

Interventional Cardiac Electrophysiology

Catheter Ablation of Paroxysmal Supraventricular Tachycardia

Hiroshi Nakagawa, MD, PhD; Warren M. Jackman, MD

Radiofrequency (RF) catheter ablation became first-line therapy for supraventricular tachycardia in patients with Wolff-Parkinson-White syndrome^{1–5} and atrioventricular (AV) nodal reentrant tachycardia (AVNRT)^{6–11} in the early 1990s. High ablation success (>90%) and low complication rates (<5%) were found by numerous investigators.^{1–11} This article describes these ablation techniques with a focus on approaches to ablation of the forms of accessory AV pathways and AVNRT that are difficult to ablate, as well as the less common variants.

Part 1: Catheter Ablation of Accessory AV Pathways

Between 1989 and 2005, we performed catheter ablation at the University of Oklahoma Health Sciences Center in 1702 patients with 1728 accessory pathways. Acute ablation success was achieved in 1707 (98.8%) of the 1728 accessory pathways. At the 1-year follow-up, conduction (ventricular preexcitation and/or tachycardia) recurred in 92 (5.4%) of the 1707 accessory pathways that had achieved acute success. The recurrence rate was highest for anteroseptal accessory pathways (17 of 116, 14.7%), intermediate for posteroseptal accessory pathways (48 of 476, 10.1%), and lowest for left and right free-wall accessory pathways (17 of 756, 2.2%; and 10 of 359, 2.8%, respectively). Successful repeat ablation was achieved in 71 of the 92 accessory pathways, for a 1-year success of 1686 (97.6%) of the total 1728 accessory pathways.

Of the 1702 patients, 904 (53%) had undergone 1 to 4 prior failed catheter and/or surgical ablation procedures at other institutions. From our experience in these 904 patients, we classify the causes for accessory pathway ablation failure into 3 groups: accessory pathway localization error (60%); unusual and unsuspected accessory pathway location (5%); and technical difficulties, including locations that are difficult to access with the ablation catheter, ablation at sites of low blood flow (limiting RF power), and ablation at sites associated with a high risk of injury to a coronary artery or the AV node (35%).

Causes of Accessory Pathway Ablation Failure

Mapping/Localization Error

The most common cause of ablation failure is incorrect localization of the accessory pathway, usually resulting from

an oblique course. Accessory pathways have been thought to course perpendicular to the AV groove. Therefore, the site exhibiting the shortest local ventriculoatrial (VA) interval during retrograde accessory pathway conduction (orthodromic AV reentrant tachycardia or ventricular pacing) or the site with the shortest local AV interval during antegrade accessory pathway conduction has often been considered the optimal target for ablation.^{12–15} However, most accessory pathways have an oblique course,^{16,17} which can shift the sites of shortest local VA or AV interval away from the accessory pathway (Figure 1A through 1F). With an oblique course, a ventricular wave front propagating from the direction of the ventricular end of the accessory pathway (concurrent direction) produces an artificially short local VA interval at the site of earliest atrial activation (Figure 1A and 1C). Because ventricular and accessory pathway activation (AP) is propagating parallel and simultaneously, the ventricular potential overlaps and masks the AP potential and often overlaps the atrial potential near the atrial end of accessory pathway, masking earliest atrial activation (electrograms CS_d through CS₃ in Figure 1E). The shortest local VA interval often is recorded beyond the atrial end of the accessory pathway (CS_d and CS₂ in Figure 1E), where ablation is unlikely to be successful.

Reversing the direction of the ventricular wave front (countercurrent direction) increases the local VA interval all along the accessory pathway, exposing the AP potential and atrial activation sequence, because the ventricular wave front passes the accessory pathway before reaching and activating its ventricular end (Figure 1B, 1D, and 1F).

During atrial pacing, a concurrent atrial wave front (from the direction of the atrial insertion) shortens the local AV interval at the site of earliest ventricular activation (local AV) and produces overlapping atrial and ventricular potentials, often masking the AP potential and site of earliest ventricular activation. The shortest local AV interval often is recorded beyond the ventricular end of the accessory pathway, where ablation is unlikely to be successful. Reversing the direction of the atrial wave front (countercurrent direction) lengthens the local AV and exposes the AP potential and ventricular activation sequence. The presence of an oblique course of accessory pathways also can be demonstrated histologically.¹⁷

From the Cardiac Arrhythmia Research Institute and Department of Medicine, University of Oklahoma Health Sciences Center, Oklahoma City. Correspondence to Hiroshi Nakagawa, MD, PhD, Cardiac Arrhythmia Research Institute, University of Oklahoma Health Sciences Center, 1200 Everett Dr, ET-6E103, Oklahoma City, OK 73104. E-mail hiroshi-nakagawa@ouhsc.edu (*Circulation*. 2007;116:2465–2478.)

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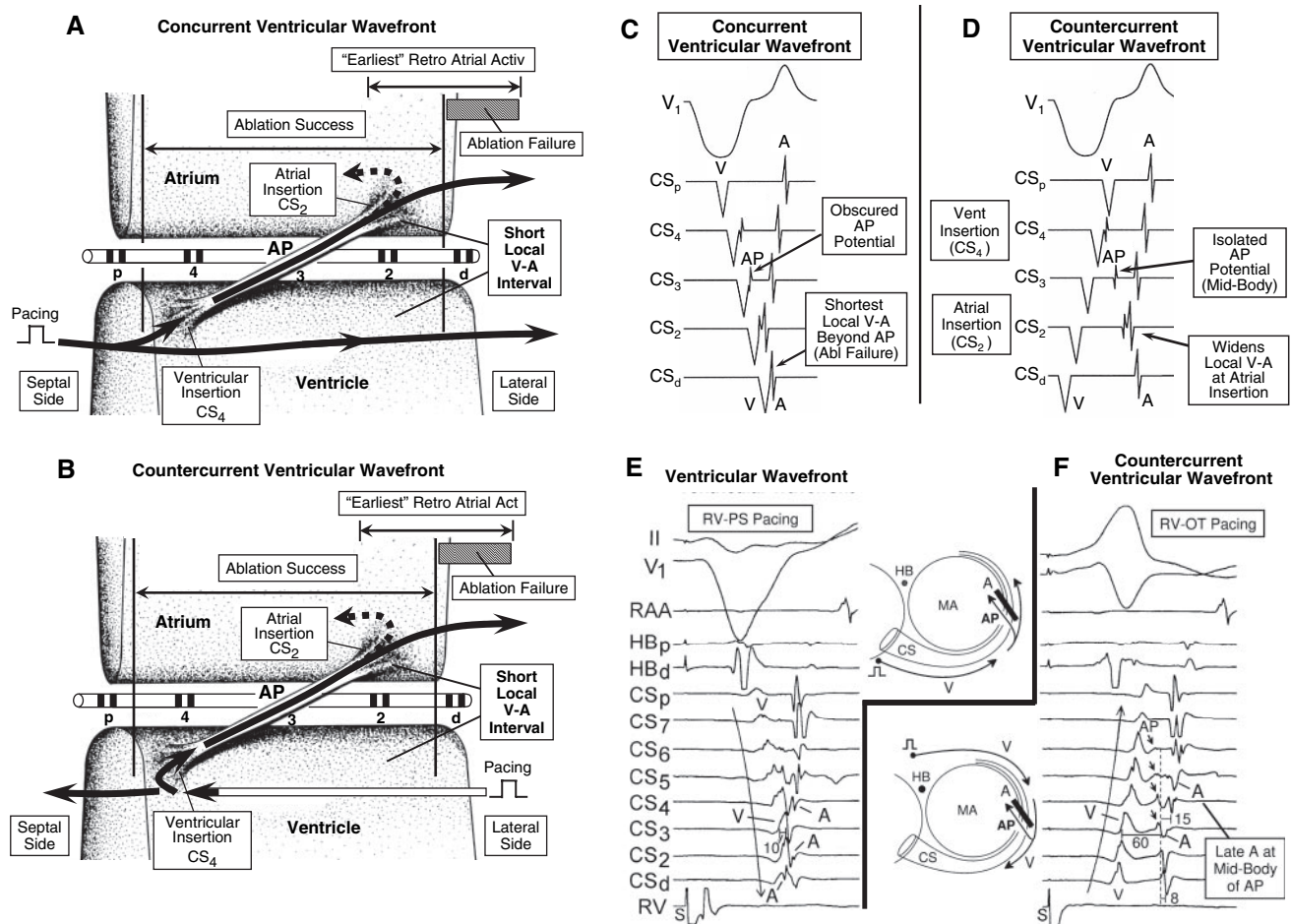


Figure 1. Effects of the oblique course in a left free-wall accessory pathway on the timing of ventricular (V), atrial (A), and AP potentials by reversing the direction of the ventricular wave front. A through D, Schematic representations. E and F, Recordings from a patient with a left lateral accessory pathway. Reversing the ventricular wave front from the concurrent direction (E; posteroseptal basal RV pacing [RV-PS]) to the countercurrent direction (F; distal RV outflow tract pacing [RV-OT]) increased the local VA interval at the site in the coronary sinus of earliest atrial activation (electrogram CS₃) from 10 to 60 ms and exposed the AP potential. The ventricular insertion (left) was located 15 mm septal to the atrial insertion (right). RAA indicates right atrial appendage. C through F, Modified with permission from Otomo et al.¹⁶ Copyright © 2006, The American Heart Association.

