Atrioventricular (AV) Nodal Reentry Associated with 2:1 Infra-His Conduction Block during Tachycardia in a Patient with AV Nodal Triple Pathways

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SUMMARY

We report a patient with atrioventricular (AV) nodal reentry in which a 2:1 infra-His conduction block was demonstrated during tachycardia. The electrocardiogram (ECG) at the time of attack showed two types of supraventricular tachycardias. The first type was a narrow QRS tachycardia associated with 1:1 AV conduction at a rate of 170 beats/minute. The second type was a narrow QRS associated with 2:1 AV block at a rate of 85 beats/minute. Electrophysiological study revealed AV nodal reentry based on AV nodal triple pathways. The AV conduction curve obtained by atrial premature stimulation showed two discontinuous points at two different basic cycle lengths (500 msec, 400 msec) and from two different pacing sites (high right atrium, distal coronary sinus). These two types of tachycardias were induced by both atrial premature and overdrive stimulation. In the first type, the impulse conducted in the slow pathway antegradely with 1:1 AV conduction and in the fast pathway retrogradely. In the second type, the impulse was conducted beat-to-beat by either a slow pathway or a very slow pathway antegradely with the retrograde limb being the fast pathway and 2:1 infra-His conduction block. Only when the impulse was conducted in the slow pathway antegradely was the infra-His conduction block observed during the tachycardia. The tachycardia in this patient was drug refractory and controlled by an anti-tachycardia pacemaker. (Jpn Heart J 35: 241–248, 1994)

Key words: Atrioventricular nodal reentry AV nodal triple pathways Infra-His block Anti-tachycardia pacemaker

SUPRAVENTRICULAR tachycardia (SVT) due to AV nodal reentry is the most common form of SVT, accounting for approximately 50% of the cases.1-4) The mechanism of this tachycardia is considered to be related to AV

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nodal dual pathways. AV nodal reentrant tachycardia is also seen in patients with AV nodal triple pathways.\textsuperscript{5,6} However, it is relatively rare in patients with AV nodal triple pathways to observe the antegrade impulse being conducted alternately from one pathway to another during tachycardia. We examined electrophysiologically a patient with AV nodal triple pathways who had AV nodal reentrant tachycardia and a 2:1 infra-His conduction block during the tachycardia.

**Case Report**

A 71-year-old woman reported the onset of intermittent palpitations about twenty years ago. Since the attacks subsided spontaneously in about 1 hour whenever they occurred, they were left untreated. The attacks had occurred at a frequency of once every 1 or 2 months thereafter, and she visited a local hospital

![Figure 1](image-url)  
**Figure 1.** Electrocardiogram during sinus rhythm at a rate of 86 beats/minute. PQ and QRS intervals were 0.12 sec and 0.11 sec, respectively.
Figure 2. Electrocardiograms during the tachycardia associated with 1:1 AV conduction (A) and with 2:1 AV conduction (B). (A) The tachycardia rate was 170 beats/minute. QRS morphology was almost the same as in sinus rhythm (Figure 1) and the P wave is seen as a slurring of the terminal QRS. (B) The ventricular rate was 85 beats/minute. The QRS morphology was almost the same as in sinus rhythm. The P was negative in leads II and III. Furthermore, the P wave was also seen in a slurring of the terminal QRS. The P rate was 170 beats/minute, suggesting supraventricular tachycardia associated with 2:1 AV block.

and was treated whenever an attack did not subside spontaneously. In 1992, the attacks began to occur once or twice a day. She was admitted to our hospital on February 7, 1992. The patient was 150 cm tall, weighed 50 kg, had a blood pressure of 122/56 mmHg and a regular heart rate of 86 beats/minute. Other physical findings were normal. On chest x ray, CTR was 49%, and the lung fields were clear.

Figure 1 shows an electrocardiogram (ECG) during sinus rhythm at a rate of 86 beats/minute. The PQ interval was 0.12 sec and the QRS duration was 0.11 sec.

