SUPRAVENTRICULAR TACHYCARDIA

CLINICAL DECISION-MAKING

Ablation of Atrioventricular Nodal Reentrant Tachycardia: Multiple Pathways, Multiple Mechanisms

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ABSTRACT. A middle-aged female triathlete with recurrent, symptomatic paroxysmal supraventricular tachycardia underwent electrophysiology study and was found to have multiple forms of atrioventricular nodal reentry. Here, we describe the types of reentry circuits and the approach to ablation.

KEYWORDS. Atrioventricular nodal reentry, intracardiac electrophysiology, radiofrequency ablation, supraventricular tachycardias.

Case presentation

A 38-year-old female triathlete underwent electrophysiology study for highly symptomatic, supraventricular tachycardia (SVT), initiated with physical activity, limiting her ability to compete. SVT stopped with vagal maneuvers (occasionally) or reproducibly with adenosine or verapamil. A 12-lead electrocardiogram showed narrow-complex tachycardia with RR intervals of 400 ms, inverted P waves in the inferior leads and upright P waves in the right precordial leads (Figure 1a).

During ventricular pacing, retrograde conduction was concentric and slowed progressively without “jumps”. During atrial pacing, the antegrade AV node function curve showed progressively slowed conduction followed by a “jump”. Single premature atrial extrastimuli initiated dramatic AH interval prolongation and narrow complex tachycardia (cycle length (CL) 400 ms, ventriculoatrial (VA) interval 70 ms) with concentric atrial activation and earliest atrial electrograms in a proximal coronary sinus (CS) channel (CS 7,8). Stable position of the CS catheter and CS 9,10 electrodes position in the CS ostium was confirmed by fluoroscopy. The differential diagnosis for tachycardia included 1) typical slow-fast AV nodal reentrant tachycardia (AVNRT), 2) orthodromic AV reentrant tachycardia (AVRT) using a posteroseptal accessory pathway, 3) septal AT, and 4) AV junctional automatic tachycardia. Ventricular pacing during SVT failed to show entrainment as it terminated repeatedly. His-refractory premature ventricular stimuli from the basal right ventricular septum could not advance atrial activation during SVT, making an accessory pathway and AVRT less likely. Premature beats from the CS or the high right atrium advanced the next V and reset SVT without an additional A, consistent with reentry. This sign and induction with programmed pacing excluded automatic junctional tachycardia. The VA interval in the advanced beat remained constant, indicating that atrial activation depended upon AV nodal conduction excluding the possibility of atrial tachycardia (AT) (Figure 1b). Slow–fast AVNRT was suspected and radiofrequency (RF) lesions applied to the right atrial posterior septum (slow pathway region) initiated an accelerated junctional rhythm (CL 600 ms, VA, 30 ms, Figure 1c). Repeat programmed atrial pacing with and without isoproterenol infusion induced multiple SVTs with different CLs, VA intervals and regularity (Figures 1d, 2a,b). What is going on?
After the first several RF lesions were applied, delivery of atrial extrastimuli induced SVT that looked similar, but faster than the first SVT (CL 310–370 ms, VA 60 ms); it was irregular. Changes in HH intervals preceded, and predicted, changes in AA intervals, suggesting alternation of antegrade conduction (Figure 1d). Tachycardia initiation with atrial extrastimuli excluded an automatic mechanism. The single most likely mechanism for this tachycardia was AVNRT. RF lesions were applied to the posteroseptal region and then up to triple atrial extrastimuli and burst atrial pacing failed to induce SVT. Isoproterenol was next infused and a third SVT was then induced with triple atrial extrastimuli. This SVT was slow and irregular (CL 520–580 ms, VA 50 ms). The HH intervals predicted the AA intervals and the earliest atrial activation moved to the mid-CS (CS 5,6) (Figure 2a). This apparent slow–fast AVNRT became non-inducible after additional RF applications in the slow AV nodal pathway region. Then, a fourth SVT initiated spontaneously with no isoproterenol infusion. This tachycardia was irregular (CL 330–480 ms, VA 30 ms) and showed the earliest atrial activation at CS 7,8 (the left atrial septum) is induced by programmed atrial pacing with triple extrastimuli. During CL fluctuations, changes in HH intervals precede, and predict, changes in AA intervals.

