Incidence and Mechanism of Dislocated Fast Pathway in Various Forms of Atrioventricular Nodal Reentrant Tachycardia

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Background  The incidence and mechanism of the dislocated antegrade fast pathway (A-FP) were examined in various forms of atrioventricular nodal reentrant tachycardia (AVNRT).

Methods and Results  To localize the A-FP, 5 atrial sites comprising the inferior coronary sinus ostium (CSOS), apex of the triangle of Koch (A-TOK), and 3 equidistant sites on the atrioventricular junction extending from A-TOK to CSOS (site S, M, and I) were pace mapped at 100 beats/min in 71 patients with slow-fast (n=49), fast-slow (n=7) and slow-intermediate (n=15) forms of AVNRT. The site with the shortest interval between the stimulus and His potential recorded at the A-TOK (shortest St-H) was defined as the A-FP site. The A-FP was located at A-TOK in 31 patients (nondislocated group), and inferior to A-TOK in 40 patients (site S in 26, M in 13, and I in one patient; dislocated group). There was no significant difference in the location of the A-FP among the 3 forms of AVNRT. Although the shortest St-H did not differ between groups, the St-H at A-TOK in the dislocated group was significantly longer than that in the nondislocated group. Additionally, the His potential preceding that of the A-TOK was observed more frequently inferior to the A-TOK in the dislocated group than in the nondislocated group, suggesting that the A-FP dislocation was accompanied by displacement of the His bundle.

Conclusions  Dislocated A-FP was frequently and uniformly observed among various forms of AVNRT, and is probably caused by inferior displacement of the entire atrioventricular node–His bundle apparatus. (Circ J 2007; 71: 1099–1106)

Key Words: Atrioventricular node; Electrophysiology; Mapping

The prevailing clinical understanding of atrioventricular nodal reentrant tachycardia (AVNRT) is based on a model of dual atrioventricular nodal (AVN) pathways that are functionally and anatomically distinct, with a fast pathway located superior to the compact AVN and a slow pathway located in the infero-septal right atrium.1,2 The basis for catheter ablation is this spatial segregation of the fast and slow pathways.3–7 Although selective slow-pathway ablation is performed at the inferior aspect to the triangle of Koch (TOK), far from the usual site of the compact AVN, the risk remains of inadvertent atrioventricular (AV) block related to injury of the antegrade fast pathway (A-FP).7 A possible explanation for impairment of AV conduction following slow-pathway ablation is atypical location of the A-FP.8,9 However, the location of the A-FP remains incompletely understood. In the present study, we used pace mapping10,11 to localize the A-FP in various forms of AVNRT and systematically defined the incidence of dislocated A-FP. Furthermore, we elucidated the characteristics and the mechanism of dislocated A-FP by analyzing the clinical and electrophysiological variables of patients with dislocated and nondislocated A-FP.

Methods

Patients  Seventy-one patients with AVNRT (34 males, 37 females; mean age, 51 years, range, 11–87 years) who were referred for curative ablation were included in this study. The underlying mechanisms of AVNRT were the slow-fast form in 49 patients, fast-slow form in 7 patients, and slow–intermediate form in 15 patients. Diagnosis of AVNRT was based on previously published standard criteria.12–14 The slow–intermediate form of AVNRT was diagnosed if (1) the tachycardia was initiated with an antegrade jump in the AH interval, and (2) retrograde atrial activation with the earliest atrial activation recorded at the inferoseptal right atrium and exhibiting decremental conduction properties. All anti-arrhythmic drugs were withdrawn for at least 5 half-lives before the electrophysiological study. Written informed consent for the study and ablation was given by each patient. The protocol was approved by the Hospital Human Research Committee.

Electrophysiological Study  The study was performed with patients in a fasting, unsedated state. Two 6-Fr quadripolar electrode catheters (USCI, Billerica, MA, USA) were inserted percutaneously.
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via the right femoral vein and positioned in the right ven- tricular apex and the apex of the TOK (A-TOK) where the His potential was observed. A 6-Fr decapolar electrode catheter (Daig Corp, Minnetonka, MN, USA) was inserted via the subclavian vein and positioned in the coronary sinus. A 7-Fr large-tip (4-mm long), deflectable quadrupolar electrode catheter with a 2-mm interelectrode distance (Biosense Webster, Inc, Diamond Bar, CA, USA) was introduced into the right femoral vein and advanced to the right intra-atrial septum for atrial mapping, pacing, and ablation. Bipolar electrograms from the coronary sinus, His bundle region, right ventricular apex, and sequential right intra-atrial septum were filtered at 50–600 Hz and recorded with the surface ECG using a polygraph (RMC-2000, Nihon Kohden, Tokyo, Japan, or EP-workmate, EP Med systems, Inc, Mt Arlington, NJ, USA). The right atrium was paced at an output of twice the diastolic threshold and a pulse width of 2 ms with a cardiac stimulator (SEC-3102, Nihon Kohden, Tokyo, Japan).

