Narrow QRS Complex Ventricular Tachycardia

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7.1 Case Summary

A 19-year-old male with episodic palpitations since age 12, usually precipitated by exercise, was referred for evaluation. Some episodes terminated with Valsalva maneuver. The response to adenosine was variable. An electrophysiology study (EPS) was performed at a tertiary medical center at age 14. By report, a narrow QRS complex tachycardia was induced with atrial pacing. Although there was no evidence of dual AV nodal pathways, a slow pathway modification was performed with no tachycardia inducible at the end of the procedure. Shortly thereafter symptoms of tachycardia recurred, with increased frequency. A second EPS was performed at the same medical center 9 months later. Modification of the AV node slow pathway was performed again. However, tachycardia remained inducible at the end of the procedure. He was discharged on verapamil with significant improvement in the frequency of the tachycardia. An ECG recorded during recurrent tachycardia while taking verapamil showed a RBBB, left axis tachycardia with QRS duration 110 ms (Fig. 7.1). What is the differential diagnosis of the tachycardia?

7.2 Results of EPS

Review of the 12 lead ECG (Fig. 7.1) shows AV dissociation (best seen in leads aVR and aVF). In the EP lab in the baseline state atrial stimulation showed no evidence of dual AV node pathways. The baseline HV interval was 50 ms (Fig. 7.2). VA conduction was absent. Programmed atrial and ventricular stimulation induced no tachycardia. With infusion of isoproterenol at 1.0 mcg/min VA conduction was present. VA Wenckebach occurred at a paced cycle length (CL) of 520 ms. After 1 mg of atropine, tachycardia was reproducibly induced by atrial pacing at cycle length (CL) of 340–350 ms as well as with single APDs—400/330 ms (Fig. 7.3). There was distinct change in QRS morphology to a RBBB, left axis with onset of tachycardia. QRS width was 110 ms; VA dissociation, sinus capture and sinus fusion beats were noted during tachycardia (Fig. 7.4). Initiation of the tachycardia was not dependent on a critical degree of AH prolongation. The HV interval during tachycardia was 22 ms. Single ventricular premature depolarizations as well as RVA overdrive pacing were able to terminate the tachycardia reproducibly. Based on the above, the tachycardia was determined to be most likely idiopathic ventricular (fascicular) tachycardia. Two decapolar catheters were placed along both the RV and LV septum to record Purkinje activation. However, the tachycardia became non-inducible after the placement of the LV catheter. Pace mapping of sites along the LV septum in regions demonstrating Purkinje potentials was done. The closest pace maps were along the inferior mid-LV septum. Five radiofrequency lesions were delivered at those sites. The tachycardia remained non-inducible at the end of the procedure. Off medication, patient has not had recurrence of the tachycardia over the following 13 years.

7.3 Case Discussion

This case illustrates several points. First, examination of the ECG recorded during the spontaneous tachycardia shows AV dissociation. Although we occasionally observe AV dissociation during AV node reentrant SVT, this usually occurs only briefly, during the onset of induced tachycardia. The presence of AV dissociation during the tachycardia induced in the EP lab, confirms the interpretation of the ECG recorded during spontaneous tachycardia. Furthermore, the fact that even during isoproterenol infusion VA conduction block occurred at a paced CL of 520 ms (in the presence of a tachycardia CL of 340 ms) makes any SVT unlikely. It is important to know that VT can have relatively narrow QRS

