

Articles

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Articles

Para-Hisian Pacing

A New Method for Differentiating Retrograde Conduction Over an Accessory AV Pathway From Conduction Over the AV Node

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Abstract

Background Differentiation between ventriculoatrial (VA) conduction over an accessory AV pathway (AP) and the AV node (AVN) may be difficult, especially in patients with a septal AP.

Methods and Results A new pacing method, para-Hisian pacing, was tested in 149 patients with AP and 53 patients without AP who had AV nodal reentrant tachycardia (AVNRT). Ventricular pacing was performed adjacent to the His bundle and proximal right bundle branch (HB-RB), initially at high output to capture both RV and HB-RB. The output was then decreased to lose HB-RB capture. The change in timing and sequence of retrograde atrial activation between HB-RB capture and noncapture was examined. Loss of HB-RB capture without change in stimulus-atrial (S-A) interval or atrial activation sequence indicated exclusive retrograde AP conduction. An increase in S-A interval without change in His bundle-atrial interval or atrial activation sequence indicated exclusive retrograde AVN conduction. A change in atrial activation sequence indicated the presence of both retrograde AP and AVN conduction. Para-Hisian pacing correctly identified retrograde AP conduction in 132 of 147 AP patients, including all septal and right free wall APs. Retrograde AVN conduction masked AP conduction in 9 of 34 patients with a left free wall AP and 6 of 9 patients with the permanent form of junctional reciprocating tachycardia. Para-Hisian pacing correctly excluded AP conduction in all 53 patients with AVNRT.

Conclusions Para-Hisian pacing reliably identifies retrograde conduction over septal and right free wall APs, but AVN conduction may mask APs located far from the pacing site or with a long retrograde conduction time.

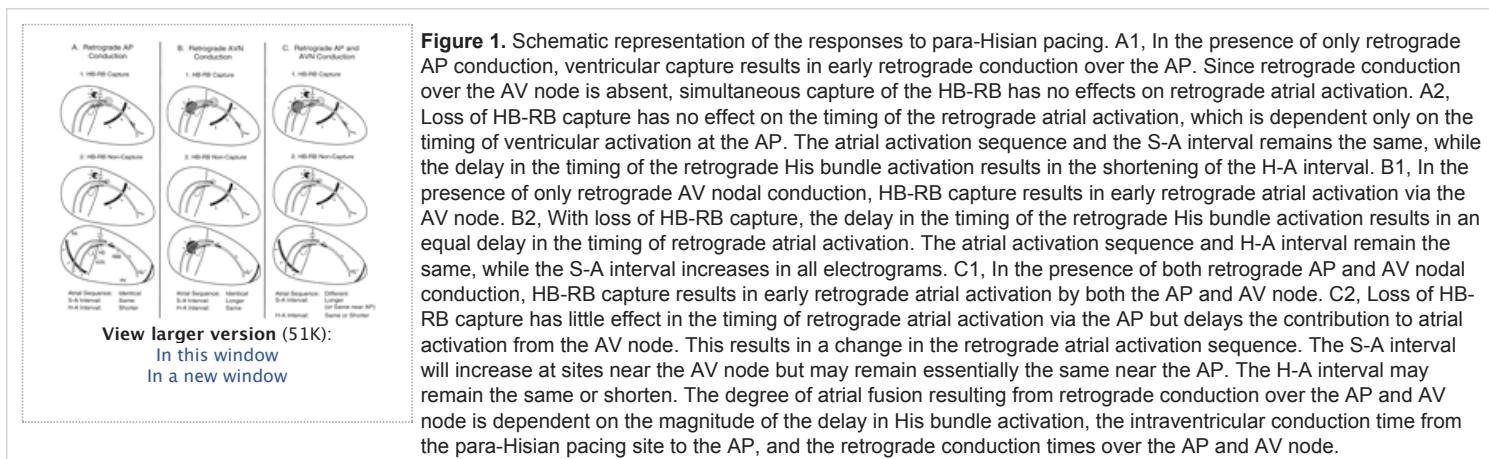
Key Words: pacing • conduction • atrioventricular node • electrophysiology • accessory AV pathway

Introduction

Radiofrequency catheter ablation of an accessory AV pathway has become the primary therapy for patients with Wolff-Parkinson-White syndrome.^{1 2 3 4 5 6 7 8 9} During these procedures, it is necessary to distinguish retrograde conduction over an AP from retrograde conduction over the fast or slow AV nodal pathway. The presence or absence of retrograde AP conduction can be determined by delivering a late ventricular extrastimulus during supraventricular tachycardia in an attempt to advance the timing of atrial activation at a time when the His bundle is refractory^{9 10 11 12 13 14 15 16} or during programmed ventricular stimulation by an early ventricular extrastimulus to determine the presence or absence of significant delay in the timing of retrograde atrial activation when the timing of His bundle activation is delayed. However, it may be difficult to differentiate between retrograde AP conduction and retrograde AV nodal conduction when sustained supraventricular tachycardia cannot be induced, especially when retrograde conduction is present only at long pacing cycle lengths. One approach is to move the right ventricular pacing catheter from the apex toward the site of earliest retrograde atrial activation.^{17 18 19} The stimulus-atrial interval will shorten with retrograde AP conduction. With retrograde AV nodal conduction, the stimulus-atrial interval will lengthen as the pacing catheter moves farther from the peripheral inputs to the His-Purkinje system.

A new approach, para-Hisian pacing, uses right ventricular pacing close to the His bundle or proximal right bundle branch. The pacing output is altered to produce ventricular capture with intermittent His bundle or right bundle branch capture to selectively alter the timing of His-bundle activation without changing the timing of local ventricular activation.²⁰ With retrograde conduction over an AP, the loss of His bundle capture should not change the timing of retrograde atrial activation or the retrograde atrial activation sequence (Fig 1A \blacklozenge). With retrograde AV nodal conduction, the delay in timing of retrograde His bundle activation should produce an equal delay in the timing of retrograde atrial activation without changing the retrograde atrial activation sequence (Fig 1B \blacklozenge). A mixture of these two responses (delay in the timing of atrial activation in the His bundle electrogram with a change in the retrograde atrial activation sequence) would be expected with retrograde

conduction over both an AP and the AV node (Fig 1C➔). The degree of contribution to atrial activation by the AP and the AV node (atrial fusion) should depend on the distance of the AP from the para-Hisian pacing site and the retrograde conduction times over the AP and AV node. The purpose of this study was to determine the ability of para-Hisian pacing to differentiate retrograde conduction over the AP from the AV node as a function of the location of the AP and the conduction properties of the AP and AV node.