Study Protocol

To localize the A-FP, 5 atrial sites comprising the inferior portion of the coronary sinus ostium, A-TOK where the His potential was recorded, and 3 equidistant sites on the AV junction extending from the A-TOK to the coronary sinus ostium (sites S, M, and I) were pace mapped during sinus rhythm (Fig 1).

Pace mapping was performed using a 7-Fr large-tip, deflectable quadrupolar electrode catheter. The anatomical location of these pace mapping sites suggestive of an AV annulus of the A-TOK was confirmed by simultaneous recording of atrial and ventricular electrograms, relative to the coronary sinus catheter and the images obtained by biplane contrast right atrioography. Pace mapping was performed at a rate of 100 beats/min. The catheter positions during pacing were checked by biplane fluoroscopy. The pacing protocol was performed at least twice at each site. The last 2–3 beats that were stably paced were used for the calculations. The interval between the stimulus artifact and the onset of the His potential deflection recorded by the reference 6-Fr quadrupolar electrode catheter positioned at the A-TOK (St-H) was measured. The A-FP site was de- fined as the site where the shortest St-H was recorded. The dislocated A-FP group was defined as the patients in whom the shortest St-H was observed at the sites inferior to the A-TOK (sites S, M, and I, and the inferior portion of the coronary sinus ostium). The nondislocated A-FP group was defined as the patients in whom the shortest St-H was observed at the A-TOK. The slow and/or intermediate pathway was ablated after localization of the A-FP.

To characterize patients with dislocated A-FP, we compared their clinical and electrophysiological variables to those with a nondislocated A-FP: age, gender, AH and HV intervals during sinus rhythm, and antegrade Wenckebach rate, as well as the shortest St-H interval and the St-H interval at the A-TOK. To clarify whether a dislocated A-FP is associated with dislocation of the His bundle, the presence or absence of His potential at each mapping site was compared between groups. Furthermore, the AH intervals during slow-pathway conduction were compared between groups to examine whether inferior dislocation of A-FP is associated with dislocation of the His bundle and thus a shortened slow-pathway conduction interval. The AH intervals during slow-pathway conduction were collected from patients with the slow–fast and slow–intermediate forms of AVNRT, in whom slow-pathway conduction was antegrade during tachycardia.

Catheter Ablation

Radiofrequency energy was delivered by an energy generator as a continuous, unmodulated sine waveform at 500 kHz in a unipolar mode between the tip of the ablation catheter and a large skin electrode placed under the patient’s back, using a radiofrequency energy generator (CABL-IT, Central Inc Ichikawa, Chiba, Japan). In patients with the slow–fast form of AVNRT, slow-pathway ablation was instituted in a stepwise fashion. The slow-pathway ablation site was defined anatomically, irrespective of the presence or absence of slow and/or slow pathway potential. Radiofrequency energy (20 W for 30 s at each site) was delivered along the tricuspid annulus, starting at the level of the inferior portion of the coronary sinus ostium, with the ablation catheter positioned to record an atrial to ventricular electrogram ratio of <0.5. If the slow–fast form of AVNRT was induced after energy application, the catheter tip was then advanced superiorly to the adjacent site in a stepwise fashion. In patients with the fast–slow form of AVNRT, the earliest atrial activation site of the retrograde slow pathway was targeted as the ablation site. In patients with the slow–intermediate form of AVNRT, slow-pathway ablation was performed initially in a stepwise fashion, starting at the level of the inferior portion of the coronary sinus ostium. If additional tachycardia using an intermediate pathway was induced after elimination of the antegrade slow-pathway conduction, ablation of the intermediate pathway was performed. Radiofrequency energy was not delivered to the A-FP site because we wanted to avoid impairing fast-pathway conduction.

The endpoint of the ablation procedure was inability to induce any type of AVNRT, even after administration of 10 μg/min of isoproterenol. After ablation, the relative location of the A-FP and the successful slow or intermediate pathway ablation sites were compared among the slow–fast, fast–slow, and slow–intermediate forms of AVNRT.

