Junctional rhythm associated with ventriculoatrial block during slow pathway ablation in atypical atrioventricular nodal re-entrant tachycardia

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Received 10 March 2008; accepted after revision 19 May 2008; online publish-ahead-of-print 9 June 2008

Aims We assessed responses to slow pathway ablation with respect to the appearance of ventriculoatrial (VA) block during junctional rhythm in both typical and atypical types of atrioventricular nodal re-entrant tachycardia (AVNRT).

Methods and results The 31 subjects included 16 patients with slow–fast type of typical AVNRT and 15 patients with atypical AVNRT (9 patients with fast–slow type and 6 patients with slow–slow type). During atypical AVNRT, the HA interval was prolonged (>70 ms) and the earliest atrial activation was located around the coronary sinus (CS) ostium. The difference in atrial activation times at the CS ostium and His-bundle area (A(CS–His)) during AVNRT was measured. Slow pathway ablation was performed using a classical electro-anatomical approach. In typical AVNRT, A(CS–His) was 21.3 ± 3.4 ms, and the HA interval was 34 ± 14 ms. During slow pathway ablation, all patients with typical AVNRT had junctional rhythm with retrograde atrial conduction. In contrast, in patients with atypical AVNRT, A(CS–His) was 12 ± 19.3 ms and the HA interval was 189 ± 77 ms. In 13 of the 15 patients with atypical AVNRT, slow pathway ablation induced junctional rhythm, which was not associated with retrograde atrial conduction. After ablation, AVNRT became non-inducible and antegrade atrioventricular (AV) conduction was preserved in all patients.

Conclusion In patients with atypical AVNRT, junctional rhythm with VA block during slow pathway ablation is commonly observed and indicates the success of the ablation of retrograde slow pathway conduction, but has no relation to the risk of subsequent AV block. During junctional rhythm, occasional appearance of the sinus beats with intact antegrade AV conduction is essential for safety of ablation.

KEYWORDS
Ablation; Atroventricular nodal re-entry; Junctional rhythm; Slow pathway; Ventriculoatrial block

Introduction

In patients with slow–fast type of typical atrioventricular nodal re-entrant tachycardia (AVNRT), ablation of the slow pathway area can make most AVNRT events non-inducible. During ablation, thermal injury of the slow pathway may enhance automatic activity of the posterior extension of the atrioventricular (AV) nodal cells and induce junctional rhythm that is conducting to the atrium through the retrograde fast pathway. Hence, the appearance of junctional beats associated with ventriculoatrial (VA) block during slow pathway ablation has been suggested as a marker of injury to the fast pathway, which could induce AV block. However, loss of VA conduction during slow pathway ablation is not always associated with AV conduction block.

It is possible that different types of AVNRT have different responses to slow pathway ablation depending on their re-entrant circuits. Lee et al. reported that all of 7 patients with fast–slow type AVNRT and 1 of 11 patients with slow–intermediate type AVNRT had junctional rhythm with VA block during slow pathway ablation. Heidbüchel et al. observed VA block during junctional rhythm in 67% of patients with fast–slow type and 53% of patients with slow–slow type of atypical AVNRT and none of these patients developed AV block. The aim of this study was to assess the relation between electrophysiological properties of AVNRT and responses to the slow pathway ablation with respect to the appearance of VA block during junctional rhythm.
Methods

Patients

On the basis of the AH and HA intervals at the His-bundle electrogram (HBE) measured during tachycardia, the AVNRT is mainly classified into two types: atypical and typical. During atypical AVNRT, the HA interval was prolonged by >70 ms and the earliest atrial activation was located around the coronary sinus (CS) ostium, and negative P-wave after QRS was often recognized at leads II, III, and aVF. Atypical AVNRT with the AH interval ≥200 ms was classified as slow-slow type. Among 100 consecutive patients with AVNRT, who underwent electrophysiological study and slow pathway ablation at our institution from 2002 to 2006, 15 patients (8 men and 7 women; age 51.0 ± 20.0 years) had atypical AVNRT. Among the remaining 85 patients with typical AVNRT, 16 patients (7 men and 9 women; age 50.7 ± 17.0 years) were randomly included to match atypical type in age and gender.

