AV Nodal Reentrant Tachycardia with Mahaim Fiber Conduction

Chikayoshi Komatsu, M.D., Katsuro Ito, M.D., Takanari Ishinaga, M.D., Yasutaka Tokuhisa, M.D., Takashi Tanoiri, M.D., Hiroshi Makino, M.D., Jun Nomoto, M.D., Tetsutaro Tamura, M.D., and Tetsuo Okamura, M.D.

SUMMARY

Paroxysmal tachycardia with widened QRS complexes was found in a 46-year-old woman. In sinus rhythm, the patient had electrocardiographic evidence of type B preexcitation with a left bundle branch block pattern. The resting PR interval (160 msec) and A-H interval (100 msec) were within normal limits, but the H-V interval (10 msec) was abnormally short. Programmed atrial extrastimuli at progressively shorter coupling intervals resulted in sudden prolongation of the A-H interval from 120 msec to 250 msec, and the His bundle activities became incorporated just after initiation of the QRS complexes. The QRS morphology was changed but the change was minimal, and atrial echo beats or sustained tachycardia with wide and preexcited QRS complexes were elicited. It is postulated that the site of reentry is within the AV node with preexcitation occurring as the result of conduction in an anomalous nodo-ventricular pathway.

Additional Indexing Words:
AV nodal reentrant tachycardia Mahaim fiber Nodo-ventricular pathway Fasciculo-ventricular pathway Accessory pathway Preexcitation AV node dual pathway WPW syndrome Wide QRS tachycardia Innocent bystander PSVT

PREEXCITATION of the ventricles may result from conduction over the atrioventricular bypass tract of Kent or via an accessory nodo-ventricular or His-ventricular pathway of the Mahaim type.\textsuperscript{1,2} We report a case in which there was electrocardiographic evidence of right ventricular preexcitation in association with recurrent episodes of paroxysmal tachycardia with a widened, preexcited QRS pattern.

\textsuperscript{1}From the Fourth Department of Internal Medicine, Jikei University School of Medicine, Tokyo, Japan.

Address for reprints: Chikayoshi Komatsu, M.D., Fourth Department of Internal Medicine, Jikei University School of Medicine, 3-25-8, Nishishinbashi, Minato-ku, Tokyo 105, Japan.

Received for publication October 13, 1988.

Accepted May 24, 1989.
Patients and Methods

An otherwise normal 46-year-old female was admitted to our hospital with frequent episodes of paroxysmal tachycardia from a young age. Since January, 1976, the duration and frequency of the tachycardia have increased. An electrocardiogram (Fig. 1) revealed a regular tachycardia at 190 beats/min with widened QRS complexes and a left bundle branch block pattern, but no detectable P waves. Intravenous administration of procainamide (500 mg) restored sinus rhythm. An electrocardiogram during sinus rhythm indicated a PR interval of 0.16 sec, and QRS complexes of type B preexcitation with a left bundle branch block pattern (Fig. 2). The QRS morphology in sinus rhythm and during tachycardia did not differ significantly. The physical examination, chest x ray, blood count, urinalysis and blood chemistry were normal. Subsequently, the patient underwent electrophysiological studies. A tripolar electrode catheter was passed percutaneously through a femoral vein and placed across the tricuspid valve for His bundle recording. A second hexapolar catheter was placed at the right ventricular apex through an antecubital vein. The distal two electrodes (tip) were utilized for ventricular pacing or recording of ventricular electrograms, the middle two electrodes (10 cm from the tip) were used for recording of right atrial electrophysiology.

![Fig. 1. ECG during paroxysmal tachycardia attack.](image-url)
grams, and the proximal two electrodes (13.5 cm from the tip) were designated for right atrial pacing. A filtered bipolar esophageal electrode was utilized for recording the left atrial electrograms. Multiple surface electrocardiographic leads (I, II, III, V1, V6) and intracardiac electrograms were recorded simultaneously on a multichannel jet recorder (Nihon Kohden, RM-6000) at a paper speed of 100 or 200 mm/sec. Stimuli were provided by a programmable digital stimulator (Cardiac Stimulator, SEC-2102), at an intensity approximately twice the diastolic threshold and a duration of 2 msec.