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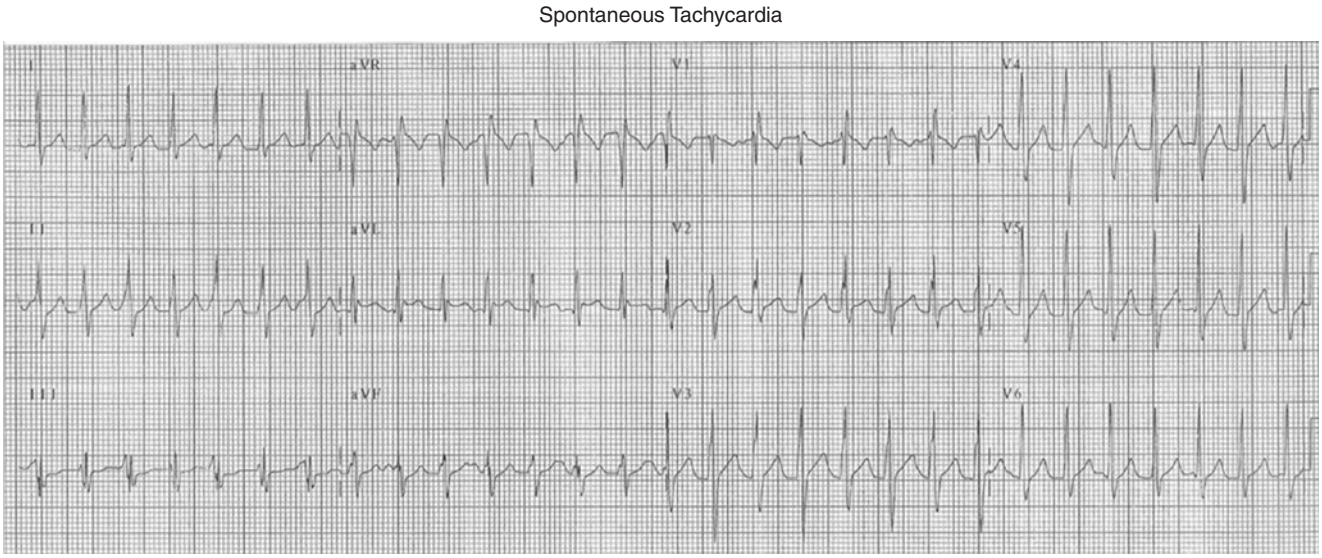

Fig. 7.1 12 lead ECG showing RBBB, left axis tachycardia and AV dissociation seen in multiple leads

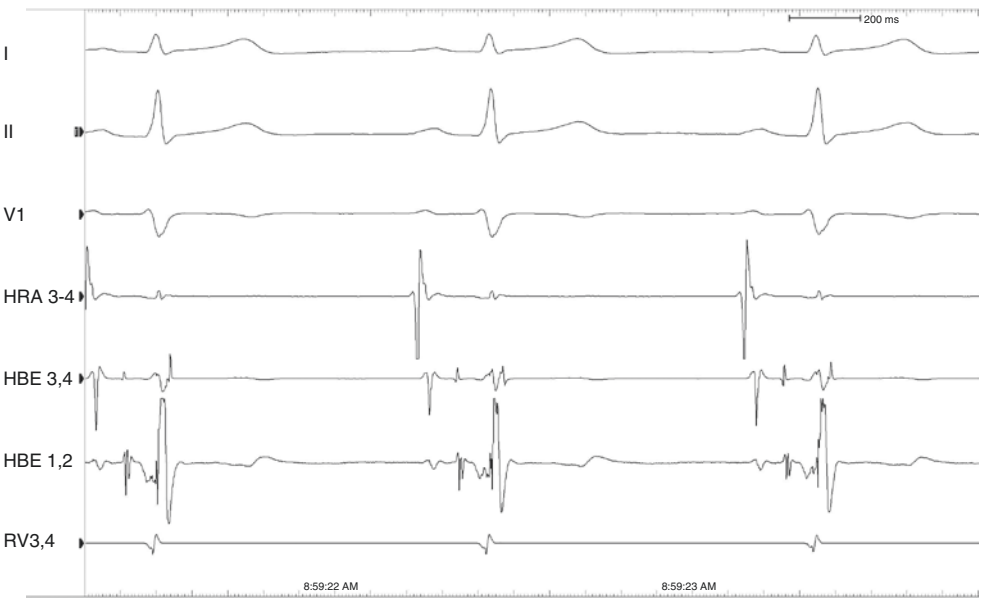

Fig. 7.2 Shown are ECG leads I, II and V1 and intracardiac electrograms from the high right atrium (HRA), His bundle (HBE), and the right ventricular apex (RV3,4). The HV interval is 50 ms

