COMPLEX CASE STUDY

The Use of Atrial Pacing to Distinguish Atrioventricular Nodal Re-entry Tachycardia from Junctional Tachycardia

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ABSTRACT. Following slow pathway modification for typical slow–fast atrioventricular nodal re-entrant tachycardia (AVNRT) in a 20-year-old man, two narrow complex tachycardias with similar cycle lengths and atrial activation patterns were induced. Herein, we describe the value of responses to appropriately timed atrial extrastimulation to differentiate typical slow–fast AV nodal re-entry from a junctional tachycardia.

KEYWORDS. supraventricular tachycardia, atrioventricular nodal re-entrant tachycardia, junctional tachycardia.

Case report

A 20-year-old man with a history of palpitations presented to the electrophysiology (EP) laboratory for EP study and ablation. The EP test was performed in the fasting state, and the patient’s written informed consent was obtained before sedation. Quadripolar electrode catheters were inserted into the femoral vein and positioned in the right ventricular (RV) apex and the anteroseptal tricuspid valve (His-bundle recording). A 20-pole catheter was inserted into the femoral vein and positioned distally in the coronary sinus and proximally in the high right atrium. All 12 electrocardiographic leads and intracardiac electrograms were recorded and stored on a digital recording system (Prucka Engineering, Inc., Houston, TX). Bipolar intracardiac electrograms were filtered between 30 and 500 kHz and recorded from the proximal electrode pair at a speed of 100–200 mm/s. Bipolar pacing was performed at twice the diastolic threshold from the distal electrode pair by using a programmable stimulator. The onset of burst pacing was timed to begin based on sensing from the RV catheter.

At baseline, there was concentric decremental ventriculo-atrial (VA) conduction to 600 ms. With isoproterenol, VA conduction improved and was present to 400 ms. Programmed atrial stimulation induced an abrupt increase in the A–H interval and a narrow complex tachycardia with a cycle length of 400 ms and a septal VA time of 0 ms. Pacing in the right ventricle accelerated the atrial cycle length to the pacing cycle length and resulted in a ventricular–atrial–ventricular (V–A–V) response suggestive of typical slow–fast atrioventricular nodal re-entrant tachycardia (AVNRT). Radiofrequency (RF) ablation was delivered to the low septum in the region of the slow pathway. RF ablation was associated with junctional rhythm with 1:1 ventricular and atrial responses.

Following RF ablation, a narrow complex tachycardia was still inducible with a cycle length of 470 ms and septal VA time of 0 ms. To differentiate AVNRT from junctional tachycardia (JT), single atrial extrastimuli were delivered. An atrial extrastimulus delivered immediately prior to the His depolarization had no effect on the immediate QRS but did advance the subsequent His and QRS (Figure 1). An earlier atrial extrastimulus terminated the tachycardia without affecting the immediate H–H or V–V intervals (Figure 2). This suggested termination in the anterograde limb of the AVNRT circuit (slow pathway). Further RF was performed in the slow pathway region, during which junctional rhythm with 1:1 ventricular:atrial response was observed.

Following the second ablation, tachycardia remained inducible with a cycle length of 480 ms and septal VA time of 0 ms. Single atrial extrastimuli in close proximity to the His deflection had no effect on the tachycardia. However, an earlier extrastimulus advanced the immediate His and QRS without terminating the tachycardia.
Figure 1: Programmed single atrial extrastimuli has no effect on the immediate His/QRS but advances subsequent His/QRS. H indicates His, A2 is atrial extrastimuli.

Figure 2: Programmed single atrial extrastimuli terminates the tachycardia. H indicates His, A2 is atrial extrastimuli.

Figure 3: Programmed single atrial extrastimuli advances the immediate His/QRS and tachycardia resumes. H indicates His, A2 is atrial extrastimuli.
This suggested JT as the etiology, and no further slow pathway ablation was performed (Figure 3).

Discussion

JT is an uncommon tachycardia seen mostly in pediatric and postoperative patients.1–3 It can also be seen in patients with AVNRT after slow pathway ablation.4 In our patient, differentiating JT from AVNRT was critical since AVNRT required further ablation in the slow pathway region, whereas JT is unlikely to be of clinical significance. JT is often difficult to distinguish from AVNRT,4,5 especially when patients are given isoproterenol to facilitate arrhythmia induction, as in this case. JT is an automatic focal tachycardia originating from the region of the compact AV node whereas AVNRT is a re-entrant circuit involving the slow pathway and fast pathway inputs to the AV node.1,6 Atrial extrastimuli can be used to distinguish the rhythms by exploiting this mechanistic difference.4

In our patient, following ablation of the initial typical slow–fast AVNRT, tachycardia was still inducible. This could have either been JT or a slower AVNRT, suggesting modification but not elimination of the slow pathway. An atrial stimulus delivered immediately prior to the His electrogram did not affect the immediate His deflection or QRS. However, the premature atrial stimulus conducted anterograde in the slow pathway and thus advanced the subsequent His/QRS without terminating the tachycardia. This proved the presence of a slow pathway but not necessarily its participation in the tachycardia. The ability of the earlier atrial stimulus to terminate tachycardia confirmed participation of the slow pathway in the tachycardia.

The third induction resulted in tachycardia with a similar cycle length and activation pattern. Atrial extrastimuli advanced the immediate His/QRS with no effect on the subsequent His/QRS. This indicates that slow pathway function was eliminated with ablation since premature atrial stimuli now affect only the anterograde fast pathway conduction. The differential diagnosis for this response also includes the rare possibility of a 2:1 atrial:ventricular response to the atrial extrastimulus. This is unlikely for two reasons: 1) H–H interval following advancement of the His was fixed and similar to the tachycardia cycle length regardless of prematurity of A2 (10 ms decrement in A2 from 330 to 240 ms); 2) In our patient, a 2:1 atrial:ventricular response was never observed during atrial extrastimulation in sinus rhythm. The case described illustrates a common clinical scenario where an automatic JT with atrial activation similar to typical AVNRT can be provoked on isoproterenol following slow pathway modification in the same patient. In our patient, these arrhythmias had similar tachycardia cycle lengths and atrial activation, and the use of atrial extrastimulation helped differentiate between the two possible mechanisms and thus avoided unnecessary further ablations.

References