Statistical Analysis

The values for all data are expressed as mean±SD. Dif-
Results

Location of A-FP

The A-FP site was localized by atrial pace mapping in all patients. The atrial to ventricular electrogram ratio of the pace mapping site at the A-TOK, sites S, M, and I, and the inferior portion of the coronary sinus ostium was 3.6±2.5, 3.2±2.5, 3.0±2.6, 3.2±2.7 and 3.4±2.7, respectively. There was no significant difference in the ratio among these pace mapping sites, suggesting that this mapping was performed uniformly with respect to positioning of the tricuspid valve. The shortest St-H site, defined as the A-FP site, was observed at a single site in all patients. The A-FP was observed at the A-TOK in 31 patients (44%; nondislocated group), but was located inferior to the A-TOK in the dislocated group in 29 patients (40%).

Fig 2. Location of the antegrade fast pathway (A-FP) identified by atrial pace mapping. (Left) A-FP distribution in all patients. (Right) A-FP distribution in patients with the slow–fast (SF-), fast–slow (FS-), and slow–intermediate forms of atrioventricular nodal reentrant tachycardia (SI-AVNRT). Closed and open circles denote the dislocated and nondislocated A-FP groups, respectively. Abbreviations are the same as in Fig 1.

Fig 3. Changes in the St-H intervals of the nondislocated (Panel A) and dislocated (Panels B–D) A-FP groups. Ordinates: the difference between the St-H interval at each pace mapping site and the shortest St-H interval. Abscissa: the pace-mapping site. *p<0.0001 between pace mapping sites. Abbreviations are the same as in Figs 1,2.

Differences between the clinical and electrophysiological parameters were analyzed using Student’s t-test for quantitative data or the chi-squared statistic for qualitative data. A value of p<0.05 was considered statistically significant.
remaining 40 patients (56%; dislocated group) (Fig 2, Left). Overall, the A-FP was observed at site S in 26 patients (37%), site M in 13 patients (18%), and site I in 1 patient (1%) (Fig 2, Left). The percentage of nondislocated A-FPs in patients with the slow–fast, fast–slow, and slow–intermediate forms of AVNRT was 41%, 71%, and 40%, respectively, and thus that of the dislocated A-FPs was 59%, 29%, and 60%, respectively (Fig 2, Right). There was no significant difference in the distribution of the A-FP among the 3 forms of AVNRT (Fig 2, Right). Fig 3 shows the change in St-H interval between patients of the nondislocated and dislocated A-FP groups. In the nondislocated A-FP group, the shortest St-H was observed at the A-TOK and was significantly shorter than those at the other sites (Fig 3A). In the dislocated A-FP group, however, the shortest St-H site was observed at a single site in all patients and was surrounded by sites with longer St-H intervals (Figs 3B–D).

Catheter Ablation
The slow pathway was successfully eliminated by the application of radiofrequency energy in all patients. The mean number of energy applications was 5±4. The successful slow-pathway ablation site was at the inferior portion of the coronary sinus ostium in 55 patients (77%), at site I in 10 (14%), and at site M in 6 (8%) (Fig 4); this site was adjacent to the A-FP in 9 patients (13%) (7 slow–fast, 1 fast–slow, and 1 slow–intermediate forms of AVNRT; adjacent group), but was not in the remaining 62 patients (87%) (42 slow–fast, 6 fast–slow, and 14 slow–intermediate forms of AVNRT; nonadjacent group) (Fig 4).

After successful ablation of the antegrade slow pathway, no additional tachycardia was induced in patients with the slow–fast or fast–slow form of AVNRT. However, sustained or nonsustained fast–intermediate form of AVNRT was induced after elimination of the antegrade slow pathway conduction in 12 of 15 patients with the slow–intermediate form of AVNRT. Intermediate pathway ablation was thus performed in these 12 patients, targeting the earliest atrial activation site of the retrograde intermediate pathway. The intermediate pathway was successfully eliminated at site I in 11 patients and site M in 1 patient (Fig 5). Among these 12 patients in whom the intermediate pathway was ablated, the A-FP was adjacent to the successful intermediate-pathway ablation site in 5 patients (42%) (Fig 5). The percentage of patients in whom the A-FP was adjacent to the successful intermediate-pathway ablation site was significantly higher than in those in whom the A-FP adjacent to the successful slow-pathway ablation site was...
Although there was no significant difference, the AH interval was slightly prolonged following slow and/or intermediate pathway ablation from 82±12 to 85±13 ms in the adjacent group (n=14; p=NS). However, the AH interval was slightly shortened from 90±27 to 88±28 ms after ablation in the nonadjacent group (n=57; p=NS). Transient impairment of AV conduction was observed following slow-pathway ablation in 2 patients with the slow-fast form of AVNRT in the adjacent group, but not in the nonadjacent group. In 1 patient of the adjacent group, the Wenckebach AV block rate was decreased from 160 to 120 beats/min and the AH interval was transiently prolonged from 80 to 95 ms, but both recovered several minutes later. In another patient in the adjacent group, transient Wenckebach AV block was observed during sinus rhythm, but recovered.

