Bundle Branch Reentrant Tachycardia Treated by Electrical Ablation of the Right Bundle Branch

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A 66 year old man presented with multiple episodes of tachycardia. Some had QRS complexes with a right bundle branch block configuration identical to those of sinus beats. The onset of the tachycardia was preceded by premature His bundle depolarizations. There was a His potential before each QRS complex of the tachycardia. Atrial activity was dissociated. Occasionally the appearance of sinus beats with a left bundle branch block pattern announced a tachycardia with an identical configuration and atrioventricular dissociation. His bundle activity occurred before the QRS complex and was followed by a right bundle branch deflection. A reentrant mechanism within the bundle branch system was invoked. One 200 J shock was delivered through an electrode catheter to the site of the right bundle branch. The postprocedure course was uneventful (follow-up 10 months).

Case Report

Clinical data. The patient was a 66 year old man with aortic regurgitation and left ventricular failure. On August 23, 1984 he underwent replacement of the aortic valve. Forty-eight hours later he had episodes of regular tachycardia that recurred continuously during the following 2 weeks. Two types of arrhythmia alternated: one at a rate of 220 beats/min with a pattern similar to that of sinus beats (right bundle branch block and right-axis deviation), the other at a rate of 250 beats/min with a left bundle branch block pattern (Fig. 1). Per fusions of various antiarrhythmic drugs such as amiodarone, lidocaine, procainamide and bretylium were unsuccessful.

Electrophysiology. An electrophysiological study was carried out on September 6, 1984. The patient was receiving at this time a combination of procainamide, 3g, and bretylium, 700 mg/day, given intravenously. Five electrode catheters were introduced percutaneously through the femoral veins. Two were positioned in the right atrium, two others in the region of the tricuspid valve to obtain the His bundle potential (H) and the right bundle branch potential (RB) and the fifth in the right ventricular apex. Recordings were made on an eight channel ink-jet recorder (Elema, Sweden). Cardiac pacing was performed by a modular programmable stimulator (Janssen, Belgium) providing square impulses of 1.5 ms duration, the intensity being twice the diastolic threshold. All data were stored on magnetic tape (Hewlett-Packard).

Basic values at the beginning of the study were as follows: sinus cycle length 700 ms, AH interval 110 ms, HV interval 95 ms, duration of the QRS complex (incomplete right bundle branch block pattern) 140 ms. The sinus rhythm was frequently interrupted by atrial premature beats associated
Figure 1. Twelve lead electrocardiograms. **Left panel.** In sinus rhythm (SR). **Middle panel.** During ventricular tachycardia (VT) with right bundle branch block configuration. **Right panel.** During ventricular tachycardia with a left bundle branch block configuration.

Figure 2. Effect of atrial pacing at increasing frequency. S indicates the stimulus artifact. A, H and V represent the atrial, His bundle and ventricular activities, respectively, recorded in the atrioventricular junction. The incomplete right bundle branch block pattern progressively disappears. At a pacing cycle length (PCL) of 500 ms, the paced QRS complexes are narrow (**right panel**). At the same time, the interval between the right bundle branch potential (arrows) and V increases from 0 to 30 ms. Note that the QRS deformation is again apparent after the pause ending the atrioventricular nodal Wenckebach phenomenon. A = bipolar right atrial lead; HIS = His bundle lead; RBB = right bundle branch lead; SR = sinus rhythm.

Figure 3. His bundle premature depolarizations resulting in a 2:1 atrioventricular block (**upper panel**). Delivery of a ventricular extrastimulus (S) before the QRS complex is able to advance the concealed Hisian excitation (**middle panel**). After earlier extrastimuli, His bundle bigeminy disappears and normal sinus rhythm resumes (**lower panel**). Abbreviations as in Figure 2.
with a normalization of the QRS complex. Rapid atrial pacing provoked the same response (Fig. 2). At a rate of 120 beats/min, the right bundle branch block disappeared and this was associated with an increase of the RB-V interval from 0 to 30 ms. The HV interval remained unchanged.

