Case Reports

Slow-fast Form of Atrioventricular Nodal Reentrant Tachycardia with Eccentric Retrograde Left-sided Activation

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SUMMARY

A case of atypical AV nodal reentrant tachycardia (AVNRT) with eccentric retrograde left-sided activation, masquerading as tachycardia using a left-sided accessory pathway, is reported. Initially, it appeared that the tachycardia was a typical slow-fast form of AVNRT. The earliest retrograde activation, however, was registered at a site approximately 3 cm from the coronary sinus orifice (left atrial free wall), indicating atypical AVNRT. Atrial tachycardia and orthodromic AV reciprocating tachycardia using an accessory AV pathway were excluded. Slow pathway ablation at the posteroseptal right atrium eliminated the tachycardia. It was suggested that the anterograde limb of the tachycardia circuit was a slow AV nodal pathway with typical posteroseptal location, whereas the retrograde limb was a long atrionodal pathway connecting the compact AV node and the left atrial free wall near the mid-coronary sinus. (Jpn Heart J 1999; 40: 655–664)

Key words: AV nodal reentrant tachycardia, Eccentric retrograde atrial activation, Slow AV nodal pathway, Atrionodal pathway, Perinodal transitional cell, Slow pathway ablation

A typical atrioventricular (AV) nodal reentrant tachycardia (AVNRT) with eccentric retrograde left-sided activation, masquerading as tachycardia using a left-sided accessory pathway, has recently been reported. In that report Hwang et al. suggested that the left-sided perinodal transitional cells, although responsible for eccentric retrograde left-sided activation during atypical AVNRT, were not critically involved in the AVNRT circuit (i.e., bystander role), since the attempted ablation near the lateral coronary sinus (CS) leads that registered the earliest retrograde atrial activation failed to eliminate the AVNRT.
whereas the slow pathway ablation targeting the posteroseptal right atrium successfully prevented the reinduction of AVNRT. The purpose of this case report is to describe a patient with atypical AVNRT in which the left-sided perinodal transitional cells (or atrionodal pathway) were considered to form an essential component of AVNRT circuit.

**Case Report**

A 45-year-old woman with paroxysmal supraventricular tachycardia was referred to our hospital for radiofrequency catheter ablation. The ECG in sinus rhythm was normal, with a mean heart rate of 85 beats/min, a PR interval of 0.18 seconds, and a QRS duration of 0.08 seconds. The clinical tachycardia was a regular, narrow QRS complex tachycardia at a rate of 140 beats/min, with a visible pseudo r' deflection in lead V1, suggesting AV nodal reentry as a tachycardia mechanism.

Electrophysiologic study was performed after informed written consent was obtained and 72 hours after discontinuation of all antiarrhythmic drugs. Four 6F quadripolar electrode catheters were introduced percutaneously via the femoral veins and placed in the high right atrium (HRA), the posteroseptal right atrium (PS), across the tricuspid valve for recording His bundle electrogram (HBE), and the right ventricular apex (RVA). A 5F decapolar catheter was inserted into the CS through the right internal jugular vein; the most distal electrode pair (CS 1–2) was positioned approximately 5 cm from the CS orifice. Another 7-French quadripolar, deflectable tip catheter with a 4-mm distal electrode (Celebrate [Dr. Osypka GmbH, Grenzach, Germany]) was used to deliver radiofrequency current from the tip electrode to a large skin electrode positioned on the posterior chest. Stimuli of 2 msec in duration and approximately twice diastolic threshold in intensity were delivered by a digital programmed stimulator (Fukuda Denshi Co., Tokyo, Japan). Bipolar intracardiac electrograms filtered at 50 to 500 Hz along with the surface ECG leads (II, V1, V2, V3) were displayed on an oscilloscope and recorded on a thermal recorder (Fukuda Denshi Co.) at a paper speed of 100 mm/sec. The power source used for radiofrequency catheter ablation was a commercially available system (HAT 300S, Dr. Osypka GmbH) that delivered continuous unmodulated radiofrequency energy at a frequency of 500 kHz in the temperature control mode.

During baseline sinus rhythm, the mean sinus cycle length was 660 msec, the atrial-His (AH) interval was 95 msec, and the His-ventricular (HV) interval was 50 msec.

