

Case Report

**Slow-fast Form of Atrioventricular Nodal Reentrant Tachycardia with Unusual Retrograde Activation in the Right Atrium
—Possible Conduction Disturbance across the Tendon of Todaro Related to the Genesis of Positive Component of Biphasic Retrograde P Wave—**

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We report a rare case of slow-fast form of atrioventricular nodal reentrant tachycardia with delayed activation in the low septal right atrium (His bundle area). During supraventricular tachycardia (SVT), electrocardiogram showed “pseudo-positive P waves” in II, III, and aV_F leads. SVT was induced by atrial extrastimulus with marked AH prolongation (i.e., jump phenomenon). Ventricular pacing showed a decremental retrograde conduction without jump phenomenon. Double atrial potentials were observed in the His bundle area during SVT and during ventricular pacing. The first electrogram of these split potentials, which was the earliest activation during SVT and during ventricular pacing, showed a dull and small deflection, whereas the second electrogram was sharp and clear. The interval of these discrete potentials was 70 msec during SVT. After a standard slow pathway ablation, SVT could never be induced by any programmed stimuli. It was concluded that in this case, the conduction disturbance across the tendon of Todaro was likely to cause the delayed atrial activation in the His bundle area, which created the pseudo-positive (biphasic) retrograde P wave. (J Arrhythmia 2006; 22: 52–57)

Key words: Atrioventricular nodal reentry, Conduction block, Double potentials, Ablation, His bundle

Introduction

Slow-fast form of atrioventricular nodal reentrant tachycardia (AVNRT) is a common reentrant tachycardia within the so-called Koch’s triangle with the earliest retrograde activation at the low septal right

atrium (His bundle area). However, AVNRT with the earliest activation in other sites has been reported by many investigators.^{1–9)} We report a rare case of AVNRT with delayed atrial activation in the His bundle area.

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Case Report

A 57 year-old female complained of frequent palpitations. She was referred to hospital for treatment of tachyarrhythmia. Electrocardiogram on admission was normal without delta waves. P wave was not split during sinus rhythm. The width of P wave was 0.1 sec. Echocardiogram showed a normal cardiac function without any valvular disease. The clinical tachycardia was supraventricular tachycardia (SVT) at the rate of 138 bpm. "Pseudo-positive retrograde P waves"¹⁰ were recorded at the end of the QRS complex in II, III, and aV_F leads (Figure 1). P wave morphology during tachycardia was clearly documented after a ventricular premature beat that occurred by chance. It exhibited a biphasic form composed of the former negative deflection and the latter positive deflection. All antiarrhythmic medications had been discontinued for a week. After informed consent was obtained, an electrophysiological study was performed. 5 mm spacing quadripolar electrode catheters were positioned in the high right atrium (HRA), the His bundle area and the right ventricle (RV). A 2-5-2 mm spacing decapolar electrode was inserted into the coronary sinus (CS), with the proximal bipole (CS 9-10) located at the ostium. During sinus rhythm and during HRA pacing, the atrial electrogram at the CS 7-8 site and the CS 5-6 site preceded the atrial electrogram at the CS 9-10 site. This activation sequence suggested that breakthrough points from the RA to

the LA might exist at sites other than the CS ostium. SVT was easily induced by atrial extrastimulus, atrial burst pacing or ventricular extrastimulus. Atrial extrastimulus at the HRA at a basic cycle length of 600 msec demonstrated a discontinuous AV nodal conduction curve, indicating a dual AV nodal antegrade pathway (Figure 2A). During SVT, double atrial electrograms (A₁ and A₂) were recorded in the His bundle area. The earliest atrial activation (A₁) was dull and small. At this site the atrial electrogram preceded the ventricular electrogram. Under the condition of good catheter contact, the A₂ potentials activating later than the HRA were clearly recorded during SVT (Figure 2B). A single ventricular pacing delivered at the coupling interval when the His bundle was refractory never preexcited the atrium during SVT. On the other hand, RV scanning delivered at a shorter coupling interval during SVT revealed the double potentials (A₁ and A₂) in the His bundle area (Figure 3A). The A₁A₂ interval was 70 ms during SVT. Ventricular extrastimulus was performed at a basic cycle length of 600 msec at the right ventricular apex. Retrograde conduction time increased progressively and continuously at a shorter coupled extrastimulus (Figure 3B). Split atrial electrograms were also recorded in the His bundle area during RV pacing. The activation mapping by RV pacing revealed that the earliest atrial activation site was the His bundle area. The first component of the atrial electrogram in the His bundle (A₁) was constantly recorded as a small and