The optimal ablation site is near the middle of the accessory pathway ("midbody"), which is identified by pacing the atrium or ventricle on either side of the accessory pathway. One direction will widely separate the atrial and ventricular potentials (countercurrent wave front) and unmask an isolated AP potential (isoelectric segment between the AP potential and the atrial and ventricular potentials), both verifying the presence of an oblique course and localizing the midbody of the accessory pathway. An isolated AP potential can occur only with an oblique course. Otherwise, the AP potential would be fused with the atrial and ventricular potentials.

During retrograde conduction, atrial activation is misleadingly late near the midbody of the accessory pathway at the ideal ablation site (electrograms CS₄ through CS₆ in Figure 1F). The late activation time at the midbody is due to conduction delay produced by the reversal of the direction of atrial activation at the atrial insertion of accessory pathway (Figure 1A and 1B). In Figure 2F, retrograde atrial activation in the coronary sinus (CS) just a single bipolar electrode toward the midbody (CS₄) was recorded 15 ms after activa-

tion at the atrial insertion of accessory pathway (CS₃). In contrast, atrial activation propagates very rapidly away from the accessory pathway when traveling in the same direction as the accessory pathway (CS₂ in Figure 1F). Because of this rapid conduction and the relatively wide recording range of the 4-mm-tip electrode generally used for ablation, "earliest retrograde atrial activation" is recorded over a region extending from the atrial insertion to >5 mm beyond the atrial insertion (Figure 1A and 1B). This explains the ≥50% failure rate for RF applications targeting earliest atrial activation.^{12,13,18} Similarly, during antegrade accessory pathway conduction, ventricular activation is late near the midbody of the accessory pathway (Figure 1D).

An AP potential can be recorded in 89% of patients with accessory pathways by ventricular or atrial pacing from the side producing the longer local VA or local AV interval, respectively.¹⁶ In the absence of an AP potential, ablation should be targeted at the site of earliest retrograde atrial activation or earliest antegrade ventricular activation recorded from the unfiltered unipolar electrogram.¹⁹ The anat-

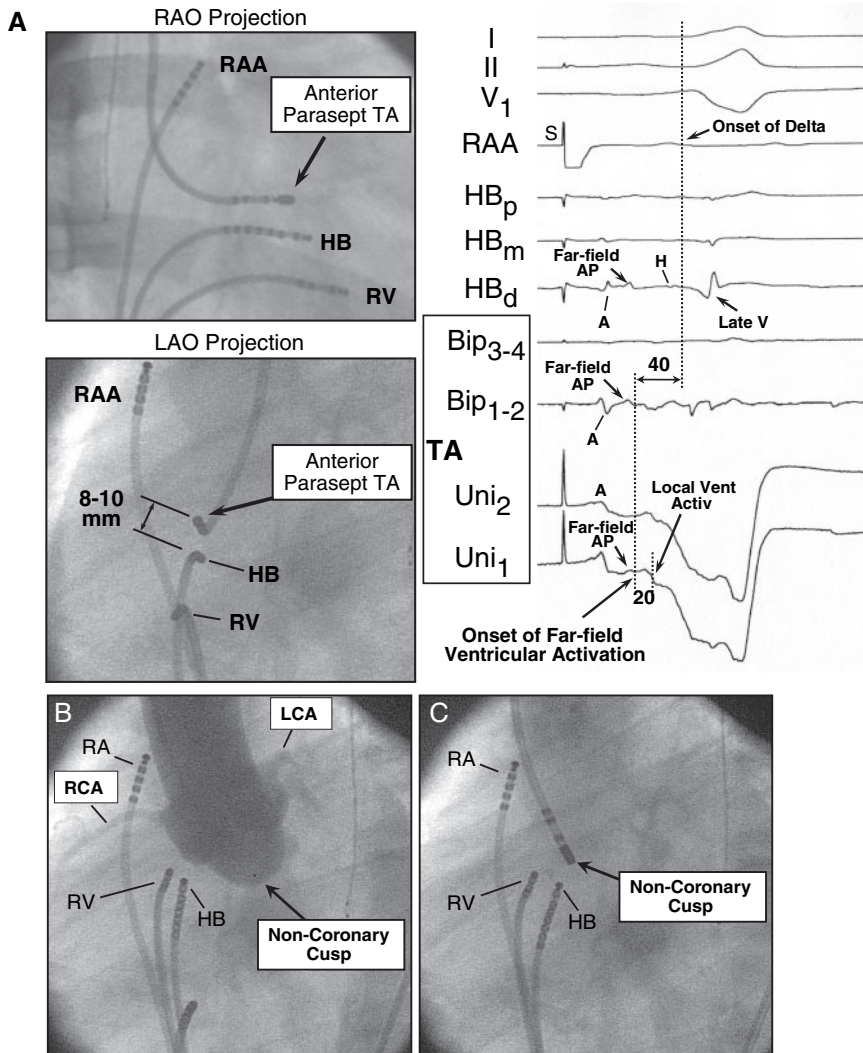


Figure 2. Ablation of an epicardial anteroseptal accessory pathway. A, Mapping the tricuspid annulus (TA) during right atrial appendage (RAA) pacing showing far-field ventricular activation beginning 40 ms before the delta wave but local ventricular activation beginning only 20 ms before the delta wave. B, Aortogram in the left anterior oblique projection. C, Mapping catheter in the noncoronary cusp of the aortic valve. Note close proximity to the HB catheter and RV catheter at the anterior paraseptal TA region. D and E, Validation of antegrade AP potential recorded in the noncoronary cusp (Ao_a) using ventricular extrastimuli. F, A single RF application in the noncoronary cusp at a site recording a large, sharp AP potential from the tip unipolar electrogram (Uni₁) eliminates accessory pathway conduction without affecting AV nodal conduction (no prolongation of A-H interval). RCA indicates right coronary artery; LCA, left coronary artery.

my of these pathways is usually less clear, and they require a median of 4.5 RF applications for ablation compared with a median of 1 RF application when an AP potential is recorded.¹⁶

Because all isolated potentials are not AP potentials, it is helpful to verify AP potentials before ablation. The accessory pathway origin of an antegrade AP potential can be verified by use of ventricular extrastimuli.^{20,21} A late ventricular extrastimulus advances the local ventricular potential without advancing the AP potential, differentiating the AP potential from local ventricular activation (Figure 2). An earlier ventricular extrastimulus advances the AP potential without advancing the local atrial potential, differentiating the AP potential from local atrial activation (Figure 2). A retrograde AP potential is differentiated from the local atrial and ventricular potentials by use of atrial extrastimuli. A late atrial extrastimulus advances the local atrial potential without advancing the AP potential, differentiating the AP potential from local atrial activation. An earlier atrial extrastimulus advances the AP potential without advancing the local ventricular potential, differentiating the AP potential from local ventricular activation.

For ablation, the catheter is positioned to record a large, sharp AP potential from the unipolar electrogram recorded from the ablation tip electrode (Figure 2). The unipolar electrogram is important because a sharp bipolar AP potential can be generated from the second (nonablation) electrode (Figure 3). We use an electrode in the inferior vena cava (25 cm from the tip of a right atrial catheter) as the reference electrode for unipolar recordings (Ablation electrode is the positive pole; inferior vena cava electrode is the negative pole; and filter settings of 1 to 500 Hz reduce baseline drift during respiration).