Figure 2 shows ECGs during tachycardia. Two types of tachycardias (Panel A and Panel B) were observed in the patient. In Panel A, a narrow QRS tachycardia at a rate of 170 beats/minute is presented in which the P wave was
seen as a slurring of the terminal QRS. In Panel B, the ventricular rate was 85 beats/minute with a narrow QRS. The P wave was seen at a rate of 170 beats/minute and the morphology during tachycardia was negative in leads II and III.

**Electrophysiological study:** Informed consent was obtained from the patient. All medication was suspended at least 1 week before the examination. Three electrode catheters were inserted subcutaneously via the right femoral vein under radiographic guidance. One (6F 4-electrode catheter, Mansfield) was placed in the right ventricular apex (RVA), one (6F 4-electrode catheter, Mansfield) in the junction between the superior vena cava and the high right atrium (HRA), and the other (6F 2-electrode catheter, Mansfield) just below the tricuspid valve. Another catheter (7F 4-electrode catheter, Mansfield No. 5251) was advanced via the left subclavian vein to the coronary sinus (CS). In the catheters placed in the RVA and HRA, the 2 terminal electrodes were used for stimulation and the proximal 2 electrodes for recording, which was done at a paper speed of 100 mm/sec during monitoring on an oscilloscope. Stimulation was given by rectangular waves of a duration of 1.5 msec at an intensity of twice the diastolic threshold. During sinus rhythm, the AH interval was relatively short at 40 msec and the HV interval was normal at 50 msec.

The tachycardia was reproducibly induced by overdrive and extrastimuli from the HRA and the distal CS. The tachycardia was also interrupted with A-H block by atrial pacing. Figure 3 shows the AV conduction curve obtained by

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**Figure 3.** AV conduction curve obtained by atrial premature stimulation from the high right atrium (basic cycle length = 500 msec). The coupling interval of atrial premature stimulation (A1-A2) is plotted on the horizontal axis, and the resultant H1-H2 interval is plotted on the vertical axis. Note the jump in H1-H2 interval occurred at a coupling interval of 310 msec and also 280 msec. At coupling intervals from 280 to 240 msec (*), A-V nodal reentry was induced.
Figure 4. Electrocardiographic lead V1 recorded simultaneously with the intracardiac electrograms during tachycardia. Panel (A) showed the tachycardia associated with 1:1 AV conduction. Tachycardia cycle length was 350 msec. The earliest activation site of the atria was the low atrial septum, followed by activation of the HRA, proximal CS, and distal CS. The Ae-H interval during tachycardia was constant at 260 msec. Panel (B) shows the tachycardia associated with 2:1 infra-His conduction block. The atrial cycle length and atrial sequence during tachycardia were the same as in (A). The Ae-H interval changed alternately between 260 msec and 300 msec. The infra-His conduction block occurred when the Ae-H interval was at 260 msec. Abbreviations: HRA=high right atrium; CSp=proximal coronary sinus; CSd=distal coronary sinus; HBE=His bundle electrogram; RV=right ventricle; A(e)=atrial potential; H=His potential; V=ventricular potential. All numbers are in msec.
the tachycardia. When the basic cycle length was 400 msec, the AV conduction curve obtained by extrastimuli from the HRA showed a discontinuous conduction curve at two different coupling intervals as seen at the basic cycle length of 500 msec. Furthermore, the AV conduction curve obtained by extrastimuli from the distal CS showed the same result. These data indicate that the AV nodal triple pathways were present in this patient.