(42% vs 12%, p<0.05).

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Comparison of Patients With Dislocated and Nondislocated A-FP

The clinical and electrophysiological variables of the patients in the dislocated and nondislocated A-FP groups are summarized in Table 1. There was no significant difference in mean age, gender, AH and HV intervals during sinus rhythm, and antegrade Wenckebach rate. The shortest St-H interval did not differ between the 2 groups; however, the St-H interval at the A-TOK in the nondislocated group was significantly shorter than that of the patients in the dislocated group. This finding suggests the presence of an anatomically prolonged propagation route in the dislocated group when pacing was performed at the A-TOK. The His potential that preceded that of the A-TOK by $-10\pm10$ ms was more frequently observed at the sites inferior to the A-TOK in the dislocated group than in the nondislocated group (63% vs 10%, p<0.01), suggesting that the A-FP displacement was accompanied by the His bundle apparatus. Although the successful slow-pathway ablation site did not differ between the dislocated (site M in 6 patients, site I in 5 patients, and the inferior portion of the coronary sinus ostium in 29 patients) and the nondislocated (site I in 5 patients and the inferior portion of the coronary sinus ostium in 26 patients) groups (p=NS), the AH interval during slow-pathway conduction in the dislocated group was significantly shorter than that in the nondislocated group (Table 1).

Fig 6 shows the recordings from a patient with the slow–fast form of AVNRT in whom the A-FP was observed at site M. During sinus rhythm, the His potential was observed at sites S and M (Fig 6A), although the His potential at sites S and M preceded that at the A-TOK by 15 and 20 ms, respectively (Fig 6A). The St-H intervals with the mapping catheter positioned at the A-TOK, sites S, M, and I, and at the inferior portion of the coronary sinus ostium were 115, 110, 95, 115, and 120 ms, respectively (Fig 6B). These results indicate an inferiorly displaced AVN–His bundle apparatus at site M in this patient.

Discussion

Using atrial pace mapping we found that the A-FP was dislocated inferior to the A-TOK in 56% of patients with AVNRT. Dislocation was observed uniformly among the 3 forms of AVNRT. The finding that transient impairment of AV conduction occurred only in patients in whom the A-FP was adjacent to the successful ablation site further confirmed the accuracy of A-FP localization using atrial pace mapping. Furthermore, our results show that the A-FP and successful ablation site were adjacent more often during intermediate-pathway ablation than during slow-pathway ablation, which suggests a potential risk of AV block during intermediate-pathway ablation.

Several investigators have reported that successful slow-pathway ablation with a posterior approach may be accompanied by inadvertent injury of the fast pathway. Jackman et al reported damage to the fast pathway in a patient who underwent radiofrequency energy application to the coronary sinus ostium and Langberg et al reported that 14% of patients had unintended injury of the fast pathway during slow-pathway ablation with a posterior approach. Williamson et al further suggested that radiofrequency application near the coronary orifice might be sufficiently close to the compact AVN to injure its structure. Chen et al showed that successful modification of the AV junction using radiofrequency energy at the mid/posteroseptal area not only eliminated the slow pathway but also injured the compact AVN. Engelstein et al reported that in 7 patients with AVNRT who had evidence of a posterior fast pathway near the coronary sinus ostium, fast-pathway conduction was abolished following radiofrequency ablation in the posteroseptal region in 6 of them. Taken together, these findings suggest that ablation of the posteroseptal region introduces a potential risk of inadvertent fast-pathway injury and dislocation of the A-FP to the mid/posteroseptal area, which is consistent with the mapping data from the present study. We also delineated the limitations of an anatomically guided slow-pathway ablation approach and emphasized the importance of accurate localization of the A-FP before slow- or intermediate-pathway ablation.

There were no significant differences in the clinical char-
Dislocation of the A-FP was not infrequent in patients with AVNRT in this study and was uniformly observed among the various forms of AVNRT. Considered together, the results of our study suggest that dislocation of A-FP is caused by inferior displacement of the entire AVN–His bundle apparatus. Because atrial pace mapping is useful in identifying a dislocated A-FP, this simple method should be performed prior to slow- and/or intermediate-pathway ablation in patients with AVNRT in order to avoid causing AV block.

References


