Ventriculatrial Block During Atrioventricular Nodal Reentrant Tachycardia Suggesting Existence of an Upper Common Pathway

Sedat Kose,1 MD, Basri Amasyali,1 MD, Kudret Aytemir,1 MD, Ayhan Kilic,1 MD, Atila Iyiso,1 MD, Turgay Celik,1 MD, Hurkan Kursaklioglu,1 MD, and Ersoy Isik,1 MD

SUMMARY

Studies on the mechanisms of atrioventricular nodal reentrant tachycardia (AVNRT) have yet to clarify whether the slow and fast pathways connect directly with the atria or via an upper common pathway. Although a “final common pathway” connecting the slow and fast pathways to the proximal His bundle was thought to be part of the reentrant circuit, debate on the presence of an upper common pathway continues. We report a case of AVNRT continuing despite the occurrence of ventriculoatrial block, thus supporting the existence of an upper common pathway. (Int Heart J 2005; 46: 333-338)

Key words: Atrioventricular nodal reentrant tachycardia, Ventriculoatrial block, Upper common pathway

Since the original description of atrioventricular nodal reentrant tachycardia (AVNRT), controversy continues with respect to whether the slow and fast pathways connect directly with the atria or via an upper common pathway.1) After defining the concept of dual atrioventricular (AV) nodal conduction, although a “final common pathway” connecting the slow and fast pathways to the proximal His bundle was thought to be part of the reentrant circuit, it was suggested that atrial myocardium provides the necessary link between the dual pathways to complete the reentrant circuit.2) We report a case of AVNRT continuing despite the occurrence of ventriculoatrial (VA) block, supporting the existence of an upper common pathway.

From the 1Department of Cardiology, Gulhane Military Medical Academy, Ankara, Turkey.
Address for correspondence: Basri Amasyali, MD, Department of Cardiology, Gulhane Military Medical Academy, 06018, Etilk, Ankara, Turkey.
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CASE REPORT

A 22-year-old man was referred to our hospital for ablation of recurrent narrow QRS tachycardia consistent with the common type of AVNRT refractory to multiple antiarrhythmic drugs, including oral verapamil, metoprolol, and propafenone (Figure 1). The results of a physical examination, resting ECG, chest X-rays, and echocardiography were all normal.

After obtaining informed consent, the electrophysiological study was performed in the fasting state, with the patient free of antiarrhythmic drugs. Three multipolar catheters were positioned at the high right atrium, His bundle region, and coronary sinus. Normal baseline AV conduction and concentric decremental VA conduction were present and there was no sign of preexcitation during sinus rhythm, right atrial pacing, or coronary sinus pacing. During ventricular pacing, decremental VA conduction started at a cycle length of 520 msec and there was no sign of dual retrograde pathway physiology. Programmed atrial stimulation revealed dual AV nodal physiology with an AH jump of 75 msec followed by reproducible induction of a narrow complex regular tachycardia with a cycle length of 394 msec and an earliest retrograde atrial activation recorded at the His bundle site. The VA interval, recorded at the high right atrial catheter, was 97 msec (Figure 2). During tachycardia, it was noticed that the VA conduction was blocked transiently, without interrupting the tachycardia (Figure 3). The presence of VA block during ongoing tachycardia was interpreted as the atrium not being required for tachycardia maintenance, and therefore, atrial tachycardia and ortho-

**Figure 1.** Twelve-lead surface ECG consistent with AVNRT. As a consequence of retrograde atrial activation, leads II and aVF show a ‘pseudo S’ pattern (arrow) and V1 records a ‘pseudo R’ (arrow).
Figure 2. Slow-fast AVNRT with a cycle length of 394 ms induced by programmed atrial stimulation. The HV interval is positive (44 ms); the VA interval is 97 ms as measured from the hRA electrode and the earliest retrograde atrial activity is in the His region (arrow).

hRA = high right atrium; CS = coronary sinus; ds = distal; paper speed 100 mm/s.