Programmed atrial extrastimulus testing from the HRA (basic cycle length, 600 msec) demonstrated a discontinuous AV nodal conduction curve, indicating
Figure 1. Ventricular extrastimulus testing from the right ventricular apex (basic cycle length = 600 msec, coupling (S1S2) interval = 240 msec). The atrial electrogram from the CS 5–6 site (arrow) preceded the low septal right atrial electrogram from the His bundle (HBE) recording site by 15 msec. The H2A2 interval measured on the CS 5–6 electrogram is 100 msec, identical to the value during the tachycardia (see Figure 3). II, V1, V2, V3 = ECG lead II, V1, V2 and V3; HRA = high right atrium; HBEp, HBEd = proximal and distal HBE; PS 3–4 (1–2) = proximal (distal) posteroseptal right atrial electrogram; CS 9–10 (7–8, 5–6) = electrogram from the CS 9–10 (CS 7–8, CS 5–6) bipolar pair of electrodes (CS 9–10 pair, most proximal). Paper speed is 100 mm/sec in this and subsequent figures.

Figure 2. Fluoroscopic view showing positions of electrode catheters (60° left anterior oblique projection). Electrode catheters are placed at the high right atrium (HRA), the His bundle recording site (HBE), the posteroseptal right atrium (PS), the right ventricular apex (RVA) and within the coronary sinus (CS). CS electrode catheter is decapolar. The site of the CS 5–6 bipolar pair is approximately 3 cm from the CS orifice.
Figure 3. Induction of AV nodal reentrant tachycardia during atrial extrastimulus testing from the high right atrium (basic cycle length = 600 msec; coupling [S1S2] interval = 280 msec). The earliest retrograde atrial activation was registered at the CS 5–6 recording site (arrow). During the basic (S1) drive beats, the atrial activation along the CS catheter proceeds in a proximal-to-distal direction, indicating correct electric connections of individual CS electrodes to the recording system. Abbreviations are as in Figure 1.

Figure 4. Catheter movement-induced premature ventricular contraction during the tachycardia. Four CS electrograms (CS 9–10, CS 7–8, CS 5–6 and CS 3–4) along with HRA and HBE recordings are shown. The earliest atrial activation almost simultaneously occurs at the CS 5–6 and CS 7–8 recording site; this presumably reflects a slight movement of the electrode catheter in the CS. Abbreviations are as in Figure 1.
After ablation

Figure 5. Rapid ventricular pacing from the right ventricular apex (paced cycle length, 400 msec) after slow pathway ablation. The earliest atrial activation was registered at the CS 5–6 recording site (upward arrow), followed 15 msec later by the low septal right atrium on the His bundle electrogram (downward arrow); this eccentric retrograde atrial activation is identical to that seen before ablation. SA = stimulus–atrium interval. Abbreviations are as in Figure 1.

Dual AV nodal pathway physiology. Programmed ventricular extrastimulation was performed from the RVA at a basic cycle length of 600 msec (Figure 1). The earliest retrograde atrial activation was registered at the CS 5–6 recording site (approximately 3 cm from the CS orifice, Figure 2), followed by the CS 7–8 and CS 9–10 recording sites. The H2A2 interval measured on the CS 5–6 electrogram was 100 msec. The atrial electrogram from the CS 5–6 site preceded the low septal right atrial electrogram on the HBE recording by 15 msec. The demonstration of a consistent V-H-A sequence at any coupling intervals, as well as a progressively increased ventriculoatrial (VA) conduction interval in response to progressively premature ventricular stimulation, suggests that the retrograde conduction was by way of the His-AV node axis, thus excluding a rapidly conducting accessory pathway.

Supraventricular tachycardia was reproducibly induced with atrial extrastimulation. Induction of tachycardia was associated with a marked prolongation in the AH interval (175 msec → 550 msec, Figure 3). During tachycardia,
atrial activation occurred simultaneously with ventricular activation, with the shortest HA interval being registered at the CS 5–6 recording site (eccentric retrograde left-sided activation). The HA interval on the CS 5–6 recording was 100 msec during supraventricular tachycardia and ventricular extrastimulus testing (Figures 1, 3); this suggests that the same conducting pathway (His-AV node axis) was used for retrograde conduction both during tachycardia and ventricular extrastimulation. Further, the VA interval at the CS 5–6 recording site (earliest site) during tachycardia was 50 msec, excluding participation of an accessory AV pathway in the tachycardia circuit. Catheter movement-induced premature ventricular contraction occurred spontaneously during the tachycardia (Figure 4); it is noted that the atrial electrogram from the CS 5–6 recording site preceded the low septal right atrial electrogram on the HBE recording by 15 msec (not labelled). Ventricular stimulation from the RVA did not reset the tachycardia when the His bundle was refractory.