Methods

Study Population

The study population consisted of 202 patients with paroxysmal supraventricular tachycardia undergoing electrophysiological study and radiofrequency catheter ablation. There were 114 male and 88 female patients, 4 to 76 years old (mean, 34.6±16.4 years).

Orthodromic AV reentrant tachycardia was induced in 149 patients. The AP was located at the anteroseptal or right anterior paraseptal region in 26 patients, midseptal region (earliest retrograde atrial activation recorded posterior to the His bundle and anterior to the coronary sinus ostium) in 8 patients, posteroseptal region in 56 patients, right free wall in 16 patients, and left free wall in 34 patients. The AP exhibited conduction in the antegrade and retrograde directions in 90 patients and only in the retrograde direction in 59 patients. Nine of the 59 patients with a concealed AP had incessant orthodromic AV reentrant tachycardia related to an AP in the posteroseptal region (8 patients) or left posterolateral region (1 patient) that had a long retrograde conduction time and decremental conduction properties consistent with PJRT.

Fifty-three patients were found to have AVNRT. Earliest retrograde atrial activation during tachycardia was recorded at the anterior septum close to the His bundle (slow/fast AVNRT) in 45 patients and at the posterior septum close to the coronary sinus ostium (fast/slow and slow/slow AVNRT) in 8 patients.

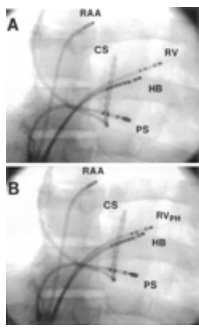
Electrophysiological Study

Electrophysiological study was performed with the patients in the fasting state under sedation with fentanyl and midazolam.^{2 21} Five multipolar electrode catheters were inserted percutaneously into the right subclavian vein, right femoral vein, and left femoral vein and were positioned in the right atrial appendage, His bundle region, right ventricle, and coronary sinus. The fifth catheter was used for mapping the tricuspid annulus and proximal coronary sinus. Mapping of the tricuspid annulus and coronary sinus was performed during supraventricular tachycardia and during ventricular pacing to identify the site(s) of earliest retrograde atrial activation. The presence of retrograde AP conduction was confirmed or excluded by standard techniques, including late ventricular extrastimuli during supraventricular tachycardia and programmed ventricular stimulation, both performed as close as possible to the site of earliest retrograde atrial activation.¹⁹

Para-Hisian Pacing

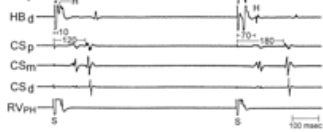
The His bundle electrogram was recorded with a 7F deflectable octapolar catheter with 2-mm interelectrode spacing (four close bipolar electrograms) to localize the His bundle and proximal right bundle branch. For para-Hisian pacing, a 7F deflectable quadripolar catheter was positioned at the anterobasal right ventricular septum 1 to 2 cm anterior and apical to the His bundle catheter (Fig 2A➔). Bipolar ventricular pacing was performed through the distal pair of electrodes (2-mm spacing) at a long pacing cycle length (>500 ms) and high output (10 mA and 2- to 6-ms pulse width). During pacing, the catheter was slowly withdrawn toward the pair of electrodes on the His bundle catheter, which recorded activation of the HB-RB until the width of the paced QRS complex shortened, indicating direct HB-RB capture (Fig 2B➔). The pacing output and pulse width were then decreased until the paced QRS complex lengthened, which was associated with a delay in the timing of the retrograde His bundle potential, indicating loss of HB-RB capture (Fig 3➔). The pacing output was increased and decreased to gain and lose HB-RB capture, respectively, while local ventricular capture was maintained. The response to para-Hisian pacing was determined by the change in the following variables between HB-RB capture and HB-RB noncapture: (1) atrial activation sequence; (2) the S-A interval in each electrogram, including an electrogram recorded close to the site of earliest retrograde atrial activation during tachycardia; and (3) H-A interval measured in the His bundle electrogram (Fig 1 ➔). These variables were examined before and after AP ablation.

Figure 2. Radiographs in the right anterior oblique projection illustrating the catheter positions used for para-Hisian pacing. A, A deflectable octapolar catheter (2-mm spacing) was positioned to record activation of the His bundle or proximal right bundle branch from the distal pair of electrodes (HB). A deflectable quadripolar catheter with 2-mm spacing between the distal pair of electrodes (RV) was positioned ≈1 cm anterior and apical to the His bundle. During continuous pacing at high output between the distal pair of electrodes, the RV catheter was slowly withdrawn to the position in B, which resulted in direct HB-RB capture in addition to ventricular capture. This para-Hisian pacing site (RV_{PH}) was located ≈5 mm anterior and apical to the His bundle. RAA indicates electrode catheter in the right atrial appendage; CS, catheter with eight groups of orthogonal electrodes positioned within the coronary sinus and great cardiac vein; and PS, mapping catheter positioned at the posteroseptal tricuspid annulus.



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Figure 3. Para-Hisian pacing demonstrating retrograde conduction over the slow AV nodal pathway (AVN/AVN pattern). The pacing stimulus (S) in the left complex resulted in ventricular capture and HB-RB capture, producing a relatively narrow QRS complex and early activation of the His bundle (H). HB-RB capture was lost in the right complex, resulting in widening of the QRS complex and a 60-ms increase in the S-H interval from 10 to 70 ms. This was also associated with a 60-ms increase in the S-A interval from 120 to 180 ms, without a change in the retrograde atrial activation sequence. The constant H-A interval (110 ms) and atrial activation sequence indicate that retrograde conduction was dependent on His bundle activation and not on local ventricular



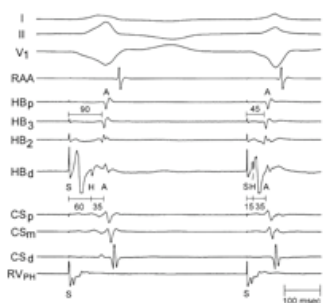
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activation, indicating retrograde conduction exclusively over the AV node. Earlier atrial activation in the proximal coronary sinus electrogram (CS_p) than in the His bundle electrogram (HB_p) suggests retrograde conduction over the slow AV nodal pathway. This tracing was recorded after ablation of a left lateral accessory AV pathway.