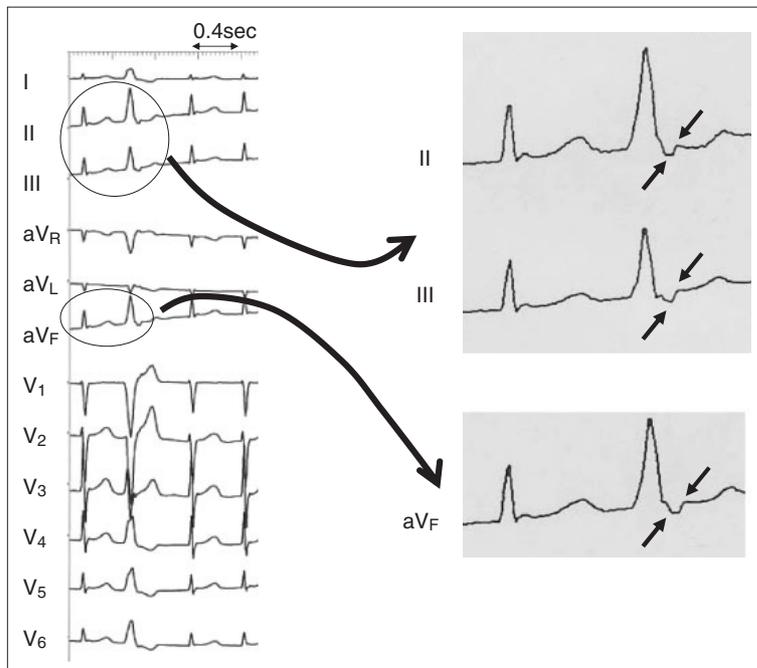


Figure 1 Electrocardiogram during palpitation attack showed a narrow QRS tachycardia with "pseudo-positive P waves" at the terminal of QRS complexes in II, III and aV_F leads.

The premature ventricular beat revealed biphasic (negative-positive) retrograde P waves during the tachycardia (arrow).