Unusual and Unexpected Accessory Pathway Locations

Accessory pathways are occasionally located at sites that are unexpected and not usually explored during the mapping procedure. These include epicardial anteroseptal pathways, epicardial pathways coursing anterior or posterior to the aortic root, epicardial connections between the right or left atrial appendage and epicardial surface of the ventricle, and sites where atrial myocardium is thought to be absent, including the left fibrous trigone and along the anteroseptal and midseptal mitral annulus (Figure 4).²²⁻²⁴ These sites should be suspected when an AP potential is not recorded

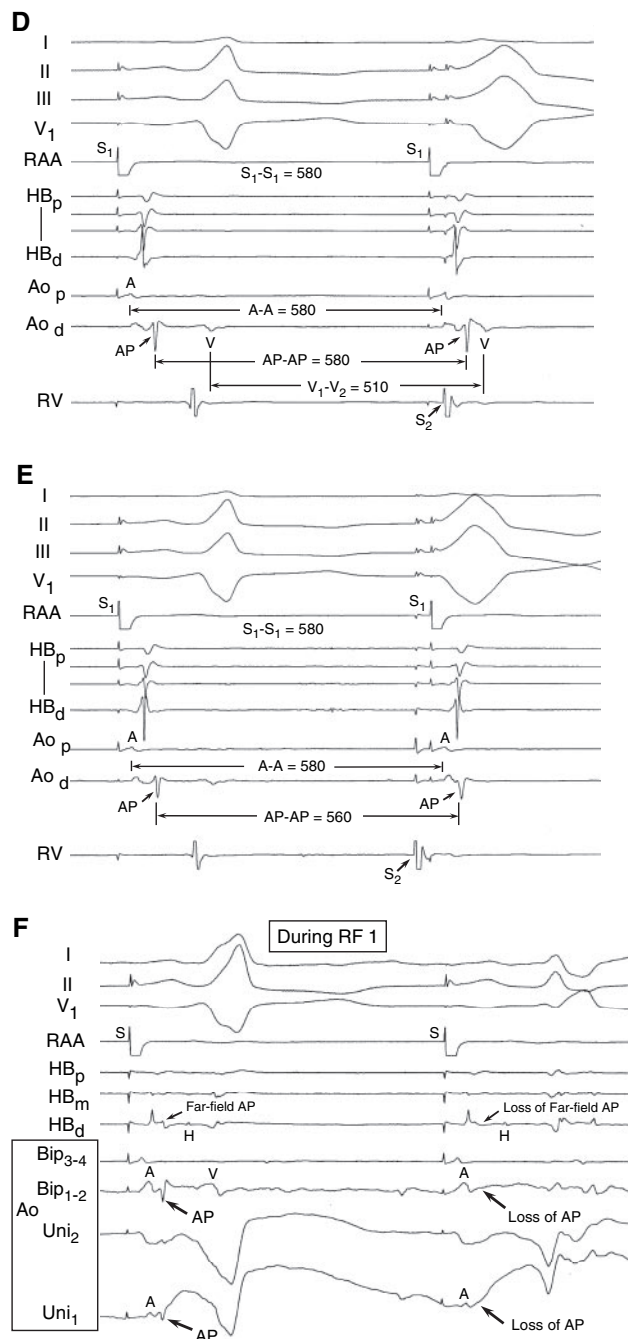


Figure 2 (Continued).

endocardially along the tricuspid annulus and the usually examined regions of the mitral annulus (anterolateral, lateral, posterior, and posteroseptal).

Epicardial Anteroseptal Accessory Pathways

An epicardial location for an anteroseptal accessory pathway should be suspected when endocardial recordings exhibit only far-field early antegrade ventricular activation and only far-field early retrograde atrial activation. In these patients, unfiltered unipolar recordings along the tricuspid annulus show local activation (rapid downstroke) beginning at least 20 ms after the onset of the far-field potential (Figure 2A) and may exhibit a tiny far-field AP potential. RF applications at

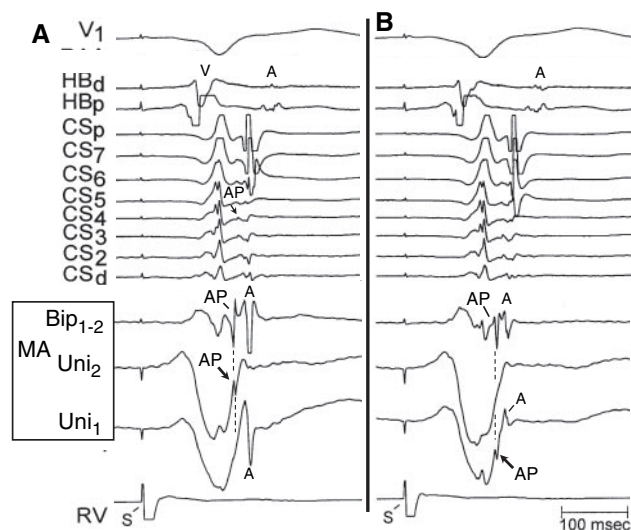


Figure 3. AP potential recorded in the unipolar and bipolar electrograms from the distal 2 electrodes on an ablation catheter positioned across the mitral annulus (MA) using the retrograde transaortic approach in a patient with a left posterolateral accessory pathway. A, During RV pacing, the sharp bipolar AP potential (Bip₁₋₂) was generated by the second electrode (Uni₂). B, The catheter was withdrawn toward the ventricle. The bipolar AP potential was then generated from the tip electrode (Uni₁). One RF application there eliminated accessory pathway conduction.

these sites usually fail to (or only transiently) eliminate accessory pathway conduction. The noncoronary cusp of the aortic valve is located just opposite the anteroseptal and anterior paraseptal tricuspid annulus (Figure 2B and 2C). Mapping within the noncoronary cusp may disclose a sharp AP potential, indicating close proximity to the accessory pathway (Figure 2D and 2E). Ablation at the site within the noncoronary cusp recording a sharp unipolar AP potential is generally successful in eliminating accessory pathway conduction (Figure 2F). The risk of AV block for ablation in the noncoronary cusp appears low, similar to the low risk of block with endocardial ablation >5 mm anterior to the His bundle (HB; right anterior paraseptal accessory pathways).

The risk of AV block is generally considered to be high for ablation of accessory pathways at the site recording a sharp HB potential (anteroseptal accessory pathway) or posterior to the HB and anterior to the CS (midseptal accessory pathway). Some investigators have advocated the use of cryoablation to reduce this risk.²⁵ However, we have found that positioning the ablation catheter on the ventricular side of the tricuspid annulus so that the unipolar tip electrogram records a sharp AP potential with little or no atrial potential has allowed the use of RF current with no AV block (or junctional extrasystoles) in patients with right anteroseptal and midseptal accessory pathways. Right bundle-branch block generally occurs with ablation when a sharp right bundle-branch potential is recorded in the unipolar tip electrogram. However, this is not clinically significant and is preferable to the risk of AV block with ablation on the atrial side of the tricuspid annulus.

Several tools are helpful for ablation of accessory pathways in general and especially useful for anteroseptal and