Results

Para-Hisian pacing (HB-RB capture and noncapture) was successfully performed in 200 of 202 patients. Intermittent loss of HB-RB capture was often obtained without alteration of the pacing output because of slight changes in the location of the pacing electrode with respiration. In 2 patients, proximal right bundle branch capture failed to produce early retrograde His bundle activation because of proximal right bundle branch block resulting from a previous unsuccessful attempt at radiofrequency ablation of an anteroseptal AP.

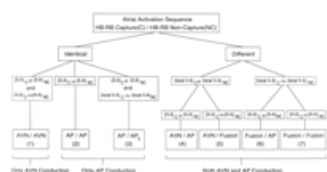
The His bundle electrogram during para-Hisian pacing exhibited a narrow ventricular potential beginning shortly after the pacing stimulus (Fig 3 \blacklozenge). The timing of the local ventricular potential became progressively earlier as the pacing electrode was moved closer to the His bundle. The narrow early ventricular potential allowed consistent identification of the local atrial and His bundle potentials (Figs 3 and 4 \blacklozenge).



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Figure 4. Para-Hisian pacing demonstrating retrograde conduction over the fast AV nodal pathway (AVN/AVN pattern) in a patient with AVNRT. The pacing stimulus (S) in the left complex did not produce HB-RB capture, reflected by the wide QRS complex and relatively late His bundle activation (S-H=60 ms). HB-RB capture was achieved in the right complex reflected by narrowing of the QRS complex and shortening of the S-H interval to 15 ms. The 45-ms shortening in S-H interval was matched by a 45-ms shortening in the S-A interval from 90 to 45 ms, without a change in the atrial activation sequence. The constant H-A interval (35 ms) and atrial activation sequence indicate that retrograde conduction was dependent on activation of the His bundle and not on the local ventricular myocardium.

Seven patterns of response to para-Hisian pacing were observed in the 200 patients (Fig 5 \blacklozenge and Table 1 \blacklozenge). In pattern 1, retrograde conduction occurred exclusively over the AV node. Loss of HB-RB capture resulted in an increase in the S-A interval in all electrograms equal to the increase in the S-H interval, with no change in the atrial activation sequence. The H-A interval remained essentially the same (Figs 3 and 4 \blacklozenge). This response indicated that retrograde conduction was dependent on His bundle activation and not on local ventricular activation. This AVN/AVN pattern (retrograde AV nodal conduction during HB-RB capture/retrograde AV nodal conduction during HB-RB noncapture) was observed in all 53 patients with AVNRT and was not observed in any patient with a septal or right free wall AP (Table 1 \blacklozenge). However, this pattern was observed in 9 of 34 patients with a left free wall AP and in 6 of 9 patients with PJRT, in which retrograde AV nodal conduction masked the presence of retrograde AP conduction.



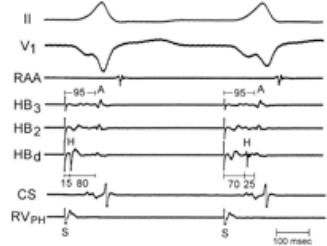
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Figure 5. Schema for interpretation of the response to para-Hisian pacing. See text for details. (S-A)_C indicates S-A interval during HB-RB capture; (S-A)_{NC}, S-A interval during HB-RB noncapture; (local V-A)_C, local VA interval recorded close to the AP during HB-RB capture; (local V-A)_{NC}, local VA interval recorded close to the AP during HB-RB noncapture; (H-A)_C, H-A interval recorded in the His bundle electrogram during HB-RB capture; and (H-A)_{NC}, H-A interval recorded in the His bundle electrogram during the HB-RB noncapture.

Table 1. Patterns of Response to Para-Hisian Pacing

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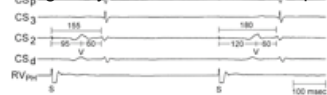
Patterns 2 and 3 resulted from retrograde conduction occurring exclusively over an AP. In pattern 2 (AP/AP pattern), the S-A interval was identical during HB-RB capture and noncapture (Fig 6 \blacklozenge), indicating that retrograde conduction was dependent on local ventricular activation and not on His bundle activation. In pattern 3, loss of HB-RB capture was associated with a delay in the timing of ventricular activation close to the AP. This resulted in an increase in the S-A interval in all electrograms, with no change in the atrial activation sequence (Figs 7 \blacklozenge and 8). The local VA interval, recorded close to the AP, remained approximately the same. The increase in S-A interval was less than the increase in the S-H interval. Therefore, the H-A interval shortened with loss of HB-RB capture, indicating that retrograde conduction could not be occurring over the AV node. Two mechanisms were identified for the delay in timing of ventricular activation close to the AP. First, activation of the His-Purkinje system resulted in earlier ventricular activation near some APs located far from the para-Hisian pacing site, such as left lateral or anterolateral APs (Fig 7 \blacklozenge). Second, decreasing the pacing output to lose HB-RB capture occasionally resulted in a small delay in ventricular activation close to the pacing site (Fig 8 \blacklozenge). Pattern 3 is referred to as the AP/AP_L pattern, where AP_L refers to a lengthening of the S-A interval with loss of HB-RB capture.



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Figure 6. Para-Hisian pacing demonstrating retrograde conduction only over an anteroseptal accessory AV pathway (AP/AP pattern). HB-RB capture in the left complex resulted in an S-H interval of 15 ms. Loss of HB-RB capture in the right complex resulted in a 55-ms increase in S-H interval to 70 ms. The S-A interval remained fixed at 95 ms and the atrial activation sequence remained identical, indicating that retrograde conduction was dependent on the timing of ventricular activation and not on the timing of retrograde His-bundle activation.

Figure 7. Para-Hisian pacing demonstrating retrograde conduction over a left free wall AP with S-A prolongation on loss of HB-RB capture (AP/AP_L pattern). HB-RB capture is present in the left complex (S-H=5 ms) and lost in the right complex (S-H increased to 70 ms with widening of the QRS complex). The retrograde atrial activation sequence is identical in the two complexes, with earliest atrial activation recorded from the coronary sinus (CS₂ electrogram). Loss of HB-RB capture was associated with a 25-ms increase in S-A from 155 to 180 ms caused by a 25-ms delay in the timing of ventricular activation close to the AP (S-V_{AP} increased from 95 to 120 ms). The local VA remained constant at 60 ms. The S-V interval in the Hb_d electrogram, close to the para-Hisian pacing site, was unchanged by the loss of HB-RB capture, indicating that the His-Purkinje system was responsible for earlier activation of the ventricle close to the AP (CS₂



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electrogram) during HB-RB capture. The prolongation in S-V_{AP} was due to the longer intraventricular conduction time without the aid of the Purkinje system. Note that the decrease in H-A interval from 150 to 110 ms without a change in the atrial activation sequence indicates that the AV node did not participate in retrograde atrial activation during either HB-RB capture or noncapture.

