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

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Introduction

It is well known as Coumel's sign[1] 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.

KEYWORDS Accessory atrioventricular pathway; Coumel's sign; Electrophysiology; Orthodromic atrioventricular reentrant tachycardia; Paradoxically premature atrial capture; Radiofrequency catheter ablation; Right mid-septum; Supraventricular tachycardia; Ventriculoatrial interval prolongation
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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

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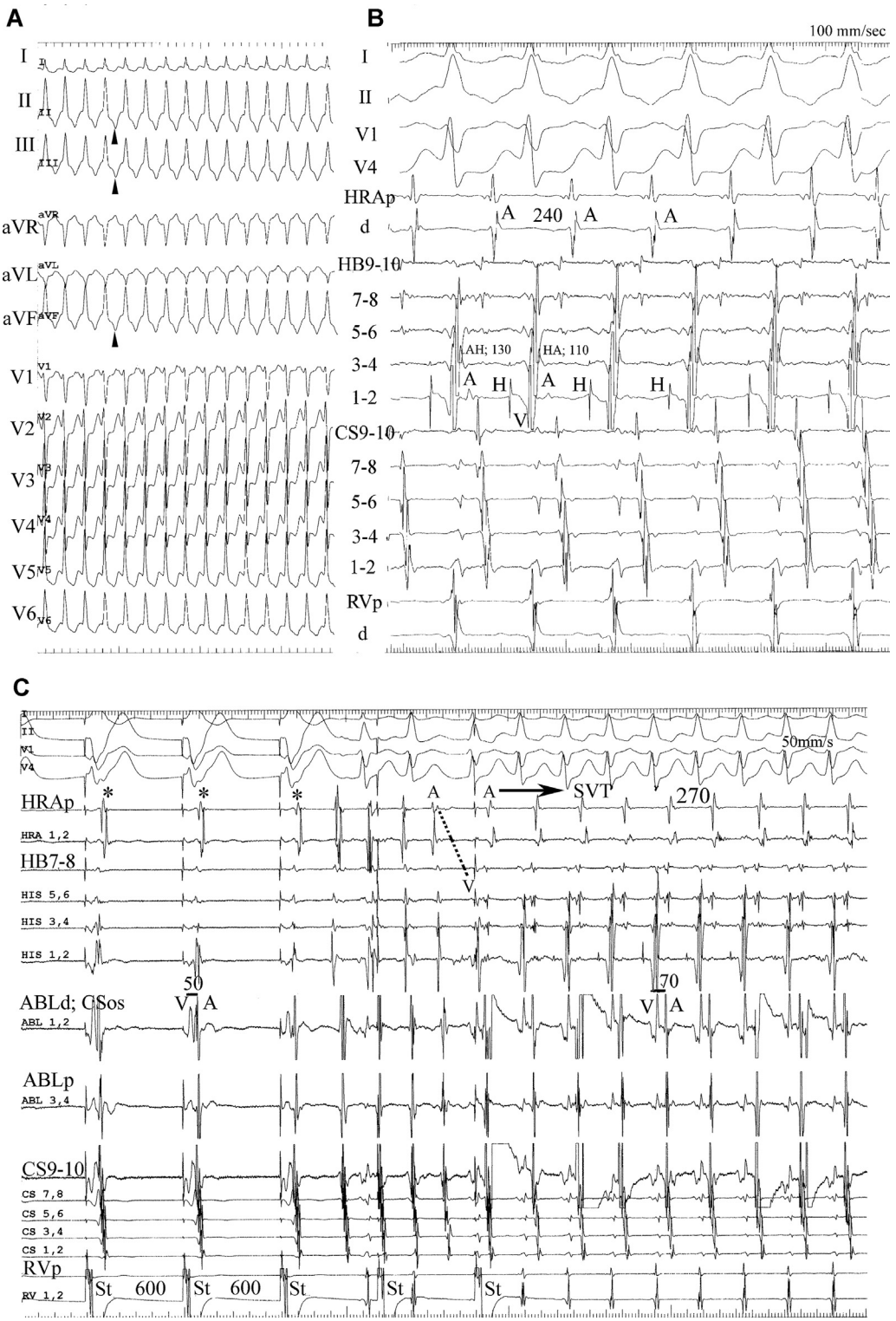

Figure 1 A supraventricular reentrant tachycardia (SVT). **A:** Electrocardiogram (ECG). Arrowheads indicate negative P waves in inferior leads. **B:** Intracardiac recordings. A, H, and V indicate atrial, His bundle, and ventricular depolarizations, respectively. Tracings from top to bottom indicate surface ECG leads I, II, V_1, V_4, 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. ABLd 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).