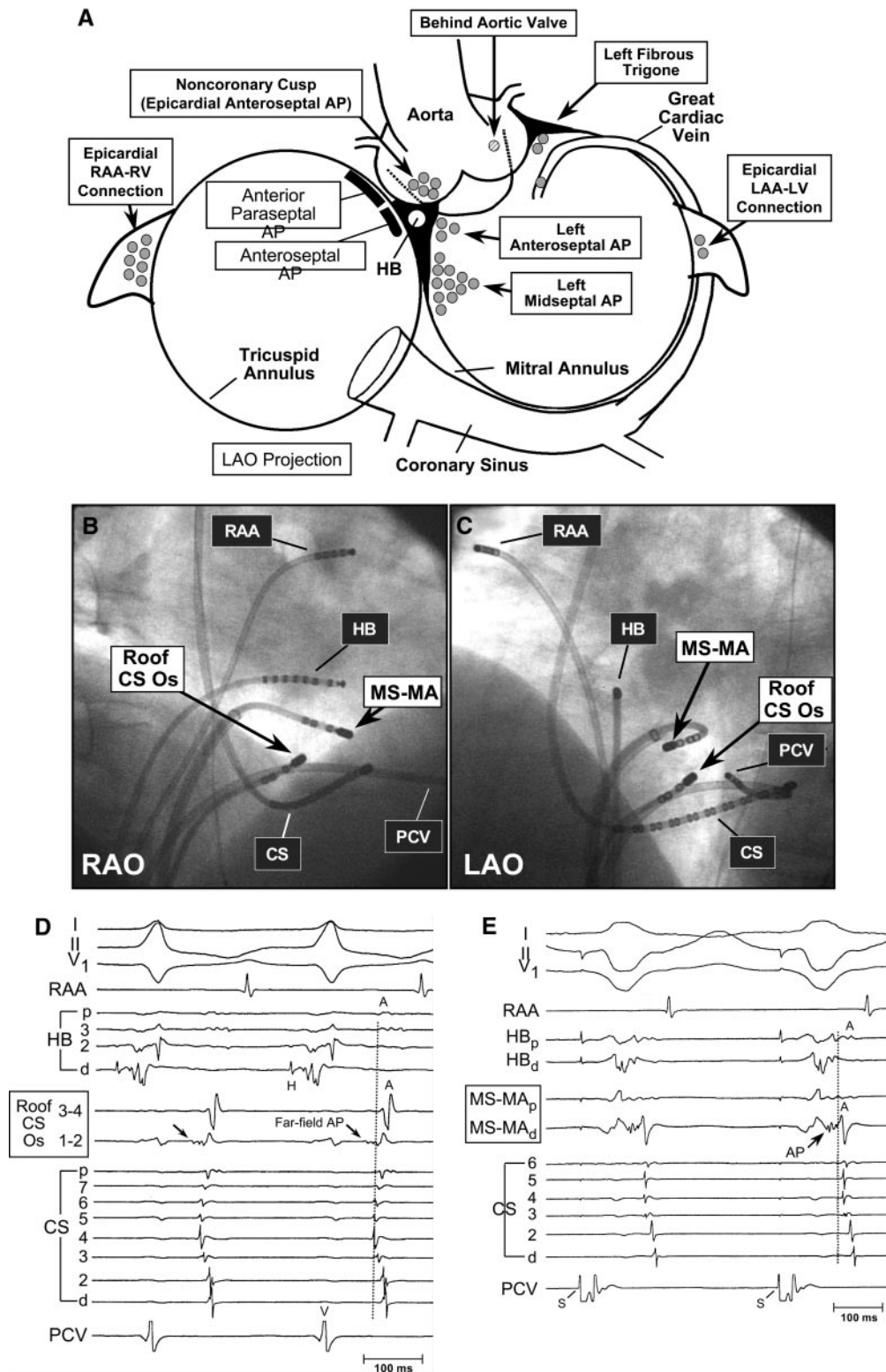


Figure 4. A, Schematic of unusual and unexpected locations of accessory pathways. Circles represent the successful ablation site for individual patients. B through E, Mapping and ablation of a left midseptal accessory pathway. Catheter positions in right atrial oblique (RAO; B) and left atrial oblique projections (LAO; C). D, Retrograde atrial activation is similar in timing in the HB and proximal CS electrograms. Mapping the roof of CS ostium (Os) identifies a small, far-field AP potential. E, Ablation catheter positioned at the midseptal mitral annulus (MS-MA) records a sharp AP potential. One RF application there eliminated accessory pathway conduction. RAA indicates right atrial appendage; LAA, left atrial appendage; LV, left ventricular; and PCV, posterior coronary vein.

midseptal accessory pathways. The use of general anesthesia with a paralytic agent allows 1- to 2-minute periods of apnea to help stabilize the catheter position during mapping and ablation. Another tool is parahissian pacing.^{26,27} Right ventricular (RV) pacing close to the HB with intermittent HB capture is useful to differentiate between retrograde conduction over the septal accessory pathway and retrograde conduction over the AV node. Ventricular pacing at that site without HB capture delays retrograde activation of the HB, allowing selective retrograde conduction over the accessory pathway for mapping and ablation. Another is curving the ablation catheter underneath the anterior leaflet (right subclavian venous approach) or the septal leaflet (femoral venous approach) of the tricuspid valve and maneuvering the catheter tip to the annulus while remaining underneath the leaflet. This provides a stable catheter position on the ventricular side of the annulus during ablation of anteroseptal and midseptal accessory pathways, respectively, with a low risk of AV block.^{2,28,29}

Ablation of left anteroseptal accessory pathways may be associated with a higher risk of AV block. We used the retrograde transaortic approach for ablation of 3 left anteroseptal accessory pathways. Even with apnea, significant movement of the catheter occurred during each cardiac cycle. AV block (resolving after several months) occurred in 1 of the 3 patients.

Left Midseptal Accessory Pathways

A hallmark of accessory pathways located at the midseptal mitral annulus is the recording of “earliest” retrograde atrial activation nearly simultaneously in the HB and proximal CS electrograms (Figure 4B through 4D), suggesting that activation originated at a site equally distant from the HB and proximal CS. By directing a mapping catheter vertically into the roof of the CS ostium, a far-field AP potential preceding atrial activation often is recorded (Figure 4B through 4D). Finding the far-field AP potential should be followed by mapping the mitral annulus because ablation with vertical pressure in the roof of the CS ostium (close to the atrial side of the mid-septal mitral annulus) usually fails to eliminate or only transiently eliminates accessory pathway conduction and is associated with a risk of AV block. In 11 patients with this finding, ablation along the mitral annulus (posterior to the HB and anterior to the CS) at the site that recorded a large, sharp AP potential eliminated accessory pathway conduction without AV block (Figure 4B, 4C, and 4E).²²

Right and Left Appendage Connection

Accessory pathway conduction can be produced by an epicardial connection between the right or left atrial appendage and the underlying ventricle (Figure 4A). These connections are located ≥ 1 cm apical to the tricuspid or mitral annulus.^{30–32} Therefore, endocardial mapping along the annulus records local activation (rapid downstroke of the unfiltered unipolar electrogram) up to 30 to 40 ms after the onset of the far-field potential. Retrograde atrial activation is recorded earlier on the atrial wall, close to the orifice of the appendage, but still long after the onset of far-field activation. Antegrade ventricular activation is recorded earlier at sites 1 to 3 cm apical of the annulus but also long after the onset of far-field

activation. Maneuvering a mapping catheter inside the atrial appendage locates earliest endocardial activation, usually beginning within 10 ms of the onset of far-field retrograde atrial and antegrade ventricular activation. An AP potential is not recorded because these pathways result from a direct connection between the atrial appendage and the ventricular myocardium. Ablation within the right atrial appendage was successful in 6 of 7 patients but generally required isolation of a segment of the atrial appendage surrounding the attachment to the ventricle using a large number of RF applications (4 to 17; median, 8). Using a saline-irrigated ablation electrode was helpful because of low blood flow around the electrode in the heavily trabeculated appendage. A steam pop was complicated by perforation of the appendage in 1 patient, suggesting that RF power should be limited (15 to 25 W). Ablation was unsuccessful in the right atrial appendage in the first of 7 patients and in both patients from the left atrial appendage. Surgical ablation was successful in these 3 patients by separating the atrial appendage from the epicardial surface of the ventricle.^{30,31} A percutaneous epicardial approach may prove useful for catheter ablation of these connections.³³

Inability to Safely Deliver RF Energy

RF energy should be delivered cautiously near the AV node or within the CS and its branches. For coronary venous ablation, significant risk of stenosis exists in coronary arteries located within 2 to 3 mm, especially when the ablation catheter is pushing against the artery (Figure 5).^{34–36} Coronary arteriography should be considered before coronary venous ablation. Cryoablation is preferred for coronary venous sites within 4 to 5 mm of a significant artery because the risk of coronary artery stenosis is low.³⁷

The most common form of epicardial accessory pathway results from a connection between an extension of the CS myocardial coat along the middle cardiac vein, posterior coronary vein, or neck of a CS diverticulum and the left ventricular epicardium (epicardial posteroseptal accessory pathway, Figure 5A and 5B).³⁸ This anatomy is a frequent cause of ablation failure. We found this anatomy in 144 of 306 patients (47%) with a posteroseptal or left posterior accessory pathway and ≥ 1 previous failed ablation procedures compared with only 42 of 212 patients (20%) with no prior attempted ablation. The finding of a steep negative delta wave in ECG lead II in a patient with a posteroseptal accessory pathway (V_1 to V_2 transition and negative delta wave in lead aVF) is specific but only moderately sensitive (70%) for an epicardial location.^{38,39}

A characteristic pattern (3 distinct potentials) is recorded from the coronary venous system during retrograde conduction over epicardial posteroseptal accessory pathways (Figure 5B and 5C).³⁸ The first potential (1 in Figure 6C) is recorded in the middle cardiac vein (or other coronary vein or CS diverticulum) and is generated by retrograde activation of the CS myocardial extension. The second potential (2) is small and recorded along the floor of the proximal CS as a result of leftward activation of the CS musculature. Because of fiber orientation,⁴⁰ the CS myocardium activates the left atrium at a location 2 to 4 cm left of the orifice of the middle cardiac