_Electrophysiological abbreviations:_

HRA, LSRA, LA, H, V1 are the high right atrial, low septal right atrial, left atrial, His bundle, and ventricular electrograms of sinus or driven beats (S1), respectively. HRA2, LSRA2, LA2, H2, V2 are the electrograms in response to the extrastimulus (S2).

---

Fig. 2. ECG during sinus rhythm.
RESULTS

Intracardiac electrograms during sinus rhythm revealed a P-A interval of 50 msec, an A-H interval of 100 msec and an H-V interval of 10 msec (Fig. 3). Atrial extrastimulus testing was performed at an atrial driven cycle length of 400 msec. When A1-A2 coupling intervals were decreased from 320 msec to 310 msec, there was a sudden prolongation of the A-V interval from 170 msec to 260 msec, and the His bundle activity was incorporated just after initiation of the QRS complexes (Fig. 4). The AV conduction curves thus

![Fig. 3. His bundle electrogram during sinus rhythm.](image)

![Fig. 4. Discontinuous AV conduction curve. When the coupling interval decreased from 320 msec to 310 msec A-V interval increased suddenly from 170 msec to 260 msec.](image)
became discontinuous, the characteristic feature of dual AV nodal pathways conduction. A coupling interval of 260 msec resulted in further prolongation of the A-H interval to 370 msec, and an atrial echo beat and sustained tachycardia with preexcited QRS complexes ensued (Fig. 5). Beat-to-beat changes in the degree of preexcitation were seen, but the changes were minimal. The His bundle activities appeared just after initiation of the QRS complexes.

Fig. 5. Induction of tachycardia by a single right atrial premature beat (coupling interval 260 msec) after 8 right atrial beats at a BCL of 400 msec. Following a sudden increase in A-H and A-V intervals, paroxysmal tachycardia was induced. His bundle activities appeared just after the initiation of QRS complexes. The QRS contours are preexcited and wide.

Fig. 6. Disappearance of delta wave during His bundle pacing (third and fourth beat).
Fig. 7. Atrial flutter and fibrillation. Rapid atrial pacing produced atrial flutter and fibrillation. At the beginning of this strip, the atrial electrograms are irregular and are thought to show atrial fibrillation. However, atrial electrograms gradually became more regular (atrial flutter). After a short duration of atrial flutter, sinus rhythm was re-established. The beat-to-beat change of the degree of preexcitation during atrial flutter and fibrillation was minimal.

complexes and the V-H interval was variable (Fig. 5). Mapping of the atrial activation sequence during tachycardia revealed the earliest atrial activation at the LSRA. Further shortening of the coupling interval resulted in atrial refractoriness. Disappearance of the delta wave and normalization of the QRS complexes were not observed. However, His bundle pacing resulted in disappearance of the delta wave (Fig. 6). After the intravenous administration of procainamide (500 mg), the delta wave also disappeared.

Rapid atrial pacing (250/min) resulted in atrial flutter and fibrillation. A beat-to-beat change in the degree of preexcitation was detected, but the change was minimal, and disappearance of the delta wave and normalization of the QRS complexes were not observed during atrial flutter and fibrillation (Fig. 7). The ventricular rate was below 140 beats/min, and the shortest R-R interval was more than 310 msec. After an atrial flutter of short duration the sinus rhythm was restored spontaneously (Fig. 7). Right ventricular pacing was performed during programmed ventricular extrastimuli at a basic cycle length of 800 msec. Progressively shorter coupling intervals produced prolongation of the retrograde conduction time.
DISCUSSION

In our patient, a progressive shortening of the A1-A2 interval produced a prolongation of the A-V interval and a small increase in preexcitation, and the His bundle activities became incorporated within the QRS complexes. Furthermore, His bundle pacing resulted in disappearance of the delta wave (Fig. 6). Disappearance of the delta wave was also seen after administration of procainamide (500 mg) intravenously.