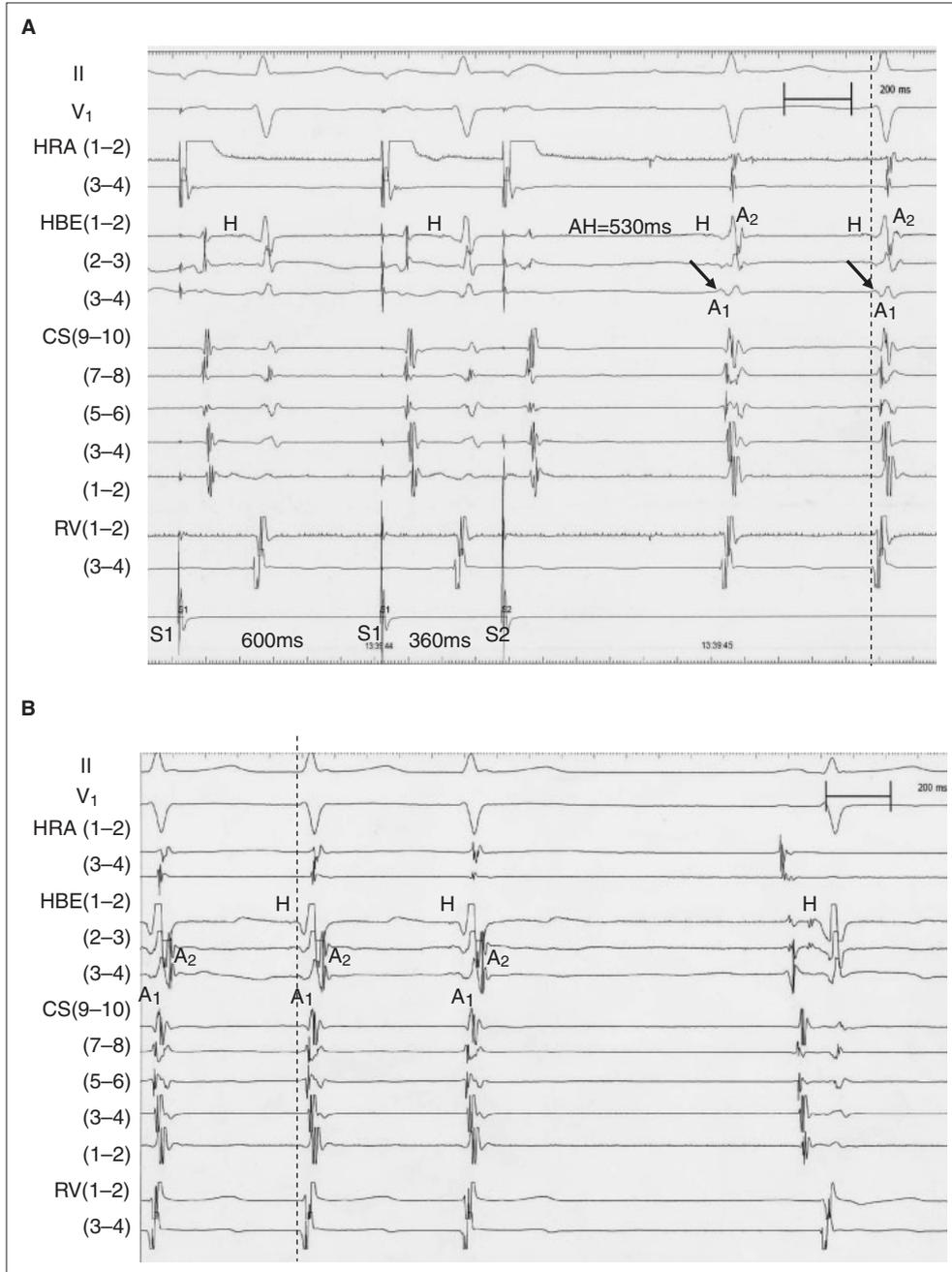


Figure 2

A. Supraventricular tachycardia (SVT) was induced by atrial extrastimulus at a basic cycle length of 600 ms and a coupling interval of 360 ms. SVT was initiated by marked AH jump with the earliest atrial echo in the His bundle area (A₁), which was followed by the ventricular electrogram (arrow). The second atrial electrogram in the His bundle area (A₂) was observed later than the ventricular electrogram.

B. Under good catheter-contact, the sharp atrial electrograms (A₂) activating later than the HRA were clearly recorded in the His bundle area during SVT. The atrial electrogram at the CS 7-8 site and at the HRA preceded the A₂ potential at the His bundle area by 50 msec and 30 msec, respectively. The dull deflections (A₁) were also recorded in the His bundle area.

HRA, high right atrium; HBE, His bundle electrogram; CS, coronary sinus; RV, right ventricle; H, His bundle deflection.