**Premature stimulation of the right ventricle was carried out** using basic cycle lengths of 600 and 500 ms. The delivery of one or two extrastimuli was able to produce the following phenomena. If the postpacing interval was sufficiently long (at least 1,070 ms) the subsequent sinus beat showed a further increase in the HV interval (values ranging from 110 to 150 ms) associated with a complete right bundle branch block pattern (QRS duration 170 ms; RB potential unseen). This was invariably followed by a premature His bundle depolarization accounting for the atrioventricular (AV) nodal blockade of the following P wave (2:1 pseudo AV block) (Fig. 3). The VH interval was 340 ms. The delivery, after the basal H potential, of ventricular stimulation anticipating the sinus QRS complex was capable of advancing the occurrence of the concealed His bundle excitation. When delivered earlier the extrastimulus suppressed His bundle bigeminy and restored normal sinus rhythm.

**At times the premature H potential was followed by a ventricular response and this was the prelude to a sustained tachycardia.** The tachycardia (Fig. 4) was regular at a cycle of 300 ms and was composed of QRS complexes with a complete right bundle branch block pattern identical to that of spontaneous episodes. Each QRS complex was preceded by a His bundle deflection (HV interval = 150 ms); it was then buried in the ventricular electrogram. The right bundle branch potential was recorded 90 ms before each QRS complex of the tachycardia (except for the first two beats, which had RB-V intervals measuring 75 and 130 ms, respectively). The arrhythmia stabilized at a cycle of 260 ms and was accompanied by AV dissociation. Because this attack was poorly tolerated, it was terminated by electrical counter-shock.

**Catheter ablation.** These data strongly suggested a bundle branch reentrant mechanism. It was then decided to electrically ablate the right bundle branch. Using a standard defibrillator and pentothal anesthesia, one 200 J shock was delivered through the distal electrode of the right bundle branch catheter, the discharge being directed toward a back paddle. This immediately produced a complete infraHis AV block with escape beats successively showing a left and a right bundle branch block pattern (Fig. 6). Then, 1:1 AV conduction resumed 20 minutes later associated with an HV interval of 95 ms and complete right bundle branch block (RB potential unseen). No ventricular arrhythmia recurred.

**Follow-up.** An electrophysiologic follow-up study was carried out 3 months later. The AH and HV intervals measured, respectively, 90 and 75 ms. No right bundle branch potential was recorded. Rapid atrial pacing showed persistence of the complete right bundle branch block pattern (Fig.

**Figure 4.** Initiation of bundle branch reentrant tachycardia. The sinus QRS complex exhibits a complete right bundle branch block configuration. It is followed by a His bundle premature depolarization. Tachycardia ensues with a similar pattern and a His bundle deflection occurring before each subsequent beat. A right bundle branch potential is not seen (the tiny deflection at the beginning of the QRS complex being attributed to the ventricular depolarization; see Fig. 2). Atrial activity is dissociated. Note the shortening of the VH interval associated with the tachycardia development. Abbreviations as in Figure 2.
Figure 5. The appearance of left bundle branch block in sinus rhythm is the prelude to a tachycardia with the same configuration (left panel). A right bundle branch potential (indicated by arrows) now precedes each QRS complex. The His bundle deflection is visible before the first tachycardia beat. It is then buried within the ventricular activity. Atrioventricular dissociation is present. The arrhythmia becomes faster and stabilizes at a cycle length of 260 ms (right panel). Abbreviations as in Figure 2.

Figure 6. Complete infraHisian atrioventricular block immediately after the electrical shock delivered to the area of the right bundle branch. The escape beats successively exhibit a left bundle branch block pattern (upper panel), then a right bundle branch block pattern (middle panel). Twenty minutes later 1:1 atrioventricular conduction resumes (lower panel). Abbreviations as in Figure 2.

Figure 7. Effect of rapid atrial pacing 3 months after ablation. There is complete right bundle branch block. Moreover, the intraventricular conduction disturbance persists at all the pacing rates used. Abbreviations as in Figure 2.
There was no inducible arrhythmia. The patient was still free of tachycardia in July 1985. Holter electrocardiographic recordings or exercise tests were not performed during the follow-up period.

**Discussion**

**Role of bundle branch reentry.** The data collected in our patient can be analyzed as follows. The onset of the tachycardias with a right bundle branch block pattern seemed linked to the suppression of all conduction in the right bundle branch. This change followed a critical lengthening of the cardiac cycle suggesting a phase 4 block mechanism (10). The associated increase in the HV interval implied further conduction impairment within the His bundle or the left bundle branch. During the concealed Hisian bigeminy preceding the tachycardias, the response to premature ventricular stimulation gave support to a reentrant mechanism through the bundle branches. The recording of earlier Hisian excitations allowed us to assume that the ventricular activity had reached and depolarized the His bundle. Because the sinus impulse was simultaneously well on its way in the left bundle branch, the right bundle branch was likely to constitute the return pathway to the His bundle. These data were in favor of a functional dissociation within the intraventricular conduction system which predisposed to reentry. When more premature, the electrically induced ventricular response would become incapable of exciting the still refractory His bundle, resulting in suppression of the junctional bigeminy.

