Atrioventricular (AV) Reciprocating Tachycardia Utilizing Left Lateral AV Bypass Pathway with a Long Retrograde Conduction Time

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SUMMARY

A 52-year-old man had suffered from attacks of palpitation for 10 years. The frequency of the attacks had been increasing since June 1991. The electrocardiogram (ECG) at the time of attack showed a heart rate of 175 bpm and $RP'/P'R>1$, indicating long RP' tachycardia. Electrophysiological examination of the heart revealed an accessory pathway in the left lateral position. During the tachycardia, PVCs from the right ventricular apex (RVA) captured the atria. On the basis of these findings the patient was diagnosed as having had atrioventricular (AV) reciprocating tachycardia (AVRT). Ventriculoatrial (VA) conduction indicated a decremental conduction curve by single premature stimulation from the RVA, and the atrial cycle length following PVC during tachycardia was prolonged (paradoxical delay). When verapamil was administered intravenously, tachycardia induced by the premature stimulation showed prolongation of the VA interval, associated with an increased tachycardia cycle length. Tachycardia in this patient was completely controlled by administration of verapamil. (Jpn Heart J 34: 109–115, 1993.)

Key Words:
Atrioventricular reciprocating tachycardia Left lateral accessory pathway Verapamil Long retrograde conduction time

LONG RP' tachycardia is relatively rare. The mechanism of this tachycardia is considered to be related in some patients to AVRT utilizing an accessory pathway, and showing a long retrograde conduction time.1) Gallagher et al11-2) called the accessory conducting pathway the “accessory AV node” because it is always located in the posterior interatrial septum and has the property of decremental conduction. However, some re-
ports\textsuperscript{3,4) have shown that a similar accessory pathway that shows long retrograde conduction time is present between the left atrium and left ventricle. The pathway is therefore not necessarily an accessory AV node. We examined electrophysiologically a patient with long RP' tachycardia, and studied the effects of verapamil on atrioventricular reciprocating tachycardia (AVRT) in the patient, who had tachycardia utilizing the left lateral accessory pathway with a long retrograde conduction time.

**CASE REPORT**

A 52-year-old man reported the onset of intermittent palpitations about 10 years ago. Since the attacks subsided spontaneously in about 10 to 20 min whenever they occurred, they were left untreated. The attacks occurred at a frequency of once every 1 or 2 months thereafter, and he visited a local hospital and was treated whenever an attack did not subside spontaneously. In 1991, the attacks began to occur two or three times a day. He experienced syncope in June 1991. He was admitted to our hospital on July 30, 1991. The patient was 154 cm tall, weighed 55 kg, and had a blood pressure of 130/84 mmHg and a regular heart rate of 72 bpm. Other

![Electrocardiogram](image)

**Fig. 1.** Electrocardiogram during sinus rhythm (A) and tachycardia (B). (A) Normal sinus rhythm, normal axis deviation. (B) RP' interval of 0.17 sec and P'R interval of 0.16 sec, RP'/P'R was 1.06.
physical findings were normal. On chest x ray, CTR was 44%, and the lung fields were clear.

Figure 1 shows ECGs during sinus rhythm (A) and tachycardia (B). During tachycardia the P wave corresponded to the QRS at a 1:1 ratio at a P'R interval of 0.16 sec and RP' interval of 0.17 sec. The RP'/P'R ratio was 1.06. From these findings, this tachycardia was diagnosed as long RP' tachycardia.

Electrophysiological examination of the heart:

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 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. In

![ECG diagram](image)

Fig. 2. ECG leads I, II, and V1 recorded simultaneously with the intracardiac electrograms during tachycardia. Tachycardia cycle length was 350 msec. The earliest site of activation of the atria was the distal coronary sinus, followed by activation of the proximal coronary sinus, the low atrial septum, and high right atrium. Abbreviations: HRA=high right atrium; CSp=proximal coronary sinus; CSp=distal coronary sinus; HBE=His bundle electrogram; RV=right ventricle; A=atrial potential; H=His bundle potential; V=ventricular potential; AH=AH interval; HV=HV interval.
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 at an intensity twice the diastolic threshold and a duration of 1.5 msec. During sinus rhythm, the AH and HV intervals were normal, at 80 and 50 msec, respectively.

