Effect of Atrioventricular Sequential Pacing in Patients With No Ventriculoatrial Conduction

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Candidates for the dual chamber “universal” (DDD) pacemaker are frequently tested for the presence of intact ventriculoatrial (VA) conduction to identify those at risk for developing endless loop tachycardia. However, recent reports have cited instances where clinical endless loop tachycardia has occurred even when no VA conduction could be demonstrated during ventricular pacing. A pacing protocol was designed to assess the effect of atrioventricular (AV) sequential pacing on VA conduction in 13 patients who showed no evidence of VA conduction during routine electrophysiologic testing. The absence of VA conduction was inferred by pacing the ventricle at several cycle lengths without obtaining a retrograde atrial capture. With the AV sequential method, which consisted of an AV sequential drive with a programmed AV interval of 100 to 160 ms, the presence or absence of VA conduction was tested utilizing a premature ventricular stimulus (V2) over a wide range of coupling intervals. During the AV sequential method, the V2 effectively propagated to the atria in 5 of 13 patients with V2-A2 intervals ranging from 200 to 460 ms (mean 304 ± 97).

It is concluded that in patients showing absent VA conduction during routine testing, the ability of a paced ventricular impulse to propagate retrogradely can be demonstrated in a significant number of cases with AV sequential pacing. Although the exact mechanism could not be determined, it is postulated that as compared with ventricular pacing alone, a longer input into the AV node (first anterogradely during the AV sequential drive and then retrogradely with V2) may be partly responsible for the facilitative effect of the AV sequential method.

Recently it has been demonstrated that AV sequential pacing exerts a facilitative effect on retrograde conduction and that the conventional methods utilizing ventricular pacing followed by a premature ventricular stimulus consistently underestimate the VA conduction time of the premature ventricular beat (8). That study, however, was limited to patients with intact VA conduction.

The effect of AV sequential pacing on retrograde conduction in patients who have no demonstrable VA conduction during routine electrophysiologic evaluation is unknown. Consequently, a pacing protocol was designed to evaluate the facilitative effect, if any, of AV sequential pacing on the retrograde conduction of a premature ventricular beat in such a group of patients. The premature ventricular beat in the setting of AV sequential pacing was designed to simulate conditions under which pacemaker arrhythmias are known to occur (9,10).

Methods

Study patients. The study group was selected from the patients referred to the electrophysiology laboratory for diagnostic studies. It comprised 13 consecutive patients (9 men and 4 women) with intact atrioventricular (AV) but
absent ventriculoatrial (VA) conduction (see below). All patients were in sinus rhythm and their ages ranged from 42 to 71 years (mean 59 ± 9). Ten patients had coronary artery disease, one had mitral valve prolapse and two had no underlying heart disease. The patients were studied in a nonsedated postabsorptive state and all cardioactive drugs were stopped 48 to 72 hours before the study. The procedure was explained and signed consent obtained for all patients.

**Electrophysiologic studies.** After local anesthesia, quadripolar electrode catheters were percutaneously introduced through anteceubital and femoral veins and positioned under fluoroscopic guidance. The catheter in the region of tricuspid valve permitted the recording of the His bundle potential, and those in the high right atrium and right ventricle recorded the local electrograms and were used for electrical stimulation.

Intracardiac electrograms (filtered at 30 to 500 Hz), surface electrocardiographic leads and time lines were simultaneously displayed on a multichannel oscilloscope (Electronics for Medicine VR-16) and recorded on a magnetic tape (Honeywell model 101). For analytic and illustrative purposes, recordings were subsequently reproduced on photographic paper at 100 mm/s speed.

**Intracardiac electrical stimulation** was performed using a custom designed DTU 101 digital stimulator (Bloom Associates, Ltd.) capable of delivering premature stimuli after a basic drive of AV sequential pacing, with adjustable AV intervals. Although complete electrophysiologic studies using the previously described techniques (11–13) were carried out in these patients, the pacing protocol relevant to this study is described in detail as follows.