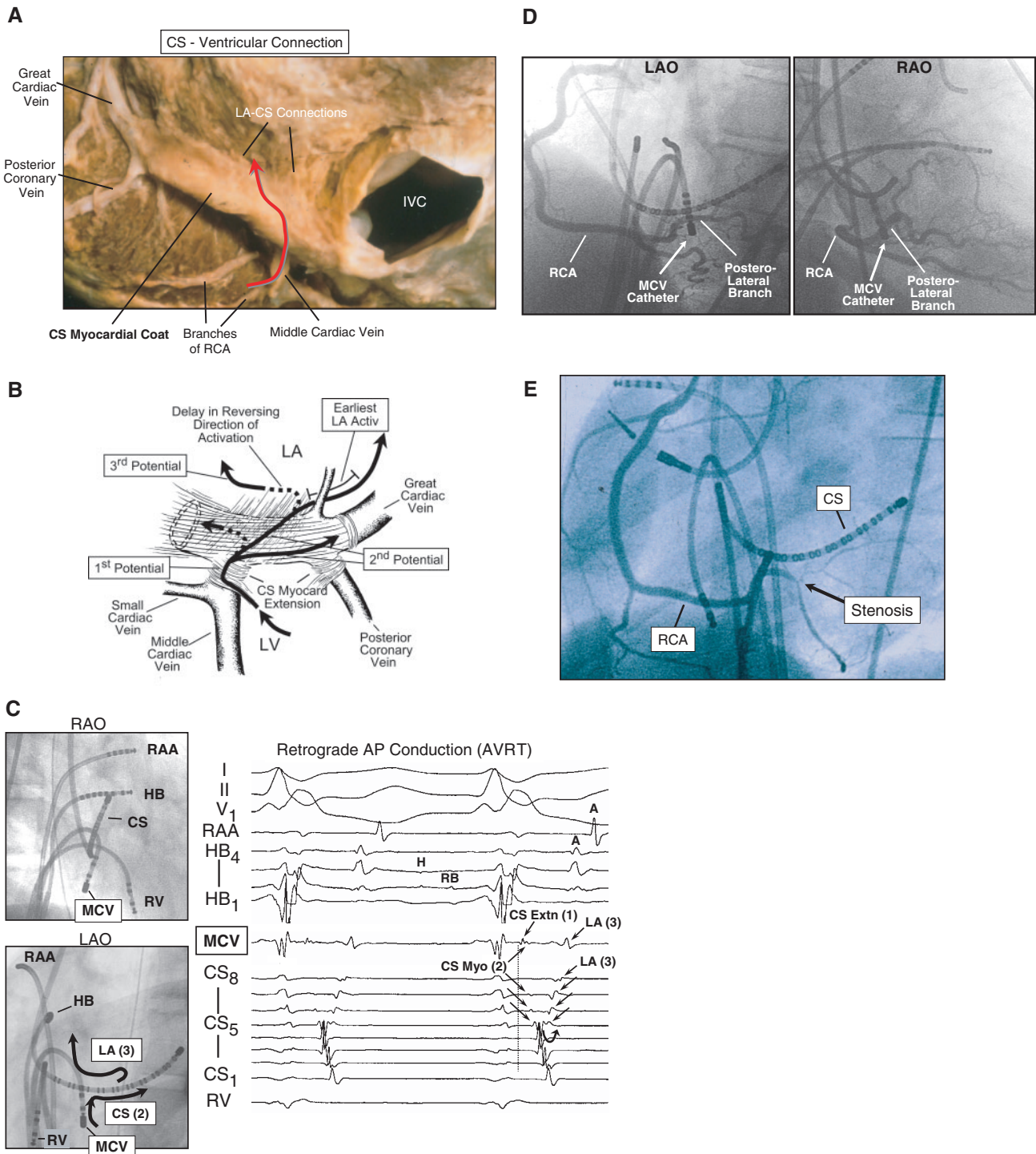


Figure 5. Epicardial posteroseptal accessory pathway resulting from connection between an extension of CS myocardium along the middle cardiac vein (MCV) and epicardial left ventricle (LV). A, Photograph showing CS myocardial coat, left atrial (LA)–CS connections, and relationship between MCV and distal branches of right coronary artery (RCA) located very close to the coronary artery. Photograph courtesy of Anton E. Becker. B and C, Schematic (left atrial oblique [LAO] projection) and recordings of retrograde conduction demonstrating characteristic pattern of 3 potentials. A through C modified from Jackman et al⁴⁸ and used with permission from the publisher. Copyright © 2007, Blackwell Publishing. D, Right coronary arteriography with ablation catheter in the MCV. The ablation electrode (AP recording site) is located within 2 mm of the posterolateral branch of the RCA. Cryoablation was performed from the MCV to avoid arterial injury. E, Distal RCA stenosis in a 14-year-old boy who underwent RF ablation at the floor of the CS ostium 5 years earlier at another hospital. IVC indicates inferior vena cava; RAO, right anterior oblique; and RAA, right atrial appendage.

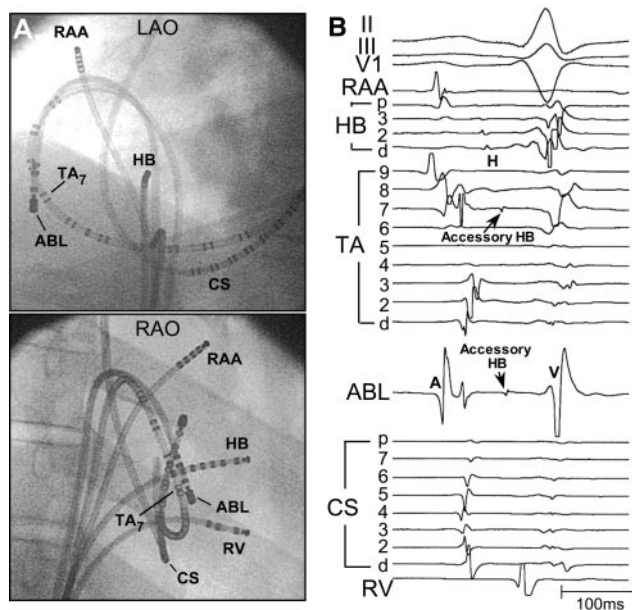


Figure 6. RAF ablation. A and B, The accessory HB potential is recorded along the lateral tricuspid annulus in the circular tricuspid annulus catheter (TA₇) and ablation catheter (ABL). RAA indicates right atrial appendage; LAO, left atrial oblique; and RAO, right atrial oblique.

vein and results in rapid left atrial activation in the leftward direction. Left atrial activation in the rightward (septal) direction is delayed as a result of slowing of conduction during the reversal in direction of activation (Figures 1A and 5B). The third potential (3) is generated by the late left atrial activation near the orifice of the middle cardiac vein.

During antegrade conduction, earliest endocardial ventricular activation is usually recorded ≥ 25 ms after the onset of far-field activation and nearly simultaneously on the right and left sides of the interventricular septum, 1 to 3 cm apical to the tricuspid and mitral annuli. Earliest ventricular activation (usually ≤ 15 ms after the onset of far-field activation) is recorded from the middle cardiac vein (or other coronary vein or CS diverticulum) and is preceded by a distinct potential resulting from antegrade activation of the CS myocardial extension.³⁸

CS angiography (balloon occlusion technique) is useful for delineating the coronary venous anatomy associated with an epicardial posteroseptal accessory pathway. A CS diverticulum or distorted coronary vein is found in only 20% and 10% of patients, respectively. The accessory pathway is associated with a structurally normal middle cardiac vein (or other coronary vein) in 70% of patients.³⁸

Because of the extensive connections between the CS myocardium and left atrium, the optimal ablation site is within the middle cardiac (or other coronary vein or neck of CS diverticulum) at the site recording the largest, sharpest unipolar potential generated by the CS myocardial extension. Saline-irrigated RF ablation is recommended when coronary arteriography shows this site to be located ≥ 5 mm from the closest significant coronary artery. We usually begin at 10 to 15 W and increase power as required up to 25 W. An impedance rise usually occurs, even with saline irrigation. It is important

to terminate the RF application as quickly as possible when the impedance rise occurs (3- to 5- Ω increase above the lowest value) to prevent adherence of the electrode to the vein. Accessory pathway conduction is usually eliminated by 1 or 2 RF applications, with a low long-term recurrence.

When the optimal ablation site in the vein is located within 4 mm of a significant coronary artery, cryoablation is recommended (Figure 5D and 5E). In our early experience, we found short-term efficacy using cryoablation alone in only 9 of 12 patients (75%). A large number of cryoapplications (1 to 40; median, 9) was required, with frequent transient accessory pathway conduction block lasting up to 60 minutes. In 2 of the remaining 3 patients, cryoablation blocked activation between the ventricular connection and the CS, but an epicardial connection to the right atrium remained. RF ablation in the right atrium at the site of earliest far-field retrograde atrial activation, far from the tricuspid annulus, ultimately eliminated accessory pathway conduction for short-term ablation success in 11 of the 12 patients. Accessory pathway conduction returned in 2 of the 11 patients, for long-term success in only 9 of the 12 patients (75%). Short-term success has improved with large cryoablation electrodes, but recurrence remains higher than with RF ablation.

Other Variants: Right Atriofascicular and AV (Mahaim) Accessory Pathways

Right atriofascicular (Mahaim) accessory pathways (RAFs) are a duplication of the normal AV conduction system, with an accessory AV node (located anywhere along the anterolateral to posterolateral tricuspid annulus) connected to an isolated bundle of Purkinje fibers. This accessory HB and right bundle branch extends to the apical region of the RV free wall; it is unknown whether the HB connects with the distal right bundle branch at the moderator band or inserts into the apical RV free wall near the moderator band. This anatomy produces unique antegrade conduction properties, including a long conduction time, decremental properties, and activation of the ventricles via the distal right bundle branch.⁴¹ Ventricular preexcitation usually is not present during sinus rhythm because of the long RAF conduction time. Because RAFs do not exhibit retrograde conduction, they present with a preexcited AV reentrant tachycardia using either the AV node (antidromic AVRT; 90% to 95% of patients) or a concealed accessory pathway for retrograde conduction (5% to 10%). The hallmarks of antidromic AVRT using an RAF are (1) QRS complex identical to left bundle-branch block (sharp QS pattern in V₁); (2) earliest antegrade ventricular activation recorded at the apical RV free wall and preceded by a right bundle-branch-type potential^{40–43}; (3) early retrograde activation of the right bundle branch, resulting in recording the retrograde HB potential within 30 ms of the QRS onset; (4) early retrograde atrial activation (short VA interval); (5) long AV interval; and (6) an increase in VA interval and tachycardia cycle length with right bundle-branch block.^{41–44}

The optimal ablation site is along the tricuspid annulus where the RAF produces an accessory HB potential (Figure 6).⁴⁰ If the ablation catheter cannot be stabilized at the

tricuspid annulus, ablation can be performed along the basal or mid RV free wall where a high-frequency RAF potential (appearing similar to the right bundle-branch potential) is recorded. Locations close to the apex should be avoided to prevent ablation of the distal right bundle branch (proximal to the RAF). This increases antidromic AVRT cycle length, occasionally producing incessant tachycardia.