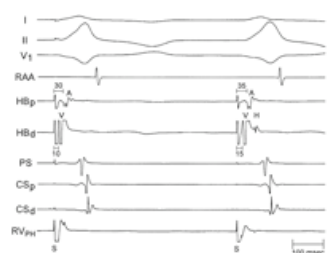


Figure 8. Para-Hisian pacing demonstrating retrograde conduction over an anteroseptal AP with slight prolongation in the S-A interval (AP/AP_L pattern). Loss of HB-RB capture (right complex) was associated with a 5-ms increase in S-A interval caused by a 5-ms delay in local ventricular activation (S-V_{AP} increased from 10 to 15 ms). Unlike Fig 7 \blacklozenge , this delay in local ventricular activation was not related to the loss of activation of the Purkinje system but was a result of a delay in ventricular activation at the pacing site as the pacing output was decreased to lose HB-RB capture. Note the short S-A caused by the close proximity of the ventricular pacing site to the AP (negligible S-V_{AP}). The short S-A interval resulting from conduction over the AP causes retrograde activation of the atrium before retrograde activation of the His bundle during loss of HB-RB capture.

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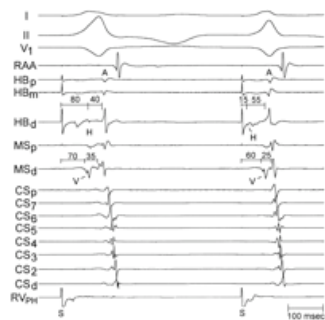
Patterns 2 and 3, indicating that retrograde conduction occurred exclusively over an AP, were observed in 75 of 147 patients with an AP (Table 1 \blacklozenge). However, retrograde AV nodal conduction was present after AP ablation in 32 of the 75 patients. In 25 of these 32 patients, the S-A interval during retrograde conduction over the AP was shorter than the S-A interval resulting from retrograde conduction over the AV node, even during HB-RB capture, either because the AP was located close to the para-Hisian pacing site (8 patients) or because retrograde AV nodal conduction occurred only over the slow AV nodal pathway (17 patients; Table 2 \blacklozenge). The remaining 7 patients had retrograde conduction over the fast AV nodal pathway after ablation, with an S-A interval during HB-RB capture that was shorter than the S-A interval resulting from retrograde conduction over the AP before ablation. This suggested that retrograde conduction over the fast AV nodal pathway was absent during para-Hisian pacing before ablation, possibly because of a change in autonomic tone or catheter trauma to the fast AV nodal pathway.²²

Table 2. Classification of Retrograde AV Nodal Conduction After Accessory Pathway Ablation in Patients Without Retrograde AV Nodal Conduction on Para-Hisian Pacing Before Ablation

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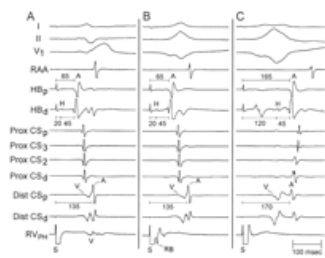
In patients in whom retrograde conduction occurred over both the AV node and an AP during para-Hisian pacing, the amount of atria activated by each of the two pathways (atrial fusion) was dependent on four variables: (1) S-H interval, (2) the retrograde conduction time over the AV node (H-A interval during HB-RB capture), (3) the intraventricular conduction time from the para-Hisian pacing site to the ventricular end of the accessory pathway (S-V_{AP}), and (4) the retrograde conduction time over the accessory pathway [(V-A)_{AP}]. The amount of atria activated by the AV node was greater during HB-RB capture (minimal S-H interval). Loss of HB-RB capture was associated with an increase in the amount of atria activated by the AP, resulting in a change in the retrograde atrial activation sequence. Therefore, a change in the retrograde atrial sequence with loss of HB-RB capture always indicated the presence of retrograde conduction over both an AP and the AV node (Fig 5 \blacklozenge). There were four such patterns (patterns 4 through 7).

In patterns 4 and 5, HB-RB capture was associated with activation of the atria exclusively by retrograde conduction over the AV node. In pattern 4, loss of HB-RB capture was associated with activation of the atria exclusively by the AP (AVN/AP pattern). Loss of HB-RB capture resulted in an increase in S-A interval and local VA interval in all electrograms, with the least increase occurring in the electrogram closest to the AP (Fig 9 \blacklozenge). The H-A interval shortened, indicating that the atrium near the AV node was activated by the AP before retrograde conduction over the AV node was complete (Fig 9 \blacklozenge). In pattern 5, loss of HB-RB capture resulted in activation of part of the atria by the AV node and part by the AP (AVN/fusion pattern). Loss of HB-RB capture was associated with an increase in S-A interval and local VA interval in all electrograms (Fig 10B and 10C \blacklozenge). However, the H-A interval remained constant, indicating that part of the atria was still activated by the AV node (Fig 10 \blacklozenge).



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Figure 9. Para-Hisian pacing demonstrating retrograde conduction over both a midseptal AP and the AV node (AVN/AP pattern). During loss of HB-RB capture (left complex), retrograde conduction occurred exclusively over the midseptal AP, as evidenced by the shortening in the H-A interval from 55 to 40 ms, whereas during HB-RB capture (right complex), retrograde conduction occurred exclusively over the AV node, as evidenced by the shortening in the local VA interval recorded in the electrogram close to the AP (MS_d) from 35 to 25 ms. Note the change in retrograde atrial activation sequence with loss of HB-RB capture, indicating the presence of retrograde conduction over both the AP and the AV node.



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Figure 10. Para-Hisian pacing demonstrating an AVN/fusion pattern in a patient with retrograde conduction over both a left anterolateral accessory AV pathway and the AV node. The pacing stimulus captured only the proximal right bundle branch in A, the proximal right bundle branch and local ventricular myocardium (HB-RB capture) in B, and only the local ventricular myocardium (HB-RB noncapture) in C. The H-A interval was constant (45 ms) in all three panels, indicating the presence of retrograde AV nodal conduction in all three pacing responses. C, The loss of HB-RB capture was associated with a change in the retrograde atrial activation sequence, indicating that parts of the atria were being activated by the AP. The S-A interval in the HB electrograms increased by 100 ms (65 to 165 ms), equal to the 100-ms increase in the S-H interval (20 to 120 ms). However, the S-A interval in the DistCS_p electrogram, close to the AP, increased by only 35 ms (135 to 170 ms). The increase in S-A interval being less than the increase in S-H interval indicates the emergence of retrograde conduction over an AP. Note that the presence of right bundle branch block did not prevent early retrograde activation of the His bundle during HB-RB capture (A and B), indicating that the block was located distal to the para-Hisian pacing site.

