Termination of tachycardia with resolution of left bundle branch block: What is the mechanism?

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Introduction

Correctly diagnosing supraventricular tachycardia (SVT) in the electrophysiology (EP) laboratory is often challenging. It may be possible to reach the correct diagnosis by closely examining tachycardia termination. We present an intracardiac electrocardiographic tracing of nonsustained SVT termination that correctly identifies the SVT mechanism.

Case report

A 34-year-old woman with a history of palpitations for 10 years presented to the EP laboratory for an EP study and possible catheter ablation. Her symptoms generally resolved with vagal maneuvers, and no episodes occurred during 2 weeks of electrocardiographic recording using an adhesive patch. She had no other significant medical history and declined medical therapy for her symptoms. A baseline electrocardiogram did not demonstrate ventricular preexcitation. A decapolar catheter was placed in the coronary sinus (CS), with the proximal bipoles at the CS ostium. Quadripolar catheters were placed in the right ventricular apex, in the high right atrium, and across the tricuspid annulus at the His bundle position.

Ventricular pacing resulted in eccentric atrial activation. Figure 1 shows 3 beats of the tachycardia initiated by atrial extrastimuli from the high right atrium. The first 2 beats of the tachycardia exhibit a left bundle branch block (LBBB) morphology and eccentric distal to proximal CS atrial activation. LBBB resolves on the third beat; the same eccentric atrial activation is then observed; and the tachycardia finally terminates. This series of events was reproducible. When tachycardia initiated without bundle branch block while the patient was on isoproterenol, tachycardia was sustained (Figure 2). Ventricular overdrive pacing was performed during sustained tachycardia to differentiate the SVT mechanism. What is the mechanism of spontaneous tachycardia termination?

KEY TEACHING POINTS

- The presence of an accessory pathway and the participation of the pathway in supraventricular tachycardia can be demonstrated by a prolongation of the retrograde ventriculoatrial time with ipsilateral bundle branch block and termination of the tachycardia with resolution of bundle branch block.
- Eccentric ventriculoatrial activation during tachycardia can be seen with a left-sided accessory pathway, atrioventricular nodal reentrant tachycardia due to a left posterior extension of the atrioventricular node, or atrial tachycardia.
- Understanding the pattern of termination of supraventricular tachycardia can make a precise diagnosis.

Discussion

The differential diagnosis of a 34-year-old woman with no structural heart disease presenting with a regular narrow complex tachycardia with 1:1 atrioventricular (AV) conduction includes AV nodal reentrant tachycardia, AV reentrant tachycardia, and atrial tachycardia. Eccentric atrial activation during both ventricular pacing and tachycardia is most consistent with retrograde ventriculoatrial (VA) conduction over a left lateral accessory pathway. However, neither atrial tachycardia nor atypical AV nodal reentrant tachycardia using left posterior extensions of the AV node can be excluded on the basis of those findings alone.[1]

As seen in Figure 1, the VA time from the onset of the surface QRS complex to the atrial signal in the distal CS channel was 77 ms longer during LBBB vs during a narrow QRS (123 ms vs 46 ms, respectively). This observation strongly suggests that the mechanism of the tachycardia was an orthodromic AV reentrant tachycardia using a left lateral accessory pathway.[2] The first 2 beats of the

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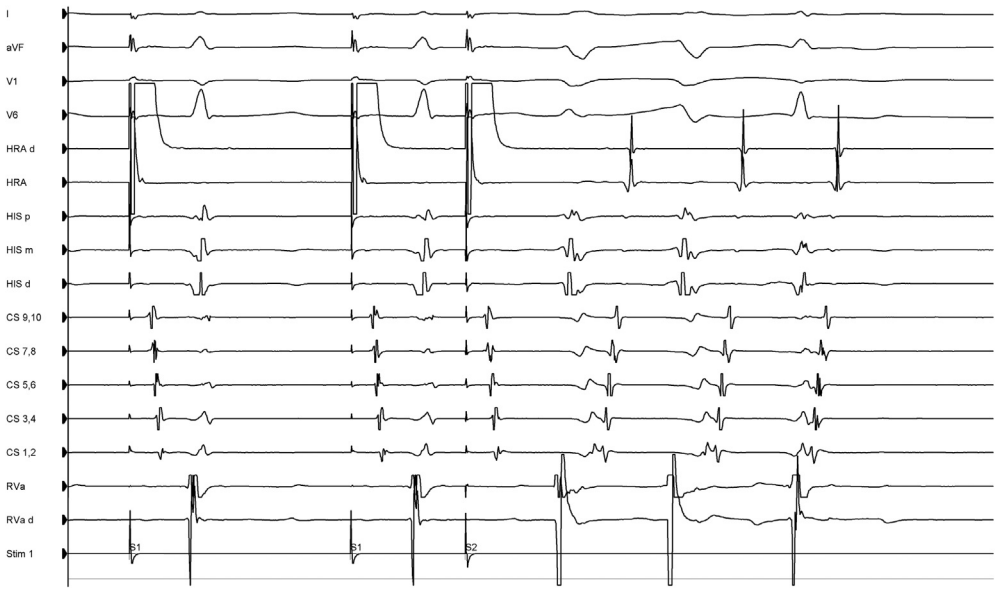