Electrophysiological study

All electrophysiological studies were performed using standard methods after obtaining written informed consent. All antiarrhythmic agents had been discontinued for at least five half-lives before the study. Catheterization was performed under local anesthesia via the right and left femoral veins and the left antecubital vein. Electrode catheters were positioned at the high right atrium (HRA), the HBE region, the right ventricle (RV), and the CS, with the proximal bipole of the CS catheter positioned at the CS ostium.

Bipolar intra-cardiac electrograms filtered between 30 and 500 Hz were simultaneously recorded and stored digitally on a CardioLab system (Prucka Engineering, Houston, TX, USA) with a 12-lead surface electrocardiogram. All measurements with the CardioLab system were made at screen speeds of 100-200 mm/s using on-screen digital calipers. Stimuli were delivered from a programmable stimulator (Nihon-Kohden SEC3102, Tokyo, Japan) as rectangular pulses of 2 ms duration and twice the diastolic threshold. Baseline electrophysiological evaluations and tachycardia inductions were performed during incremental pacing and extrastimulation from the RV and HRA. The diagnosis of AVNRT was made on the basis of the standard criteria.

Earliest retrograde atrial activation

The earliest retrograde atrial activation site during AVNRT and the RV pacing was localized by mapping the right side of the inter-atrial septum and the CS using the ablation catheter. The HA interval was measured from the onset of the most proximal His-bundle potential to the onset of the earliest atrial potential at the HBE during stable tachycardia. When His-bundle potential was obscured during AVNRT, the timing of the onset of His-bundle potential was inferred from the HV interval during sinus rhythm. The measurement of HA interval or VA interval at the HBE during tachycardia was helpful in differentiating the typical from the atypical type of AVNRT, and the HA interval >70 ms or VA interval >60 ms has been reported as atypical AVNRT. Comparison of the HA interval measured from the end of the most proximal His-bundle potential to the onset of atrial potential during ventricular pacing similar to the tachycardia cycle length with the HA interval during tachycardia was made to evaluate the presence of the lower common pathway. The presence of retrograde fast pathway conduction was evaluated using RV stimulation before ablation.

To clarify the electro-anatomical relationship between the anterior and posterior connections of the AV node to the atrium, the difference in the atrial activation times at the CS ostium and the HBE area [A(CS–His)] during AVNRT was measured. When the atrial potential at the CS ostium preceded that of the HBE area, the value of A(CS–His) became positive.

Radiofrequency catheter ablation of the slow pathway

Ablation was performed during sinus rhythm in all patients with typical AVNRT and nine patients with atypical AVNRT at the classic slow pathway region between the CS ostium and the tricuspid annulus using the electro-anatomical approach. In six patients with atypical AVNRT, ablation was performed during tachycardia. A 7 Fr deflectable catheter with a 4 mm ablation tip (EP Technologies, Sunnyvale, CA, USA) was inserted through the right femoral vein. Ablation was performed between the distal electrode and a skin electrode with a 550 KHz unmodulated radiofrequency (RF) current from a generator with temperature monitoring (EP Technologies, Sunnyvale, CA, USA). The RF energy was applied in a temperature-controlled mode, with an upper temperature limit of 55°C, maximal power output of 30 W, and duration of 40 s for each application. The inducibility of AVNRT was assessed after each application. Successful ablation was defined as the elimination of AVNRT induction associated with suppression of either antegrade or retrograde slow pathway conduction. The ablation position was moved upwards gradually along the right septal region, if the ablations at the right inferiorseptal region were unsuccessful. All episodes of junctional rhythm caused by slow pathway ablation were analysed for VA conduction.

Results

The relationship between the AH and the HA interval at the HBE during tachycardia in 15 patients with typical AVNRT (9 patients with fast–slow type and 6 patients with slow–slow type) and 16 patients with slow–fast type of typical AVNRT is shown in Figure 1, left panel. The HA interval during tachycardia in patients with typical AVNRT was significantly shorter than that in patients with atypical AVNRT (36 ± 19 vs. 189 ± 77 ms, P < 0.001). The AH interval during tachycardia in patients with typical AVNRT was significantly longer than that in patients with atypical AVNRT (339 ± 65 vs. 196 ± 127 ms, P < 0.001). The tachycardia cycle length did not differ between typical and atypical AVNRT (373 ± 63 vs. 385 ± 99 ms).

