Concealed Accessory Pathways with a Single Ventricular and Two Discrete Atrial Insertion Sites

RYAN T. KIPP, M.D.,* RAED ABU SHAM'A, M.D.,† ITO HIROYUKI, M.D.,‡ FREDERICK T. HAN, M.D.,§ MARWAN REFAAT, M.D., PH.D.,¶ JONATHAN C. HSU, M.D., M.A.S.,# MICHAEL E. FIELD, M.D.,* DOUGLAS E. KOPP, M.D.,* GREGORY M. MARCUS, M.D., M.A.S.,‡ MELVIN M. SCHEINMAN, M.D.,‡ and KURT S. HOFFMAYER, M.D.#,**

From the *Division of Cardiology, Section of Electrophysiology, University of Wisconsin School of Medicine and Public Health, Madison, Wisconsin; †Cardiac Pacing and Electrophysiology, University of Ottawa Heart Institute, Ottawa, Ontario, Canada; ‡Division of Cardiology, Section of Electrophysiology, University of California, San Francisco, California; §Division of Cardiovascular Medicine, Section of Electrophysiology, University of Utah, Salt Lake City, Utah; ¶Department of Internal Medicine, Cardiovascular Medicine/Cardiac Electrophysiology, American University of Beirut Faculty of Medicine and Medical Center, Beirut, Lebanon; #Division of Cardiology, Section of Electrophysiology, University of California, San Diego, California; and **Division of Cardiology, Section of Electrophysiology, VA San Diego Healthcare System, San Diego, California

Background: *Atrioventricular reciprocating tachycardia (AVRT) utilizing a concealed accessory pathway is common. It is well appreciated that some patients may have multiple accessory pathways with separate atrial and ventricular insertion sites.*

Methods: *We present three cases of AVRT utilizing concealed pathways with evidence that each utilizing a single ventricular insertion and two discrete atrial insertion sites.*

Results: *In case one, two discrete atrial insertion sites were mapped in two separate procedures, and only during the second ablation was the Kent potential identified. Ablation of the Kent potential at this site remote from the two atrial insertion sites resulted in the termination of the retrograde conduction in both pathways. Case two presented with supraventricular tachycardia (SVT) with alternating eccentric atrial activation patterns without alteration in the tachycardia cycle length. The two distinct atrial insertion sites during orthodromic AVRT and ventricular pacing were targeted and each of the two atrial insertion sites were successfully mapped and ablated. In case three, retrograde decremental conduction utilizing both atrial insertion sites was identified prior to ablation. After mapping and ablation of the first discrete atrial insertion site, tachycardia persisted utilizing the second atrial insertion site. Only after ablation of the second atrial insertion site was SVT noninducible, and VA conduction was no longer present.*

Conclusions: *Concealed retrograde accessory pathways with discrete atrial insertion sites may have a common ventricular insertion site. Identification and ablation of the ventricular insertion site or the separate discrete atrial insertion sites result in successful treatment. (PACE 2017; 40:255–263)*

catheter, ablation, accessory pathways

Introduction

Accessory pathways (APs) most frequently occur with retrograde unidirectional conduction facilitating the development of atrioventricular (AV) reciprocating tachycardia (AVRT).[1–5] Previously described retrograde APs have single atrial insertion sites.[1–3] It is well appreciated that some patients may have multiple APs with separate atrial and ventricular insertion sites.[1–4,6–18] We present three cases each with orthodromic AVRT utilizing an AP with a single ventricular but two separate atrial insertion sites.

Methods

From 2008 to 2014, three cases were identified with evidence of concealed APs with two distinct

Author Disclosures: Frederick T. Han has received Honoraria from Biotronik and research support from Boston Scientific, St Jude Medical. Jonathan Hsu has received honoraria from St. Jude Medical, Medtronic and Biotronik, and has served on Advisory Boards for Janssen Pharmaceuticals and Bristol-Myers Squibb. Melvin M. Scheinman has received honoraria for lectures from St Jude Medical, Biotronick, Medtronic, and Boston Scientific, and is on a data monitoring committee for Gilead. Other authors declare no conflict of interest.

Address for reprints: Kurt S. Hoffmayer, M.D., Division of Cardiology, Section of Electrophysiology, University of California, San Diego, 9434 Medical Center Drive, La Jolla, CA 92037-1300. Fax: 858-657-8814; e-mail: kurt.hoffmayer@gmail.com

Received September 13, 2016; revised November 27, 2016; accepted December 11, 2016.

doi: 10.1111/pace.13024

atrial and one ventricular insertion site from three centers. An electrophysiology (EP) study was performed in each with catheters placed in the right atrium, right ventricle, His-bundle region, and coronary sinus (CS). In each case, the CS proximal electrodes were positioned near the ostium. Specific diagnostic and ablation catheter selection as well as ablation technique was performed at the discretion of the electrophysiologist. Three-dimensional (3D) mapping was used in two of the three cases. Patient information was de-identified. The clinical findings and EP studies are reported below.

Results

Case One

A 39-year-old female with a history of palpitations was found to have a short RP tachycardia that was refractory to medications. Due to the incessant nature of these arrhythmias, she was referred for EP study and ablation. The HV interval was 48 ms and there was no evidence of manifest preexcitation. Supraventricular tachycardia (SVT) with a narrow QRS complex with eccentric atrial activation and a cycle length of 380 ms was spontaneously induced during catheter manipulation (Fig. 1A). The earliest atrial signal was located at the middle of the CS catheter. His synchronized premature ventricular contractions (PVCs) delayed the next atrial electrogram. Following ventricular overdrive pacing, a V-A-H-V response was recorded. Using differential site pacing, the stimulus to atrium timing from the apex was 173 ms and from the base was 168 ms. These findings proved presence of an AP and its participation in the circuit for orthodromic AVRT.