In patterns 6 and 7, HB-RB capture resulted in atrial activation over both the AV node and the AP. In pattern 6, loss of HB-RB capture resulted in activation of the atrium exclusively by the AP (fusion/AP pattern). Loss of HB-RB capture was associated with no change in the S-A or local VA interval recorded near the AP (Fig 11 \blacklozenge). In the His bundle electrogram, the S-A interval increased, but not as much as the S-H interval, leading to a decrease in the H-A interval, indicating that the atrial myocardium in that region was no longer activated by the AV node (Fig 11 \blacklozenge). In pattern 7, the atria continued to be activated by both the AV node and the AP during loss of HB-RB capture, with a greater amount of the atria activated by the AP than during HB-RB capture (fusion/fusion pattern). Like pattern 6, loss of HB-RB capture was associated with minimal change in the S-A or local VA interval recorded close to the AP, but the H-A interval remained essentially the same, indicating that part of the atria was still activated by the AV node (Fig 12 \blacklozenge).

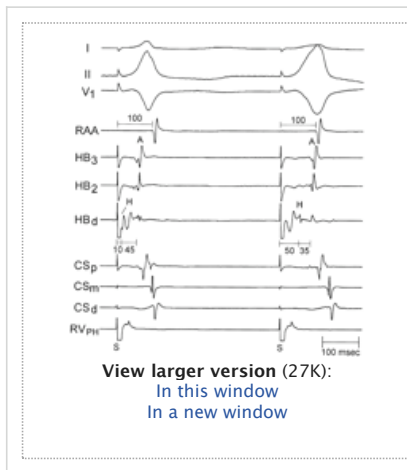


Figure 11. Para-Hisian pacing demonstrating a fusion/AP pattern in a patient with a right anterior accessory AV pathway. The S-A interval recorded from the right atrial appendage (RAA) remained constant (100 ms), with loss of HB-RB capture (right complex), indicating that this region of the atria was activated by the AP during HB-RB capture and during loss of HB-RB capture. With loss of HB-RB capture, the H-A interval shortened by 10 ms, from 45 to 35 ms, with a change in the atrial activation sequence, indicating loss of retrograde atrial activation by the AV node. Therefore, the atrium was activated by both the AV node and the AP (fusion) during HB-RB capture and by only the AP during HB-RB noncapture.

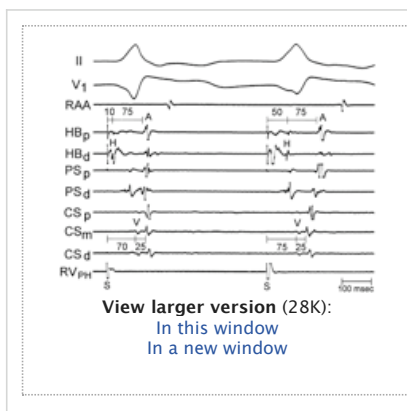


Figure 12. Para-Hisian pacing demonstrating a fusion/fusion pattern in a patient with a left lateral accessory AV pathway. HB-RB capture was present in the left complex (S-H=10 ms) and absent in the right complex (S-H=50 ms). The loss of HB-RB capture was associated with prolongation of the S-A interval in the His bundle electrogram equal to the prolongation in S-H interval (constant H-A interval of 75 ms), indicating retrograde activation of the atrial septum by the AV node during HB-RB capture and noncapture. Loss of HB-RB capture was associated with a shift in the retrograde atrial activation sequence, with a relatively constant local VA interval (25 ms) recorded in the CS_m electrogram, indicating that this region of the left atrium was activated by the AP during HB-RB capture and noncapture. Therefore, the atria were activated by both the AV node and the AP during both HB-RB capture and noncapture. The degree of fusion changed, with a larger amount of the atria being activated by the AP during loss of HB-RB capture.

After AP Ablation

After AP ablation, VA conduction was present in 104 of the 147 patients. Para-Hisian pacing produced an AVN/AVN pattern (pattern 1) in all 104 patients, confirming that retrograde conduction occurred exclusively over the AV node.

Discussion

Para-Hisian pacing (with HB-RB capture and noncapture) correctly identified the presence of retrograde AP conduction in all 88 patients with an anteroseptal, midseptal, or posteroseptal AP and all 16 patients with a right free wall AP but in only 25 of 34 patients with a left free wall AP and in 3 of 9 patients with PJRT (Table 1 \blacklozenge). In the remaining 15 patients, retrograde AP conduction was masked by retrograde AV nodal conduction. Para-Hisian pacing also correctly identified the presence of only retrograde AV nodal conduction in all 53 patients with AVNRT.

Concept of Para-Hisian Pacing

The para-Hisian pacing site is unique because it is anatomically close but electrically far from the His bundle. Para-Hisian pacing at high output simultaneously captures the His bundle or proximal right bundle branch, as well as the adjacent ventricular myocardium. At lower output, direct HB-RB capture is lost and retrograde activation of the His bundle is delayed because the His bundle and right bundle branch are insulated from the adjacent myocardium and the peripheral inputs to the Purkinje system are located far from the para-Hisian pacing site. By maintaining local ventricular capture while intermittently losing HB-RB capture, retrograde conduction can be classified as dependent on the timing of local ventricular activation (AP), His bundle activation (AV node), or both (fusion).

