Parsing a perplexing paroxysmal pathway

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Introduction

Catheter ablation is an effective and often preferred treatment for supraventricular tachycardia (SVT). Successful ablation is predicated on a precise diagnosis of the arrhythmia mechanism.[1] The initial pacing maneuvers during electrophysiologic study (EPS) are often able to narrow the differential diagnosis immediately.[2] We report an unusual case of SVT in which the initial findings at EPS were inconsistent with the eventual mechanism of the tachycardia.

Case report

A 34-year-old man with a history of high blood pressure and hyperlipidemia and 5 years of intermittent self-limited palpitations presented to the emergency room with an SVT at 220 beats per minute (Figure 1). His symptoms included chest pain and dyspnea. The tachycardia terminated spontaneously, and the patient was admitted to the hospital for further evaluation. His workup showed no structural cardiac abnormalities. Given his youth, otherwise good health, and rapid tachycardia with moderately severe symptoms, he was referred for catheter ablation.

Electrode catheters were placed within the right atrium (RA), right ventricle (RV), His-bundle position (HIS), and coronary sinus (CS). The baseline conduction intervals included a sinus cycle length of 680 milliseconds, an atrial-His interval of 67 milliseconds and His-RV interval of 32 milliseconds. Pacing the RV at both the apex and base revealed ventriculoatrial (VA) dissociation at cycle lengths from 350 to 650 milliseconds (Figure 2A). Programmed atrial stimulation did not reveal ventricular preexcitation or dual atrioventricular (AV) nodal physiology. The AV Wenckebach cycle length was 280 milliseconds. Tachycardia was easily induced with either rapid ventricular pacing or atrial premature extrastimuli. The atrial activation sequence during tachycardia was distal to proximal in the CS leads (Figure 2B).

Pacing maneuvers were performed in an attempt to elucidate the mechanism of the tachycardia. Premature ventricular complexes (PVCs) were able to terminate the tachycardia without activating the atrium (Figure 2C). Entrainment of the tachycardia with RV pacing demonstrated a VAV response on termination of pacing (Figure 2D). Notably, during multiple terminations of the tachycardia and immediate ventricular burst pacing, VA dissociation persisted. When isoproterenol was infused and ventricular pacing repeated, only a concentric and decremental retrograde atrial conduction pattern was present, which was consistent with AV nodal conduction.

Fortuitously, it was noted that spontaneous PVCs during sinus rhythm would occasionally conduct in an eccentric activation pattern, similar to the activation sequence observed during the tachycardia. This phenomenon was further examined and replicated using programmed PVCs during sinus rhythm, which revealed eccentric retrograde activation of the atrium within a window of coupling intervals or during ventricular reentrant beats (Figures 3A and 3B). Finally, during the manipulation of catheters within the left ventricle (LV) near the mitral annulus, VA conduction with an eccentric pattern was also seen (Figure 3C).

Given these contradictory findings, what was the mechanism of the tachycardia?

Discussion

The common differential diagnosis of SVT in a patient without structural heart disease is AV nodal reentrant tachycardia (AVNRT), AV reentrant tachycardia (AVRT), or atrial tachycardia (AT). Convention at electrophysiologic study for SVT dictates that RV pacing be performed first, as the nature of retrograde atrial conduction lends immediate insight into the mechanism of the tachycardia. AVRT, in which retrograde conduction up an accessory pathway is a necessary part of the circuit, is essentially eliminated when there is lack of retrograde conduction, and AVNRT becomes less likely as well.

KEYWORDS Atrioventricular reciprocating tachycardia; Lateral bypass tract; Orthodromic reciprocating tachycardia; Retrograde conduction; Supraventricular tachycardia

ABBREVIATIONS **AV** = atrioventricular; **CS** = coronary sinus; **ECG** = electrocardiogram; **EPS** = electrophysiological study; **HIS** = His bundle; **LV** = left ventricle; **PVC** = premature ventricular complex; **RA** = right atrium; **RV** = right ventricle; **SVT** = supraventricular tachycardia; **VA** = ventriculoatrial (Heart Rhythm Case Reports 2015;1:453–456)

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KEY TEACHING POINTS

- Right ventricular pacing eliciting ventriculoatrial dissociation does not necessarily rule out atrioventricular reentrant tachycardia in unique cases in which the sinus conduction may arrive at the accessory pathway at the same time, resulting in summation.
- Programmed premature ventricular complexes during sinus rhythm may be used for mapping the bypass tract in unique cases in which there is baseline ventriculoatrial dissociation.
- In rare cases of atrioventricular reentrant tachycardia, paroxysmal retrograde conduction along the bypass tract can be seen.