Figure 4 shows intracardiac electrograms obtained during tachycardia. The earliest site of atrial activation during tachycardia was the low atrial septum (His bundle recording site), followed by activation of the HRA, the proximal CS and the distal CS. Figure 4A shows the tachycardia associated with 1:1 AV conduction. The tachycardia cycle length was 350 msec. The Ae-H intervals and H-Ae intervals were 260 msec and 90 msec, respectively. The H-V intervals during tachycardia were constant at 50 msec. Although the tachycardia continued until it was interrupted by pacing, it was associated with 1:1 AV conduction that converted spontaneously into a tachycardia associated with 2:1 H-V block and vice versa. The premature ventricular stimulation during tachycardia when the His bundle was refractory did not capture the atrium. These data indicate that the ventricle was not involved in the maintenance of tachycardia. During the tachycardia associated with 2:1 infra-His conduction block, the ventricular cycle length was 700 msec. The Ae-H intervals were 300 msec when associated with infra-His conduction and 260 msec when associated with infra-His conduction block.

To determine the effects of antiarrhythmic agents on these tachycardias, we used verapamil, propranolol, and procainamide on different days. However, after intravenous administration of verapamil (10 mg), propranolol (8 mg), and procainamide (500 mg), the tachycardia was still inducible by premature stimulation, and was sustained until it was interrupted by pacing. During electrophysiological study, H-V block was not induced by atrial overdrive and extrastimulus pacing during sinus rhythm when the 1:1 A-H conduction was preserved. In this patient, an anti-tachycardia pacemaker was implanted.

**DISCUSSION**

Although the AV nodal triple pathways are relatively rare in patients with AV nodal reentrant tachycardia, the diagnosis for the AV nodal triple pathways need the following criteria: two discontinuous points on the AV conduction curve obtained by atrial premature stimulation are present at least at two different basic cycle lengths and also from at least two different pacing sites. In our present case, all of these criteria were involved. As for AV nodal reentry utilizing triple nodal pathways, however, there have been reports of only a few cases that
have demonstrated the participation of all of these pathways during AV nodal reentry. Dopirak et al\textsuperscript{7} described a woman with paroxysmal tachycardia in whom atrial extrastimulus testing demonstrated doubly discontinuous AV nodal conduction curves. Tachycardia was not induced in this patient during the electrophysiological study. Kühlkamp et al\textsuperscript{8} reported a patient with AV nodal reentrant tachycardia using three different AV nodal pathways. However, they could not show doubly discontinuous curves obtained by atrial extrastimulus. Recently, Sublett et al\textsuperscript{6} demonstrated a case with two distinct forms of AV nodal reentry that can utilize three nodal pathways.

In the present study, we demonstrated two types of tachycardias in the patient. The first tachycardia associated with 1:1 AV conduction utilized the slow pathway antegradely (Ae-H interval = 260 msec) and the fast pathway retrogradely. The second tachycardia associated with 2:1 infra-His conduction block utilized the very slow pathway antegradely (Ae-H interval = 300 msec) and the fast pathway retrogradely. The impulse conducted through the His-Purkinje system and the ECG showed 1:1 AV conduction. When the impulse conducted through the slow pathway antegradely (Ae-H interval = 260 msec) and the fast pathway retrogradely, 2:1 AV block occurred because the His-Purkinje system refractoriness showed AV block. Although the coupling interval (H1-H2) was the same as in the first type of tachycardias when the H-V block occurred, the preceding H-H interval in the second type of tachycardia (H-H interval = 300 msec) was longer when compared with the first type of tachycardia (H-H intervals = 260 msec). This condition is known as the Ashman phenomenon\textsuperscript{9} in atrial fibrillation and is also explained as Akhtar’s phenomenon in the His-Purkinje system.\textsuperscript{10} In addition, although conduction block during AV nodal reentry (i.e. bundle branch block) is also involved, it has been demonstrated to be present transiently for a short period of initiation of tachycardia. It is of interest that infra-His block during tachycardia in our patient was sustained until the tachycardia was interrupted by atrial pacing.

In order to prevent initiation of these tachycardias, we used three different drugs intravenously (verapamil, propranolol, and procainamide) during the electrophysiological studies. Two other drugs were also tested orally (disopyramide 300 mg/day, flecainide 150 mg/day) by Holter monitoring. None of these drugs were effective for prevention of the tachycardia. These tachycardias were completely controlled by the implantation of an anti-tachycardia pacemaker device.

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