Rapid atrial pacing resulted in paroxysmal atrial flutter and fibrillation (Fig. 7). In patients displaying atrial flutter and fibrillation in conjunction with Mahaim fiber conduction arising from the proximal His bundle, the ventricular rate during atrial flutter and fibrillation depends on the functional properties of the AV node. In particular, the QRS contour may be identical to the sinus rhythm, and beat-to-beat changes in the degree of preexcitation may not occur. On the other hand, in patients with Mahaim fiber conduction arising from the lower part of the AV node, the beat-to-beat changes in the degree of preexcitation will depend upon the conduction delay distal to the origin of Mahaim fibers. In the present case, beat-to-beat changes in the degree of preexcitation were seen but they were minimal. These data all suggest the existence of Mahaim fiber conduction arising from the lower part of the AV node (nodo-ventricular pathways).

On the other hand, programmed atrial stimulation induced discontinuous AV conduction curves (A1A2, H1H2). The H1H2 curves in response to A1A2 were discontinuous, due to a sudden increase in AV conduction time (A2H2) at a coupling interval of 310 msec (Fig. 4). This is a characteristic feature of conduction via dual AV nodal pathways, reflecting the failure of anterograde conduction over a fast AV nodal pathway, with resultant slow AV nodal pathway conduction. A coupling interval of 260 msec resulted in further prolongation of the A-H interval to 370 msec, and an atrial echo beat and sustained tachycardia with preexcited QRS complexes appeared. The His bundle activity was incorporated just after initiation of the QRS complexes (Fig. 5). During induced tachycardia, a normal retrograde activation sequence was recognized, with the LSRA being activated earlier than other atrial recording sites (Fig. 5). Progressive shortening of ventricular pacing (V1-V2) resulted in a gradual prolongation of the V-A interval, suggesting retrograde AV nodal conduction. These data indicate the existence of dual AV nodal pathways, Mahaim fibers, which originate from the lower part of the AV node, and reentrant tachycardia within the AV node.

The prevalence of dual AV pathways was about 10 to 25% in a large study of patients with conduction system disease, but the association of
Mahaim fiber conduction and AV nodal reentrance is particularly rare. In patients with Mahaim fiber conduction, participation of Mahaim fibers within a tachycardia circuit is difficult to prove.\(^{12,13}\)

We cannot exclude the possibility that the impulses were conducted via Mahaim fibers anterogradely and then conducted retrogradely via the His bundle and the AV node. Wellens et al reported a case of Mahaim fiber conduction associated with dual AV nodal pathways. They speculated that the reentrant circuit is composed of anterograde conduction through Mahaim fibers and retrograde conduction via the His-Purkinje system.\(^{14}\) In their case, the QRS complexes of the tachycardia were followed by a His bundle electrogram. In our case, though, the His bundle electrogram appeared just after initiation of the QRS complexes, and the V-H intervals during tachycardias were variable (Fig. 5). These results suggest that the Mahaim pathways and the His bundle did not participate in the reentrant circuit and that reentry was within the AV node in our case.

Ward et al\(^8\) reported 3 cases of nodo-ventricular pathway. In 2 patients, sustained tachycardia with preexcited QRS complexes were detected. The accessory pathway did not appear to be involved in the tachycardia circuit, and the site of reentry was postulated to be within the AV node. In common with the cases of Ward et al, the site of reentry in our case was thought to be within the AV node, with preexcited QRS complexes reflecting conduction in an anomalous nodo-ventricular pathway.

Bardy and co-workers described six characteristic ECG features during tachycardia in patients with nodo-ventricular fibers.\(^9\) These include a QRS axis between 0 and -75°, a QRS duration of 0.15 sec or less, an R wave in limb lead I, an rS in precordial lead V\(_1\), a transition in the precordial lead from a predominantly negative to a predominantly positive QRS complex after V\(_4\) or later, and a cycle length between 220 and 450 msec.\(^9\) Our case fulfilled all of these criteria, which are useful in distinguishing patients with nodo-ventricular fibers from those with other wide QRS tachycardia. In patients with such ECG features during a tachycardia attack, one should consider the existence of nodo-ventricular fibers.

**References**