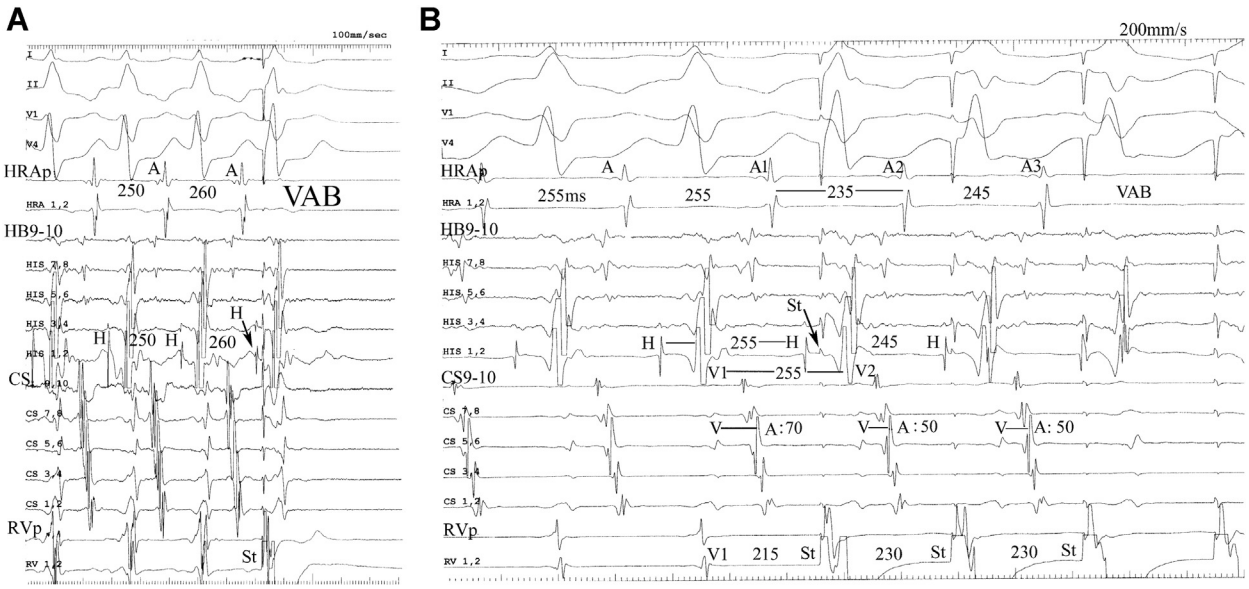

Figure 2 Termination of tachycardia. **A:** A premature stimulus (St) at the right ventricular apex (RV) delivered immediately after registration of antegradely depolarized His bundle (H; *arrow*) can terminate tachycardia due to ventriculoatrial block (VAB). **B:** RV overdrive pacing with a cycle length of 230 ms. A1/A2 and V1/V2 indicate atrial and ventricular depolarizations during tachycardia before (1)/after (2) the first St (*arrow*) from the left, respectively. A3 indicates an atrial depolarization owing to the second St. A1-A2 interval (235 ms) is shortened without change of V1-V2 (and H-H) interval (255 ms). Also, the second reduces a VA interval and succeedingly the third can result in VAB. **C:** Ablation fluoroscopic images. ABL = radiofrequency ablation; CS = coronary sinus; HB = His bundle; HRA = high right atrium; LAO = left anterior oblique view; RAO = right anterior oblique view; RV = right ventricular apex catheter. **D:** Ablation energy application. ABLd indicates distal electrode pair of ablation catheter. Asterisks indicate putative accessory pathway potentials. Vertical line indicates commencement of radiofrequency catheter ablation (RF). Tachycardia terminates instantaneously by RF.