Figure 1 Nonsustained tachycardia induced by atrial extrastimuli from the high right atrium with a drive train of 600 ms and an extrastimulus at 310 ms. The ventriculoatrial time during the first 2 beats (with left bundle branch block) is 123 ms, and the ventriculoatrial time during the last (narrow complex) beat is 46 ms. CS = coronary sinus; d = distal; HRA = high right atrium; p = proximal; RVa = right ventricular apex.

tachycardia differ slightly in QRS morphology, which raises the possibility of fusion beats. Regardless, this change in the VA interval (from LBBB to no LBBB) resulted in a "long-short" sequence in the atrium. The resultant "premature" atrial depolarization then encountered a refractory AV node, resulting in tachycardia termination. Conversely, in the absence of LBBB, there was no substantial variability in the atrial cycle length, enabling consistent conduction down the AV node and sustained SVT.

Overdrive pacing demonstrated a V-A-V response, excluding atrial tachycardia. If ventricular overdrive pacing had been nondiagnostic, another option to rule out atrial tachycardia would have been differential atrial overdrive pacing to demonstrate VA linking.[3]

Therefore, resolution of LBBB was itself causal in the mechanism of tachycardia termination, demonstrating that understanding the pattern of termination can be a useful tool in making a precise diagnosis.

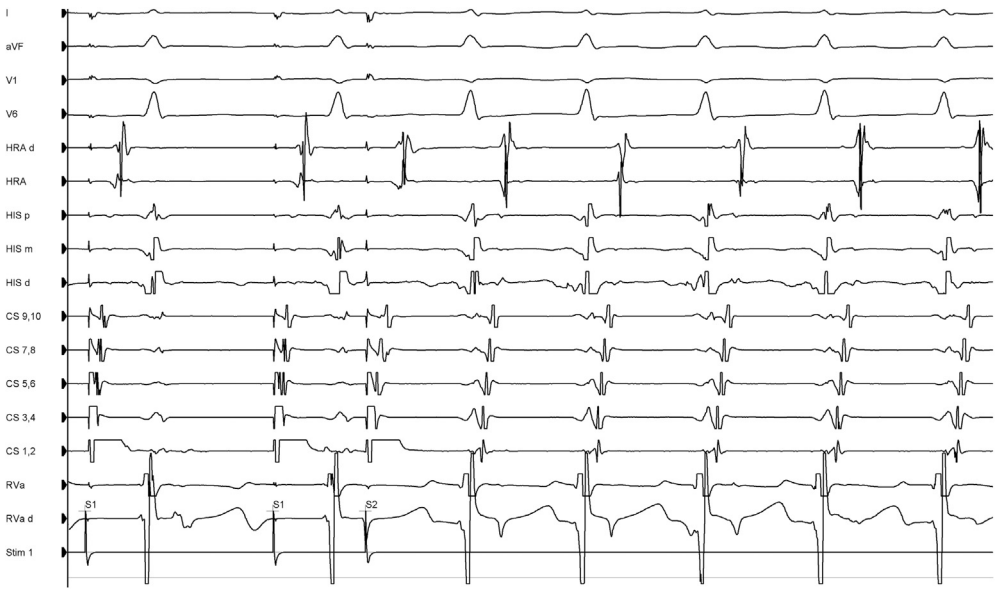

Figure 2 Sustained narrow complex tachycardia with a cycle length of 320 ms and eccentric distal to proximal coronary sinus activation. The tachycardia was induced with a drive train of 500 ms and an extrastimulus at 250 ms and isoproterenol at 1 μg/min. CS = coronary sinus; d = distal; HRA = high right atrium; p = proximal; RVa = right ventricular apex.

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