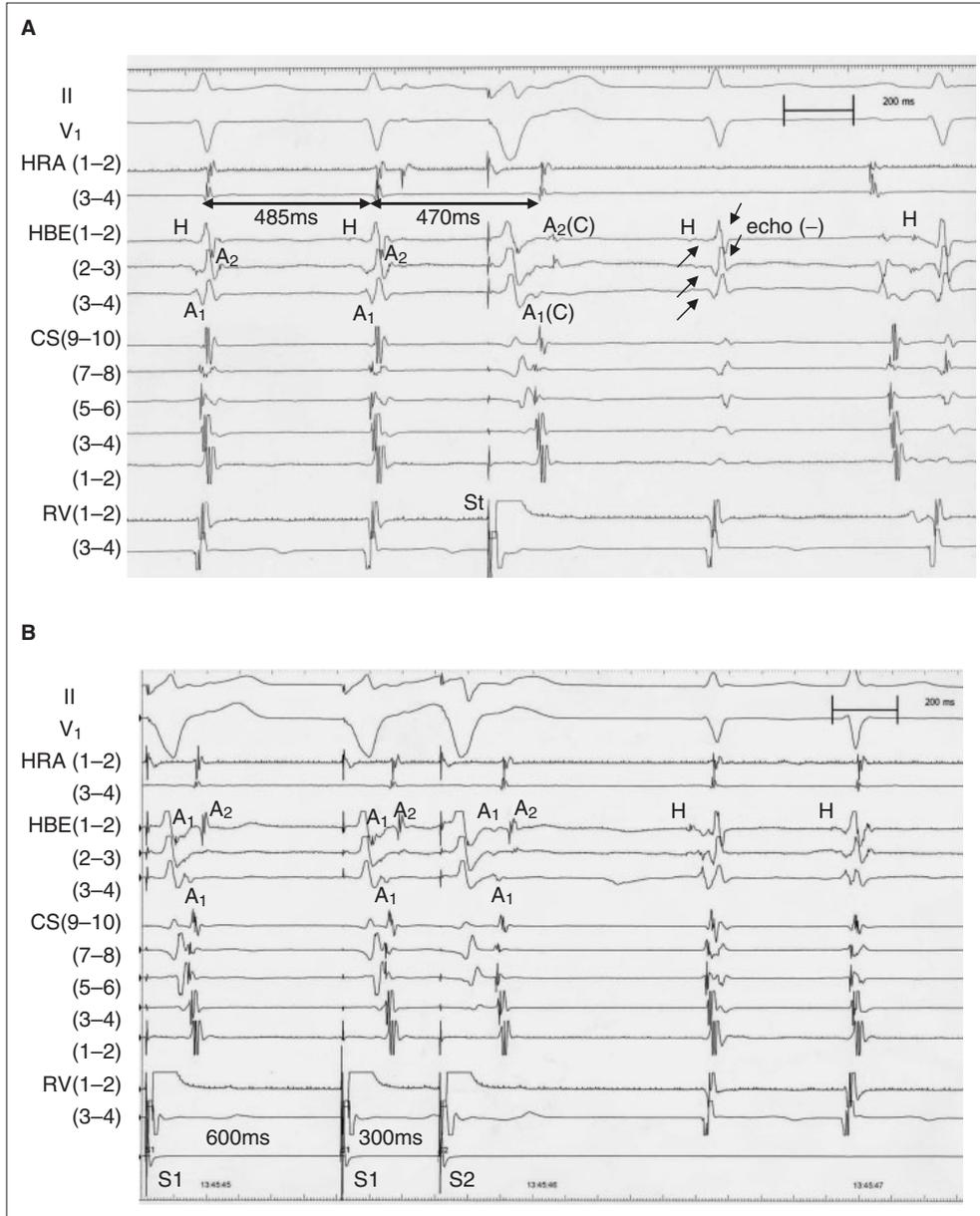


Figure 3

A. RV pacing during supraventricular tachycardia (SVT) obviously revealed double atrial electrograms in the His bundle area. The first atrial electrogram (A₁) in the HBE (1-2) was dull and small. The second electrogram (A₂) was sharp. The A₁A₂ interval was 70 msec during SVT. A single ventricular pacing captured the atrium via retrograde conduction, which terminated SVT. These captured atrial electrograms were also split (A₁(C) and A₂(C)). The A₁ and A₂ potentials vanished after the termination of SVT (arrows).