The RAF is easily bumped during mapping, eliminating the accessory HB. The accessory HB can be located with a minimum risk of traumatic block by a circular multielectrode catheter (such as Halo, Biosense Webster, Inc, Diamond Bar, Calif) positioned around the tricuspid annulus (Figure 6A and 6B). If the RAF is bumped, ablation can be performed empirically at the bump site and slightly atrial to the bump site where a tiny proximal accessory HB is recorded. Of 51 patients with RAF, an accessory HB was recorded in 50 patients. Ablation targeting the accessory HB (including a tiny proximal accessory HB after bumped RAF in 13 patients) eliminated RAF conduction in all 50 patients using 1 to 12 (median, 2) RF applications. In the 1 remaining patient, ablation targeting the tricuspid annulus site with the shortest stimulus-ventricular interval during resetting of antidromic AVRT successfully eliminated RAF conduction. RAF conduction has not recurred after ablation in any of the 51 patients.

A variant of RAF inserts into the right ventricle, close to the tricuspid annulus and far from the distal right bundle branch. Of 17 patients with this AV variant, an accessory HB potential was recorded in 10 patients. Ablation at the accessory HB in these 10 patients and at the site of earliest ventricular activation in the remaining 7 patients eliminated accessory pathway conduction in all 17 patients with no recurrence.

Part 2: Catheter Ablation of the Various Atrioventricular Nodal Reentrant Tachycardias

Reentrant Circuits of AVNRT

Most forms of AVNRT are created by reentry between 2 (or more) atrial connections to the AV node. At least 3 atrial connections are evident anatomically and electrophysiologically. In the previous section on ablation of accessory pathways, we used the traditional anatomic coordinates. For the following description of AVNRT, we use the recently proposed coordinate system in which the anterior-posterior directions are replaced by superior-inferior directions and the superior-inferior directions are replaced by anterior-posterior directions (Figure 7).⁴⁵

The fast AV nodal pathway (shortest conduction time) is formed by transitional cells crossing the tendon of Todaro superiorly.⁴⁶ During retrograde fast pathway conduction, earliest atrial activation is recorded simultaneously on the right and left sides of the interatrial septum, posterior to the tendon of Todaro at a height approximately one third of the distance from the HB to CS roof (Figure 8).^{6,47} Atrial activation at this site precedes activation at the site recording the HB potential by a mean of 11 ± 8 ms.⁴⁷

Two slow AV nodal pathways are formed by the rightward and leftward inferior extensions of the AV node (Figure 7).⁴⁷⁻⁴⁹ The rightward inferior extension has the longest conduction time and participates in most forms of AVNRT. During retrograde slow pathway conduction over the rightward inferior extension, the earliest high-frequency potential usually is recorded between the tricuspid annulus and CS ostium (A_{SP} potential in Figure 7E).^{6,50} This is followed serially by activation of the floor of CS ostium and leftward activation along the floor of the proximal CS and the left atrium at the inferior mitral annulus (Figure 7E). The retrograde conduction time over the leftward inferior extension is shorter, with earliest activation usually recorded from the roof of the CS ≈ 2 to 4 cm from the CS ostium (Figure 7D).^{48,50}

Slow/Fast AVNRT

Typical Slow/Fast AVNRT

We define typical slow/fast AVNRT as AVNRT using the rightward inferior extension of the AV node for antegrade slow pathway conduction and the fibers crossing the tendon of Todaro for retrograde fast pathway conduction. We propose the following circuit⁴⁸: Retrograde conduction over the fast pathway activates the right and left sides of the interatrial septum (red arrows in Figure 7B). Right atrial activation is blocked along the eustachian ridge (green lines). Left atrial activation propagates inferiorly and laterally (green arrows) and activates the roof of the CS. The CS myocardium propagates the impulse to the floor of the CS ostium (orange arrows). Activation of the atrial myocardium between the tricuspid annulus and the CS ostium in the superior direction generates the relatively late A_{SP} potential (straight blue arrow) and activates the atrial end of the rightward inferior extension of the AV node (zigzag blue arrow).

Mapping the right atrium and CS during AVNRT is used to differentiate slow/fast AVNRT from slow/slow AVNRT. In 650 patients with AVNRT exhibiting a short H-A interval (≤ 120 ms), earliest retrograde atrial activation was recorded posterior to the tendon of Todaro (slow/fast AVNRT) in 543 patients (83%) and within the triangle of Koch or CS (slow/slow AVNRT) in 109 patients (17%).^{47,51,52}

Two atrial potentials often are recorded close to the HB during slow/fast AVNRT.⁵³ The first potential originates posterior to the tendon of Todaro (fast pathway). The second potential is generated by superiorly directed activation in the triangle of Koch. In some patients, the HB electrogram records only the second potential, mimicking slow/slow AVNRT (Figure 8). In these patients, mapping posterior to the tendon of Todaro records earlier activation, confirming slow/fast AVNRT (Figure 8).

Our approach for ablation of typical slow/fast AVNRT is to interrupt the atrial end of the rightward inferior extension (slow pathway) either by delivering RF energy at sites between the tricuspid annulus and CS ostium recording an A_{SP} potential during sinus rhythm or by creating a linear lesion between the tricuspid annulus (at the level of the middle of the CS ostium) and the anterior (apical) edge of the CS ostium (hatched area in Figure 7B).^{6,48} The electrogram recording the A_{SP} potential during sinus rhythm has an initial small, far-field atrial potential (generated by the right atrium

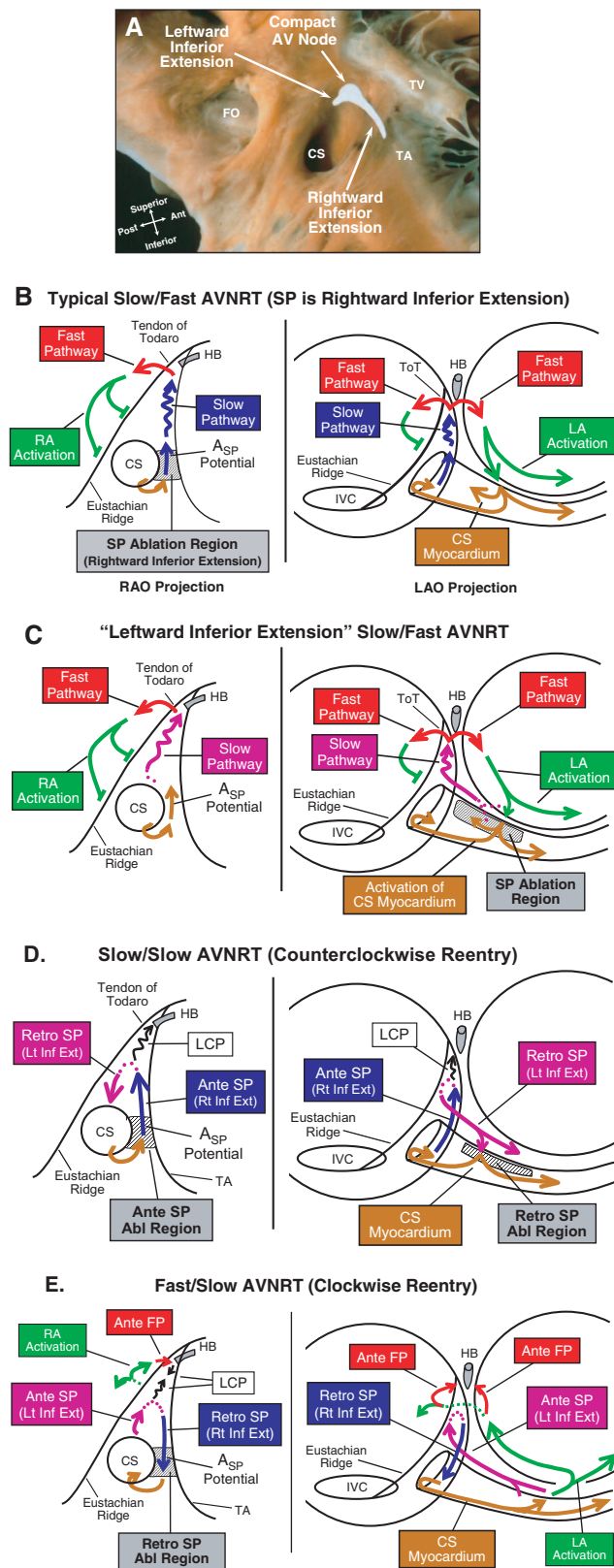


Figure 7. A, Anatomic location of the compact AV node and the rightward and leftward inferior extensions of the AV node in a human heart. B through E, Schematic representations of AVNRT reentrant circuits. Red arrows indicate fast AV nodal pathway; blue arrows, slow pathway formed by rightward inferior extension; purple arrows, slow pathway formed by leftward inferior extension; orange arrows, CS myocardial activation; black arrows, lower common pathway (LCP); and green arrows, atrial

posterior to the eustachian ridge), followed by an isoelectric interval, the sharp A_{SP} potential, and a large, sharp ventricular potential (generated by the ventricular myocardium underneath the atrial myocardium in the muscular AV septum).⁶ The late timing of the A_{SP} potential during sinus rhythm (after activation in the proximal CS) may be explained by conduction block at the eustachian ridge with the sinus impulse entering the triangle of Koch either from the inferior right atrium (extension of activation from the crista terminalis) or from the left atrium via the CS.⁴⁷

