A short RP tachycardia with alternating ventriculoatrial interval, but identical atrial activation sequence: What is the mechanism?

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Introduction
The critical change of ventriculoatrial (VA) interval during supraventricular tachycardia (SVT) is due to the existence of multiple retrograde conduction pathways and atrial tachycardia. The electrophysiological study during oscillation of VA interval is often challenging. We present an intracardiac recording at the timing of SVT termination by ablation that may help to elucidate the mechanism of SVT with different VA intervals.

Case report
A 67-year-old woman with sustained palpitations for 5 days was referred to our institution for a catheter ablation. Physical examination and transthoracic echocardiographic study did not show any abnormality. A surface electrocardiogram of sinus rhythm did not show any pre-excitation. The surface electrocardiogram during tachycardia showed narrow complex tachycardia with short RP interval (heart rate 174 beats/min). The tachycardia was terminated spontaneously but soon resumed after several beats of sinus rhythm (incessant form). Electrophysiological study was urgently performed to relieve palpitation. Decapolar catheters were placed in the coronary sinus and tricuspid annulus. Quadrupolar catheters were placed in the right ventricle (RV) and across the tricuspid annulus at the His bundle position. The intracardiac recording revealed that atrial activation sequence during tachycardia and burst pacing from the RV was identical and the earliest activation site was located at the distal coronary sinus (left anterolateral region). Occasionally, a second-degree VA block was observed during burst pacing from the RV, but the critical prolongation of the VA interval was not observed (Figure 1A). The atrioventricular (AV) conduction curve and VA conduction curve were not fully evaluated owing to an incessant form of tachycardia. The first VA interval at initiation of the tachycardia (long RP tachycardia) was obviously longer (about 100 ms) than the VA interval during the subsequent short RP tachycardia, but the atrial activation sequence was identical (Figure 1B). The long RP tachycardia was observed only in the several beats immediately after the initiation of tachycardia and the longer VA interval was not observed by RV burst pacing and programmed stimulus. The short RP tachycardia was advanced by His refractory ventricular premature depolarization from the base of the left ventricle. What is the mechanism of the narrow complex tachycardia?

Discussion
The differential diagnosis of a short RP tachycardia with the earliest atrial activation in the left anterolateral region includes atrial tachycardia originating from the left atrium, left variant atrioventricular nodal reentrant tachycardia (AVNRT), and orthodromic reciprocating tachycardia via left-sided accessory pathway. The finding that overdrive...
pacing from the RV terminated tachycardia within the fusion period could exclude atrial tachycardia. As shown in Figure 2, the finding that His refractory ventricular premature depolarization advanced the tachycardia was diagnosed as orthodromic reciprocating tachycardia via left-sided accessory pathway. On the other hand, the differential diagnosis of a long RP tachycardia with the earliest atrial activation in the left anterolateral region includes left-sided atrial tachycardia, atypical AVNRT, accessory pathway automaticity, and orthodromic reciprocating tachycardia using a slowly conducting accessory pathway like a permanent form of junctional reciprocating tachycardia (PJRT). The findings that VA interval at initiation of long RP tachycardia was always constant could exclude abnormal automaticity and atrial tachycardia as a mechanism of long RP tachycardia. Because electrophysiological study for long RP tachycardia has not been performed owing to the extremely short duration, it was difficult to distinguish any more. But AVNRT can also be excluded because there is no report that the earliest activation site of atypical AVNRT was located at the anterolateral region in left atrium. Therefore, the mechanism of long RP tachycardia is diagnosed as slow conducting accessory pathway.

The 2 tachycardias use a single pathway via the AV node as antegrade limb and 2 available accessory pathways as retrograde limb and had exactly the same atrial activation sequence. That is, these retrograde conduction pathways were anatomically identical but electrophysiologically different. There are 2 possibilities to account for the mechanism of these discrete VA intervals: (1) functional longitudinal dissociation in a single accessory pathway or (2) 2 different accessory pathways in close proximity. Denes and colleagues reported orthodromic reciprocating tachycardia with discrete VA interval, but with identical atrial activation sequence. The authors adopted the possibility of functional

\[ \text{Figure 1} \quad \text{A: Right ventricular pacing (600 ms). Ventriculoatrial (VA) conduction showed second-degree VA block. B: Spontaneous initiation of tachycardia immediately after sinus rhythm (\ast). CS = coronary sinus; CSp = paroxysmal coronary sinus; HBE = His bundle electrogram; HRA = high right atrium; RV = right ventricular. Asterisk (\ast) = sinus beat.} \]