Using 3D electroanatomic mapping (CARTO, Biosense Webster, Diamond Bar, CA, USA) during ventricular pacing and orthodromic AVRT, the earliest atrial signal was mapped to 5 o'clock on the mitral annulus in the left anterior oblique position(left posterolateral pathway). A Kent potential was not identified. However, the earliest atrial activation was fused with the ventricular electrogram. Retrograde conduction over this pathway was abolished within 5 seconds after RF application.

A short-RP tachycardia was reinduced with a cycle length of 360 ms with concentric atrial activation. Following entrainment pacing from the ventricle, a V-A-H-V response occurred. The postpacing interval minus the tachycardia cycle length from the right ventricular apex was 68 ms. Stimulus to atrium minus ventricular to atrium was 46 ms. Based on these findings, a diagnosis of AVRT was once again made. Using electroanatomical mapping during orthodromic AVRT, the earliest atrial signal was mapped to 8 o'clock on the mitral annulus where a Kent potential was identified (Fig. 1B). Retrograde conduction with the same activation sequence was abolished within 3 seconds of a second radiofrequency (RF) lesion after which SVT could not be reinduced. Thereafter, retrograde conduction during ventricular pacing showed concentric atrial activation.

She represented several months later with recurrent SVT with a cycle length of 380 ms. A second EP study was performed, and analogous to the first procedure, it showed two different atrial activation sequences during SVT (Fig. 2A). The two atrial activation sequences changed from one to the other during SVT with the same cycle length. Right ventricular entrainment showed V-A-V response; stim-A (265 ms) minus V-A (217 ms) was 48 ms, postpacing interval (PPI) (528 ms) minus tachycardia cycle length (TCL) (380 ms) was 148 ms, and a corrected PPI-TCL was 101 ms (corrected for AV delay). The earliest atrial activation site during SVT was located at posteroseptal and posterolateral site on the CS catheter. Based on these findings, the SVT was diagnosed as orthodromic AVRT using left-sided APs. During extensive mapping of the mitral annulus, a Kent potential was identified at 7 o'clock on the mitral annulus (Fig. 2B). One application of RF at this site terminated retrograde conduction over both APs, after which SVT could not be reinduced.

In this case, a single RF lesion resulted in the elimination of retrograde conduction over both pathways, suggesting the elimination of a single ventricular insertion for both atrial exit sites, a "stalked pathway."

Case Two

A 52-year-old male with a history of palpitations and previously documented SVT was referred to the EP lab for EP study (EPS) and ablation. On arrival to the EP lab, the HV interval was 42 ms. VA conduction was only present following isoproterenol infusion with a concentric pattern of atrial activation with earliest atrial activation at the His-bundle location. With atrial extra stimuli, a narrow complex tachycardia with a cycle length of 306 ms and eccentric atrial activation was induced and was incessant after isoproterenol infusion. During tachycardia, there was beat-to-beat variability in the eccentric atrial activation pattern without change in the tachycardia cycle length (Fig. 3A). There were two distinct eccentric atrial activation patterns seen with earliest activation in the distal CS[1,2] and at times earliest in more proximal CS.[7,8] Ventricular overdrive pacing