The response to para-Hisian pacing can be determined by comparing the following three variables between HB-RB capture and noncapture: (1) the atrial activation sequence; (2) the S-A interval recorded at multiple sites, including close to the site of earliest atrial activation during supraventricular tachycardia; and (3) the H-A interval recorded in the His bundle electrogram (Fig 13 \blacklozenge). An identical retrograde atrial activation sequence indicates that retrograde conduction is occurring over the same system during HB-RB capture and noncapture (AP or AV node). If the S-A or local VA interval at any site remains essentially the same and the H-A interval shortens, retrograde conduction is occurring only over an AP. If the S-A interval increases in all electrograms while the H-A interval remains essentially the same, retrograde conduction is occurring only over the AV node. A change in retrograde atrial activation sequence with loss of HB-RB capture indicates the presence of retrograde conduction over both an AP and the AV node. The degree of change in the retrograde atrial activation sequence with loss of HB-RB capture, ie, magnitude of the increase in the amount of atria activated by the AP (patterns 4 through 7 in Fig 5 \blacklozenge), is dependent on four variables: (1) the magnitude of the delay in retrograde activation of the His bundle (increase in S-H interval), (2) the retrograde conduction time over the AV node (H-A interval), (3) the intraventricular

conduction time from the para-Hisian pacing site to the ventricular end of the accessory pathway ($S-V_{AP}$), and (4) the retrograde conduction time over the accessory pathway $[(V-A)_{AP}]$. The first two variables (S-H plus H-A) form the S-A interval resulting from retrograde conduction over the AV node, and the latter two variables $[S-V_{AP}$ plus $(V-A)_{AP}]$ form the retrograde conduction time over the AP.

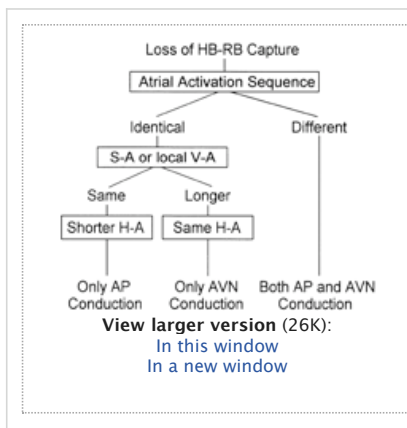


Figure 13. Algorithm for interpreting the response to para-Hisian pacing. With loss of HB-RB capture, a change in the retrograde atrial activation sequence indicates that the retrograde conduction occurs over both an AP and the AV node. An identical atrial activation sequence indicates that retrograde conduction is occurring over the same pathway during HB-RB capture and HB-RB noncapture. During loss of HB-RB capture, a constant S-A interval (or local VA interval) combined with shortening of the H-A interval indicates that retrograde conduction is occurring only over an AP. Lengthening of the S-A interval combined with a constant H-A interval indicates that retrograde conduction is occurring only over the AV node.

For anteroseptal APs, $S-V_{AP}$ is short (Fig 8♦). For APs located progressively farther from the para-Hisian pacing site, $S-V_{AP}$ increases progressively. This is not a significant factor for midseptal, posteroseptal, or most right free wall APs (Fig 11♦). Retrograde AP conduction was manifest with loss of HB-RB capture for all septal and right free wall APs in this study (Table 1♦). However, for left free wall APs, which are located far from the pacing site, $S-V_{AP}$ can be sufficiently long to have the entire atria activated by the AV node, even during loss of HB-RB capture. In this study, para-Hisian pacing produced an AVN/AVN pattern, failing to identify the presence of retrograde AP conduction in 9 of 34 patients with a left free wall AP (because of long $S-V_{AP}$) and 6 of 9 patients with PJRT [because of long $(V-A)_{AP}$]. Therefore, the location of the AP as well as the retrograde conduction time over the AP must be taken into account when we interpret the response of para-Hisian pacing.

Para-Hisian pacing produced an AP/AP or AP/AP_L pattern, failing to identify the presence of retrograde AV nodal conduction in 32 patients who were found to have retrograde AV nodal conduction after ablation of the AP. The occurrence of only retrograde AP conduction during HB-RB capture indicates that the H-A interval resulting from the conduction over the AV node was longer than the sum of $S-V_{AP}$ and $(V-A)_{AP}$. In 8 patients, the AP was located very close to the para-Hisian pacing site, making $S-V_{AP}$ negligible, and the $(V-A)_{AP}$ was shorter than the H-A interval (Table 2♦). In 17 patients, the H-A interval was long as a result of retrograde AV nodal conduction only over the slow AV nodal pathway. In the remaining 7 patients, retrograde fast AV nodal pathway conduction was absent during para-Hisian pacing before ablation but present after ablation. The absence of retrograde fast AV nodal pathway conduction before ablation may be explained by a difference in autonomic tone or catheter trauma to the fast pathway.²²

Technical Aspects

Para-Hisian pacing is optimized when very closely spaced bipolar electrodes are used for recording the His bundle electrogram. A close bipolar electrode has a small recording range. Therefore, the paced ventricular impulse propagates outside the recording range of the bipolar electrode relatively quickly, producing an early narrow ventricular potential in the His bundle electrogram. The short early ventricular potential facilitates identification of the retrograde His bundle and atrial potentials.

Para-Hisian pacing is also facilitated by use of a closely spaced bipolar electrode for pacing. The close bipolar pacing electrode results in direct stimulation of a smaller region, enhancing the ability to lose HB-RB capture as the pacing output is decreased or as the pacing electrode moves slightly with respiration.

For APs located far from the para-Hisian pacing site, especially left free wall APs, it is important to record atrial activation close to the suspected site of an AP. If para-Hisian pacing produces an AVN/fusion pattern or fusion/fusion pattern, this may appear as exclusive retrograde AV nodal conduction (AVN/AVN pattern) if only the His bundle and high right atrial electrograms are examined (Figs 10 and 12♦♦).

In 2 patients with very proximal right bundle branch block (resulting from a previous unsuccessful attempt at catheter ablation of an anteroseptal AP), right bundle branch capture failed to produce early retrograde activation of the His bundle, limiting the use of para-Hisian pacing in these patients. This suggests that HB-RB capture actually represents capture of the proximal right bundle branch and not His bundle capture. This is supported by the observation that, during HB-RB capture, the His bundle potential was often recorded 10 to 20 ms after the pacing stimulus (Figs 3, 4, 6, and 9 through 12♦♦♦♦♦♦). Importantly, para-Hisian pacing was performed successfully in many patients with more distal right bundle branch block, ie, distal to the para-Hisian pacing site (Fig 10♦).

