ARRHYTHMIA ROUNDS _

A Wide Complex Tachycardia: What is the Mechanism?

MICHAEL KUHNE, M.D., NAGIB CHALFOUN, M.D., WARANGKNA BOONYAPISIT, M.D., and KRIT JONGNARANGSIN, M.D.

From the Division of Cardiovascular Medicine, University of Michigan Health System, Ann Arbor, Michigan, USA

Case Presentation

A 33-year-old female was referred for an electrophysiologic study because of recurrent episodes of palpitations. Catheters were placed in the high right atrium (HRA), the His bundle (HIS), and the right ventricular apex (RVA). Baseline cycle length in sinus rhythm and atrial-His (AH) and His-ventricular (HV) intervals were 790, 90, and 45 ms, respectively. Spontaneous onset of a wide complex tachycardia with a left bundle branch block morphology and variable cycle length was observed on several occasions (Fig. 1). Atrial pacing at a rate of 600 ms with an extrastimulus with a coupling interval of 390 ms was conducted, as shown in Figure 2. What is the mechanism of this wide complex tachycardia?

Commentary

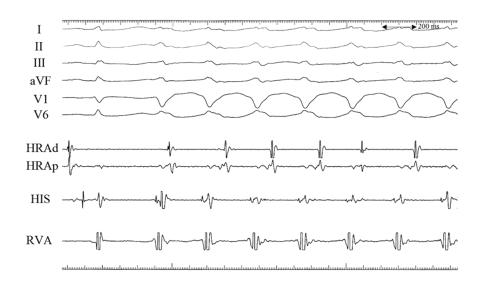
The wide complex tachycardia shown in Figure 1 has a left bundle branch block morphology with a 1:1 retrograde atrio-

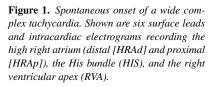
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Address for correspondence: Krit Jongnarangsin, M.D., Department of Internal Medicine, Division of Cardiovascular Medicine, 1500 E. Medical Center Drive, VAMC 111A, Box 2399, Ann Arbor, MI 48109, USA. Fax: 734-845-3270; E-mail: kritj@med.umich.edu

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ventricular node conduction. The differential diagnosis of a wide complex tachycardia includes ventricular tachycardia (VT), antidromic reciprocating tachycardia, and supraventricular tachycardia (SVT) with aberrant conduction or preexcitation with an innocent bystander accessory pathway. The baseline sinus (1st beat) has a normal QRS morphology and normal intracardiac conduction intervals. During atrial stimulation at a cycle length of 600 ms, as shown in Figure 2, the QRS complex becomes wider and HV interval is shorter. An atrial extrastimulus at a coupling interval of 390 ms (3rd beat) results in a longer AH interval and wider QRS complex with a left bundle branch block pattern resembling the QRS morphology during tachycardia. A His bundle potential is noted to be later than the onset of the QRS complex. This is due to ventricular preexcitation over an accessory pathway that does not manifest at baseline. The accessory pathway conduction appears to be slow based on the relatively long stimulus to QRS interval. Preexcitation is fully manifested following an atrial extrastimulus due to either slower AV nodal conduction or AV nodal block. The ventricle is more preexcited when the conduction time over the AV node exceeds the conduction time over the accessory connection. It is also noted that when the preexcitation fully manifests, the His bundle is activated retrogradely and before the ventricular septum. This suggests that the distal insertion of the accessory pathway is close to the His bundle. The above electrophysiologic findings are consistent with atriofascicular accessory pathway characteristics.





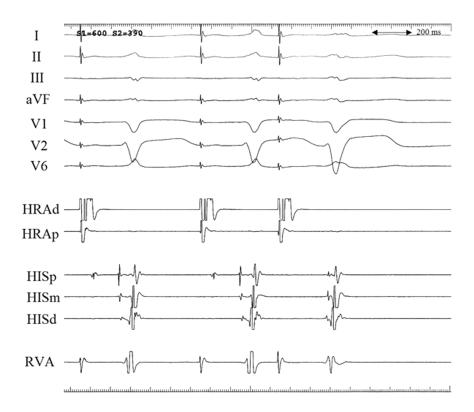


Figure 2. Corresponding surface leads and intracardiac electrograms during atrial pacing at a cycle length of 600 ms and one extrastimulus with a coupling interval of 390 ms. Abbreviations as in Figure 1.

Spontaneous occurrence of wide complex tachycardia without a preceding atrial beat makes AV reciprocating tachycardia or SVT with aberrant conduction or preexcitation with an innocent bystander accessory pathway unlikely. Because the QRS morphology during tachycardia is identical to the QRS morphology during full preexcitation, the atriofascicular accessory pathway must participate in this tachycardia. The most likely mechanism of this tachycardia is spontaneous automaticity of the atriofascicular accessory pathway. Atrial activation during tachycardia results from retrograde atrioventricular node conduction, except for the first atrial beat of the tachycardia, which is a fusion beat between sinus and retrograde conduction. Although automaticity of atrioventricular accessory pathways is commonly seen during radio frequency ablation, spontaneous automaticity of an atriofascicular accessory pathway is rare.^{1,2} The atriofascicular accessory pathway in this patient was mapped during atrial pacing and the pathway potential was found at the lateral aspect of the tricuspid annulus. Radio frequency ablation at this site resulted in disappearance of the preexcitation and spontaneous wide complex tachycardia.

References

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