**Commentary**

After the first several RF lesions were applied, delivery of atrial extrastimuli induced SVT that looked similar, but faster than the first SVT (CL 310–370 ms, VA 60 ms); it was irregular. Changes in HH intervals preceded, and predicted, changes in AA intervals, suggesting alternation of antegrade conduction (Figure 1d). Tachycardia initiation with atrial extrastimuli excluded an automatic mechanism. The single most likely mechanism for this tachycardia was AVNRT. RF lesions were applied to the posteroseptal region and then up to triple atrial extrastimuli and burst atrial pacing failed to induce SVT. Isoproterenol was next infused and a third SVT was then induced with triple atrial extrastimuli. This SVT was slow and irregular (CL 520–580 ms, VA 50 ms). The HH intervals predicted the AA intervals and the earliest atrial activation moved to the mid-CS (CS 5,6) (Figure 2a). This apparent slow–fast AVNRT became non-inducible after additional RF applications in the slow AV nodal pathway region. Then, a fourth SVT initiated spontaneously with no isoproterenol infusion. This tachycardia was irregular (CL 330–480 ms, VA 30 ms) and showed the earliest atrial activation at CS 5,6. During this SVT, some narrow complex beats were preceded by a deformed His bundle potential or no His potential at all (Figure 2b). After termination of the atrial overdrive pacing train, SVT recurred with the following sequence: St(1)–A(1)–H(1)–V(1)–H(2)–V(2)–A(2) (Figure 2c). The absence of an atrial echo-beat during the induction of SVT ruled out AT and orthodromic AVRT as mechanisms of SVT, leaving AVNRT the only possibility. This SVT was ablated successfully in the slow AV nodal pathway region in the right atrial septal location anterior to the CS.
Then the following patterns of AV conduction were observed during atrial pacing: 1) bigeminal conduction pattern with alternation of stimulus to QRS intervals. For every second cardiac cycle, there was no His bundle potential and the St-QRS interval was 180 ms. In the other beats, the St-QRS interval was 260 ms and the His bundle electrograms were visible with AH interval duration of 180 ms (Figure 3a). 2) Wenckebach-like pattern with several normally conducted beats and progressively increasing AH intervals followed by very short stimulus to QRS with no apparent His bundle electrograms (Figure 3b). 3) No “jumps” and no inducible SVT with up to triple extrastimuli with isoproterenol and ultimate elimination of all retrograde conduction. Earliest atrial activation at the septal and proximal left sided CS 7,8 or CS 5,6 electrodes (not at the ostial CS 9,10) could be explained by the individual AV nodal anatomy and possible left-sided extensions of the slow AV nodal pathway. Initial RF lesions were applied in the traditional right-sided slow pathway region and were effective to cure SVT#1 and every subsequent tachycardia. Therefore, a left atrial approach was not attempted and would have been less preferable because of its somewhat higher risks and unknown efficacy in this particular patient.

The unusual AV nodal conduction patterns with narrow QRS complexes without a preceding His potential could have several explanations: 1) dual AV nodal conduction, 2) ectopic beats from the distal part of the His bundle, 3) atrio-fascicular or nodo-fascicular pathway connecting to the distal part of the His bundle, or 4) Zhang’s phenomenon with alternating conduction over the different His bundle layers. In the case of dual antegrade conduction of the same sinus impulse over the fast and slow AV nodal pathway, both ventricular complexes should be preceded by His activation. This was not present. Ectopic beats from the part of the His bundle more distal than the location of the diagnostic catheter are also unlikely as ablation lesions were placed far from that region. There were no signs of any additional connections between atria and ventricles as found during detailed EP study before the ablation. An unstable His catheter position was a weak explanation, as the His recording was stable throughout the procedure. The most probable explanation for these unusual tracings was the rare electrophysiological phenomenon, which was described for the first time by Y. Zhang and colleagues in vitro and later verified in vivo by the same group. The main point of this hypothesis is the presence of individual approaches for