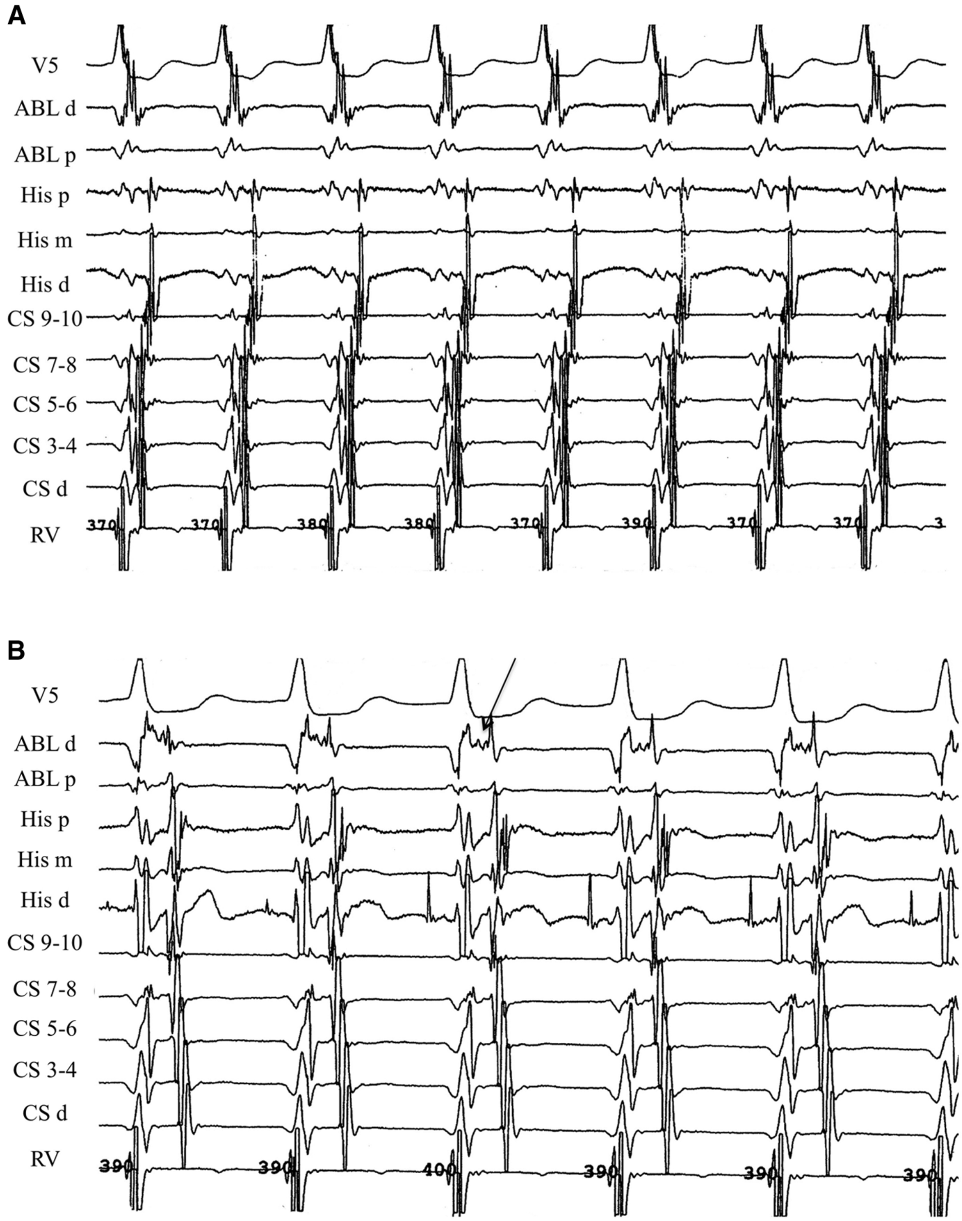

Figure 1. *(A) Successful ablation site of orthodromic reentrant tachycardia over a left posterolateral accessory pathway. Tachycardia cycle length was 380 ms and the earliest atrial activation potential was located at posterolateral (CS 5–6) site on mitral annulus. (B) Successful ablation site of orthodromic reentrant tachycardia with left posteroseptal accessory pathway. The arrow shows a possible Kent potential on the ablation catheter, which is located at 8 o'clock on mitral annulus. ABL = ablation catheter; CS = coronary sinus; RV = right ventricular.*

had a corrected PPI-TCL of 81 ms, corrected for the AV delay.[19] The $HA_{(Entrainment)} - HA_{(SVT)}$ was −26 ms.[20,21] During tachycardia, spontaneous left bundle branch block developed with an increase in the VA time from 115 ms to 159 ms (Fig. 3B). His synchronous PVCs advanced the subsequent A and reset the tachycardia cycle length. On the basis of these findings, a diagnosis of AVRT was made.

Due to the changing atrial activation sequences mapping was performed initially during ventricular pacing with verapamil. The site of earliest activation was mapped to the posteroseptal side of the mitral valve. After