For the linear lesion, we start on the ventricular side of the tricuspid annulus identified by recording an A_{SP} potential on the unipolar electrogram from the second electrode but not from the tip electrode. RF energy is delivered at each site during the pullback until the atrial potential on the tip unipolar electrogram is markedly diminished. An accelerated junctional rhythm usually begins when the tip unipolar electrogram begins to record an A_{SP} potential. RF energy is maintained at each site producing an accelerated junctional rhythm^{54–56} until 15 to 20 seconds after cessation or marked slowing of the junctional rhythm. The RF pullback is continued until the ablation electrode reaches the apical edge of CS ostium. We start with an RF power of 30 to 45 W (electrode temperature <60°C) and reduce power to 20 to 25 W when approaching the CS ostium. The tip electrode is then positioned within the apical edge of the CS ostium for completion of the ablation line. We avoid delivering RF energy near the floor of the CS ostium to prevent injury to the coronary artery.³⁶ We found that junctional extrasystoles or an accelerated junctional rhythm was present during the RF application (or line) that eliminated slow/fast AVNRT in 95 of 100 consecutive patients.⁴⁶ The loss of 1:1 retrograde fast pathway conduction during junctional rhythm may indicate injury to the AV node or fast pathway, and the RF application should be terminated immediately.^{54,55}

We try to limit RF applications to sites inferior to the level of the roof of the CS ostium, which reduces the risk of permanent AV block to <0.5%.^{6,48} Maintaining catheter contact in the inferior triangle of Koch is often difficult because the eustachian ridge pushes the catheter away from the septum during systole (seen as rightward movement of the catheter tip during systole in the left anterior oblique projection). A sharp A_{SP} potential recorded during diastole may falsely suggest stable contact. A long sheath can be used to position the catheter around the eustachian ridge.

Our end points for ablation, tested without and with isoproterenol (1 to 4 μg/min), are elimination of inducibility of AVNRT (single slow/fast atrial echo complexes are allowed) and elimination of 1:1 antegrade conduction over the slow AV nodal pathway during decremental atrial pacing. We achieved these end points with long-term success in 99.4% patients with typical slow/fast AVNRT.^{6,48} Others have reported similar high success.^{7–9}

Figure 7 (Continued). activation. ToT indicates tendon of Todaro; IVC, inferior vena cava; LA, left atrium; RA, right atrium; RAO, right anterior oblique; LAO, left anterior oblique; and Abl, ablation. Modified from Jackman et al⁴⁸ and used with permission from the publisher. Copyright © 2007, Blackwell Publishing.

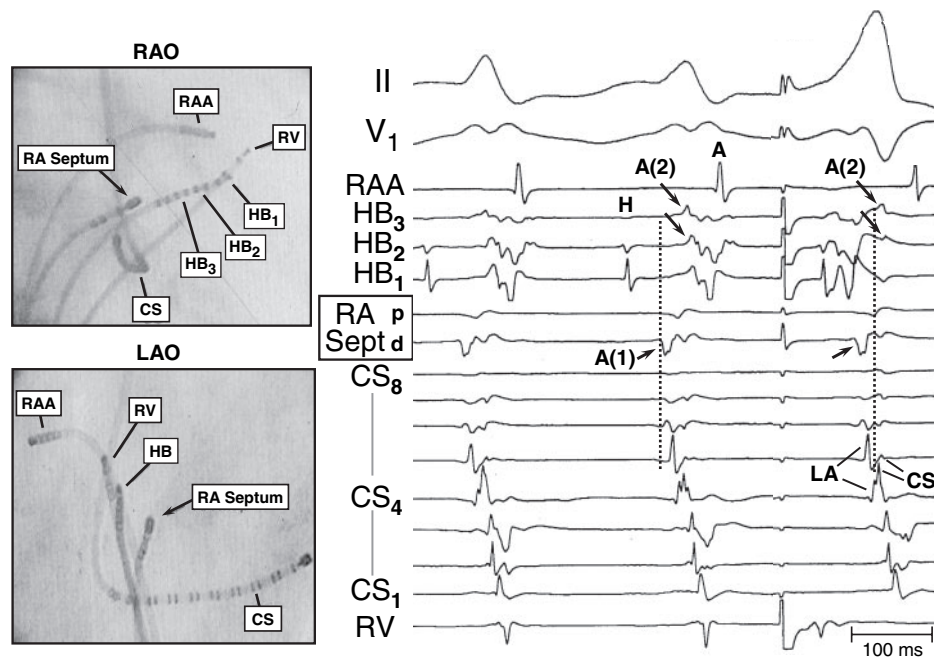


Figure 8. Two atrial potentials recorded from the septum during typical slow/fast AVNRT. The first potential, A(1), is recorded posterior to the tendon of Todaro as evidenced by leftward orientation of right atrial (RA) septum catheter in the left atrial oblique radiograph (fast pathway area). The second potential, A(2), is recorded sequentially in HB₂ and HB₃ electrograms at the superior aspect of the triangle of Koch. The first potential in CS₄ through CS₈ is left atrial activation; the second is CS myocardial activation. Note that A(2) follows CS myocardial activation (second dotted line). The absence of A(2) in HB electrograms mimics slow/slow AVNRT. RAO indicates right atrial oblique; RAA, right atrial appendage.

Cryoablation has been proposed to reduce the risk of AV block.⁵⁷ AV nodal block occurring during cryoablation often reverses if the cryoapplication is terminated immediately.⁵⁷ However, the long-term success with cryoablation is only in the range of 86%.⁵⁸ Improvement in success is expected with newer, larger cryoelectrodes.

“Leftward Inferior Extension” Slow/Fast AVNRT

Ablation between the tricuspid annulus and the anterior edge of the CS ostium (A_{SP} recording sites) produces accelerated junctional rhythm (rightward inferior extension injury) but fails to eliminate AVNRT in ≈5% of slow/fast AVNRT patients. In these patients, the leftward inferior extension of the AV node may form the antegrade slow pathway in the reentrant circuit (Figure 7C). Rather than delivering RF energy at progressively higher sites in the triangle of Koch, we prefer to target the atrial end of the leftward inferior extension along the roof of the proximal CS, between the CS ostium and 2 to 4 cm from the ostium (Figure 7C). We avoid positioning the catheter straight upward (perpendicular to the CS roof) with force during ablation close to the CS ostium because the fast pathway may be injured.

“Left Atrial” Slow/Fast AVNRT

In <1% of the patients with slow/fast AVNRT, ablation between the tricuspid annulus and the CS ostium along the roof of the proximal CS and the anterior-superior edge of the CS ostium fails to eliminate the tachycardia. In some of these patients, the atrial end of the slow pathway is located in the left atrium, close to the inferolateral mitral annulus.^{47,59–62} A left atrial insertion of the slow pathway can be identified by the resetting response. During AVNRT, a late atrial extra-

stimulus (after the onset of retrograde atrial activation) is delivered to the left atrium close to the inferolateral mitral annulus (Figure 9). Advancing the next HB potential by ≥10 ms, followed by resetting of the tachycardia (H-H interval equal to the tachycardia cycle length), indicates that the pacing site is located close to the atrial end of the slow pathway (Figure 9C). Ablation at the site of resetting frequently produces accelerated junctional rhythm with retrograde fast pathway conduction (slow pathway automaticity) and eliminates the tachycardia (Figure 9D).^{47,59} Ablation at the inferolateral mitral annulus usually is not successful if a late atrial extrastimulus fails to advance the next HB potential.

Slow/Slow AVNRT

We have proposed that slow/slow AVNRT and fast/slow AVNRT result from reentry between the rightward and leftward inferior extensions of the AV node.^{47,48,61} Slow/slow AVNRT most often uses the rightward inferior extension for the antegrade limb of the circuit and the leftward inferior extension for the retrograde limb^{47,48,61} (counterclockwise reentry as viewed in the right atrial oblique projection, Figure 7D). Fast/slow AVNRT most often uses the leftward inferior extension for the antegrade limb and the rightward inferior extension for the retrograde limb (clockwise reentry as viewed in the right atrial oblique projection, Figure 7E).