The return of the reentrant excitation to the ventricles initiated the tachycardia. The HV interval was slightly longer than in sinus rhythm, indicating a further slowing of conduction in the anterograde limb of the circuit during incomplete recovery. The VH interval in tachycardia, which is supposed to include the time of retrograde transmission to the His bundle, was shorter than that accompanying the initial sinus beat. Supernormal conduction in the right bundle branch could explain this phenomenon. This property has already been demonstrated during anterograde conduction. Another alternative implies the persistence of some degree of anterograde penetration of the right bundle branch in sinus rhythm responsible for a depression in retrograde conduction. It is postulated that the right bundle branch (RB) activity, unseen during the tachycardia, was in fact buried in the ventricular electrogram. This idea implies a marked delay between the recording zones of the RB and H potentials, a notion supported by the anterograde conduction data.

**Other hypotheses.** The other hypotheses are less convincing. A His bundle tachycardia must be invoked. However, the Hisian bigeminy could be reproducibly suppressed by a ventricular premature depolarization that did not excite the His bundle. Furthermore, the occurrence of a ventricular response after the Hisian extrasystole was a prerequisite for the tachycardia development. Reentry through accessory Mahaim fibers is an alternative possibility (11). Nonetheless, no manifest pre-excitation was ever recorded in this patient. A third possible mechanism is a circus movement involving the two ramifications of the left bundle branch and initiated by unidirectional block within the posterior fascicle. The absence of shortening of the HV interval (in comparison with sinus rhythm) is not in concordance with this notion. Finally, all these explanations do not clarify the relation between conduction disturbances in the right bundle branch and the onset of the arrhythmias.

**Occurrence of reversed reentry.** The occurrence in sinus rhythm of a left bundle branch block announced a tachycardia of the same configuration. During the basal sinus beat, the progression of the sinus impulse through the right bundle branch (RB) was proved by the recording of an RB potential 75 ms before the QRS complex. The same sequence was seen during the tachycardia, with a further increase in the RB-V interval attributed to the high rate. It was tempting to evoke bundle branch reentry once more, but the circuit would have been reversed. Initially, the blockade of the sinus impulse in the left bundle branch unmasked persistent slow conduction along the right bundle branch. When it reached the right ventricle, the excitation was assumed to cross the septum and progress to the His bundle through the left specialized intraventricular tissue. The continuation of the circus movement formed the basis of the arrhythmia. This was associated with a sudden decrease in conduction time along the retrograde limb of the circuit, the mechanism of which has been already discussed. Alternatively, the role of Mahaim fibers conducting retrogradely cannot be excluded. However, this hypothesis does not account for the role of the left bundle branch block in the arrhythmia initiation.

**Effect of procainamide.** A proarrhythmic action of procainamide in our patient is to be discussed. Previous reports (12,13) have shown that the intravenous infusion of procainamide could both facilitate and suppress bundle branch reentry, depending on critical changes in conduction and refractoriness of the His-Purkinje system. The facilitating effect was observed in lower doses (that is, 5 to 8 mg/kg). However, it should be noted that the tachycardias described in this case appeared before any drug was administered.

**Further evidence provided by the catheter ablation.** The suppression of all tachycardias by electrical ablation of the right bundle branch constitutes a convincing argument in favor of bundle branch reentry. The effect of ablation on the specialized intraventricular tissue was proved by the electrophysiologic changes in the right bundle branch. The suppression of all conduction, even during the supernormal phase, contrasted with the data before shock. It provided the only apparent sign of the electroablation. The subsequent control of the two types of tachycardia supports the view that a common mechanism was involved and that the right
bundle branch played an essential role in their genesis. This study, therefore, offers further evidence in favor of the intervention of bundle branch reentry as a possible basis for tachycardia. Endocavitary ablation was a suitable treatment in our patient once we had identified the underlying mechanism of the arrhythmia.

References