In this unique case that we report, initial RV pacing from 2 RV sites and at various cycle lengths revealed VA dissociation, thus leading one to a potential diagnosis of AT. However, entrainment maneuvers revealed a VAV response during RV pacing, which was inconsistent with AT, and the result of PVCs terminating the tachycardia without activating the atria coupled with an eccentric retrograde atrial activation sequence strongly point toward AVRT as the diagnosis.

How can one reconcile the fact that AVRT might exist in the presence of VA dissociation in the baseline state? A conduction block can be fixed or functional, and it can occur under tachycardic (Phase 3) or bradycardic (Phase 4) conditions.[3] VA conduction was evidently robust during rapid tachycardia, and the functional block that we observed was not strictly present during bradycardic conditions, arguing against these mechanisms. One could postulate that a "gap" phenomenon involving myocardial tissue intervening between the RV and accessory pathway was occurring. This mechanism might be supported by the observation in Figure 3C, where LV PVCs could conduct up the accessory pathway. However, arguing against this mechanism is the fact that the patient had no evidence of structural heart disease and so there was no reason to suspect diseased myocardial conduction. The observation of spontaneous PVCs occasionally activating the atria suggests a concealment phenomenon, where anterograde penetration of the accessory pathway during sinus rhythm precludes retrograde conduction unless the PVCs were critically timed so that the accessory pathway was not refractory from the sinus impulse. We were able to demonstrate this effect with programmed PVCs during sinus rhythm (Figures 3A and 3B). Alternatively, it can be postulated that the sinus wavefront that conducts down the AV node may arrive at the accessory pathway at nearly the same time as the wavefront conducting in a retrograde fashion from the PVC, with appropriate timing such that their combined impulses would result in summation.[4]

We proceeded to insert a mapping catheter via the retrograde aortic approach. Because of the lack of retrograde atrial activation in the baseline state, mapping occurred during tachycardia. The earliest retrograde atrial electrogram was located on the anterolateral mitral annulus. Radiofrequency energy at this site was able to terminate the tachycardia during the first few seconds of application. VA dissociation persisted after ablation and no other arrhythmias

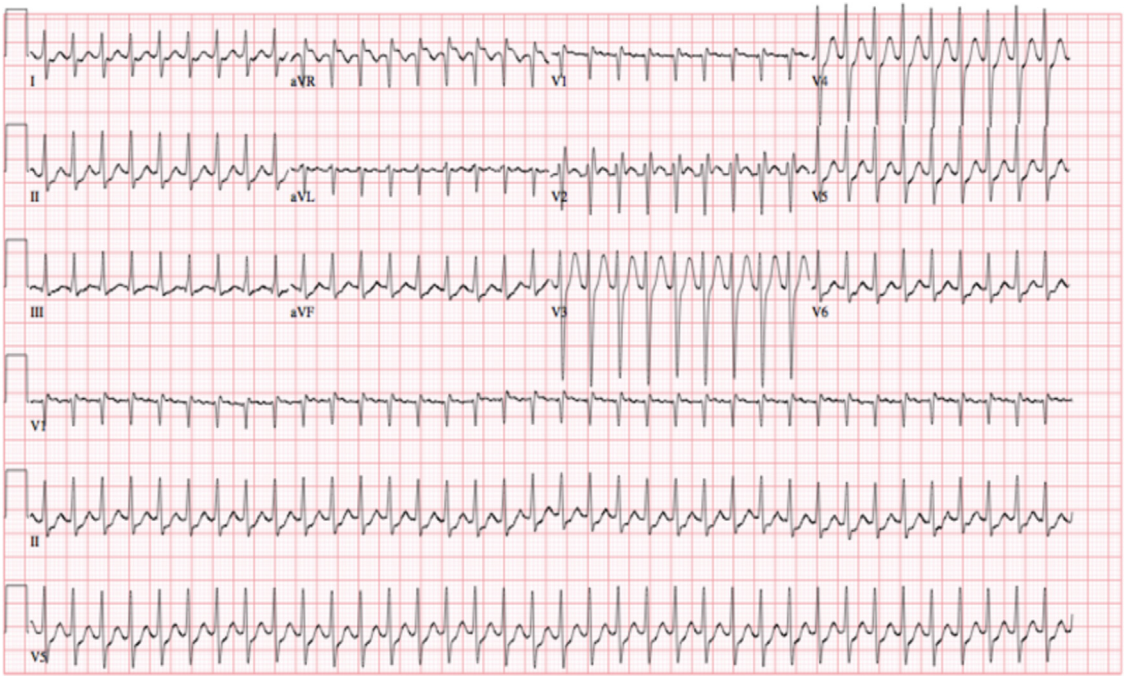

Figure 1 A 12-lead electrocardiogram of supraventricular tachycardia.