Tachycardia was induced with reproducibility by overdrive and extrastimuli from the HRA and extrastimuli from the RVA. Figure 2 shows an intracardiac electrogram obtained during tachycardia. The earliest site of activation recorded in the atria during the tachycardia was the distal coronary sinus (CSd), followed by activation of the proximal coronary sinus (CSp), the low atrial septum (His bundle recording site) and high right atrium. This finding excluded the fast-slow type of AV nodal reentrant tachycardia.

Figure 3 shows the recordings obtained during overdrive pacing from the RVA. Figure 3A shows the recording at a pacing cycle length (PCL) of 340 msec, and Fig. 3B shows it at a PCL of 310 msec. Although the on-

Fig. 3. ECG leads and intracardiac electrograms are shown as in Fig. 2. The recordings were obtained during overdrive pacing from the RV apex. Panel A shows the recording at a pacing cycle length (PCL) of 340 msec and panel B, at a PCL of 310 msec. Intraventricular conduction time (SA interval – VA interval) at a PCL of 340 msec was 110–120 msec, and that at a PCL of 310 msec was 150 msec. The prolongation of VA conduction time was not due to prolongation of intraventricular conduction time. Abbreviations are as in Fig. 2. S = stimulus artifact.
Fig. 4. ECG leads and intracardiac electrograms are shown as in Fig. 2 during right ventricular pacing at a cycle length of 300 msec. Repeated retrograde Wenckebach cycles are noted between the ventricular and atrial potentials recorded in the distal coronary sinus electrogram. All numbers are in msec. Abbreviations are as in Figs. 2 and 3.

set of the ventricular activation in the CSd was unclear, the VA interval in the CSp was 140–150 msec and 200 msec. The SA interval ranged from 250 to 270 msec at A, but 320 msec at B. In other words, the intraventricular conduction time in Fig. 3A is the result of deducting the VA interval from the SA interval, 110–120 msec. Hence, intraventricular conduction time at B was 120 msec, demonstrating that the prolongation of VA conduction was not due to prolongation of intraventricular conduction time. Figure 4 shows the recording obtained at a PCL of 300 msec during tachycardia. It indicates the conduction time from the pacing artifact S to the atrial potential recorded in the CSd. The SA interval was gradually prolonged, and VA block was observed at every 4th beat. That is, the accessory pathway in the left lateral free wall showed decremental conduction.

After intravenous administration of verapamil (10 mg), the tachycardia was still readily induced by premature stimulation, and was sustained until it was interrupted by pacing. However, the tachycardia cycle length was longer than that before verapamil administration, due to the prolongation of the retrograde ventriculoatrial conduction time (Fig. 5).
Fig. 5. ECG leads and intracardiac electrograms are shown as in Fig. 2 during atrioventricular reciprocating tachycardia induced after verapamil administration. The ventriculoatrial conduction time increased to 180 msec, compared with those before verapamil administration (Fig. 2), and was associated with an increase of tachycardia cycle length. Abbreviations are as in Fig. 2.

**DISCUSSION**

As mechanisms of long RP' tachycardia, fast-slow (uncommon type) AVNRT, permanent form of AV junctional reciprocating tachycardia (PJRT), ectopic atrial tachycardia, and AV reciprocating tachycardia utilizing the AV bypass pathway with a long retrograde conduction time are currently considered, based on ECG findings. In our present patient, fast-slow AVNRT and PJRT, which show an accessory pathway in the posterior interatrial septum, were excluded because the earliest site of activation was recorded in the atria during tachycardia. The PVC occurred during tachycardia when the His bundle was refractory, demonstrating the involvement of the ventricle in the maintenance of tachycardia. This excluded ectopic atrial tachycardia. In the present condition, the accessory pathway was present in the left lateral free wall, because the CSD was the earliest site of activation during tachycardia. The ventriculoatrial conduction seen during RVA pacing showed a Wenckebach cycle and demonstrated decremental conduction such as that seen in the AV node. The relative sequence of retrograde atrial activation by the conducted impulse was the same as that observed during the tachycardia. During the tachycardia, the atrial cycle
length following a PVC was increased, probably because the impulse was conducted more slowly over the AV bypass pathway following a PVC, leading to an increase in the cycle length of tachycardia. A similar phenomenon has been reported by Bardy et al., and they have called it "paradoxical delay".

In order to study the characteristics of the accessory AV pathway, verapamil was administered intravenously to the patient. Verapamil suppressed retrograde ventriculoatrial conduction utilizing the accessory pathway and tachycardia cycle length was prolonged. This indicates involvement of the Ca channel in slow conduction utilizing the accessory pathway. As a result, slow conduction and decremental conduction might have been present.

REFERENCES