**Method used to determine absence of VA conduction** (Fig. 1A). Several ventricular pacing cycle lengths were used. This included a cycle length 20 to 50 ms shorter than the sinus cycle length. Complete AV dissociation was demonstrated at all paced cycle lengths. The number of paced beats required to demonstrate the absence of VA conduction was calculated from the formula:

\[
\text{Sinus cycle length} - \frac{\text{Paced ventricular cycle length}}{\text{Sinus cycle length}} \times \text{Paced ventricular cycle length}
\]

Thus, if the difference between the paced ventricular cycle length and the sinus cycle length was 20 ms and the sinus rate was 800 ms, it would require 40 paced beats to ensure that at least one retrograde input of 780 ms was achieved. This method helps to determine the number of paced beats required to scan the interval between two sinus beats. The longest paced cycle length demonstrating AV dissociation was considered the longest retrograde input \(V_{1}V_{2}\) (Fig. 1A) with no demonstrable VA conduction.

In four patients, ventricular pacing caused an increase in the sinus cycle length, which necessitated the use of pacing cycle lengths 60 to 80 ms shorter than the baseline sinus rate to document complete AV dissociation.

![Figure 1](https://example.com/figure1.png)

**AV sequential method** (Fig. 1B). This method consisted of an AV sequential drive (six beats) with AV intervals varying from 100 to 160 ms. The AV intervals were selected to achieve collision of atrial and ventricular impulses in the His-Purkinje system, which has been shown to result in maximal facilitation of retrograde conduction (8). After the AV sequential drive, a paced ventricular beat \(V_{2}\) was programmed to test for VA conduction. The premature ventricular stimulation was started with the longest attainable coupling interval \(V_{1}V_{2}\). Then the \(V_{1}V_{2}\) interval \(S_{1}S_{2}\) (Fig. 1B) was progressively decreased until ventricular refractoriness was encountered.

For purpose of comparison with ventricular pacing alone, the input into the AV node with the AV sequential method (Fig. 1B) was considered to be \(S'_{1}S_{2}\) (anterogradely from the last atrial stimulus of AV sequential drive and retrogradely from the ventricular test stimulus \(S_{2}\)). As shown in Figure 1B, this value was an approximation of the actual input into the AV node, which could not be measured because a retrograde His bundle depolarization (\(H_{2}\)) was not
seen when V₂ was preceded by the AV sequential drive, even at the shortest V₁V₂ interval.

The retrograde conduction time V₂A₂ was measured from the stimulus artifact (S₂) to the initial atrial deflection of high right atrial recording.

Results

Electrophysiologic data (Table 1). Eight of 13 patients (Cases 1 to 8) showed no ventriculoatrial (VA) conduction with both methods. In the remaining five patients (Cases 9 to 13), the test stimulus (V₂) propagated retrogradely to the atrium after the atrioventricular (AV) sequential drive (Fig. 2C). In these five patients, the longest programmable coupling interval (V₁V₂) at which V₂ conducted to the atrium was limited by the sinus escape interval. However, progressive shortening of V₁V₂ intervals produced a definite zone of coupling intervals at which retrograde conduction of V₂ could be demonstrated. The shortest V₁V₂ interval with intact VA conduction can be deduced from Table 1 by subtracting the programmed AV interval from the value of "shortest AV sequential input." The longest V₂A₂ intervals were achieved at the shortest V₁V₂ coupling intervals and the values (ranged from 200 to 460 ms) (mean 304 ± 97).

Stimulus input into the AV node. Table 1 also allows a comparison of input into the AV node during ventricular pacing (longest retrograde input with no VA conduction) with that during AV sequential method (AV sequential input). Although one can be fairly certain about the input into the AV node during ventricular pacing (V₁V₁ or H₁H₁), the measurement during AV sequential method (S₁S₂) is not exact. The S₁S₂ measurement overestimates the true input by not subtracting the conduction time through the atrium, and underestimates it by not adding the retrograde conduction time through the His-Purkinje system (S₂H₂). As such, the S₁S₂ is likely to be an underestimation of the true input because the S₂H₂ is probably greater than anterograde conduction time through the atrium.

It can be seen that patients who did not conduct retrogradely with both methods (Cases 1 to 8) failed to do so despite the longer input achieved with the AV sequential method. In the remaining five patients, only the shortest input with the AV sequential method at which VA conduction could be demonstrated are given. In four of these five patients (Cases 9 to 12) the S₁S₂ was shorter (by 40 to 150 ms) than the longest retrograde input, while in the remaining patient (Case 13) it was longer.

Comparison of two groups of patients. Using the single-sided paired t test analysis to compare the two groups of patients, there appeared to be no statistical difference between the longest retrograde inputs (V₁V₁) and the sinus rates of the patients who demonstrated VA conduction with AV sequential method (Cases 9 to 13) versus those who did not (Cases 1 to 8). There was, however, a statistically significant difference in AH intervals between the two groups (Table 1).