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 at a CL 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).[4,5]

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.[4,5]

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 colleagues[2] revealed that the change of VA interval in an oblique AP could be

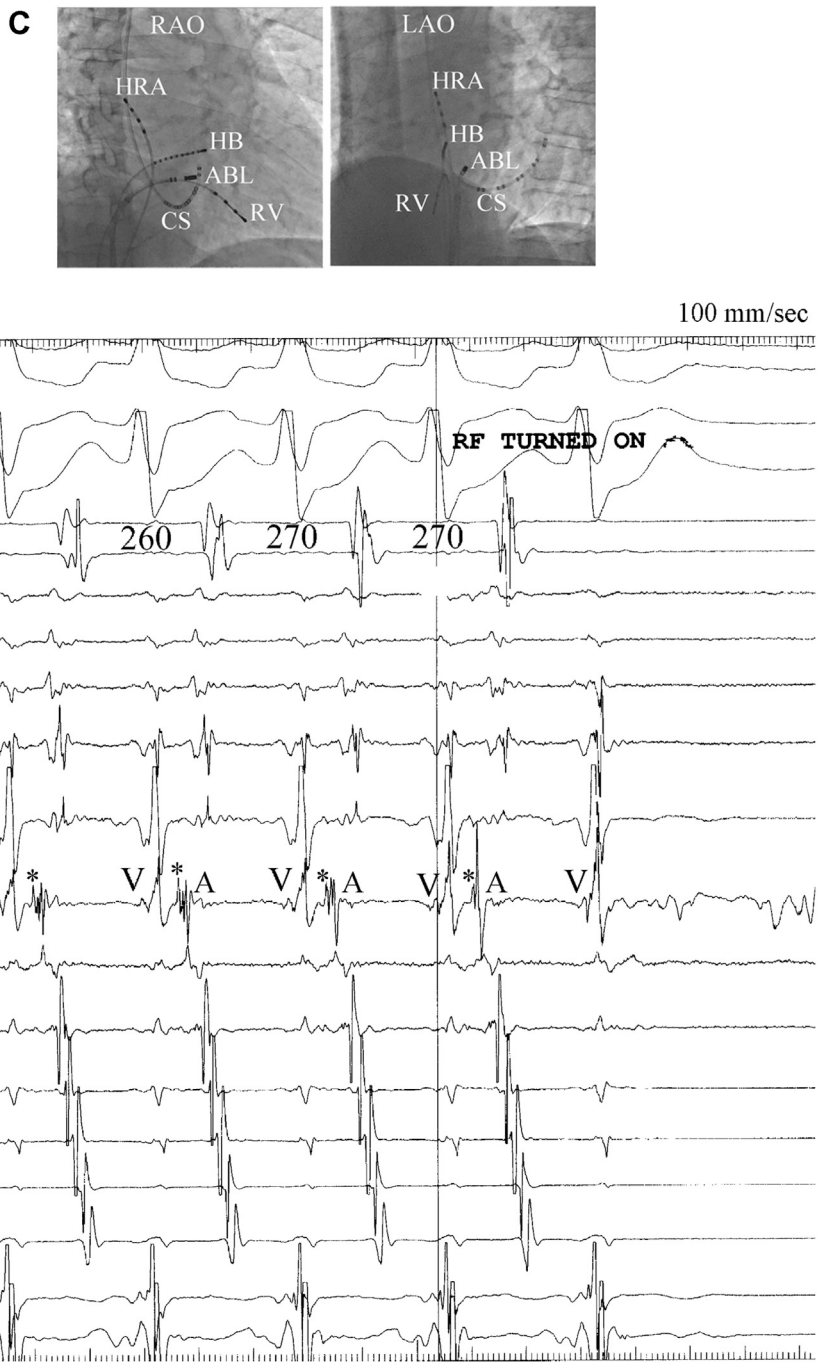

Figure 2 (*continued*).

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.[6] 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[1] without a BBB.