Figure 1  Relationship between the AH and the HA interval at the His-bundle electrogram during atrioventricular nodal re-entrant tachycardia (left) and relationship between the difference in atrial activation times at the coronary sinus ostium and the His-bundle area [A(CS–His)] and the HA interval during tachycardia (right). Left panel: patients with atypical atrioventricular nodal re-entrant tachycardia with HA interval >70 ms consisted of fast-slow type (+) and slow-slow type (°) were widely distributed, in contrast, patients with slow-fast type of typical atrioventricular nodal re-entrant tachycardia with the HA interval ≤70 ms (△) were located only right lower area. Right panel: the A(CS–His) showed significant positive correlation with the HA. Patients with atypical atrioventricular nodal re-entrant tachycardia who had both A(CS–His) ≥ −10 ms and the HA interval ≥70 ms were distributed widely.
In patients with typical AVNRT, A(CS–His) was $-21.3 \pm 3.4$ ms and the earliest atrial activation was observed at the His-bundle recording area. In patients with atypical AVNRT, the earliest atrial activation was seen around the CS ostium and the A(CS–His) was $12 \pm 19.3$ ms. The relationship between the A(CS–His) and the HA interval at the HBE during tachycardia was plotted (Figure 1, right panel). Significant positive correlation was evident between these parameters ($y = 3.47x + 128$, $r = 0.78$, $y = HA$, $x = A(CS\text{–His})$), indicating the relation of the longer retrograde AV nodal conduction time with the posterior shift of the atrial exit site; however, there was a considerable variation in atypical AVNRT. During RV pacing at a basic cycle length of 600 ms, the stimulus atrial excitation interval at the HBE was significantly longer in atypical AVNRT than in typical AVNRT ($215 \pm 54$ vs. $123 \pm 23$ ms, $P < 0.001$), and no retrograde fast pathway conduction was observed in patients with atypical AVNRT before ablation.

During slow pathway ablation, all patients with typical AVNRT had junctional rhythm with retrograde atrial conduction (Figure 2) and AVNRT became non-inducible. Junctional rhythm preceded by atrial ectopic beats from the ablation site appeared in four patients, but did not affect the inducibility of AVNRT. The mean cycle length of junctional rhythm during ablation did not differ between typical and atypical AVNRT (minimum cycle length: $532 \pm 140$ vs. $526 \pm 183$ ms and maximum cycle length: $725 \pm 123$ vs. $691 \pm 114$ ms).

In nine patients with atypical AVNRT, including four patients with slow–slow type (Figures 3 and 4) and five patients with fast–slow type (Figure 5), who had ablation during sinus rhythm, junctional rhythm appeared without retrograde atrial conduction. After slow pathway ablation, AVNRT became non-inducible and antegrade AV nodal conduction was preserved in all patients. In three patients with atypical AVNRT, the HA interval during constant RV pacing could be measured before ablation and comparison of the HA interval during tachycardia and RV pacing suggested the presence of the lower common pathway (Figure 3).

In four out of six patients with atypical AVNRT (four patients with fast–slow type and two patients with slow–slow type), slow pathway ablation terminated tachycardia with VA block and the continued RF application induced junctional rhythm without retrograde atrial conduction (Figure 6). The remaining two patients with fast–slow type had junctional rhythm preceded by atrial ectopic beats originating from the slow pathway area after termination of tachycardia. They had no VA conduction during RV pacing.

In all 31 patients, neither recurrence of AVNRT nor development of AV block was observed during a mean follow-up period of $27 \pm 24$ months. In atypical AVNRT, occasional appearance of the sinus beats with intact antegrade AV conduction is essential for the safety of ablation during junctional rhythm with VA block (Figures 4–6).

**Discussion**

In the present study, junctional rhythm without retrograde atrial conduction during slow pathway ablation was commonly observed in patients with atypical AVNRT, indicative of the successful elimination of retrograde slow pathway conduction but not related to the risk of subsequent AV block. The occurrence of VA block during slow pathway ablation may thus depend on the retrograde conduction properties of AVNRT.

Junctional rhythm occurring during slow pathway ablation is sensitive but not a specific marker of successful ablation of the slow pathway.\(^1\) Junctional rhythm may result from heat injury to the discrete slow pathway. However, the
width of the slow pathway is not so narrow that any appearance of junctional rhythm would automatically indicate the elimination of the whole slow pathway conduction. In the fast–slow type of AVNRT, most junctional rhythm conducts to the atrium through the retrograde fast pathway (Figures 2 and 7, left panel).

