there have been several reports of the occurrence of atypical varieties of atrioventricular nodal reentrant tachycardia (AVNRT) after catheter ablation of the typical AVNRT using either a fast or slow pathway approach.\(^1\)–\(^3\) However, the mechanism of this conversion is still unclear, particularly in adult patients having slow pathway ablation.\(^4\)–\(^5\)

We describe an adult patient with conversion of typical to atypical AVNRT after slow pathway ablation and we propose the electrophysiological mechanisms of this conversion.

### Case Report

The patient was a 63-year-old male with the primary complaint of palpitation, and typical AVNRT was suspected from the 12-lead surface ECG. Atypical AVNRT was not observed before the slow pathway ablation. An electrophysiological study was performed using standard electrophysiological methods after informed consent was obtained. All antiarrhythmic agents were discontinued for at least 5 half-lives before the study. Bipolar intracardiac electrograms filtered between 30 and 500 Hz were recorded and stored digitally on a Cardiolab system (Prucka Engineering) simultaneously with the 12-lead surface ECG.

Stimulation was performed with a programmable stimulator (Nihon-Kohden SEC3102). The antegrade and retrograde conduction properties were evaluated using incremental pacing and extrastimulus pacing from atrial and ventricular sites.

After determination of AVNRT as the mechanism of tachycardia, a 7F-diameter deflectable catheter with a 4-mm ablation tip (EP Technologies) was inserted. The ostium of the coronary sinus (CSos) and the posteromedial tricuspid annulus were explored in an attempt to record a slow pathway potential during sinus rhythm.\(^4\)–\(^5\) Ablation was performed with a 550-kHz unmodulated radiofrequency current from a generator with temperature monitoring (EP Technologies). During ablation, the radiofrequency energy was adjusted to obtain a catheter tip temperature between 50 and 60°C. The current was applied for 30 s if junctional tachycardia was observed during ablation.

Typical AVNRT with an His-atrial (HA) interval of 40 ms and AH/HA of 8.0 was induced by atrial extrastimulation (Fig 1). During tachycardia, the earliest atrial activation site changed from the His bundle region to the coronary sinus ostium. One additional radiofrequency current applied 5 mm upward from the initial ablation site made atypical AVNRT noninducible. These findings suggest that the mechanism of atypical AVNRT after slow pathway ablation is antegrade fast pathway conduction along with retrograde conduction through another slow pathway connected with the ablated antegrade slow pathway at a distal site. The loss of concealed conduction over the antegrade slow pathway may play an important role in the initiation of atypical AVNRT after slow pathway ablation. (\textit{Jpn Circ J} 1999; 63: 999–1001)

### Key Words:
- Atrioventricular nodal reentrant tachycardia
- Concealed conduction
- Multiple slow pathways
- Slow pathway ablation
Fig 1. Typical and atypical forms of atrioventricular nodal reentrant tachycardia (AVNRT). Three surface electrocardiographic leads and intracardiac electrograms are shown at a paper speed of 100 mm/s. (Left panel) Before slow pathway ablation: premature atrial stimulation at a coupling interval of 310 ms during a basic pacing cycle length of 600 ms initiated the typical form of AVNRT. (Right panel) After slow pathway ablation: premature atrial stimulation at a coupling interval of 300 ms during a basic pacing cycle length of 600 ms initiated the atypical form of AVNRT. After ablation, the earliest atrial excitation site changed from the anterior site near the His bundle region to the posterior site at the ostium of the coronary sinus (CS). HRA, high right atrial electrogram; HIS, His bundle electrogram; S1, basic atrial stimulation; S2, atrial premature stimulation; ABL1-2, distal pair of the ablation catheter located at the posteroseptal region; ABL3-4, proximal pair of the ablation catheter.

Fig 2. Left and right anterior oblique fluoroscopic images (LAO and RAO, respectively) demonstrating the position of the ablation catheter during typical and atypical atrioventricular nodal reentrant tachycardia (AVNRT). The successful ablation site of the retrograde slow pathway (Lower panel) was 5 mm superior to that of the antegrade slow pathway (Upper panel).

Fig 3. Schematic representation of hypothetical models of atrioventricular nodal reentrant tachycardia (AVNRT) circuit. *Ablation site. Before the ablation, atrial extrastimulation induced typical AVNRT (A) and the presence of antegrade concealed conduction of a slow pathway may prevent the retrograde conduction over another slow pathway (B). Ablation of the antegrade slow pathway decreased the antegrade concealed penetration, and allowed conduction over the retrograde slow pathway (C).
induced atypical AVNRT with an HA interval of 200 ms and AH/HA of 0.7 (Fig 1). During tachycardia, the earliest atrial activation site changed from the His bundle region to the CSos (the earliest excitation site was located at CS7-8). One additional radiofrequency current applied 5-mm upward from the initial ablation site eliminated the induction of atypical AVNRT (Fig 2).

Discussion

The mechanism of atypical AVNRT after slow pathway ablation is antegrade fast pathway conduction together with retrograde conduction through another slow or intermediate pathway connected with the ablated antegrade slow pathway at a distal site.

Langberg et al reported inducibility of atypical AVNRT after radiofrequency modification of the fast pathway and they considered this arrhythmia of little clinical significance! They postulated several mechanisms for these tachycardias. One was persistent typical AVNRT with a long ventriculo-atrial (VA) conduction time due to a damaged fast pathway. The longer conduction time through the upper common pathway may prolong the HA interval and shorten the atrio-His (AH) interval. Thus the AH/HA ratio may decrease as the atypical form of AVNRT.

Goldberg et al reported electrophysiologic characteristics of atypical AVNRT after fast pathway modification. In their study, the posterior approach, AV-node ablation of the postmodification atypical AVNRT resulted in VA block. They suggested the retrograde limb of the tachycardia is a slow pathway and not a damaged fast pathway. In contrast to these 2 reports, Silka et al demonstrated in young patients with typical AVNRT the potential for clinical recurrence of atypical AVNRT if this arrhythmia could be induced during programmed stimulation after slow pathway modification. These reports indicate that the partial modification of the anatomic substrate of AVNRT could induce another reentry, which is not clinically manifest before ablation.

The mechanism of conversion from typical to atypical AVNRT in the present patient is schematically illustrated in Fig 3. The inducibility of atypical AVNRT after radiofrequency ablation of the slow pathway is compatible with the reversal of the tachycardia (Fig 3A, C)6 The retrogradely conducting slow pathway might be the reverse of the antegrade slow pathway, but this is not necessarily true as indicated by the dissociated effect of radiofrequency ablation energy. Before the ablation, the presence of antegrade concealed conduction of a slow pathway may prevent the retrograde conduction over another slow pathway (Fig 3B). The antegrade slow pathway ablation modified and decreased the antegrade concealed penetration to the retrograde slow pathway, thereby allowing the conduction over the retrograde slow pathway (Fig 3C). The site of successful ablation of the retrograde slow pathway was different from that of the antegrade slow pathway. These findings suggest that the atrial insertions of the antegrade and retrograde slow pathways may be anatomically different.7–9 The radiofrequency modification of vagal fibers to the posteroseptal region could enhance the retrograde slow pathway conduction, resulting in the critical delay required for reentry to occur. However, we could not confirm this hypothesis because the retrograde conduction over the slow pathway could not be demonstrated before the slow pathway ablation.

In conclusion, patients with new atypical AVNRT after the slow pathway ablation may have multiple atrial insertions of functionally different slow pathways (retrograde conduction located superior to antegrade conduction in our case), and multiple slow pathway ablations may be necessary to eliminate these tachycardias.

References