B. Supraventricular tachycardia (SVT) was also induced by ventricular extrastimulus at a basic cycle length of 600 ms with the coupling interval of 300 ms. The atrial electrograms in the His bundle area (A₁ and A₂) were split during RV pacing and during SVT. The A₁ potential in the HBE (1-2) site was slightly followed by the A₁ potential in the HBE (3-4) site. The VA₁ interval increased progressively and continuously at a shorter coupled extrastimulus. During ventricular pacing and SVT, the A₁ potentials were constantly recorded in the His bundle area earlier than in any other atrial electrogram.

dull deflection. The sharp second component (A₂) followed the atrial electrogram at the CS 7-8 site and the electrogram at the HRA. Its atrial activation

sequence was concordant with the activation during SVT. During the ventricular burst pacing at a cycle length of 300 msec, Wenckebach type of VA block

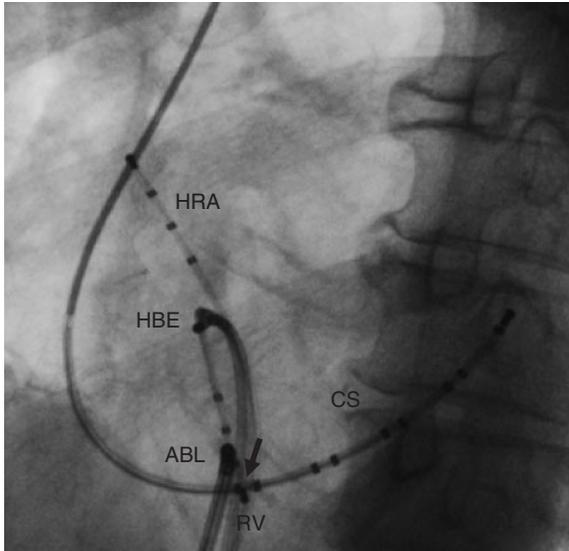


Figure 4 Catheter position seen fluoroscopically from the left-anterior oblique view (40°) during the successful slow pathway ablation. Note that CS 9–10 site was located at the CS ostium (arrow). ABL, ablation catheter.

appeared. It was concluded that SVT in this case was slow-fast form of AVNRT with delayed activation in the His bundle area. Slow pathway ablation was performed in the upper rim of the CS ostium, guided by the slow pathway potential (**Figure 4**). During the radiofrequency application, accelerated junctional rhythm was observed. During the junctional rhythm the atrial activation sequence was completely concordant with the sequence during tachycardia. After a single application, antegrade slow pathway conduction was abolished and SVT was no longer inducible despite isoproterenol infusion. The retrograde conduction was not affected after the application. The patient has been free from palpitation attacks for a year since the treatment.

Discussion

Many investigators have documented histologically and electrophysiologically that the inputs to the AV node are various and that additional connections between the AV node and the interatrial septum not involving the traditional cells are likely to exist.^{11–15} Slow-fast form of AVNRT is a common arrhythmia with the earliest activation in the His bundle area. On the other hand, there have been several reports that eccentric atrial activation was documented during AVNRT.^{1–9} Anselme et al. have reported that retrograde atrial activation within the CS earlier than at the ostium (i.e., CS breakthrough) was observed in 47% of AVNRT.⁵ These reports suggest

