CASE REPORT

Long R–P' Paroxysmal Supraventricular Tachycardia: What Is the Mechanism?

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Deciphering the electrophysiologic mechanism of a long R–P' paroxysmal supraventricular tachycardia typically requires an invasive electrophysiologic study. We present a case where analysis of a simple surface 12-lead ECG was sufficient for the diagnosis. **A.N.E. 2006;11(1):95–97**

paroxysmal supraventricular tachycardia; long R–P' tachycardia; ectopic atrial tachycardia; AV nodal reentrant tachycardia; PJRT

CASE REPORT

A 64-year-old woman with myeloproliferative disease and chronic obstructive lung disease was hospitalized because of a 3-week history of sudden-onset palpitations associated with shortness of breath, chest discomfort, and presyncope. The episodes occurred without any obvious triggers and lasted several minutes to half an hour before terminating spontaneously. During a more prolonged episode a 12-lead ECG was recorded (Fig. 1). The heart rate during tachycardia was 184 beats/min; otherwise the patient was hemodynamically stable. The tachycardia broke following the rapid intravenous administration of 6 mg adenosine. What was the mechanism of the tachycardia?

COMMENTARY

The ECG in Figure 1 demonstrates a long R–P' supraventricular tachycardia (SVT) characterized by retrograde P waves (P'), inverted in leads II, III, and aVF, which are positioned in the second half of the R–R intervals. The differential diagnosis of an SVT with inverted P waves and R–P' > P'–R intervals includes ectopic atrial tachycardia (EAT), the uncommon "fast-slow" type of AV nodal reentrant tachycardia, and an accessory pathway-dependent permanent form of junctional reciprocating tachycardia (PJRT).^{1–3} In EAT, the ec-

topic focus is frequently localized to the vicinity of the AV node. This results in retrograde atrial activation and relatively short P–R intervals. Atypical fast-slow AV nodal reentry involves anterograde conduction through a fast AV nodal pathway and retrograde conduction through a slow pathway. This again results in retrograde P wave morphology, relatively short P'–R and long R–P' intervals. As the reentrant circuit lies solely in the AV node, both atria and ventricles discharge passively and do not participate in the reentrant circuit. In PJRT, anterograde conduction is through the AV node, whereas retrograde conduction occurs through an accessory pathway that exhibits decremental conduction properties; hence, the long R–P' intervals.^{1,2} The accessory pathway in PJRT is frequently localized to the septum and conducts in the retrograde direction only. The surface ECG in the three forms of long R–P' paroxysmal SVT (PSVT) is usually identical, and thus requires electrophysiologic study to differentiate the three. This is accomplished by analyzing the inducibility and termination of the tachycardia with programmed electrical stimulation, monitoring the effects of programmed ventricular impulses on timing of retrograde P waves, and by precisely mapping the atrial activation sequence during tachycardia.³

The ECG in Figure 1 shows a seemingly homogeneous tachycardia disturbed on two occasions.

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Figure 1. Twelve-lead ECG during paroxysmal supraventricular tachycardia. Note the presence of retrograde P waves, inverted in II, III, and aVF, in the second half of the R-R intervals. An asterisk marks the absence of a P wave during tachycardia. An arrow points to a PVC. Neither event had any effect on timing of the subsequent P waves or QRS complexes.

An asterisk denotes a dropped P wave; the absence of the P wave is clearly seen in the simultaneously recorded leads V₁, V₂, and V₃. Note that the dropped P wave did not in any way alter the sequence of the QRS complexes. This finding essentially excludes EAT and PJRT. In EAT, an atrial focus drives the ventricles; the absence of an ectopic P wave therefore should result in a simultaneous drop of a QRS complex. The tachycardia circuit in PJRT involves the AV node in the anterograde direction, the ventricular myocardium, a slowly conducting accessory pathway in the retrograde direction, and the atrial myocardium that electrically connects the retrograde impulse to the AV node.^{1,2} A spontaneous retrograde block to the atrium or transient atrial refractoriness would result in a dropped P wave, but would also terminate the accessory pathway-dependent tachycardia. Therefore, atypical AV nodal reentry is the only long R-P' PSVT whereby the reentrant circuit can persist without participation of either the atria or the ventricles. A transient block distal to the AV node can result in a dropped QRS complex, and a

transient block between the AV node and the atria can result in a dropped P wave without disturbing the AV nodal reentrant process.⁴ The differential diagnosis of a narrow complex tachycardia which can have fewer P waves than QRS complexes includes AV nodal reentry, sinus rhythm with double anterograde conduction via two separate AV nodal pathways, intrahisian reentry, and junctional tachycardia involving a nodofascicular pathway. Of these, atypical AV nodal reentry is the one that would present with long R-P' characteristics.

Two additional findings in the 12-lead ECG are of interest. The 6th beat in leads aVR, aVL, and aVF (arrow) has a shorter PR interval and wider QRS complex than any of the other beats. This is a premature ventricular complex (PVC) or fusion beat. Of note, this PVC did not alter the timing of the subsequent retrograde P wave or the duration of the cardiac cycle. In accessory pathway-mediated tachycardia, premature ventricular impulses that occur at a time when the His bundle is refractory can still conduct back through the accessory pathway and may cause either an earlier or more

delayed appearance of the next retrograde P wave or termination of the tachycardia.³ The absence of atrial preexcitation, postexcitation, or tachycardia termination by the PVC in our case again suggests that there was no retrogradely conducting accessory pathway, and thus further supports AV nodal reentry. Also of interest is the subtle electrical alternans seen in V₃, V₄, and V₅ which is more conspicuous in the T waves than in the R waves. Electrical alternans during PSVT has originally been believed to indicate accessory pathway-mediated AV macroreentry tachycardia, but was subsequently found to be a phenomenon simply associated with tachycardia rate rather than tachycardia mechanism.⁵ Electrical alternans therefore is consistent with any of the listed tachycardia mechanisms.

Understanding the SVT substrate is sometimes important in clinical decision making. One of the dreaded complications of radiofrequency ablation is inadvertent AV block, necessitating the implantation of a permanent pacemaker. The risk of AV block is somewhat higher during ablation of AV nodal reentrant tachycardia compared to EAT or accessory pathway-mediated tachycardia; in the latter forms the ablation target is usually more distant from the AV node.⁶ In our patient, who had significant comorbid conditions, the noninvasive

diagnosis of atypical AV nodal reentry prompted us to recommend an initially conservative management rather than catheter ablation. The patient was started on low-dose digoxin and long-acting diltiazem. She had no documented recurrence of the arrhythmia.

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