Site of retrograde block. It needs to be stated that the site of retrograde VA block was considered to be the AV node (as opposed to the His-Purkinje system) in all 13 patients. This was based on the observation that suggested the paced ventricular impulses were being concealed in the AV

Table 1. Electrophysiologic Data

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1 to 8 793 ± 92 119 ± 28 48 ± 7 755 ± 93
9 to 13 712 ± 120 94 ± 13 51 ± 7 670 ± 116

p value NS 0.05 NS NS

All data are in milliseconds. AH = AH interval; HV = HV interval; NS = not significant; SCL = sinus cycle length; VAC = ventriculoatrial conduction.
Discussion

The importance of establishing the presence of ventriculoatrial (VA) conduction in candidates for the dual chamber universal (DDD) pacemaker is supported by several studies (5–7,9,10). A retrogradely conducting pathway can be documented in 75% of patients undergoing the standard electrophysiologic study (11). The group of patients who do not show evidence of VA conduction with the method of ventricular pacing alone sometimes demonstrate the ability to conduct retrogradely to the atrium after intervention with atropine (15) or isoproterenol (16). Thus, the reversible nature of "no VA conduction" has been observed previously using pharmacologic measures known to facilitate AV nodal conduction.

Importance of demonstrating VA conduction with AV sequential pacing. In this study, the effect of atrioventricular (AV) sequential pacing was tested in 13 patients who, on the basis of ventricular pacing alone, would be classified as having no VA conduction. In 5 of the 13 patients, VA conduction was demonstrated after an AV sequential drive. Although retrograde conduction with subsequent beats after pharmacologic intervention was not evaluated in this study, previous reports (15,16) suggest that changes in autonomic tone may enhance VA conduction, which could impart the ability to maintain a pacemaker tachycardia. Thus, the demonstration of an intact retrograde node, causing the dissociated sinus beats to block proximal to the His bundle (Fig. 2) (14).
A pathway with AV sequential pacing may identify a source of a potential clinical problem in candidates for DDD pacemakers. Changes in autonomic tone in this group of patients may explain the clinical reports of pacemaker arrhythmia in patients with no VA conduction (6,7). The relatively long V2A2 intervals obtained in the five patients give evidence for a slowly conducting retrograde pathway. Such patients may be especially at risk because a longer programmed atrial refractory period of the pacing device may be needed to prevent pacemaker arrhythmias.

Mechanism of facilitation of retrograde conduction by AV sequential pacing. The exact mechanism by which AV sequential pacing caused the facilitation of retrograde conduction in this study could not be elucidated, primarily because the true input into the AV node with the AV sequential method could not be determined. Thus, although the measured values (S1′S2) in Patients 9 to 12 are less than the longest retrograde input (V1V1 or H1H1) during ventricular pacing (Table 1), it is possible that the actual input into the AV node during the AV sequential method is still longer than the longest V1V1 interval. This would readily explain the ability of V1 to conduct retrogradely in these patients. Experimental studies (17,18) in the animal model have shown that dual excitation of the AV node shortens total activation time and results in earlier recovery and facilitation of subsequent impulse conduction. In our five patients, the earlier recovery of the AV node and subsequent facilitation of V2 probably resulted from the earlier excitation of the AV node by the anterograde (atrial) impulse during the AV sequential drive.

It can be argued that had the sinus rate been slower in these patients, it would have been possible to achieve a longer retrograde input with ventricular pacing alone, with subsequent demonstration of intact retrograde conduction. Although this may be true, it also underscores the importance of using the AV sequential pacing method to test for retrograde conduction. Because such a method can allow a longer input into the AV node, the longest retrograde input with ventricular pacing alone is, of necessity, limited by the sinus rate.

It is of interest to note that Patients 1 to 8 who did not demonstrate VA conduction by both methods had longer AH intervals at similar sinus rates. It may be possible that the relatively slower AV nodal conduction is partly responsible for lack of facilitation by the AV sequential method.

Conclusion. The AV sequential pacing protocol may be a useful additional method for determining the presence of VA conduction in pacemaker candidates. Although the exact mechanism could not be determined, it can be postulated that a longer input into the AV node may be partly responsible for the facilitative effect of the AV sequential pacing method.

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References