Novel analysis of ventriculoatrial interval prolongation during a narrow QRS tachycardia using a right atrial nondecremental-midseptal accessory pathway

Kazushi Tanaka, MD, PhD, FACC, Shinji Shiotani, MD

From the Cardiac Arrhythmia Center, Department of Medicine, Kishiwada Tokushukai Hospital, Osaka, Japan.

Introduction
It is well known as Coumel’s sign¹ that a ventriculoatrial (VA) interval is prolonged during an orthodromic atrioventricular (AV) reentrant tachycardia (AVRT) with appearance of a functional bundle branch block (BBB) ipsilateral to a free wall AV accessory pathway (AP). Also, a slant AP has a characteristic altering a VA interval dependent on ventricular pacing sites.²,³

In our case with a right atrial (RA) midseptal nondecremental concealed AV AP, a VA interval is prolonged during a supraventricular tachycardia (SVT) as compared with that during right ventricular (RV) apical pacing in sinus rhythm without the above conditions.

A novel mechanism is proposed to explain prolongation of VA interval in SVT as compared to RV pacing in non-slanted right-sided pathways.

Case report
In a 69-year-old man with palpitation since the age of 42 years and no structural heart disease or antiarrhythmic drugs, an electrocardiogram (ECG) during palpitation has an SVT followed by negative P waves in inferior leads at a rate of 205 beats/min. A surface 12-lead ECG in normal sinus rhythm had no delta wave.

Differential diagnosis
An SVT should be either an atrial reentrant tachycardia, an atrioventricular node reentrant tachycardia, an AVRT, or a reentrant tachycardia using an atrionodal (or atrio-His) AP.

KEY TEACHING POINTS
- Ventriculoatrial (VA) interval prolongation during an orthodromic atrioventricular (AV) reentrant tachycardia (AVRT) with appearance of a bundle branch block (BBB) ipsilateral to an AV accessory pathway (AP) is well known as Coumel’s sign. Otherwise, VA interval over a slant type of AP different from a perpendicular type along an annulus is considered to be changeable depending on ventricular pacing sites.
- VA interval during orthodromic AVRT using a right atrial (RA) midseptal AV-AP with no BBB can be longer than that during right ventricular (RV) pacing. The mechanism may be ascribed to the activated time lag of the endocardial interventricular septum (IVS) of the RV as compared with activation of the left ventricular IVS during AVRT rather than the slant AP.
- An RA midseptal AV-AP is located at the superficial endocardial site. Therefore, in order that ablation of this type of AP is safely achieved, it should be performed with a low energy output. Moreover, notice that appearance of junctional beats during energy applications is suggestive of a warning sign of a complication of AV block.

A standard electrophysiologic study was performed using multipolar-electrode catheters that were positioned in the high right atrium, His bundle, RV apex, and coronary sinus. At baseline, programmed RV pacing showed a retrograde VA conduction with a concentric atrial activation sequence and nondecremental fashion and any atrial pacing maneuver could induce a nonsustained SVT alone. After administration of isoproterenol (1, 2 or 4 mcg/min), programmed atrial pacing could provoke a sustained SVT similar to a clinical one. Figure 1A and 1B show an ECG and intracardiac recordings...
Figure 1  A supraventricular reentrant tachycardia (SVT). A: Electrocardiogram (ECG). Arrowheads indicate negative P waves in inferior leads. B: Intracardiac recordings. A, H, and V indicate atrial, His bundle, and ventricular depolarizations, respectively. Tracings from top to bottom indicate surface ECG leads I, II, V1, V6, and intracardiac electrograms of high right atrium (HRA, p and d; proximal and distal electrode pair), His bundle (HB, proximal 9-10 and distal 1-2), coronary sinus (CS), and right ventricle (RV). A tachycardia has a cycle length of 240 ms (atrio-His [AH] 130 ms and His-atrial [HA] interval 110 ms) and a retrograde concentric atrial activation sequence. Order of ECG leads and intracardiac recordings and abbreviations are also the same as below figures. C: Initiation by RV apex constant pacing with a cycle length of 600 ms in sinus rhythm. ABLp and p indicate distal and proximal electrode pair of an ablation catheter, respectively; CSos indicates the ostium of the coronary sinus. After 3 RV stimuli (Sts) from the left conduct to atrial myocardium (marked by asterisks) over an accessory pathway retrogradely and afterward 4 atrial depolarizations appeared, an SVT with a cycle length of 270 ms is induced following a long AV interval (shown by a broken line) and A-V-A sequence (shown by an arrow). The fourth and fifth Sts cannot capture ventricular myocardium prematurely. Note that a ventriculoatrial (VA) interval during SVT is longer by 20 ms than that during RV pacing (70 ms vs 50 ms).
of the SVT, respectively. SVT had a cycle length (CL) of 240 ms and a retrograde concentric atrial activation sequence. In Figure 1C, a sustained SVT with a CL of 270 ms could be induced by constant RV apex pacing (RV1,2) with a CL of 600 ms during sinus rhythm.

