Double Atrial Response to a Single Ventricular Extrastimulus in a Patient with Wolff-Parkinson-White Syndrome

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SUMMARY

Electrophysiological examination in a 39-year-old male disclosed an accessory pathway between the right atrium and the right ventricle and AV nodal dual pathways. Atrial and ventricular extrastimuli induced paroxysmal supraventricular tachycardia (PSVT), which was shown to be AV reciprocating tachycardia. Double atrial response was noted during ventricular extrastimuli at V1V2 of 280 msec and V1V2 of 250 msec. The first atrial response is considered to have been transmitted in a retrograde fashion in the accessory pathway, and the second atrial response similarly in the slow pathway of the AV node.

Key Words: Double atrial response WPW syndrome AV nodal dual pathways

A rare phenomenon of double ventricular responses, in which the ventricle is separately excited twice by a single atrial extrastimulus, has been reported in WPW syndrome. There is also an even rarer phenomenon in which the atrium is excited separately twice by a single ventricular extrastimulus. In a patient with PSVT, we demonstrated a right-sided AV bypass tract and AV nodal dual pathways by electrophysiological examinations. PSVT was shown to be AV reciprocating tachycardia due to antegrade conduction in the AV node and retrograde conduction in an accessory pathway. In addition, double atrial responses were elicited by a single ventricular extrastimulus in this case.

CASE REPORT

A 39-year-old male began to note attacks of palpitation of 5–10 min duration 2–3 times a year since the age of about 15 years. The condition
was not medically examined because the attacks disappeared spontaneously and were of short duration. In early July, 1989, he experienced palpitation with dizziness and unsteadiness during office work and consulted a local doctor. PSVT was demonstrated by 24-hour ambulatory electrocardiogram, and the patient was admitted to our hospital for detailed evaluation and treatment.

The patient was 177 cm tall, weighed 81 kg, and showed a blood pressure of 158/92 mmHg and a regular heart rate of 72 bpm. Other physical findings were normal. Electrocardiogram showed normal sinus rhythm at a rate of 73 bpm and normal axis. On chest X-ray, CTR was 44%, and the lung fields were clear.

**Electrophysiological examination of the heart**

All medication had been suspended from 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 remaining (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 the catheters placed in the RVA and the HRA, the 2 terminal electrodes were used for stimulation and the proximal 2 electrodes for recording, which was made at a paper speed of 100 mm/sec during monitoring on an oscilloscope. Stimulation was given by rectangular waves with an intensity twice the diastolic threshold and a duration of 1.5 msec. During sinus rhythm, the AH interval and HV interval were normal at 80 and 40 msec, respectively. During extrastimuli to the HRA at a basic cycle length of 600 msec, H₁H₂ remained 360 msec with a smooth curve until A₁A₂ became 270 msec, but it showed a sudden jump to 410 msec at A₁A₂ of 260 msec, indicating the presence of AV nodal dual pathways. When extra stimulation was given to the RVA at a basic cycle length of 450 msec, the VA conduction time remained constant at 80 msec, and the atrial sequence was the low right atrium, HRA, and left atrium, indicating the presence of an accessory pathway between the right atrium and the right ventricle. The effective refractory period was 220 msec in the atrium, 270 and 260 msec in antegrade conduction in the fast and slow pathways of the AV node, 240 msec in the ventricle, and 270 msec in the retrograde accessory pathway.

Next, when extra stimulation was given to the HRA or the RVA at a basic cycle length of 450 msec, PSVT was induced at A₁A₂ of 260–220 msec.
or at V1V2 of 260–230 msec (Fig. 1). Retrograde atrial preexcitation after a VPC introduced during reciprocating tachycardia when the His bundle is refractory to retrograde conduction was recognized. From these findings, a diagnosis of AV reciprocating tachycardia with antegrade conduction in the fast pathway of the AV node and retrograde conduction in the accessory pathway was made.

When extra stimulation was given to the RVA at a basic cycle length of 450 msec, a double atrial response was observed at S1S2 of 280 msec (Fig. 2) and 250 msec. The initial A wave of the double atrial response showed a VA interval of 80 msec (V-Ae), and atrial sequence at this time was the low right atrium, HRA, and left atrium, indicating retrograde conduction in the accessory pathway. The second A wave showed a prolonged VA interval of 380 msec (V-Ae'), and the atrial sequence was low right atrium, proximal coronary sinus, distal coronary sinus, and HRA, indicating retrograde conduction in the AV node.

DISCUSSION

There are some reports concerning double ventricular response to a single atrial extrastimulus in WPW syndrome.1) These are considered to be evidence that WPW syndrome is caused by fusion beats of the ventricle
Fig. 2. Double atrial responses to ventricular extra stimulation. Double atrial response is not observed at S1-S2 of 290 msec (upper panel) but observed at S1-S2 of 280 msec (lower panel). Retrograde conduction of S2 proceeds over both the accessory pathway (Ae) and slow pathway (Ae'). Ae' is conduction down the AV node (fast pathway) and reentry of the atria via the accessory pathway (Ae). Abbreviations are as in Fig. 1.

caused by conduction via the A-V route and an accessory pathway. As for double atrial response to a single ventricular extrastimulus, however, there have been reports of only 2 cases by Lin et al.2) One of these cases had WPW syndrome and showed a double atrial response after verapamil administration; double atrial response was caused by AV nodal dual pathways in the other. In our case, on the other hand, a right-sided accessory pathway and AV nodal dual pathways were demonstrated simultaneously.

Conditions for the occurrence of double atrial responses are as follows: (1) At least two pathways, one fast, the other slow, must be present in VA conduction. (2) After the first atrial response via the fast VA pathway occurs, the refractory period of the atrium must have ended before the second atrial response via the slow VA pathway occurs. Therefore, the conduction in the slow pathway must be delayed sufficiently. (3) There must be ante-
grade block of the first atrial response somewhere in the route of AV conduction. If not, the second activity that was transmitted by the slow VA pathway would end up in collision, and the second atrial response would be produced. In our case, the presence of the accessory pathway and the AV nodal dual pathways was consistent with the first condition. Next, the time difference (300=380–80 msec) between the first and second atrial responses, which far exceeded the atrial refractory period of 220 msec, fulfills the second condition, providing a sufficient delay of conduction for the atrium to emerge from the refractory period. As for the third condition, the site of the block could not be identified, because retrograde His bundle activities were not observed. However, blocking may have occurred in the upper common pathway of the AV node, and the retrograde conduction may have been mediated by the slow pathway, because the first pathway was still in the refractory period.

References