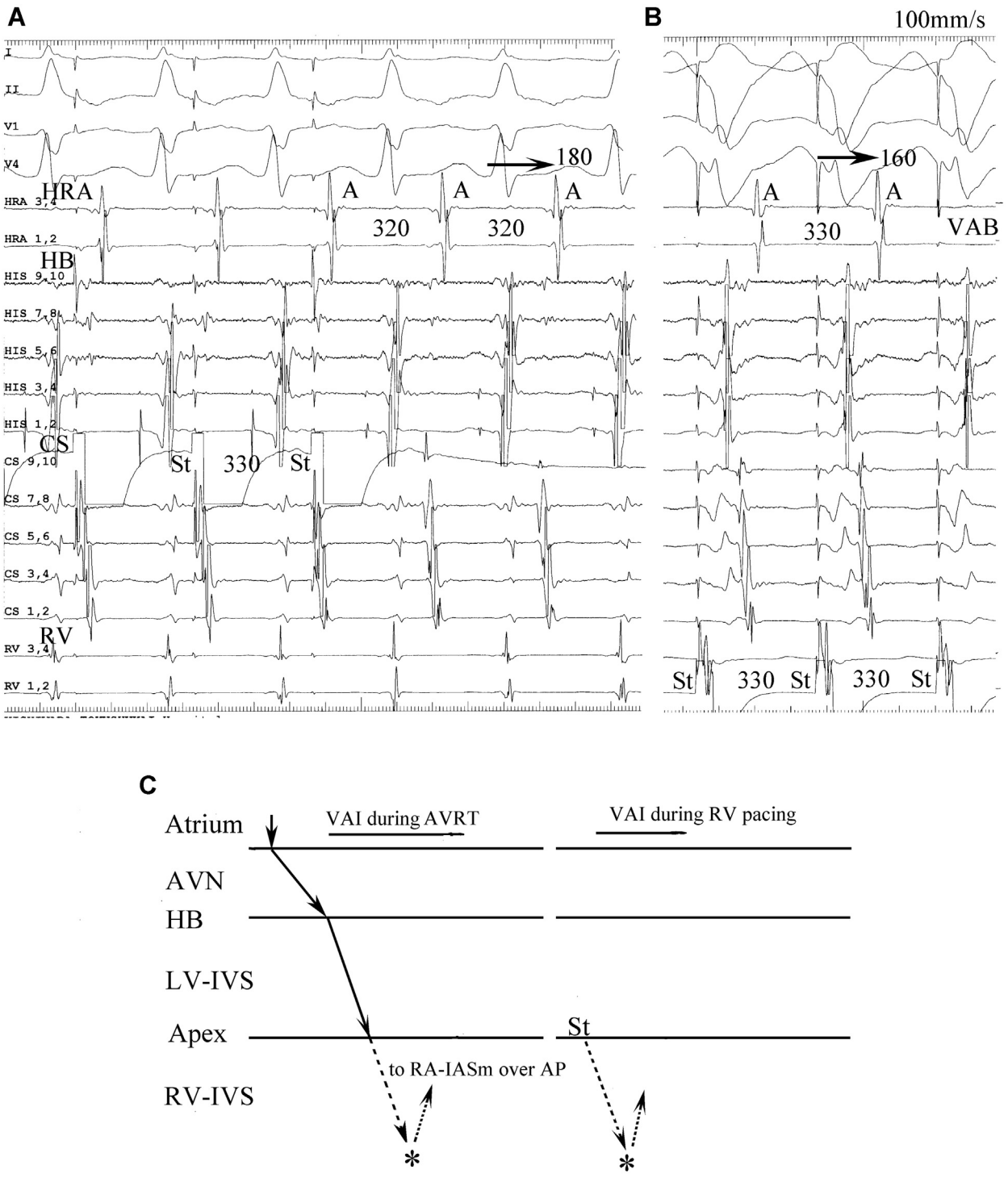

Figure 3 Comparison of ventriculoatrial (VA) intervals. **A:** A nonsustained orthodromic atrioventricular reentrant tachycardia (AVRT) with a cycle length of 320 ms. An AVRT was induced by constant pacing with a cycle length of 330 ms (St-St) from the proximal electrode pair of coronary sinus (CS9,10). 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."[1,7] 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 of the AP earlier by 20 ms. Probably, the phenomenon can substantiate evidence that the prolongation of VA interval during AVRT was mainly based on the conduction time lag of the RV-IVS.

After all, our speculation can be explained as illustrated by ladder diagrams of activation conduction sequence during AVRT (left column) and RV apex pacing (right column) in Figure 3C. VA intervals during AVRT and RV pacing were measured as conduction times from LV-IVS and stimulation 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 compatible with the conduction time from the LV-IVS endocardium to the RV apex (stimulation site) through the His-Purkinje system.[6]

Such a theory may be applicable to not only our case but also a septal RA AP, except for a posteroseptal one with a linkage between the RA and the LV.[3,8] To our knowledge, our theory has never been described in any literature.

Next, successful instantaneous elimination of the AP could be obtained immediately after RF. This might not only be produced by bumping of the ablation catheter against the septum[9] but also be explained by the anatomical feature that a septal AP exists in superficial surface.[4] At postablation study, although the antegrade AV conduction remained intact, the retrograde VA conduction disappeared.

Finally, in order to ablate a septal AP, cryothermal ablation is advocated to be superior to RF because of advantages of cryomapping and avoidance of ablation catheter dislodgement owing to adherence between the catheter tip and the endocardial tissue.[4,10]

Unfortunately, then in 2017 the usage of cryothermal ablation for a septal AP was not approved in Japan.

A limitation is that pacing at different ventricular sites to clarify whether AP ran obliquely along the TV was not performed. 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.

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