Figure 3. AVNRT continues although the fourth tachycardia complex fails to conduct to the atrium (arrow). The HV interval and the tachycardia cycle length did not change. Note also the absence of “pseudo S” in leads II, III, and aVF when the fourth tachycardia complex fails to conduct retrogradely to the atrium.

hRA = high right atrium; ds = distal; paper speed 100 mm/s.
dromic AV reciprocating tachycardia were ruled out. Thus, differential diagnosis was to be made among those tachycardias originating from the AV node or His bundle, including automatic junctional tachycardia, intra-Hisian reentrant tachycardia, reentrant tachycardia using a concealed nodoventricular or nodofascicular pathway, and AVNRT. Premature ventricular extrastimuli delivered during His bundle refractoriness did not preexcite the atrium and failed to terminate the tachycardia, thus ruling out reentrant tachycardias using a concealed nodoventricular or nodofascicular pathway. Intra-Hisian reentry was another possibility, but the tachycardia could be reproducibly induced with a critical AH interval after an AH jump but not an HV delay, which ruled out intra-Hisian reentry as the tachycardia mechanism. Automatic junctional tachycardia may also show VA conduction block but typically it cannot be initiated with a critical AH delay and its onset is usually spontaneous or after catecholamine administration.3)

After a diagnosis of typical AVNRT was made, radiofrequency ablation of the slow pathway in a typical posteroseptal location was accomplished using an ablation catheter with a 4-mm tip electrode (Marinr, Medtronic Inc., Minneapolis, MN). Runs of junctional beats were observed during radiofrequency energy application. AVNRT could not be reinduced, even after intravenous administration of atropine 1 mg, following 3 applications of radiofrequency energy. The patient has been asymptomatic without any antiarrhythmic therapy during a follow-up period of 7 months.

**DISCUSSION**

Since the original description of AVNRT by Mines, controversy continues as to whether the slow and fast pathways connect directly with the atria or via an upper common pathway.1) After defining the concept of dual AV nodal conduction, it has been suggested that the atrial myocardium provides the necessary link between the dual pathways to complete the reentrant circuit.2) Since then, some studies have demonstrated that AV nodal reentry is confined to the AV node4-13) while others support the concept that the atrial myocardium is essential for AV node reentry to occur.14-17) We report a patient with AVNRT continuing despite the occurrence of VA block, which suggests that an upper common pathway exists and that the atrial myocardium is not part of the reentrant circuit.

The concept of dual AV nodal conduction was first introduced by Moe and associates in their experimental work in dog and rabbit hearts.2) Although they suggested that a “final common pathway” connecting the slow and fast pathways to the proximal His bundle is part of the reentrant circuit, they thought that no upper common pathway existed between AV nodal pathways in the atria and that
atrial myocardium provided the necessary link between the dual pathways to complete the reentrant circuit.

Several case studies have demonstrated in different ways that the atria are not an essential part of the reentrant circuit in AVNRT. Guo, et al in their case report demonstrated irregular atrial activation during AVNRT while HH and VV intervals remained constant and suggested that conduction variation through the perinodal transitional cell envelope and intra-atrium, which are out of the AV nodal reentrant circuit, could be responsible for that irregular atrial activation pattern.4) Chen, et al reported atrial fibrillation developing in both atria during continuing AVNRT, which is also evidence that the atrium is not necessary in AVNRT.5)

VA block during AVNRT was first described by Wellens, et al in one of their 67 cases with AVNRT.6) Since then, several case studies have addressed AVNRT continuing despite the occurrence of VA block of different degrees, including intermittent complete AV dissociation,7,8) second degree 2:1 retrograde block,7,9,10) second degree type I block (Wenckebach),9-11) and as in our case transient retrograde block.8) These data suggest the existence of an upper common pathway. In our case, we observed transient VA block during AVNRT, which is also evidence suggesting that the atrium is not an essential part of the reentrant circuit in AVNRT, although participation of a small rim of low atrial tissue cannot be completely excluded by our findings and those in the other case reports mentioned above. Also, several authors, based on studies in both human subjects and experimental animal models, insist on the validity of the original concept developed by Moe and associates which stated that the anterograde and retrograde limbs of the AV nodal reentrant circuit connect directly to the atrium and that the atrium is an integral component necessary to complete the tachycardia circuit.14-17) These authors, however, have not made any comments on the implications of VA block observed in patients with AVNRT. In our opinion, the mechanism of VA block may have been the refractoriness of the perinodal atrial tissue (connection between the upper common pathway and the atrial tissue) as the electrical waveform arrives at this region. However, there remains the possibility of anatomic or functional variations causing AVNRT via different reentry circuits despite ventriculoatrial block; these variations could also be responsible for the failure of RF ablation despite multiple attempts both from the right and the left side of the atrial septum in a case reported recently.9)

REFERENCES