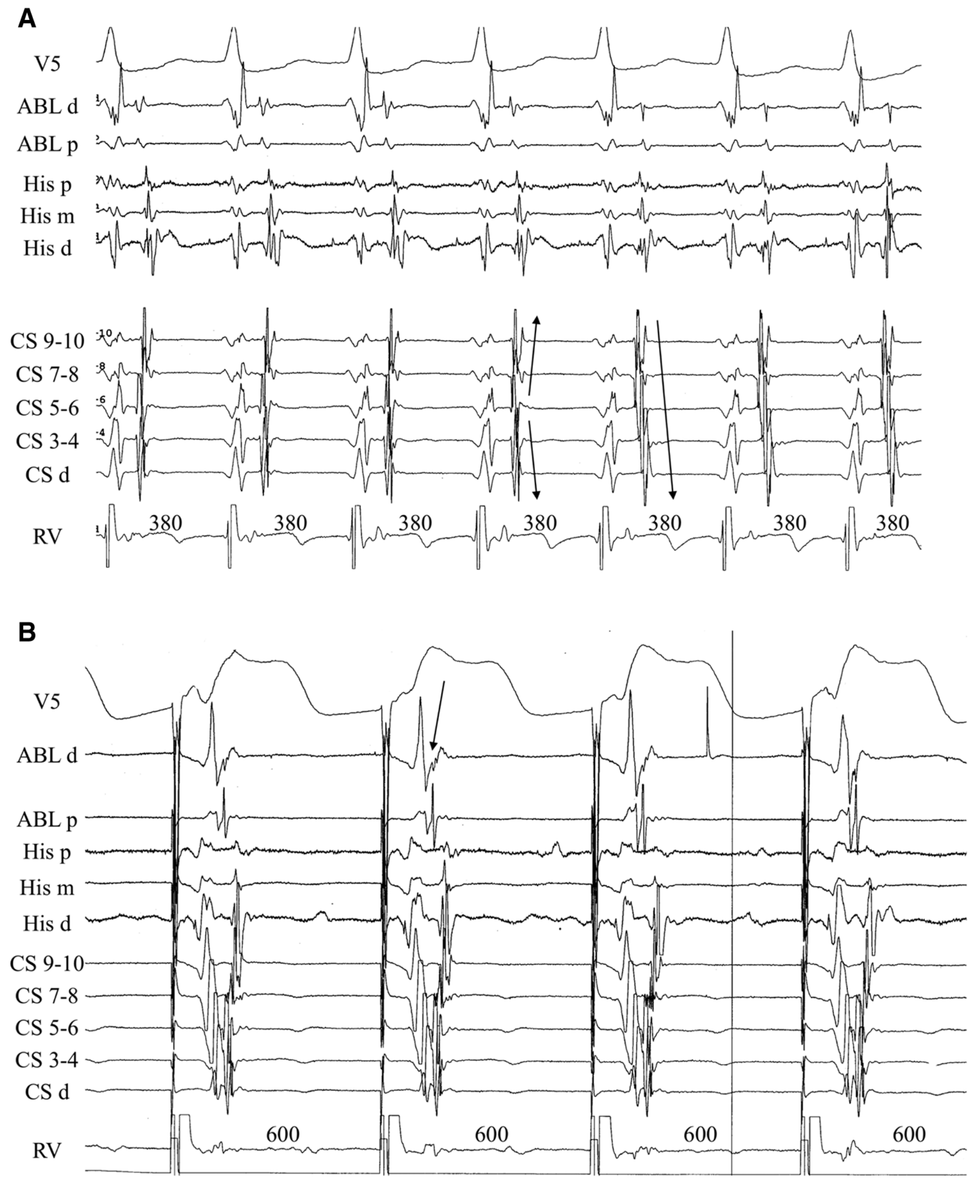

Figure 2. *(A) Orthodromic reentrant tachycardia during the second study. Tachycardia cycle length was 380 ms and the earliest atrial activation potential changed from posterolateral (CS 5–6) site to posteroseptal (CS 9–10) site on mitral annulus with the same cycle length. The arrows show the activation sequence change on coronary sinus catheter. (B) Successful ablation site during right ventricular pacing at 600 ms. The arrow shows a possible Kent potential on the ablation catheter at 7 o'clock on mitral annulus. Abbreviations as in Fig. 1.*

delivering lesions to the earliest atrial activation posteriorly, atrial activation changed to earliest at distal CS only, which was previously seen preablation. The site of earliest activation was then mapped during tachycardia to the anterolateral edge of the mitral valve where a Kent potential was identified. Successful ablation at this site resulted in the termination of tachycardia. Tachycardia could not be reinduced following ablation.

We postulate for case two that there were two discrete atrial insertion sites of a single ventricular insertion AP with two different atrial activation sequences before ablation but with constant tachycardia cycle length. We believe it would be unlikely that two different AVRT circuits

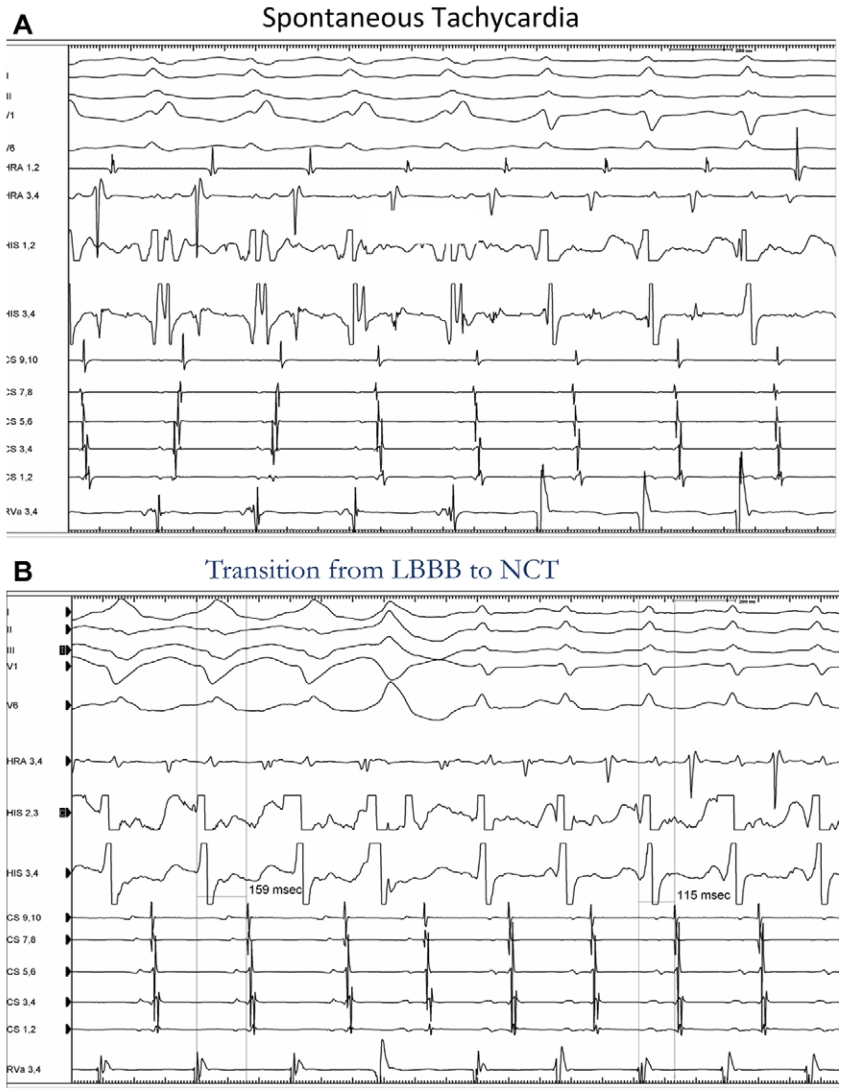

Figure 3. *(A) During tachycardia there was beat-to-beat variability in the eccentric atrial activation pattern without change in the tachycardia cycle length. There were two distinct eccentric atrial activation patterns seen with earliest activation in the distal CS[1,2] and at times earliest in more proximal CS.[7,8] Note, there was spontaneous right bundle branch block (RBBB) development with no significant change in the VA time.*
(B) During tachycardia, spontaneous LBBB developed with an increase in the VA time from 115 ms to 159 ms. LBBB = left bundle branch block; NCT = narrow complex tachycardia. Other abbreviations as in Fig. 1.
[Color figure can be viewed at wileyonlinelibrary.com]

utilizing two different concealed APs would have by chance shared identical electrophysiological properties. Both atrial insertions were targeted for ablation and after successfully mapping the earliest atrial activation to the posteroseptum and anterolateral annulus, tachycardia could not be reinduced and only concentric (septal) retrograde activation with earliest A in HIS was seen.