Determination of SVT

Figure 1C illustrates that the SVT with a CL of 270 ms was initiated following a long AV interval (shown by a broken line) and an atrial-ventricular-atrial (A-V-A) sequence. These findings are inconsistent with atrial tachycardia. Also, a VA interval during SVT with no BBB was longer than that during RV pacing with a CL of 600 ms in sinus rhythm, which might be seemingly suggestive of a retrograde VA conduction with a decremental property. Further analysis regarding the mechanism of this difference was described afterward.

Next, in Figure 2A, a premature RV stimulus introduced immediately after the His bundle depolarization (H) could terminate SVT. Additionally, Figure 2B indicates RV overdrive pacing with a cycle length of 230 ms during SVT with a CL of 255 ms. The first stimulus (shown by St with an arrow) introduced immediately after the H potential could capture only atrial myocardium prematurely, with neither change of the ventricular CL and the retrograde atrial activation sequence nor termination of SVT. These findings strongly suggested that SVT was an orthodromic AVRT but not AV nodal reentrant tachycardia.

Radiofrequency catheter ablation

Mapping into the Koch triangle during the AVRT using an ablation catheter indicated that the AP existed in the RA midseptal portion, where the earliest retrograde atrial activation site was confirmed (Figure 2C, 2D).²,⁵

At first, radiofrequency catheter ablation (RF) using a 4 mm irrigated-tip catheter (FlexAbility, D-D curve; Abbott, Plymouth, MN) was achieved at the ostial roof of the coronary sinus during AVRT in order to avoid AV block, which could result in transient termination alone. Next, RF on conditions of a low energy output of 20 W, a maximum temperature of 42°C, and a flow rate of 17 mL/s was achieved at the RA midseptal site with an AP potential (shown by asterisks in Figure 2D), so that an instantaneous termination of AVRT owing to the AP conduction block could be accomplished. However, energy delivery was stopped for 60 seconds because some junctional beats occurred followed by no AV block. No bonus RF was added.²,⁵

Discussion

So far, it has been considered to be suggestive of a slant AP that the difference between VA intervals during an AVRT with no BBB and RV pacing is 15 ms or more.²,³ Similarly, in our case, the same phenomenon could be observed. At baseline, Figure 3A and 3B showed VA intervals during a nonsustained AVRT with a CL of 320 ms and constant RV pacing with a CL of 330 ms in sinus rhythm, respectively. As well, a VA interval of AVRT was longer than that during RV pacing, as observed in Figure 1C. The finding shows that the VA interval prolongation during AVRT in our case was independent of variety of a CL.

On one hand, Otomo and colleagues² revealed that the change of VA interval in an oblique AP could be
provoked by reversing the direction of the activation wavefronts paced at different ventricular sites along the valvular annulus from counterclockwise to clockwise, or vice versa.

On the other hand, the normal conduction within both ventricles of human heart in sinus rhythm without a BBB is as follows. The first excitation starts at the interventricular septum (IVS) of the endocardial left ventricle (LV), followed by the anterior side of the RV apex through the His-Purkinje system and succeedingly the endocardial RV-IVS, requiring total conduction time of 20–30 ms (known as the septal Q waves). Such an activation pattern within the RV should be considered identical during both an AVRT with no BBB and the RV apex pacing.

Therefore, it is very difficult to conclude that in our case, both activation wavefronts during AVRT and RV apex pacing could reverse the direction along the tricuspid annulus. Thus, we speculate that the difference between both VA intervals in our case might be based on not the slant but the conduction time lag from the LV-IVS until the RV-IVS, like masquerading as Coumel’s sign without a BBB.