\[ \text{Figure 2} \quad \text{His bundle refractory ventricular premature depolarization (VPD) from the base of left ventricular advanced tachycardia. Details are described in text. Abbreviations as in Figure 1.} \]
longitudinal dissociation as an explanation for these electrophysiological findings. Accessory pathways have been thought to conduct in “all-or-none” fashion. However, the findings that some accessory pathways contain AV node–like tissue² can provide an explanation for the AV node–like function (functional longitudinal dissociation) in the accessory pathway. A discontinuous conduction curve suggestive of functional longitudinal dissociation had been demonstrated in the patients with atriofascicular fibers, nodoventricular fibers, and accessory pathway of PJRT.²,³ In an autopsy case of PJRT, the length of the accessory pathway was shown to be long and tortuous.² An accessory pathway with a long course may explain slow conduction and decremental conduction property. In this case, the atrial insertion of the accessory pathway is located at the base of the left atrial appendage away from the mitral annulus and the length of the accessory pathway seems to be long. In addition, since the retrograde VA conduction with decremental conduction property has suggested the involvement of AV node–like tissue, the discrete retrograde conduction of this case can be explained by the functional longitudinal dissociation.

Differentiating the second possibility is impossible unless histological studies were performed in detail. Therefore, the favored explanation for these retrograde conduction is accessory pathway with functional longitudinal dissociation like AV node.

The retrograde earliest activation site mapped during tachycardia via fast retrograde conduction was observed at the base of the left atrial appendage opposite the left pulmonary vein (Figure 3A). It was confirmed by contrast injection through the irrigation lumen of the ThermoCool SmartTouch SF (Biosense Webster, Inc, Diamond Bar, CA) (Figure 3B). Ablation at this site terminated the tachycardia in 2 seconds (Figure 3C). After the ablation, no VA conduction was observed and no tachycardia had been induced.

Focusing on the local electrogram recorded by the distal tip of the ablation catheter at termination of the tachycardia (Figure 3D), it was observed that the atrial sequence of fast retrograde conduction (beat ①) and slow retrograde conduction (beat ③) was not exactly identical (the preceding time of local electrogram of ablation catheter for distal coronary arterioles was 24 ms). The tachycardia was terminated by VA block after beats ② and ④. ABL = ablation catheter; AP = anterior posterior position; RAO = right anterior oblique position. Other abbreviations as in Figure 1.
sinus, 26 ms and 51 ms, respectively). The first ablation eliminated the retrograde fast conducting pathway (beat②), but the retrograde slow conducting pathway that remained in beat③ was eliminated in beat④. These findings indicated that the atrial insertion in these 2 retrograde conductions was very closely located but distinct.

The patient has not complained of palpitations and has been free of arrhythmias for 30 months.

Conclusion
We present a rare case of orthodromic reciprocating tachycardia with alternating VA intervals, but with identical retrograde atrial activation sequence. The tachycardia with discrete VA interval can be explained by functional longitudinal dissociation (fast and slow retrograde conduction), but we could electrophysiologically demonstrate that each atrial insertion of the accessory pathway with functional longitudinal dissociation was very closely located but distinct.

References
1. Denes P, Kehoe R, Rosen KM. Multiple reentrant tachycardias due to retrograde conduction of dual atroventricular bundles with atrioventricular nodal-like properties. Am J Cardiol 1979;44:162–170.
2. Becker AE, Anderson RH, Durrer D, Wellens HJ. The anatomical substrates of Wolff-Parkinson-White syndrome. A clinicopathologic correlation in seven patients. Circulation 1978;57:870–879.
3. Okishige K, Friedman PL. New observations on decremental atriofascicular and nodofascicular fibers: implications for catheter ablation. Pacing Clin Electrophysiol 1995;18:986–998.
4. Arribas F, Lopez-Gil M, Nunez A, Cosio FG. Wolff-Parkinson-White syndrome presenting as the permanent form of junctional reciprocating tachycardia. J Cardiovasc Electrophysiol 1995;6:132–136.
5. Critelli G, Gallagher JJ, Monda V, Coltorti F, Scherillo M, Rossi L. Anatomic and electrophysiologic substrate of the permanent form of junctional reciprocating tachycardia. J Am Coll Cardiol 1984;4:601–610.