Junctional rhythm during ablation for AVNRT followed by AV block can be characterized by the appearance of a shorter cycle length of junctional rhythm and loss of VA conduction. In the present study, the cycle length of slow–fast type of AVNRT, most junctional rhythm conducts to the atrium through the retrograde fast pathway (Figures 2 and 7, left panel).

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conduction through the fast pathway, indicating that fast–slow type is not the simple reversal of slow–fast type. Hintringer et al. reported that only 11 of 46 episodes of junctional rhythm during pacing from the successful slow pathway ablation were followed by AV block. These findings suggest that the occurrence of VA block during slow pathway ablation may be dependent on the type of AVNRT.

Detailed atrial mapping revealed that the retrograde atrial activation of junctional beats was conducted through the retrograde fast pathway and differed from the atrial activation during pacing from the successful slow pathway ablation. Other investigators demonstrated discordance in the earliest retrograde atrial activation during AVNRT and ablation-induced junctional rhythm in more than half of the examined patients. In the present study, during junctional rhythm, the earliest retrograde atrial conduction was seen at the HBE area in patients with typical AVNRT but VA conduction was not seen in patients with atypical AVNRT. These observations suggest that in patients with slow–fast type of typical AVNRT, the junctional rhythm may conduct to the atrium through the retrograde fast pathway. However, in patients with the fast–slow type, junctional rhythm could not conduct to the atrium because there was no retrograde conduction through the fast pathway, indicating that fast–slow type is not the simple reversal of slow–fast type (Figure 7, middle panel).

In patients with slow–slow type of atypical AVNRT, it is possible that junctional rhythm could conduct to the atrium through the retrograde slow pathway. But junctional rhythm induced by the slow pathway ablation did not show VA conduction, which means the presence of retrograde fast pathway conduction is essential for the appearance of VA conduction during junctional rhythm. After successful retrograde slow pathway ablation, antegrade slow pathway conduction remained in patients with slow–slow AVNRT (Figure 7, right panel).

Our observations have following clinical implications. In patients with slow–fast type of AVNRT, the occurrence of VA block during ablation-induced junctional rhythm is indicative of an impending AV block and is an indication that delivery of RF current should be stopped. However, in the atypical type of AVNRT, it actually means the successful elimination of the retrograde slow pathway conduction. In the present study, we could differentiate the atypical type from the typical type using the relationship between the HA interval and the A(CS–His) interval (Figure 1). It is possible that some patients with atypical AVNRT having the longer lower common pathway show the shorter HA interval similar to the typical type. Thus, increased attention should be paid as to whether AVNRT belongs to typical or atypical type before slow pathway ablation.

We measured the A(CS–His) interval during AVNRT, which was positively correlated with the HA interval. The longer A(CS–His) reflects the more posteriorly located retrograde slow pathway associated with the longer retrograde conduction time. The positive correlation between the A(CS–His) and HA intervals means that patients with typical AVNRT were located in a relatively narrow area, whereas patients with atypical type were widely distributed, indicating variability of electro-anatomical properties of the retrograde slow pathway.

At present, we were fortunate not to have any patient with AV block after slow pathway ablation. However, it is possible that patients with atypical AVNRT having posterior-shifted fast pathway could exhibit slow pathway ablation, which could in turn induce AV block. Another reliable marker other than careful observation of the sinus beats with intact antegrade conduction may be necessary for safety of ablation in atypical AVNRT, such as atrial pacing faster than junctional rhythm.

This study has several limitations. First, subjects of this retrospective study consisted of 15 patients with atypical AVNRT and 16 randomly selected patients with typical AVNRT. Although the number of patients was limited, we believe the characteristic response to slow pathway ablation in atypical AVNRT could be clarified. Secondly, although attempts were made to assess the lower common pathway in all patients with atypical AVNRT, the presence was confirmed only in three patients because of difficulty in recognition of the retrograde His-bundle potential during ventricular stimulation. Further studies are needed to establish anatomical and functional characteristics of the re-entrant circuit of atypical AVNRT.

In summary, we conclude that, in patients with atypical AVNRT, junctional rhythm without retrograde atrial conduction during slow pathway ablation is common and indicates the success in elimination of retrograde slow pathway conduction but not a risk of impending AV block.

Conflict of interest: none declared.

Funding
This work was supported in part by a Grant-in-Aid for Scientific Research (C-20590816) from the Japan Society for the Promotion of Science.
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