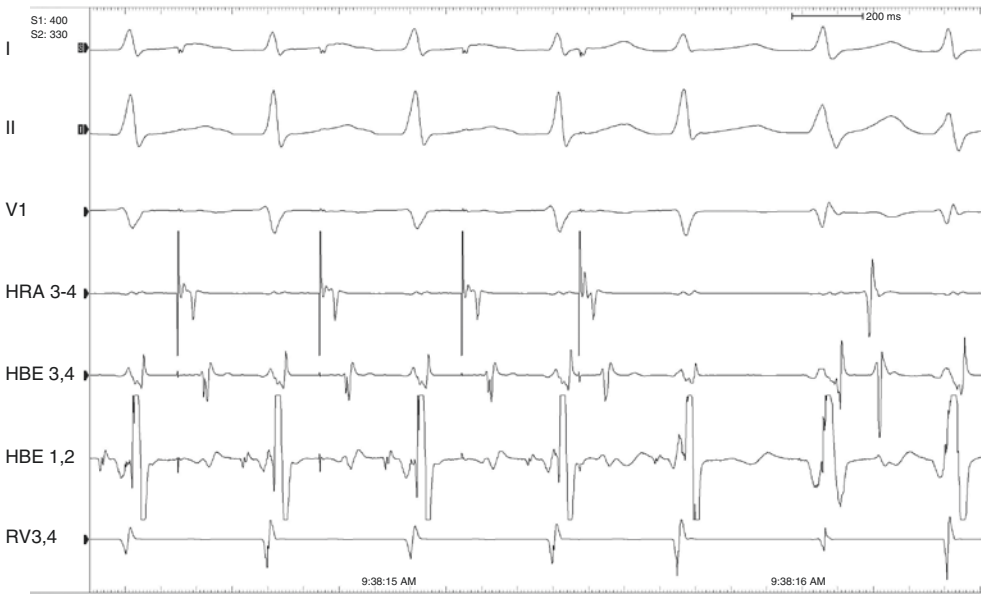

Fig. 7.3 Induced tachycardia by a single APD (coupling interval 330 ms) at paced CL 400 ms. Shown are surface electrograms from leads I, II and V1 and the intracardiac electrograms from the high right atrium (HRA), His bundle (HBE), and the right ventricular apex (RV3,4). During the first two beats of tachycardia a small His deflection is seen with an "HV interval" of 22 ms

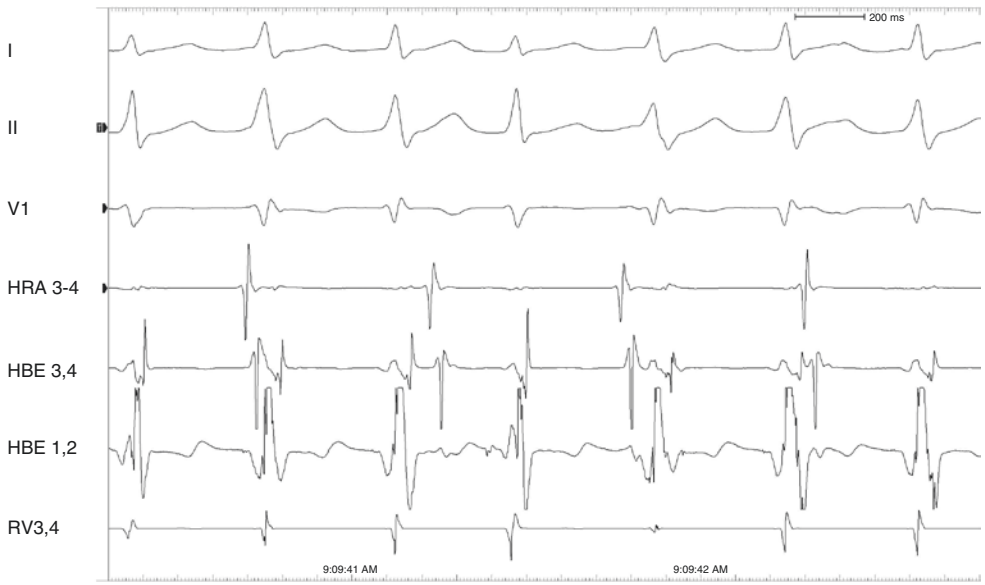

Fig. 7.4 Induced tachycardia with VA dissociation. The fourth QRS complex results from a sinus capture beat (arrow). Shown are surface electrograms from leads I, II and V1 and the intracardiac electrograms from the high right atrium (HRA), His bundle (HBE), and the right ventricular apex (RV3,4). Note that the "HV" interval during tachycardia is significantly shorter than the sinus HV interval (it is actually a VH interval). Also note that the sinus capture beat does not reset the tachycardia (the interval surrounding the sinus capture is fully compensatory). The sinus capture beat is associated with a short AH interval (effectively ruling out typical AV node reentry)

morphology, especially in young people without structural heart disease. In a retrospective study of 106 patients with VT by Hayes et al. [1], five patients had VT with a QRS duration ≤110 ms and three of these five patients were previously incorrectly diagnosed as having SVT. Most VT with relatively narrow QRS morphology are idiopathic left ventricular VT though there have been case reports of VT of relatively narrow QRS morphology in patients with myocardial infarction [2–4] Finally, this case illustrates that regardless of previous EP reports, every case must be examined completely and in an objective, unbiased fashion.

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