Atrioventricular Node Re-entry of the Common Variety*

Unusual Location for the Fast Pathway
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Radiofrequency ablation was attempted in a 17-year-old man with atrioventricular node re-entry of the common variety. Energy was delivered to three sites around the ostium of the coronary sinus. The third attempt resulted in a sudden increase in AH interval with loss of retrograde conduction. The arrhythmia was no longer inducible. These observations may suggest the unusual location of the fast pathway. (Chest 1994; 105:1669-70)

With the advent of radiofrequency ablation for atrioventricular (AV) node re-entry tachycardia, therapy for this arrhythmia has been dramatically changed.1,2 By creating only a small area of tissue injury, this procedure has allowed a person to speculate on the mechanism underlying this tachycardia. With this approach, the fast pathway is generally ablated near the AV node-His area,1 whereas the slow pathway is located around the orifice of the coronary sinus.1,2 Therefore, these pathways appear to be located at fairly specific sites. We describe a case in which the fast pathway was unusually located to the site of the slow pathway.

Case Report
A 17-year-old man was referred for evaluation of symptomatic paroxysmal supraventricular tachycardia. There was no history to suggest organic heart disease. Physical examination, chest radiography, and echocardiography were normal. A 12-lead electrocardiogram during sinus rhythm was normal. An electrophysiologic study was performed with the patient in the postabsorptive state under mild sedation after informed consent had been obtained. The study was performed in the absence of antiarrhythmic drug therapy. The techniques for electrophysiologic study of patients with supraventricular tachycardia have been described previously.3 In essence, multipolar electrode catheters were introduced percutaneously and were positioned in the high right atrium, coronary sinus, right ventricular apex, and across the tricuspid valve to record the His bundle potential. Intracardiac electrocardiograms and multiple surface electrocardiographic leads were recorded at paper speeds of 100 to 250 mm/s. The heart was stimulated at twice diastolic threshold current with a pulse width of 2 ms. During atrial extrastimulus testing, anterograde atrioventricular (AV) nodal curves were discontinuous, suggesting the presence of dual AV nodal pathway physiology. At a pacing cycle length of 600 ms, the effective refractory period (ERP) of the fast pathway was 390 ms; and that of the slow pathway was 290 ms (Fig 1). During atrial straight pacing, 1:1 ratio AV conduction occurred to a pacing cycle length of 320 ms. Retrograde discontinuity in AV nodal curve was not demonstrable. During ventricular straight pacing, 1:1 ratio ventriculoatrial (VA) conduction occurred to a pacing cycle length of 380 ms. A sustained narrow QRS complex tachycardia with a cycle length of 280 ms was reproducibly induced by atrial extrastimuli. The mechanism of this tachycardia was AV node re-entry of the common variety by the following criteria: (1) initiation of the arrhythmia only after the ERP of the fast pathway had been

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FIGURE 1. AV nodal conduction curves before and after radio-frequency ablation. Dual AV nodal pathways were present before ablation (open circles). The AV nodal re-entry (open triangles) was reproducibly inducible after the ERP of the fast pathway had been reached. After ablation (solid circles), fast pathway conduction has been abolished and the ERP is determined by the slow pathway. The AV node re-entry is no longer inducible. A1A2=atrial deflection of His’ bundle electrocardiogram; AV=atrioventricular; ERP=effective refractory period; A2H2=AH interval for atrial extrastimu-

 reached; (2) central atrial activation sequence with the earliest atrial activation recorded in the His bundle electrogram; (3) retrograde VA conduction time too short for re-entry using an accessory AV connection (VA conduction time in the His’ bundle electrogram was 0 ms); and (4) failure to advance atrial activation during His refractoriness with ventricular extrastimuli. Radiofrequency ablation was attempted after the mechanism had been identified. The high right atrial catheter was replaced by a steerable catheter with a 4-mm tip electrode for ablation. A bipolar electrogram with a frequency response of 50 to 500 Hz was recorded from the distal pair of the ablation catheter. Radiofrequency energy was delivered between the 4-mm tip electrode and a back plate. Slow pathway ablation was attempted by placing the catheter near the orifice of the coronary sinus (Fig 2). After two unsuccessful attempts, the final burn created a sudden increase in AH interval (Fig 3). The atrial electrogram was sharp, and no pathway-type potential was recorded at that site. No junctional rhythm was induced in energy application. At this point, stimulation protocol was repeated. In atrial extrastimulus testing, dual AV nodal pathway was no longer present (Fig 1). Ventricular pacing showed no evidence of VA conduction. Neither tachycardia nor echo beats due to AV node re-entry could be induced even under the influence of isoproterenol. During a 5-month follow-up, the patient continued to show a prolonged P-R interval with no evidence of clinical tachycardia.

Discussion
It is generally accepted that AV node re-entry needs dual pathways.4 Classically, the entire re-entrant circuit was thought to be confined to the AV node with the upper and lower common pathways.4 Recent surgical cures of this arrhythmia, however, suggest that perinodal tissue may be critically involved in the circuit, and the upper common pathway may not be present.4,6 Furthermore, the advent of radiofrequency catheter ablation has resulted in selective ablation of these pathways.1,2 Generally, the fast pathway is ablated in the anterior and superior aspect of the tricuspid annulus where the His bundle potential is barely recorded.1 On the other hand, the slow pathway is located near the orifice of the coronary sinus.2 Therefore, these pathways appear to be site-specific.

Our case is unique in that the fast pathway was ablated.
near the orifice of the coronary sinus, ie, usual location for the slow pathway. Jackman et al\(^2\) described a patient with AV node re-entry in whom the fast pathway was located to the anterior wall of the proximal coronary sinus. No extra-potential was recorded at that site. It can be argued that the ablation catheter might have moved inadvertently toward the superior-anterior position during ablation in our case. Careful analysis of cine films in energy delivery, however, showed no convincing evidence of catheter tip displacement. It is also conceivable that aberrant location of the coronary sinus might have made the usual fast pathway location look unusual. The catheter was placed just outside the orifice of the coronary sinus (Fig 3). Thus, even if the location of the coronary sinus is unusually high, the catheter position appears very unusual for the fast pathway ablation.

Jackman et al\(^2\) showed that extrapotentials could be recorded around the sites where the slow pathway was ablated. In their study, potential-guided ablation did not induce fast pathway ablation or AV block. On the other hand,

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**REFERENCES**


**FIGURE 2.** Radiograph of ablation catheter position during ablation of the fast pathway. Right anterior oblique projection is shown in top panel and left anterior oblique projection is shown in bottom panel. The ablation catheter with a large tip electrode is positioned just outside the orifice of the coronary sinus.

**FIGURE 3.** Basic intervals during sinus rhythm before (top panel) and after (bottom panel) radiofrequency ablation. There is a dramatic increase in AH interval postablation. Recordings from top to bottom are Surface ECG lead V1, His bundle (HB) electrogram, A= Atrial electrogram; H= His deflection; V= Ventricular electrogram.

No such potentials have been reported for fast pathway ablation.\(^1\) During our study, we specifically searched for these extrapotentials without success. Actually, the site where the fast pathway was ablated recorded sharp atrial and ventricular electrograms without extrapotentials. It is not certain whether these potentials are directly associated with specific pathways or the fast pathway is different from the slow pathway in regard to its electrophysiology and anatomy. Furthermore, ablation guided by recording extrapotentials may be more advantageous than direct anatomical approach if a person considers slow pathway ablation. Finally, careful mapping of the fast pathway in tachycardia may be needed to avoid the destruction when the potentials cannot be recorded in sinus rhythm.