Use of Para-Hisian Pacing in Catheter Ablation Studies

In this study, retrograde conduction was present after AP ablation in 104 of 147 patients. Para-Hisian pacing confirmed that the residual retrograde conduction was occurring over the AV node in all 104 patients. Combined with ventricular pacing close to the site of the AP,^{17 18 19} para-Hisian pacing may be the preferred method to assess whether retrograde conduction over an AP has been eliminated by an ablation procedure. Other techniques that have been used include ventricular extrastimulus testing (to produce functional retrograde right bundle branch block to delay retrograde activation of the His bundle and determine whether retrograde atrial activation follows the timing of ventricular activation or His bundle activation) and the administration of adenosine²³ or other agents likely to produce selective retrograde block in the AV node. However, residual AP conduction after ablation may have a relatively long refractory period causing retrograde block at long extrastimulus coupling intervals (preventing differentiation between retrograde AP and AV nodal conduction). Adenosine may produce retrograde block in AP conduction, especially after injury.²⁴ In addition, adenosine may not produce retrograde AV nodal block in some patients.²⁴ The response to para-Hisian pacing is independent of the conduction properties of the AP, since this can be performed at long ventricular pacing cycle lengths.

Selected Abbreviations and Acronyms

AP =accessory AV pathway
AV =atrioventricular
AVNRT =AV nodal reentrant tachycardia
H-A =His bundle-atrial
HB-RB =His bundle or proximal right bundle branch
PJRT =permanent form of junctional reciprocating tachycardia
S-A =stimulus-atrial
S-H =stimulus-His bundle
S-V_{AP} =interval from the pacing stimulus to the ventricular potential recorded in the electrogram closest to the AP
VA =ventriculoatrial
(V-A)_{AP}=interval from the ventricular potential to the atrial potential recorded in the electrogram closest to the AP

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References

1. Warin JF, Haissaguerre M, Lemetayer P, Guillem JP, Blanchot P. Catheter ablation of accessory pathways with a direct approach: results in 35 patients. *Circulation*. 1988;78:800-815.[\[Abstract/Free Full Text\]](#)
2. Jackman WM, Wang X, Friday KJ, Roman CA, Moulton KP, Beckman KJ, McClelland JH, Twidale N, Hazlitt HA, Prior MI, Margolis PD, Calame JD, Overholt E, Lazzara R. Catheter ablation of accessory atrioventricular pathways (Wolff-Parkinson-White syndrome) by radiofrequency current. *N Engl J Med*. 1991;324:1605-1611.[\[Medline\]](#) [\[Order article via Infotrieve\]](#)
3. Calkins H, Sousa J, El-Atassi R, Rosenheck S, Buitelir M, Kou WH, Kadish AH, Langberg JJ, Morady F. Diagnosis and cure of the Wolff-Parkinson-White syndrome or paroxysmal supraventricular tachycardias during a single electrophysiologic test. *N Engl J Med*. 1991;324:1612-1618.[\[Medline\]](#) [\[Order article via Infotrieve\]](#)
4. Kuck KH, Schluter M, Geiger M, Siebels J, Duckeck W. Radiofrequency current catheter ablation of accessory atrioventricular pathways. *Lancet*. 1991;337:1557-1561.[\[Medline\]](#) [\[Order article via Infotrieve\]](#)
5. Lesh MD, Van Hare GF, Schamp DJ, Chien W, Lee MA, Griffin JC, Langberg JJ, Cohen TJ, Lure KG, Scheinman MM. Curative percutaneous catheter ablation using radiofrequency energy for accessory pathways in all locations: results in 100 consecutive patients. *J Am Coll Cardiol*. 1992;19:1303-1309.[\[Abstract\]](#)
6. Schwartz JF, Tracy CM, Fletcher RD. Radiofrequency endocardial catheter ablation of accessory atrioventricular pathway atrial insertion sites. *Circulation*. 1993;87:487-499.[\[Abstract/Free Full Text\]](#)
7. Saul JP, Hulse JE, Wang D, Weber AT, Rhodes LA, Lock JE, Walsh EP. Catheter ablation of accessory atrioventricular pathways in young patients: use of long vascular sheaths, the transseptal approach and a retrograde left posterior parallel approach. *J Am Coll Cardiol*. 1993;21:571-583.[\[Abstract\]](#)
8. Kay GN, Epstein AE, Dailey SM, Plumb VJ. Role of radiofrequency ablation in the management of supraventricular arrhythmias: experience in 760 consecutive patients. *J Cardiovasc Electrophysiol*. 1993;4:371-389.[\[Medline\]](#) [\[Order article via Infotrieve\]](#)
9. Coumel P, Attuel P. Reciprocating tachycardia in overt and latent preexcitation: influence of functional bundle branch block on the rate of the tachycardia. *Eur J Cardiol*. 1974;1:423-436.[\[Medline\]](#) [\[Order article via Infotrieve\]](#)
10. Spurrell RAJ, Krikler DM, Sowton E. Retrograde invasion of the bundle branches producing aberration of the QRS complex during supraventricular tachycardia studied by programmed electrical stimulation. *Circulation*. 1974;50:487-495.[\[Abstract/Free Full Text\]](#)
11. Zipes DP, DeJoseph RL, Rothbaum DA. Unusual properties of accessory pathways. *Circulation*. 1974;49:1200-1211.[\[Abstract/Free Full Text\]](#)
12. Neuss H, Schlepper M, Thormann J. Analysis of re-entry mechanisms in three patients with concealed Wolff-Parkinson-White syndrome. *Circulation*. 1975;51:75-81.[\[Abstract/Free Full Text\]](#)
13. Wellens HJJ, Durrer D. The role of an accessory atrioventricular pathway in reciprocal tachycardia: observations in patients with and without the Wolff-Parkinson-White syndrome. *Circulation*. 1975;52:58-72.[\[Abstract/Free Full Text\]](#)
14. Tonkin AM, Gallagher JJ, Svenson RH, Wallace AG, Sealy WC. Antegrade block in accessory pathways with retrograde conduction in reciprocating tachycardia. *Eur J Cardiol*. 1975;3:143-152.[\[Medline\]](#) [\[Order article via Infotrieve\]](#)
15. Gallagher JJ, Gilbert M, Svenson RH, Sealy WC, Kasell J, Wallace AG. Wolff-Parkinson-White syndrome: the problem, evaluation and surgical correction. *Circulation*. 1975;51:767-785.[\[Abstract/Free Full Text\]](#)
16. Sellers TD Jr, Gallagher JJ, Cope GD, Tonkin AM, Wallace AG. Retrograde atrial preexcitation following premature ventricular beats during reciprocating tachycardia in the Wolff-Parkinson-White syndrome. *Eur J Cardiol*. 1976;4:283-294.[\[Medline\]](#) [\[Order article via Infotrieve\]](#)
17. Benditt DG, Benson DW Jr, Dunnigan A, Gornick CC, Ring WS, Almquist A, Tobler HG, Milstein S. Role of extrastimulus site and tachycardia cycle length in inducibility of atrial preexcitation by premature ventricular stimulation during reciprocating tachycardia. *Am J Cardiol*. 1987;60:811-819.[\[Medline\]](#) [\[Order article via Infotrieve\]](#)
18. Martinez-Alday JD, Almendral J, Arenal A, Ormaetxe JM, Pastor A, Villacastin JP, Medina O, Peinado R, Delcan JL. Identification of concealed posteroseptal

