pathways of the reentrant circuit. While the reentrant process was facilitated in some cases, the overall effect was prevention of clinical tachycardia (see Results). In most instances the electrophysiological factors responsible for failure of reentry were quite apparent (Figs. 4 and 7). In other instances, mechanisms (previously unknown) were postulated after comparing refractory period curves obtained during control method with those after retrograde preexcitation.

Role of conduction delay in AV junctional reentry: its relationship to site of block. It has been reported that the mechanism of prevention of reentry with dual chamber pacing is primarily the failure to achieve sufficient conduction delay in the anterograde pathway (9, 27, 28). While mitigation of conduction delay may play a role, it is not the only factor that prevents reentry. Figs. 8 and 9 demonstrate that with V-A sequential pacing, AV junctional reentry may be prevented even when anterograde conduction delay is greater than that which was sufficient for reentry during the control study. This phenomenon could best be explained by postulating that V-A sequential pacing produced a shift in the site of block (of A_2) in the retrograde pathway. It is not unreasonable to postulate that V-A sequential pacing decreased refractoriness of the retrograde pathway and caused A_2 to block more distally at a given A_1-A_2 interval (Fig. 3). In turn, the distal shift in the site of block (compared with control method) would require greater

A_2-H_2 delay to permit recovery of excitability in the distal retrograde pathway. This phenomenon occurred even when there was no manifest anterograde conduction over the accessory pathway (Fig. 6, patient 11), suggesting an increasingly distal site of block related to progressively greater retrograde preexcitation. Such an electrophysiological effect could be related to the fact that some accessory pathways may have a branching anatomy (29), and V-A sequential pacing may cause shifts in site of block within the complex structure. The overall frequency with which such a shift prevents reentry during retrograde preexcitation is unknown. In a clinical setting, autonomic changes, or drugs, or both, may affect refractoriness in the retrograde pathway and thus influence the degree of conduction delay necessary for reentry. Such functional changes in retrograde pathway may not be immediately evident on surface or intracardiac electrograms, which typically measure anterograde conduction.

Utility of V-A sequential pacing in determining presence of retrograde conduction in the anterograde pathway. Knowledge of presence or absence of retrograde conduction in the AV node can be of help during the study or intraoperative mapping of septal accessory pathways. Such information, however, is usually obtained after surgical transection of the Kent-bundle (30). During analysis of our data, it became clear that the improvement of anterograde AV nodal conduction after retrograde preexcitation was a simple way of demonstrating the presence of retrograde AV nodal conduction. Similarly, the lack of improvement of AV nodal conduction (Figs. 6 and 8) signals the absence of demonstrable retrograde AV nodal conduction in a patient with accessory pathway.

In patients with AV nodal reentry the need to know about retrograde conduction in the anterograde or slow pathway is mostly a scientific curiosity (31). In all five patients there was a decrease in anterograde AV nodal conduction (A_2-H_2) after retrograde preexcitation. As shown in Figs. 4 and 5, when AV nodal reentry occurred (with shorter A_2-H_2 intervals) during retrograde preexcitation, it indicated that V-A sequential pacing preexcited and improved conduction in the slow pathway; it was an indirect demonstration of retrograde conduction in the so-called slow pathway.

Clinical significance

It is clear from our results that the occurrence of AV junctional reentry depends on a complex interplay between conduction and refractoriness in both the anterograde as well as retrograde pathway. It would appear that the functional characteristics of a potential reentrant circuit, as demonstrated by the extrastimulation technique, are dependent to a large extent on the activation sequence during basic drive. No pacing protocol may completely simulate the clinical milieu, but methods such as retrograde preexcitation may reproduce clinical conditions that improve conduction and decrease refractoriness. As such, this pacing method may complement another that decreases conduction and increases refractoriness (24, 25). Used together, such pacing protocols may give valuable insight into how changes in conduction and refractoriness interact to permit or prevent clinical reentry.

Limitations

A possible criticism of this study could be that increased sympathetic tone during V-A sequential pacing may explain the observed results. This problem was addressed in a previous study in which preexciting only the last beat of the basic drive produced similar improvement in A-V conduction (28). In addition, in studies (19) where A-V sequential pacing was compared with ventricular pacing, the former method (preexcitation protocol) produced similar changes in AV nodal conduction and refractoriness. This occurred despite the fact that sympathetic tone would be expected to decrease with A-V sequential pacing as compared to ventricular pacing alone.

Acknowledgments

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