Slow pathway ablation was performed using a standard right atrial septum approach, with the ablation catheter positioned at the postero septal right atrium, guided by the slow pathway potential. A single 90-second application of radiofrequency energy was delivered at this site. During radiofrequency application an accelerated junctional rhythm was observed. After a single application,
anterograde slow pathway conduction was abolished and the tachycardia was no longer inducible. The retrograde atrial activation over the fast pathway still remained eccentric, with the earliest atrial activation being registered at the CS 5–6 recording site (Figure 5). At the end of the ablation procedure an additional application of radiofrequency energy ("insurance burn") was attempted just outside the CS orifice. During application of radiofrequency energy, the ablation catheter inadvertently moved and fell into the CS orifice. Although the energy application was immediately stopped, VA conduction through the retrograde fast pathway was found to be interrupted (Figure 6). However, the AH and HV intervals were, respectively, 100 msec and 50 msec at a mean sinus cycle length of 700 msec, indicating intact anterograde fast pathway conduction.

**Discussion**

**Differential diagnosis:** The possible mechanisms of the supraventricular tachycardia recorded in this patient included: (1) AVNRT; (2) atrial tachycardia arising in the left atrial free wall (CS 5–6 recording site) where the earliest atrial activation was registered; (3) AV reciprocating tachycardia using a slowly conducting, concealed accessory pathway; and (4) AV reciprocating tachycardia using a slowly conducting accessory pathway.

AV reentry using a slowly conducting accessory pathway was readily excluded because atrial activation during tachycardia occurred simultaneously with ventricular activation.

AV reentry using a rapidly conducting, left-sided accessory pathway was also ruled out since the demonstration of progressively increased VA interval in response to progressively premature stimuli during ventricular extrastimulus testing (Figure 1) excluded the presence of a rapidly conducting accessory pathway in this patient. In addition, the VA interval at the CS 5–6 recording site (earliest site) during tachycardia was 50 msec. Benditt et al. demonstrated that the intervals between onset of ventricular activation and earliest recorded atrial activity (V-Amin) during tachycardia were consistently > 60 msec in patients with orthodromic tachycardia using accessory AV pathways; the V-Amin of 50 msec in this patient provides strong evidence that an accessory AV pathway was not involved in the tachycardia circuit.

Atrial tachycardia arising in the left atrial free wall was ruled out by the observation that ventricular pacing in sinus rhythm resulted in the same eccentric pattern of atrial activation as that recorded during the tachycardia (Figures 1, 3, 4). The latter finding indicates that the VA conducting pathway formed part of the tachycardia circuit. The possibility of atrial tachycardia in the left atrial free wall was further excluded by the fact that the slow pathway ablation at the
postero septal right atrium, which was remote from the CS 5–6 recording site where the earliest atrial activation was registered, successfully prevented the reinduction of supraventricular tachycardia. After excluding several tachycardia mechanisms as described above, the diagnosis of atypical AVNRT with eccentric retrograde left-sided activation\(^1\) was made.

**Classification of AV nodal reentry:** The AVNRT was classified according to intracardiac recordings into the slow-fast form, fast-slow form and slow-slow form, based on the measurements of the AH and HA intervals.\(^1,2\) The slow-fast form was diagnosed if the retrograde conduction during AVNRT was associated with a short VA interval (< 60 msec). The fast-slow form was diagnosed if the HA interval was longer than the AH interval and the retrograde VA interval during AVNRT was > 60 msec. The slow-slow form was diagnosed if the AH interval was greater than the HA interval and the retrograde VA interval during AVNRT was > 60 msec. Slow-slow form of AVNRT was also designated as slow-intermediate form of AVNRT,\(^8\) or posterior AV junctional reentrant tachycardia.\(^5\) Hwang et al.\(^1\) demonstrated that all atypical AVNRTs with eccentric retrograde left-sided activation were classified into either the slow-slow or fast-slow forms, because the retrograde VA intervals during atypical AVNRT were all > 60 msec. The AVNRT recorded in this patient demonstrated eccentric retrograde left-sided activation and a short VA interval (\(V – A_{\text{min}} = 50\) msec). Therefore, unlike the report by Hwang et al.,\(^1\) this tachycardia can be categorized as the slow-fast form of AVNRT.