Case Three

A 64-year-old female with a history of SVT despite use of atenolol and diltiazem was referred for EP study and ablation. An electrocardiogram was obtained during SVT identified a long-RP tachycardia with narrow QRS complex at a rate of 117 beats/min. Baseline EPS revealed a HV interval of 50 ms. During ventricular pacing at

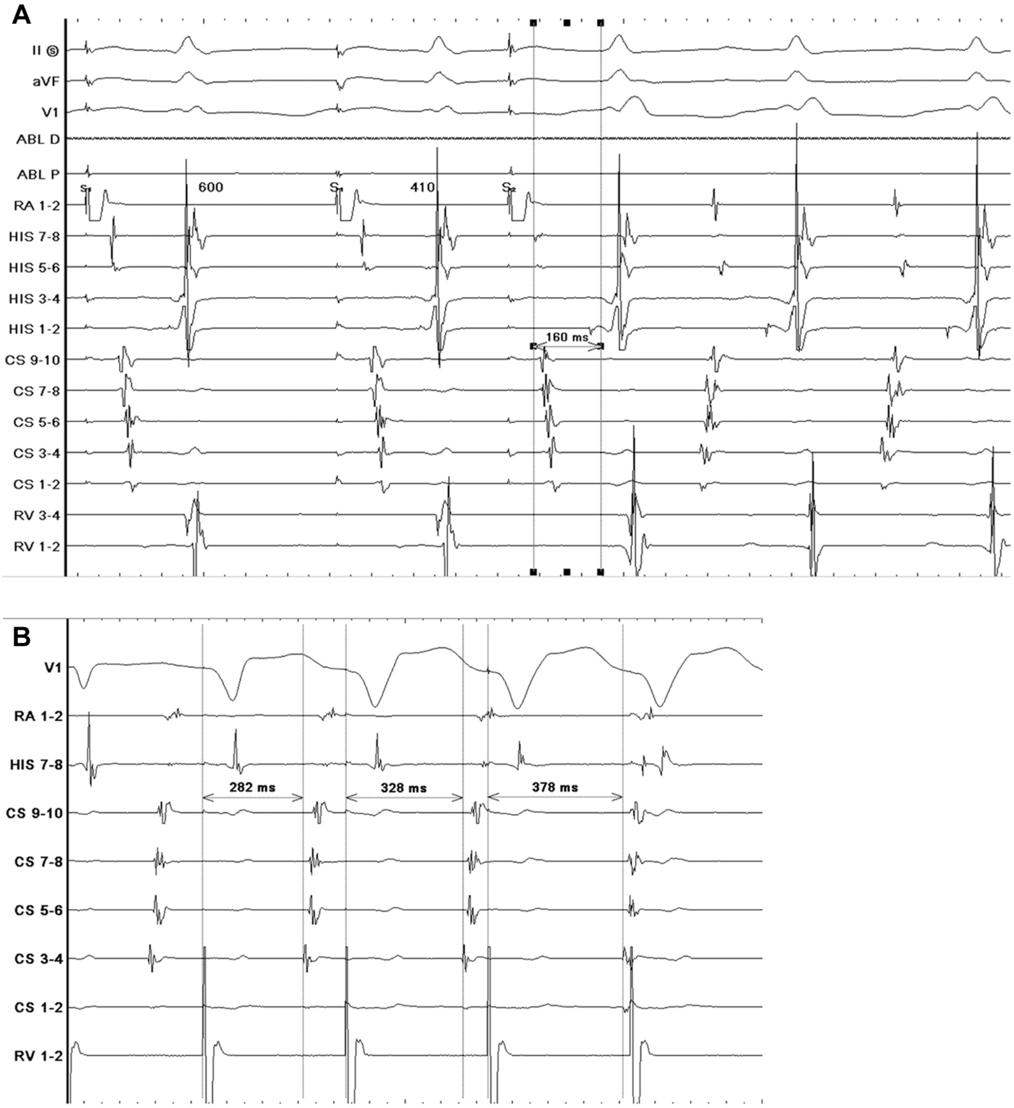

Figure 4. *(A) During atrial extra stimuli with at 600–410 ms with induction of tachycardia with a cycle length of 430 ms, atrial activation was eccentric with earliest activation at CS 1–2. (B) Ventricular pacing at decreasing cycle lengths caused incremental lengthening of the eccentric VA conduction time. RA = right atrial. Other abbreviations as in Fig. 1.*

380 ms from the apex, she had 2:1 VA conduction with the earliest atrial activation occurring in the proximal CS (9–10). During ventricular pacing at 350 ms, atrial activation became concentric with 2:1 VA conduction. During atrial extra stimulation, no AH jump was identified. During atrial extra stimuli with at cycle length of 600–410 ms, she had induction of tachycardia with a cycle length of 430 ms. Atrial activation was eccentric with earliest activation at CS (1–2) (Fig. 4A). During ventricular overdrive pacing, the acceleration of the atrial electrogram was in the transition zone, before the third fused beat. Following ventricular entrainment, there was a V-A-V response with a PPI minus TCL of 172 ms, and a corrected PPI-TCL[19] of 106 ms (corrected for AV delay). His refractory PVCs delayed the subsequent QRS complex. Ventricular pacing at decreasing cycle lengths caused incremental lengthening of the eccentric VA conduction time (Fig. 4B). A diagnosis of AVRT with a concealed decremental pathway was made.

Using electroanatomic mapping during orthodromic reentrant tachycardia (ORT), the site of earliest atrial activation was mapped to 3 o'clock on the mitral annulus. A Kent potential was not identified at this site. RF was applied during SVT with the termination of the rhythm. During ventricular pacing immediately following ablation, supra-ventricular tachycardia was reinduced. The SVT had a narrow QRS complex with a CL of 440 ms and concentric atrial activation, with proximal CS earliest (Fig. 5A). During ventricular overdrive pacing, the second fully fused beat brought in the subsequent atrial impulse. Following entrainment from the right ventricular apex, the corrected PPI-TCL was 109 ms. As with the first tachycardia, ventricular pacing at decreasing cycle lengths caused incremental lengthening of the eccentric VA conduction time (Fig. 5B). Mapping of the left atrium during ORT identified the site of earliest atrial activation at 10 o'clock on the mitral annulus. A Kent potential was identified at this site (Fig. 5C). RF was applied to this site with the termination of the arrhythmia. Following RF, VA conduction was no longer present at 600 ms. SVT could no longer be diagnosed.