Slow/slow AVNRT is defined when the A-H interval is significantly longer than the H-A interval (≥200 ms), but unlike slow/fast AVNRT, earliest retrograde atrial activation is recorded along the roof of the proximal CS (62% of slow/slow AVNRT patients, Figures 7D and 10) or between

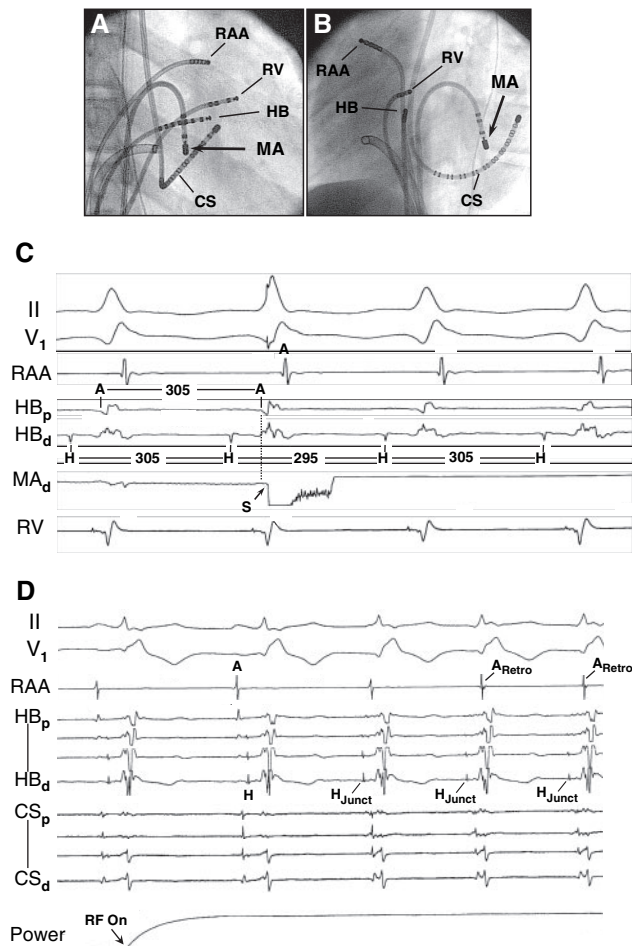


Figure 9. Catheter ablation of “left atrial” slow/fast AVNRT. Radiographs in right atrial oblique (A) and left atrial oblique (B) projections show the left atrial mapping catheter near the inferolateral mitral annulus (MA). C, Resetting slow/fast AVNRT. A late left atrial extrastimulus (S) delivered near the inferolateral mitral annulus (does not advance retrograde atrial activation in HB_p electrogram) advances the next HB potential by 10 ms (H-H=295 ms) and resets tachycardia (next H-H=305 ms). D, One RF application at the left atrial resetting site produced an immediate accelerated junctional rhythm (1:1 retrograde fast pathway conduction [A_{Retro}]) consistent with heating-induced automaticity of the slow pathway and eliminated tachycardia. RAA indicates right atrial appendage. Modified from Jackman et al⁴⁸ and used with permission from the publisher. Copyright © 2007, Blackwell Publishing.

the inferoseptal tricuspid annulus and CS ostium (38% of slow/slow AVNRT patients).^{47,48,61} Because of the presence of a long lower common pathway, the H-A interval during ventricular pacing is significantly longer than the H-A interval during tachycardia,⁶² and the H-A interval during tachycardia may vary and may be very short (<20 ms) or even negative. The range of H-A interval during tachycardia is wide (<20 to 315 ms; mean, 96 ± 65 ms).⁵²

For ablation of slow/slow AVNRT, we initially target retrograde slow pathway conduction and then target antegrade slow pathway conduction. For ablation of retrograde slow pathway conduction, RF energy is delivered to the site of earliest retrograde atrial activation, which is usually at the

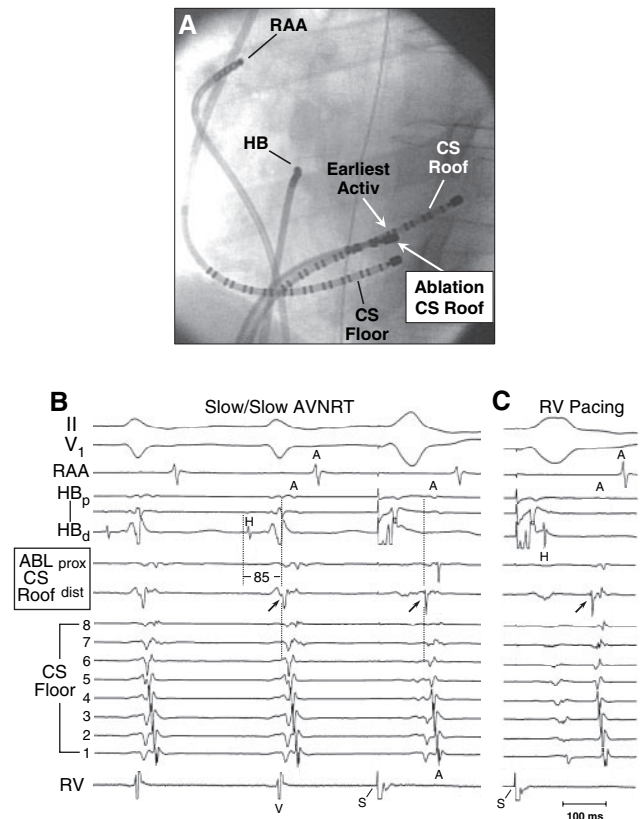


Figure 10. Ablation (ABL) of slow/slow AVNRT. A, Radiograph (left atrial oblique projection) showing the ablation site of retrograde slow pathway in slow/slow AVNRT. Two multielectrode catheters were positioned along the CS roof and CS floor for mapping. Earliest retrograde atrial activation was recorded from the fourth bipolar electrogram on the CS roof. B, Slow/slow AVNRT with short H-A interval (85 ms) mimicking slow/fast AVNRT. A late extrastimulus (S) delivered to the superior-basal RV septum was used to advance the ventricular potentials and to unmask the atrial activation sequence. Earliest activation was recorded in the roof of the proximal CS (arrows). C, During superior-basal-septal RV pacing, retrograde conduction occurred only over the retrograde slow pathway used in AVNRT. Retrograde fast pathway conduction was absent. RAA indicates right atrial appendage. Modified from Jackman et al⁴⁸ and used with permission from the publisher. Copyright © 2007, Blackwell Publishing.

roof of the proximal CS (leftward inferior extension, Figure 10A through 10C). Antegrade slow pathway conduction ablation usually involves ablation of the rightward inferior extension between the tricuspid annulus and CS ostium. During ablation at this site, accelerated junctional rhythm frequently is associated with VA block because retrograde fast pathway conduction is either absent or poor in most patients with slow/slow AVNRT (Figure 10C).

Fast/Slow AVNRT

Fast/slow AVNRT is defined when the H-A interval is significantly longer than the A-H interval. Earliest retrograde atrial activation is recorded in the region between the inferoseptal tricuspid annulus and CS ostium (recording the retrograde A_{SP} potential, Figure 7E) in 73% of patients and at the roof of CS ostium in 27% of patients.^{47,48,61} The H-A interval has a wide range (165 to 365 ms; mean, 266 ± 66 ms).⁵¹ The

A-H interval often is shorter than during sinus rhythm. The short A-H interval does not imply that the fast pathway is part of the reentrant circuit. The short A-H interval may be explained by the mechanism shown in Figure 8E. Retrograde conduction over the rightward inferior extension activates the CS myocardium and left atrium, which then activate the leftward inferior extension in the antegrade direction. Simultaneously, left atrial activation propagates to the atrial septum to activate the fast pathway and to produce the short A-H interval. In this mechanism, the fast pathway does not participate in the reentrant circuit.

For ablation of fast/slow AVNRT, we initially target the retrograde slow pathway of the tachycardia circuit and then ablate the antegrade slow pathway conduction if present. RF energy is delivered to the site of earliest retrograde atrial activation, usually the site recording the retrograde A_{SP} potential in the region between the inferoseptal tricuspid annulus and CS ostium (Figure 7E). Earliest activation near the floor of CS ostium indicates retrograde conduction over the rightward inferior extension. Further mapping along the triangle of Koch usually will disclose a small retrograde A_{SP} potential. We prefer to avoid ablation along the floor of the proximal CS to prevent injury to the coronary artery.³⁷ Short-term ablation success is achieved in essentially all fast/slow AVNRT patients. The recurrence rate for fast/slow AVNRT in our experience is 1.2%, intermediate between slow/fast AVNRT (0.4%) and slow/slow AVNRT (6%).

Disclosure

None.

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