Kent pathways by comparison of ventriculoatrial intervals from apical and posterobasal right ventricular sites. *Circulation*. 1994;89:1060-1067.[[Abstract/Free Full Text](#)]

19. Singh A, Beckman K, McClelland J, Wang X, Nakagawa H, Heidbuchel H, Arruda M, Thibault B, Abdalla I, Gonzalez M, Sweiden R, Widman L, Lazzara R, Jackman WM. Selection of optimal RV pacing site for mapping retrograde accessory pathway conduction. *Pacing Clin Electrophysiol*. 1994;17:741. Abstract.

20. Jackman WM, Beckman KJ, McClelland J, Wang X, Hazlitt A, Moulton K, Prior M, Twidale N, Calame J, Lazzara R. Para-Hisian RV pacing for differentiating retrograde conduction over septal accessory pathway and AV node. *Pacing Clin Electrophysiol*. 1991;14:670. Abstract.

21. Jackman WM, Beckman KJ, McClelland JH, Wang X, Friday KJ, Roman CA, Moulton KP, Twidale N, Hazlitt HA, Prior MI, Oren J, Overholt ED, Lazzara R. Treatment of supraventricular tachycardia due to atrioventricular nodal reentry by radiofrequency catheter ablation of slow-pathway conduction. *N Engl J Med*. 1992;327:313-318.[[Medline](#)] [[Order article via Infotrieve](#)]

22. Chiang CE, Chen SA, Wu TJ, Yang CJ, Cheng CC, Wang SP, Chiang BN, Chang MS. Incidence, significance, and pharmacological response of catheter-induced mechanical trauma in patients receiving radiofrequency ablation for supraventricular tachycardia. *Circulation*. 1994;90:1847-1854.[[Abstract/Free Full Text](#)]

23. Keim S, Curtis AB, Epstein M, Staples ED, Belardinelli L. Adenosine induced antegrade and retrograde atrioventricular block as a marker for successful surgical ablation of accessory pathways. *Pacing Clin Electrophysiol*. 1990;13:503. Abstract.

24. Engelstein ED, Wilber D, Wadas M, Lippman N, Stein KM, Lerman BB. Limitations of adenosine in assessing efficacy of radiofrequency catheter ablation of accessory pathways. *Circulation*. 1993;86(suppl I):I-295. Abstract.

Articles citing this article

The characteristics of verapamil-sensitive idiopathic left ventricular tachycardia combined with a left accessory pathway and the effect of radiofrequency catheter ablation

Europace. 2011;0:eur330v1-eur330,

[Abstract](#) | [Full Text](#) | [PDF](#)

Determination of Inadvertent Atrial Capture During Para-Hisian Pacing

Circ Arrhythm Electrophysiol. 2011;4:510-514,

[Abstract](#) | [Full Text](#) | [PDF](#)

Atrioventricular Nodal Reentrant Tachycardia

Circulation. 2010;122:831-840,

[Full Text](#) | [PDF](#)

Tachycardia and the AV Nodal Region: Guilt by Association?

Circ Arrhythm Electrophysiol. 2010;3:2-6,

[Full Text](#) | [PDF](#)

Paradoxical increase of stimulus to atrium interval despite His-bundle capture during para-Hisian pacing

Europace. 2009;11:1702-1704,

[Abstract](#) | [Full Text](#) | [PDF](#)

Entrainment from the para-Hisian region for differentiating atrioventricular node reentrant tachycardia from orthodromic atrioventricular reentrant tachycardia

Europace. 2008;10:1205-1211,

[Abstract](#) | [Full Text](#) | [PDF](#)

Ventricular Pump Function and Pacing: Physiological and Clinical Integration

Circ Arrhythm Electrophysiol. 2008;1:127-139,

[Full Text](#) | [PDF](#)

Catheter Ablation of Paroxysmal Supraventricular Tachycardia

Circulation. 2007;116:2465-2478,

[Full Text](#) | [PDF](#)

Intermittent fasciculoventricular pathway: ECG and electrophysiologic findings, clinical implications

Europace. 2007;9:702-705,

[Abstract](#) | [Full Text](#) | [PDF](#)

A New Paradigm for Physiologic Ventricular Pacing

J Am Coll Cardiol. 2006;47:282-288,

[Abstract](#) | [Full Text](#) | [PDF](#)

Classification and differential diagnosis of atrioventricular nodal re-entrant tachycardia.

Europace. 2006;8:29-36,

[Abstract](#) | [Full Text](#) | [PDF](#)

Characterization of subforms of AV nodal reentrant tachycardia

Europace. 2004;6:316-329,

[Abstract](#) | [Full Text](#) | [PDF](#)

Coronary Sinus-Ventricular Accessory Connections Producing Posteroseptal and Left Posterior Accessory Pathways: Incidence and Electrophysiological Identification

Circulation. 2002;106:1362-1367,

[Abstract](#) | [Full Text](#) | [PDF](#)

Multiple AV nodal pathways in patients with AV nodal reentrant tachycardia -- more common than expected?

Europace. 2002;4:375-382,

[Abstract](#) | [PDF](#)

Differentiation of atypical atrioventricular node re-entrant tachycardia from orthodromic reciprocating tachycardia using a septal accessory pathway by the response to ventricular pacing

J Am Coll Cardiol. 2001;38:1163-1167,

[Abstract](#) | [Full Text](#) | [PDF](#)

Reversing the Direction of Paced Ventricular and Atrial Wavefronts Reveals an Oblique Course in Accessory AV Pathways and Improves Localization for Catheter Ablation

Circulation. 2001;104:550-556,

[Abstract](#) | [Full Text](#) | [PDF](#)

Diagnostic value of tachycardia features and pacing maneuvers during paroxysmal supraventricular tachycardia

J Am Coll Cardiol. 2000;36:574-582,

[Abstract](#) | [Full Text](#) | [PDF](#)

Long-Term His-Bundle Pacing and Cardiac Function

Circulation. 2000;101:836-837,

[Full Text](#) | [PDF](#)