The evidence suggests, based on these findings, that a bifurcating concealed decremental pathway was present. There were two different atrial activation patterns diagnosed during tachycardia at similar cycle lengths (10 ms apart) with both tachycardia mechanisms proving to be AVRT by standard pacing maneuvers. The atrial insertion sites were mapped to 3 o'clock and 10 o'clock on the mitral annulus. An alternative explanation of two different concealed decremental pathways is less plausible as they would need to share the same electrophysiologic properties (decremental conduction) to allow for the minimal change in cycle length.

Discussion

We present three cases of AVRT utilizing concealed pathways with evidence that each utilizes a single ventricular insertion and two discrete atrial insertion sites.

Main Findings

In case one, two discrete atrial insertion sites were mapped in two separate procedures, and only during the second ablation was the Kent potential identified. Ablation of the Kent potential at this site remote from the two atrial insertion sites resulted in the termination of the retrograde conduction in both pathways. A single ablation lesion eliminated retrograde conduction in both pathways argues for a single ventricular insertion (Fig. 6).

Case two presented with SVT with alternating eccentric atrial activation patterns without alteration in the tachycardia cycle length. The tachycardia was diagnosed as orthodromic AVRT

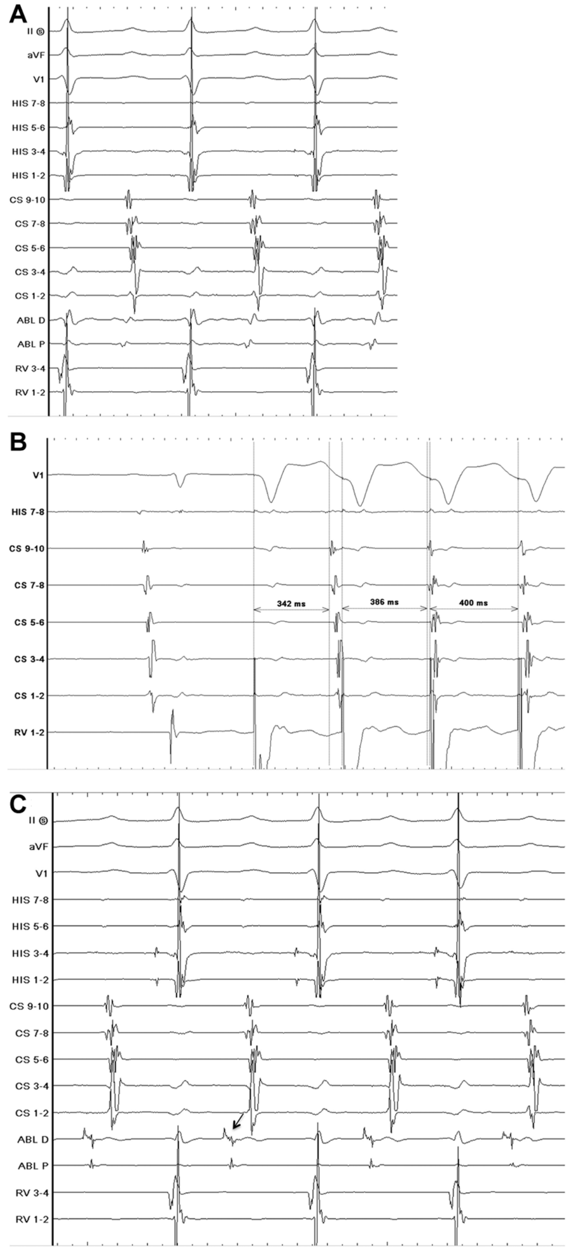

Figure 5. *(A) Second tachycardia after successful catheter ablation of first at 3 o'clock on the mitral annulus. Supraventricular tachycardia with a narrow QRS complex at a cycle length 440 ms with atrial activation, earliest in the proximal CS.*
(B) As with the first tachycardia, ventricular pacing at decreasing cycle lengths caused incremental lengthening of the eccentric VA conduction time. VA time is measured from stimulus to earliest A on proximal CS[9,10] showing decrementing VA times.
(C) Mapping of the left atrium during orthodromic reentrant tachycardia identified the site of earliest atrial activation at 10 o'clock on the mitral annulus. A Kent potential was identified at this site. Abbreviations as in previous figures.

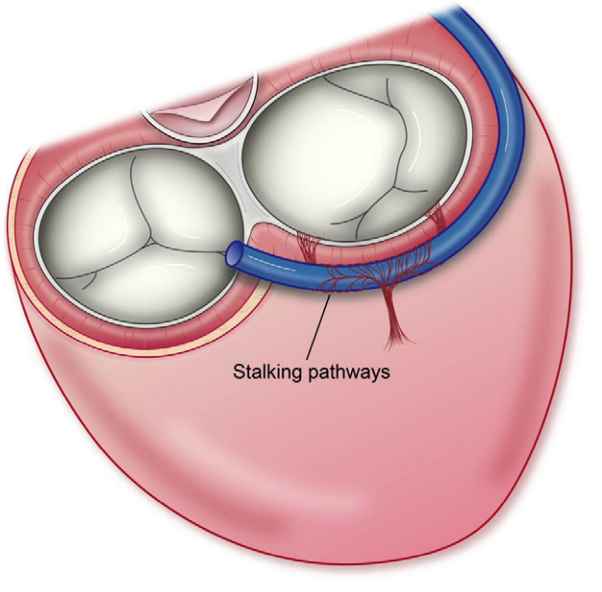

Figure 6. *Illustration showing the proposed branching pathway from case 1, with a single ventricular and two atrial insertion sites. Ablation of the Kent potential at the ventricular insertion site remote from the two atrial insertion sites resulted in the termination of the retrograde conduction in both atrial insertion sites. Illustration created by Carol Dizack, Media Solutions, University of Wisconsin, Madison, WI. [Color figure can be viewed at wileyonlinelibrary.com]*

by standard pacing techniques.[1,2,19–24] The electrophysiologists choose to target the atrial insertion site during orthodromic AVRT and ventricular pacing and each of the two atrial insertion sites were successfully mapped and ablated.