**Functional and anatomical characteristics of the tachycardia circuit**

**Anterograde limb:** Typical slow pathway, located in the postero septal right atrium, seems to constitute the anterograde limb of the AVNRT circuit in this patient because (1) programmed atrial extrastimulus testing demonstrated a discontinuous AV nodal conduction curve, consistent with dual AV nodal pathway physiology; (2) AVNRT was associated with a long AH and a short HA interval; and (3) the so-called slow pathway ablation at the postero septal right atrium abolished the slow pathway conduction and prevented the reinduction of AVNRT.

**Retrograde limb:** According to the findings during tachycardia and ventricular extrastimulus testing, a fast AV nodal pathway was identified as a sole, retrogradely conducting pathway. This retrograde fast pathway was considered to form the retrograde limb of the tachycardia circuit, since there was no evidence of any other functioning VA pathways.

Recently, a new mechanism of supraventricular tachycardia\(^6\) was proposed in which the anterograde limb was the AV node whereas the retrograde limb was a concealed atrionodod pathway,\(^9\) the atrial insertion of which was at the posterolateral tricuspid annulus; it was suggested that this pathway coursed along the
tricuspid annulus and was electrically insulated from the adjacent atrial myocardium. A similar atridonial pathway (or perinodal transitional cells) appears to form the retrograde limb of the tachycardia circuit in the present case. It is conceivable that the electrically insulated left-sided atrionodal pathway coursed in the posterior atrial septum and along the proximal CS, then entered the left atrial free wall near the mid-CS, approximately 3 cm from the orifice. Interruption of retrograde fast pathway conduction by an additional “insurance burn” delivered inside the CS orifice suggests that this long atrionodal pathway in fact coursed along the proximal CS. The atrial insertion of the atrionodal pathway may not necessarily be at the mitral annulus, since the coronary sinus is commonly remote (> 10 mm) from the mitral annulus. The retrograde atrionodal pathway in this patient may be composed of rapidly-conducting, rather than slowly-conducting, atrionodal fibers, since the HA interval during tachycardia was relatively short (100 msec on the CS 5–6 electrogram) despite the presumable long distance from the compact AV node to the CS 5–6 recording site.

Engelstein et al. recently demonstrated that in 6 patients with slow-fast form of AVNRT the radiofrequency ablation at the postero septal right atrium near the CS orifice abolished both the anterograde and retrograde fast pathway conduction and the AVNRT; this provides evidence that a single, posteriorly located fast pathway with bidirectional conduction comprised the retrograde limb of the AVNRT circuit. Our case differs from their patients in that the inadvertent radiofrequency ablation within the CS orifice only interrupted retrograde fast pathway conduction, while leaving the anterograde fast pathway conduction intact; this fortunate result suggests that an anteriorly located fast pathway was capable only of anterograde conduction whereas a distinct, posteriorly located fast pathway was operative in a retrograde direction.

**Limitations:** A limitation of this report is that the ablation within the CS orifice or at the CS 5–6 recording site, if performed first, could have successfully interrupted the retrograde VA conduction and abolished the AVNRT. Secondly, the lower turn-around site (the lower AV nodal region or the upper His bundle region) of the tachycardia circuit was not convincingly determined. It is therefore unknown whether the tachycardia mechanism was in fact due to atrionodal reentry or AV node-His-atrial reentry.

**Conclusions:** Atypical slow-fast form of AVNRT with eccentric retrograde left-sided activation, thus masquerading as tachycardia using a left-sided accessory pathway, was described. It was postulated that the anterograde limb of the tachycardia circuit was a slow AV nodal pathway with a typical postero-septal location, whereas the retrograde limb was a long atrionodal pathway connecting the compact AV node and the left atrial free wall near the mid-coronary sinus.
REFERENCES


