CASE REPORT

Bundle branch re-entry ventricular tachycardia in a patient with complete heart block

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Received 16 July 2005; accepted after revision 12 March 2006; online publish-ahead-of-print 10 July 2006

A 58-year-old male patient presented episodes of palpitations in the context of atrioventricular block treated by a dual-chamber pacemaker. Clinical and electrophysiological studies identified the tachyarrhythmia to be bundle branch re-entrant ventricular tachycardia, which was successfully treated by radiofrequency ablation of the proximal right bundle branch.

KEYWORDS
Ventricular tachycardia; Radiofrequency ablation

A 58-year-old man was admitted after two episodes of palpitations, dyspnoea, and chest pain. He had received a dual-chamber pacemaker because of symptomatic atrioventricular block and had undergone aortic valve replacement because of aortic regurgitation 8 years before. An echocardiogram revealed severely impaired left ventricular function with an ejection fraction of 15%. During cardiac monitoring, the patient had recurrent sustained wide-QRS tachycardias at a rate of 195 bpm. The episodes were triggered by isolated premature ventricular complexes with a left bundle branch morphology (Figure 1). Some episodes terminated spontaneously with premature ventricular complexes. Longer episodes could reproducibly be terminated by asynchronous pacing when the programmer head was placed over the pacemaker. The morphology of the QRS during tachycardia and the absence of ventriculo-atrial conduction during ventricular pacing at 70 bpm showed this not to be a pacemaker-mediated tachycardia.

The patient was brought to the electrophysiological laboratory after signing informed consent. Recording of His bundle depolarization was not possible as the patient had complete atrioventricular block and paced rhythm. Programmed electrical stimulation reproducibly induced ventricular tachycardia (VT). A His deflection preceded each QRS by 60 ms during tachycardia. During cycle length oscillations following VT induction, variations of HH intervals preceded variations of VV intervals, confirming the suspicion of bundle branch re-entrant VT.1, 2 Isolated premature ventricular complexes could induce and terminate VT (Figure 2A and B). His bundle electrogram recording during VT termination suggests that the retrograde pathway of the circuit is made refractory by isolated spontaneous premature ventricular complexes after a one-revolution delay (Figure 2B). The ablation catheter was moved caudally to record a right bundle potential during VT. Application of radiofrequency current was performed during VT that terminated after a few seconds, and was continued for 1 min. Following ablation, programmed stimulation failed to induce VT. VT did not recur during 12 months of follow-up.

Discussion

This patient had bundle branch re-entrant VT, which is the most common mechanism supporting monomorphic VT in the context of valvular heart disease.3 Bundle branch re-entrant VT is most often encountered in dilated cardiomyopathy, but can cause VT in patients with coronary heart disease and other structural heart disease.1, 4–6 Occasionally, it can be seen in the absence of structural heart disease.6–8 Bundle branch re-entrant VT in a patient with complete heart block has not been so far described. In our patient, heart block was due to intra-nodal block and infra-nodal conduction was only slightly prolonged during VT. As mapping of the right bundle was not possible during sinus rhythm due to AV block, mapping and radiofrequency ablation were performed during VT.2, 4, 9 The re-entrant circuit had some additional particularities. First, spontaneous inductions occurred repeatedly following isolated premature ventricular complexes and was easily inducible in the EP laboratory, whereas bundle branch re-entrant circuits are usually more difficult to induce.
Secondly, there were also repetitive arrhythmia terminations, following premature ventricular complexes during VT. Interestingly, premature ventricular complexes reproducibly provoked a slight prolongation of the next HH interval, as if a part of the re-entrant circuit had decremental conduction or as if the circuit might become larger. VT terminated not immediately following premature ventricular complexes, but with a one-cycle delay. The slight prolongation of revolution time appeared to make some portion of the retrograde limb of the circuit refractory to conduction, perhaps because the ‘delayed’ antegrade depolarization wavefront invaded a part of the retrograde limb.

In conclusion, this case is interesting because it is the first description of bundle branch re-entrant VT in a patient with complete heart block and because it illustrates the interplay between the electrophysiological substrate and the triggers that may induce and terminate ventricular re-entrant arrhythmias.

Acknowledgements
Dr Delacrétaz is supported by a grant from the Swiss National Research Foundation.

References