Figure 2 (continued).
Figure 3  Comparison of ventriculoatrial (VA) intervals. A: A nonsustained orthodromic atrioventricular reentrant tachycardia (AVRT) with a cycle length of 320 ms. An AVRT was induced by constant pacing with a cycle length of 330 ms (St-St) from the proximal electrode pair of coronary sinus (CS9,10). A VA interval during AVRT was 180 ms, measured as an interval from the onset of QRS to the positive peak of the atrial potential on high right atrium (HRA) (shown by an arrow). St = stimulus artefact; VAB = ventriculoatrial block. B: Right ventricular apex (RV) pacing with a cycle length of 330 ms in sinus rhythm. A VA interval during RV constant pacing with a cycle length of 330 ms was 160 ms. RV pacing with the shorter cycle length resulted in 2:1 VAB. C: Ladder diagrams during AVRT and RV pacing. AP = accessory pathway; AVN = atrioventricular node; HB = His bundle; LV-IVS = interventricular septum of left ventricle; RA-IASm = mid-interatrial septum of right atrium; RV-IVS = interventricular septum of right ventricle. Solid lines indicate VA intervals during AVRT and RV pacing. Solid and broken arrows indicate antegrade and retrograde activation conduction sequences, respectively. Asterisk indicates the putative insertion site of AP within RV-IVS. See text for details.
If the BBB ipsilateral to an AP was present during an orthodromic AVRT, a premature beat elicited in the ventricle of the BBB could make a VA interval shorter, namely the “paradoxically premature atrial captures.”3,7 This is explained by premature excitation of the pathway of tachycardia by the induced stimulus prior to the delayed arrival (caused by the BBB). Similarly, we consider that the VA interval shortening in Figure 2B should imply that before a tachycardia impulse would arrive at the IVS of the endocardial RV via the apex, the activation wavefront of the RV apex pacing could reach the RV-IVS insertion site to atrial potential, respectively, so that a VA interval during the former is longer by 20 ms than that during the latter, as shown in Figure 3A and 3B. The difference should be almost the same in timing for a septal AP was not approved in Japan. However, the default can be considered not to be so important as to upset our hypothesis. The reason is that although the phenomenon of VA interval prolongation observed with a slant AP is induced by reversing the direction of paced ventricular wavefront,2 in our case the prolongation during AVRT can be provoked by activation time difference between both sides of the IVS but not by reversing.

Conclusion
In a concealed nondecremental RA midseptal AP, the phenomenon that a VA interval during an orthodromic AVRT without a BBB can be prolonged as compared with that during RV pacing can be explained by our novel theory, namely the difference in timing of LV-IVS endocardial vs RV-IVS endocardial activation.

Acknowledgment
A part of the contents of this case report was presented at the 10th Asia Pacific Heart Rhythm Society Scientific Session (APHRS 2017) held in Yokohama, Japan in 2017.

References
1. 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.
2. Otomo K, Gonzalez MD, Beckman KJ, 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.
3. Kanawati J, Roberts JD, Rowe MK, et al. A simple maneuver to determine if septal accessory pathway ablation requires a left atrial approach. J Cardiovasc Electrophysiol 2020;31:3207–3214.
4. Gaita F, Riccardi R, Hocini M, et al. Safety and efficacy of cryoablation of accessory pathways adjacent to the normal conduction system. J Cardiovasc Electrophysiol 2003;14:825–829.
5. Kuck KH, Schluter M, Gursoy S. Preservation of atrioventricular nodal conduction during radiofrequency current catheter ablation of midseptal accessory pathway. Circulation 1992;86:1743–1752.
6. Durrer D, van Dam RT, Freud GF, Janse MJ, Meijler FJ, Arzbuecher RC. Total excitation of the isolated human heart. Circulation 1970;41:899–912.
7. Weiss J, Brugada P, Roy D, Bar FHWM, Wellens HJJ. Localization of the accessory pathway in the Wolff-Parkinson-White syndrome from the ventriculo-atrial conduction time of right ventricular apical extrasystoles. Pacing Clin Electrophysiol 1983;6:260–267.
8. Jazayeri MR, Dhala A, Deshpande S, Blanck Z, Sra J, Akhtar M. Posteroseptal accessory pathways: an overview of anatomical characteristics, electrocardiographic patterns, electrophysiological features, and ablative therapy. J Interv Cardiol 1995:8:89–101.
9. Bellhassen B, Viskin S, Fish R, Glick A, Glikson M, Eldar M. Catheter-induced mechanical trauma to accessory pathways during radiofrequency ablation: incidence, predictors and clinical implications. J Am Coll Cardiol 1999;33:767–774.
10. Marazzato J, Fente G, Marazzi R, et al. Efficacy and safety of cryoablation of para-Hisian and mid-septal accessory pathways using a specific protocol: single-center experience in consecutive patients. J Interv Card Electrophysiol 2019;55:47–54.