In case three, retrograde decremental conduction utilizing both atrial insertion sites was identified prior to ablation. After mapping and ablation of the first discrete atrial insertion site, tachycardia persisted utilizing the second atrial insertion site. Only after ablation of the second atrial insertion site was SVT noninducible, and VA conduction no longer present.

There have been previous reports in the literature of AVRT utilizing two or more discrete pathways.[1,2,6–11,13,14,16–18,23,25] In our case series, each case has a single AP with separate atrial insertion sites. There are several findings which support this hypothesis that there is a single ventricular insertion site and not two separate pathways. First, in case one, ablation at this Kent potential site distant from the two atrial insertion sites resulted in the termination of retrograde conduction along both pathways, indicating that there was a common stalk for these two anatomically separate pathways. The change in atrial activation pattern following RF delivery is not consistent with the creation of block within the mitral isthmus or CS muscle as previously described by Luria et al.,[26] Cheng et al.,[27] and Vijayaraman et al.[28] in which extensive ablation on the mitral annulus leads to mitral annular block with reverse change in the atrial wavefront activation of the CS. Nor would this be consistent on an oblique course of the pathways as described by Otomo et al.[29] and colleagues as the atrial insertion sites are anatomically too distant. Second, we suspect that it would be very unusual to have multiple decremental pathways in the same case with similar cycle length, suggesting the two atrial insertion sites identified in case 3 resulted from the same pathway.

Becker and colleagues[30] have performed extensive anatomical descriptions of APs showing discrete bundles. They described in some cases branching at the ventricular end of the bundle close to the ventricular insertion site where the bundle may break into a number of small branches, each of which anchors itself into the ventricular myocardium. The operative experience by Guiraudon et al. describe similar characteristics.[31,32] There has never been an anatomical description of branching pattern of the atrial insertion sites.

The most likely anatomical explanation for this finding is the presence of a single ventricular insertion utilizing the CS musculature (Fig. 6). Sun and colleagues[33] previously described CS-ventricular APs formed by the CS myocardial coat with extensive connections to the atrium. These original descriptions were not discontinuous as we describe in our cases; however, we feel this utilization of the CS musculature may allow separate pathways to course along the CS musculature and create discrete atrial insertion sites. The Kent potentials identified remote from the sites of atrial insertion most likely corresponds to the common ventricular insertion site for both atrial pathways.

Therefore, successful treatment of AVRT utilizing two discrete atrial insertion sites requires either (1) identification and ablation of the single ventricular insertion site (as was performed in case 1) or (2) ablation of both atrial insertion sites (as was performed in cases 2 and 3).

Limitations

This is a small series of only three patients from three different centers. Although this not a large series of patients the unique and seemingly rare descriptions of these pathways we feel are of interest. The pathways' potentials in these cases (case 1 and 2) were not strictly verified as previously described by Otomo and colleagues.[29] However, in case 1, it is of interest that this potential was the precise site that resulted simultaneous ablation of both atrial insertion sites.

Conclusion

Concealed retrograde APs with discrete atrial insertion sites may have a common ventricular insertion site. Identification and ablation of the ventricular insertion site, or the separate discrete atrial insertion sites results in successful treatment.