**Figure 2:** Surface leads II and aVF, endocardial electrograms from high right atrium (HRA), His bundle region (HIS), coronary sinus (CS 9,10 in the ostium to CS 1,2 on the left free wall), right ventricular apex (RV) and atrioventricular nodal slow pathway region (ABL p and ABL d). (a) Slow irregular (cycle length 520–580 ms) narrow-complex tachycardia (SVT#3) with constant ventriculoatrial (VA) interval of 50 ms induced by triple atrial extrastimuli. The HH intervals predict the AA intervals. The earliest atrial activation is in the mid-coronary sinus (CS 5,6). (b) Irregular narrow-complex tachycardia (cycle length 330–480 ms) with a VA of 30 ms and earliest atrial activation at CS 5,6 induced spontaneously (SVT#4). Some ventricular beats are preceded by a deformed His bundle potential or no His potential at all. (c) Cessation of atrial pacing during SVT#4. Tachycardia continues at the same rate with the same stable VA interval after the sequence St(1)–A(1)–H(1)–V(1)–H(2)–V(2)–A(2), which means the absence of atrial echo during re-induction of supraventricular tachycardia.
the fast and slow AV nodal pathways to the His bundle, producing functional longitudinal dissociation into two domains. When the impulse reaches the His bundle over the fast pathway, it preferentially activates the superior domain. A diagnostic catheter positioned in this region would register sharp (high-frequency) high-amplitude His electrograms. Transverse activation of the inferior His bundle domain happens slowly. If a diagnostic catheter were positioned in this region, His bundle electrograms would demonstrate low-amplitude and

Figure 3: (a,b) Surface leads II and aVF, endocardial electrograms from high right atrium (HRA), His bundle region (HIS), coronary sinus (CS 9,10 in the ostium to CS 1,2 on the left free wall), right ventricular apex (RV) and atrioventricular (AV) nodal slow pathway region (ABL p and ABL d). (a) Overdrive atrial pacing while on sinus rhythm (PCL 500 ms). Bigeminal conduction pattern with alternation of stimulus to QRS intervals. In every second cardiac cycle, there is no His bundle potential and the St-QRS interval is 180 ms. In the other beats, the St-QRS interval is 260 ms and the His bundle electrograms are visible with AH interval duration of 180 ms. (b) Overdrive atrial pacing while on sinus rhythm (PCL 500 ms). Wenckebach-like conduction pattern with several normally conducted beats and progressively increasing A-H intervals followed by very short stimulus to QRS with no apparent His bundle electrograms. (c) Schematic illustration of the AV nodal and His bundle anatomy presenting an opportunity for Zhang's phenomenon to occur. Fast AV nodal pathway is connected directly to the superior His bundle domain while the slow pathway is connected to the inferior domain. If a diagnostic catheter is positioned close to the inferior His bundle part, activation over the fast-superior pathway looks “low and slow” due to slow transverse activation from the superior to the inferior part of the His bundle. When the impulse propagates over the slow-inferior pathway it causes a “high and sharp” potential on the inferiorly located catheter. If the recording catheter was positioned at a more superior part of the His bundle, its electrograms’ characteristics would be just the opposite.
low-frequency signals. During the slow AV nodal pathway impulse propagation, the direction of transverse His bundle activation is just the opposite. A diagnostic catheter positioned over the inferior domain in this case would register sharp and high-amplitude His bundle potentials whereas if it was located more superior the electrograms would be low amplitude and low frequency. The characteristic electrogram presents His bundle potential alternans while the impulse is conducted over the fast or slow AV nodal pathway.

In conclusion, AVNRT is generally due to the presence of a slow antegrade and faster retrograde pathway connecting to the AV node. Relationships between the AV node, the atria, and these pathways remain somewhat uncertain and, in some instances, multiple pathways exist. Many patients with AVNRT respond successfully to a single application of RF energy to the slow pathway at a right posterior septal atrial location. However, AVNRT is far from simple in many instances, and may be due to complex pathway interactions with the AV node and the atria. If tachycardia is inducible after initial RF lesions, repeat differential diagnosis should be performed to prevent unnecessary additional ablation and eliminate insufficiently ablated tachycardia substrate. Zhang’s phenomenon is rare but should be kept in mind when unusual patterns of conduction are demonstrated during AVNRT ablation.

Clinically, in long-term follow-up (2 years) the described patient is doing well off all drugs, she is competing and has no SVT.

References