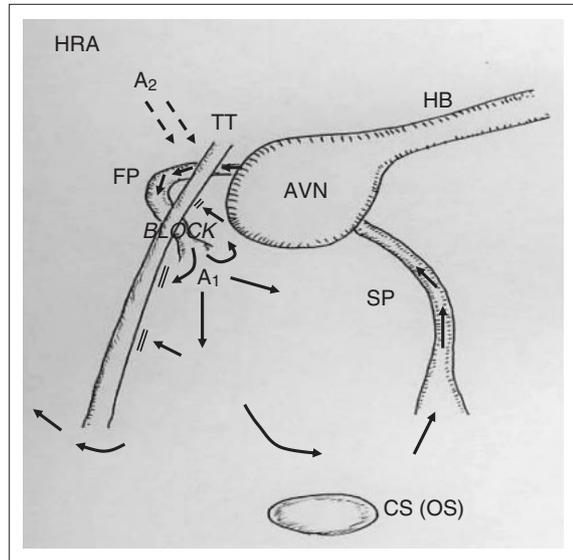


Figure 5 Speculated scheme of AVNRT.

AVN, compact atrioventricular node; HB, His bundle; TT, tendon of Todaro; CS(OS), ostium of the coronary sinus; FP, fast pathway; SP, slow pathway. S₁, first activation impulse from the fast pathway; S₂, delayed activation impulse from the upper septum.

a left-sided atrionodal connection, which is still controversial. However, there has been no previous reports that the intraatrial conduction block near the His bundle caused unusual right atrial activation sequence during AVNRT.

Double atrial potentials were recorded in the His bundle area during SVT (**Figures 2 and 3**). The first deflection (A₁), which activated earliest during SVT, was dull and small. The second deflection (A₂) was sharp and clear. Ventricular scanning efficiently differentiated these split potentials (**Figure 3A**). **Figure 3B** showed double potentials in the HBE (1–2). The dull A₁ potential in the HBE (1–2), which was likely a far-field potential of a deep subendocardial layer, preceded the A₁ potential in the HBE (3–4). On the other hand, the atrial potential in the site of HBE (3–4) showed a single deflection. Hence, it was speculated that the site of HBE (3–4) was a breakthrough point from the AV node to the right-sided atrial endocardium during SVT and RV pacing. Besides, the focal intraatrial conduction block near the His bundle made split potentials in the HBE (1–2) site. As the tendon of Todaro was likely to exist anatomically between the HBE (1–2) site and HBE (3–4) site, this focal conduction block might indicate conduction disturbance across the tendon of Todaro. As double potentials were never recorded during sinus rhythm, this conduction disturbance was supposed to be a unidirectional

conduction block from the lower to the higher septum. Any anatomical abnormalities within the Koch's triangle were not identified by catheter mapping although coronary sinus angiography was not further performed. In addition, it could not be fully ruled out that the A₁ potential in the HBE area might indicate a far-field potential of the left-sided septum, which implied the presence of the left-sided fast pathway. However, the fact that the atrial potential in the HBE (3–4) site was single during SVT and during sinus rhythm strongly suggested that the A₁ potential should indicate the right-sided potential.

During tachycardia, ECG (Figure 1) showed "pseudo-positive retrograde P waves" in II, III, and aV_F leads, which had rarely been observed. Biphasic P wave during AVNRT has been reported by Suzuki et al.¹⁰ They demonstrated that the initial negative component of retrograde P wave was masked in the QRS complexes before the positive component and that the retrograde P wave was biphasic (negative-positive). They also reported that the bidirectional blockline existed within the subeustachian isthmus in some of these cases. In our case, it was considered that the conduction disturbance across the tendon of Todaro rather than the isthmus block caused specific activation impulse from the HRA to the lower septum, which created the positive component of biphasic retrograde P waves in II, III, and aV_F leads.

Conclusion

We have reported a rare case of atrioventricular nodal reentry with biphasic retrograde P waves due to possible conduction disturbance across the tendon of Todaro. Our case report indicates that the atrial activation sequence within Koch's triangle should be interpreted prudently in AVNRT and that the conduction block across the tendon of Todaro should be taken into consideration if the HRA potential precedes the potential in the lower right atrial septum (i.e., His bundle area) during AVNRT.

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