References

1. Lee KW, Badhwar N, Scheinman MM. Supraventricular tachycardia–part I. Curr Probl Cardiol 2008; 33:467–546.
2. Lee KW, Badhwar N, Scheinman MM. Supraventricular tachycardia-Part II: History, presentation, mechanism, and treatment. Curr Probl Cardiol 2008; 33:557–622.
3. Nakagawa H, Jackman WM. Catheter ablation of paroxysmal supraventricular tachycardia. Circulation 2007; 116:2465–2478.
4. Obel OA, Camm AJ. Accessory pathway reciprocating tachycardia. Eur Heart J 1998; 19(Suppl E):E13-24, E50-1.
5. Scheinman MM. The history of the Wolff-Parkinson-White syndrome. Rambam Maimonides Med J 2012; 3:e0019.
6. Chen S, Zhou G, Liu S. Multiple manifested accessory atrioventricular pathways in a patient without obvious structural heart disease. J Cardiovasc Electrophysiol 2015; 26:448–449.
7. Gonzalez JE, Zipse MM, Nguyen DT, Sauer WH. Antidromic atrioventricular reciprocating tachycardia using a concealed retrograde conducting left lateral accessory pathway. Card Electrophysiol Clin 2016; 8:37–43.
8. Haissaguerre M, Puel V, Bekheit S, Fisher B, Dartigues JF, Egloff P, Le Metayer P, et al. Catheter ablation of accessory pathways in children. Eur Heart J 1994; 15:200–205.
9. Huang JL, Chen SA, Tai CT, Chiang CE, Lee SH, Chiou CW, Ueng KC, et al. Long-term results of radiofrequency catheter ablation in patients with multiple accessory pathways. Am J Cardiol 1996; 78:1375–1379.
10. Iesaka Y, Yamane T, Takahashi A, Goya M, Kojima S, Soejima Y, Okamoto Y, et al. Retrograde multiple and multifiber accessory pathway conduction in the Wolff-Parkinson-White syndrome: Potential precipitating factor of atrial fibrillation. J Cardiovasc Electrophysiol 1998; 9:141–151.
11. Iturralde P, Guevara-Valdivia M, Rodriguez-Chavez L, Medeiros A, Colin L. Radiofrequency ablation of multiple accessory pathways. Europace 2002; 4:273–280.
12. Jackman WM, Wang XZ, Friday KJ, Roman CA, Moulton KP, Beckman KJ, McClelland JH, et al. Catheter ablation of accessory atrioventricular pathways (Wolff-Parkinson-White syndrome) by radiofrequency current. N Engl J Med 1991; 324:1605–1611.
13. Kuck KH, Schluter M, Geiger M, Siebels J, Duckeck W. Radiofrequency current catheter ablation of accessory atrioventricular pathways. Lancet 1991; 337:1557–1561.
14. Sacher F, Wright M, Tedrow UB, O'Neill MD, Jais P, Hocini M, Macdonald R, et al. Wolff-Parkinson-White ablation after a prior failure: A 7-year multicentre experience. Europace 2010; 12:835–841.
15. Schluter M, Schluter CA, Cappato R, Hebe J, Siebels J, Kuck KH. Anatomic distribution, conduction properties and recurrences after ablation of multiple in comparison with single accessory conduction pathways. Z Kardiol 1997; 86:221–230.
16. Valentino M, Mont L, Aguinaga L, Anguera I, Eizmendi I, Sanchez J, Guillamon L, et al. Radiofrequency ablation of multiple atrioventricular accessory pathways in a patient with syncope, atrial fibrillation and fasciculoventricular fibers. Rev Esp Cardiol 1998; 51:591–595.
17. Weng KP, Wolff GS, Young ML. Multiple accessory pathways in pediatric patients with Wolff-Parkinson-White syndrome. Am J Cardiol 2003; 91:1178–1183.
18. Zachariah JP, Walsh EP, Triedman JK, Berul CI, Cecchin F, Alexander ME, Bevilacqua LM. Multiple accessory pathways in the young: The impact of structural heart disease. Am Heart J 2013; 165:87–92.
19. Gonzalez-Torrecilla E, Arenal A, Atienza F, Osca J, Garcia-Fernandez J, Puchol A, Sanchez A, et al. First postpacing interval after tachycardia entrainment with correction for atrioventricular node delay: A simple maneuver for differential diagnosis of atrioventricular nodal reentrant tachycardias versus orthodromic reciprocating tachycardias. Heart Rhythm 2006; 3:674–679.
20. Ho RT, Mark GE, Rhim ES, Pavri BB, Greenspon AJ. Differentiating atrioventricular nodal reentrant tachycardia from atrioventricular reentrant tachycardia by DeltaHA values during entrainment from the ventricle. Heart Rhythm 2008; 5:83–88.
21. Miller JM, Rosenthal ME, Gottlieb CD, Vassallo JA, Josephson ME. Usefulness of the delta HA interval to accurately distinguish atrioventricular nodal reentry from orthodromic septal bypass tract tachycardias. Am J Cardiol 1991; 68:1037–1044.
22. Knight BP, Ebinger M, Oral H, Kim MH, Sticherling C, Pelosi F, Michaud GF, et al. Diagnostic value of tachycardia features and pacing maneuvers during paroxysmal supraventricular tachycardia. J Am Coll Cardiol 2000; 36:574–582.
23. Knight BP, Morady F. Supraventricular tachyarrhythmia. Curr Treat Options Cardiovasc Med 2000; 2:297–308.
24. Michaud GF, Tada H, Chough S, Baker R, Wasmer K, Sticherling C, Oral H, et al. 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.
25. Bhatia A, Sra J, Akhtar M. Preexcitation syndromes. Curr Probl Cardiol 2016; 41:99–137.
26. Luria DM, Nemec J, Etheridge SP, Compton SJ, Klein RC, Chugh SS, Munger TM, et al. Intra-atrial conduction block along the mitral valve annulus during accessory pathway ablation: Evidence for a left atrial "isthmus." J Cardiovasc Electrophysiol 2001; 12: 744–749.
27. Cheng J, Yang Y, Ursell PC, Lee RJ, Dorostkar PC, Boahene KA, Scheinman MM. Protected circumferential conduction in the posterior atrioventricular vestibule of the left atrium: Electrophysiologic and anatomic correlates. Pacing Clin Electrophysiol 2005; 28:692–701.
28. Vijayaraman P, Wood MA, Shepard RK, Ellenbogen KA. Change in coronary sinus activation following catheter ablation: What is the mechanism? J Cardiovasc Electrophysiol 2011; 22:720–722.
29. Otomo K, Gonzalez MD, Beckman KJ, Nakagawa H, Becker AE, Shah N, Matsudaira K, et al. 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.
30. Becker AE, Anderson RH, Durrer D, Wellens HJJ. The anatomical substrates of Wolff-Parkinson-White syndrome: A clinicopathologic correlation in seven patients. Circulation 1978; 57:870–879.
31. Guiraudon GM, Klein GJ, Harma AD, Yee R, Pineada EA. Atypical posteroseptal accessory pathway in Wolf-Parkinson-White syndrome. J Am Coll Cardiol 1988; 12:1605–1608.
32. Guiraudon GM, Klein GJ, Sharma AD, Yee R, Pineada EA, McLellan DG. Surgical approach to anterior septal accessory pathways in 20 patients with the Wolff-Parkinson-White syndrome. Eur J Cardiothorac Surg 1988; 2:201–206.
33. Sun Y, Arruda M, Otomo K, Beckman K, Nakagawa H, Calame J, Po S, et al. Coronary sinus-ventricular accessory connections producing posteroseptal and left posterior accessory pathways: Incidence and electrophysiological identification. Circulation 2002; 106:1362–1367.