CASE REPORTS

Bundle Branch Reentry: A Possible Mechanism of Ventricular Tachycardia

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SUMMARY Electrophysiologic studies were performed in three patients suffering from attacks of paroxysmal tachycardia with wide QRS complexes. Two patients had atrioventricular dissociation. The arrhythmia could be initiated and terminated by premature ventricular stimulation in all three patients. One patient developed the arrhythmia after rapid atrial stimulation. In each subject, the QRS complexes during tachycardia were identical to recorded supraventricular beats (left bundle branch block pattern in two cases and right bundle branch block pattern in one). A His bundle potential was noted before the QRS complex; the HV interval was equal to or longer than that of the sinus beats. The following observations suggested the presence of a bundle branch reentry mechanism: (1) the relationship between bundle branch block development and tachycardia initiation; (2) the occurrence of tachycardia after electrically induced His-Purkinje reentry; (3) the ability of premature ventricular stimulation during tachycardia to advance the timing of the His deflection and QRS complex, with an unchanged or slightly increased HV interval; and (4) the termination of arrhythmia by premature ventricular depolarization blocked within the bundle branch system. Our results support the idea that bundle branch reentry can play a role in the genesis of ventricular tachycardia.

MOST paroxysmal tachycardias, both supraventricular and ventricular, are considered reentrant arrhythmias because they can be reproducibly initiated and terminated by programmed stimulation. Although the site of reentry has been established for most cases of paroxysmal supraventricular tachycardia,1-8 the site of reentry in ventricular tachycardia is not well defined. In patients with coronary artery disease, some ventricular tachycardias have been ascribed to reentry within the ischemic zone.9-12 Only infrequent reports have suggested the involvement of the His bundle and its branches10, 13-16 in the reentrant circuit of ventricular tachycardia, but these cases have not eliminated doubts about the obligatory incorporation of the proximal His-Purkinje system in the tachycardias. We report three patients with ventricular tachycardia in whom electrophysiologic data strongly support a bundle branch reentry mechanism.

Methods

Electrophysiologic studies were carried out with the patients in the postabsorpitive, nonsedated state. Four electrode catheters were introduced percutaneously (Seldinger technique) into a right and left femoral vein. Two catheters were positioned in the right atrium: one for recording a bipolar electrogram at the high lateral wall and the other for stimulating the septum. A tripolar catheter was used to record His bundle electrical activity17 and a bipolar catheter was placed in the apex of the right ventricle for pacing. Recordings were made on an eight-channel, direct-writing, ink-jet recorder (Elema, Mingograph 81) at a paper speed of 100-200 mm/sec. Standard techniques of programmed atrial and ventricular stimulation (Janssen) were used.18 All data were stored on magnetic tape (Hewlett-Packard). Informed consent was obtained from each patient before the studies.

Case Reports

Case 1

A 53-year-old man with aortic valve disease was admitted in October 1977 for recurrent episodes of tachycardia accompanied by faintness and chest pain. Digoxin and verapamil had been prescribed without any effect. The tachycardia was regular, with wide QRS complexes (0.15 second) showing a complete right bundle branch block pattern and left-axis deviation (QRS of -85°) (fig. 1). Atrial activity was not clearly discernible. The heart rate was 175 beats/min.

An electrophysiologic study was then undertaken. The ECG in sinus rhythm showed a PR interval of 0.20 second and narrow QRS complexes with a frontal axis of -45° (fig. 1). The AH interval was 75 msec and the HV interval 80 msec. Premature atrial stimulation at paced cycle lengths of 550 and 500 msec did not induce tachycardia. Atrial pacing at increasing rates was accompanied by lengthening of both AH and HV intervals and development of incomplete right bundle branch block (fig. 2). When the cycle was shortened to 320 msec, complete right bundle branch block occurred with an HV interval of 105 msec. After cessation of pacing, a tachycardia commenced exhibiting the same right bundle branch block pattern (fig. 2). This tachycardia was similar to spontaneous attacks. The tachycardia cycle length was 340 msec. Eccentric atrial activation occurred 35 msec before each QRS complex. In addition, a prolonged, polyphasic His
bundle electrogram preceded both atrial and ventricular activity (HV interval of 105 msec). Carotid sinus massage was ineffective. Increasingly premature stimulation of the right atrium during tachycardia, up to a coupling interval of 280 msec, was followed by a fully compensatory atrial pause and did not alter the course of the tachycardia. No premature atrial response captured the His bundle. Rapid atrial stimulation was not performed during tachycardia. We then induced premature right ventricular excitation (fig. 3). The His bundle response and succeeding ventricular complex were advanced with no change in HV interval. When the coupling interval was shortened, the corresponding atrial activity disappeared. The tachycardia was terminated by early ventricular depolarizations not followed by an H potential. During the study, tachycardia was reproducibly provoked by rapid atrial pacing. When stimulation was prematurely stopped before the complete right bundle branch block appeared, no tachycardia occurred.

Ventricular overdrive pacing to a cycle of 320 msec led to retrograde conduction to the atra. During the extrastimulus method at a paced cycle of 550 msec, retrograde conduction was blocked between V and H for coupling intervals of 280 msec and less. No bundle branch reentry was seen.

The patient was advised to undergo aortic valve replacement and demand pacemaker insertion, but he died suddenly before the operation.

Case 2

A 69-year-old man experienced paroxysmal tachycardia with wide QRS complexes. Lanatoside C (0.4 mg i.v.) was initially administered. A few hours later, cessation of the tachycardia was followed by complete atrioventricular (AV) block, necessitating temporary cardiac pacing, and the patient was transferred to our department. After stabilization, his ECG showed sinus rhythm with a PR interval of 0.18 second and two types of left bundle branch block (fig. 4). In the first type, the QRS complex measured 0.12 second, had a Q wave in lead III and a frontal axis of 60°. The second type occurred at a faster sinus rate with no change in the PR interval. The QRS complex was wider (0.17 second) and negative in leads III and aVF, and the frontal axis was 30°. During tachycardia, the recording showed regular ventricular complexes of the second type at a rate of 180 beats/min (fig. 4). Atrial activity was not clearly recognizable and the episode was terminated by 100 mg of i.v. lidocaine.

An electrophysiologic study was performed. The initial sinus rhythm was accompanied by a left bundle branch block with a vertical axis. The AH and HV intervals measured 80 and 85 msec, respectively. At paced cycle lengths of 600 and 550 msec, premature atrial extrastimuli associated with an H-H', interval of 480 msec were followed by the second type of left branch bundle block with left-axis deviation. The latter left bundle branch block pattern could also be produced by rapid atrial pacing at a rate of 120 beats/min (fig. 5). The HV interval remained unchanged regardless of the QRS form.

Right ventricular pacing was followed by retrograde
conduction to the atria. A His bundle electrogram was visible at the end of each QRS complex (SH interval = 200 msec). At 150 beats/min, 2:1 retrograde AV nodal block appeared. Increasingly premature ventricular depolarizations were then induced during regular pacing at a cycle length of 450 msec. As prematurity increased, the S1H2 interval gradually lengthened. When it reached 270 msec, reproducible His-Purkinje reentry (V3 phenomenon) resulted and the tachycardia was initiated (fig. 5). Tachycardia beats resembled V3, and were similar to the sinus complexes with a left axis. A His deflection occurred 100 msec before V3 and before each subsequent beat. The tachycardia rate was regular, approximately 200 beats/min. Atrial activity was often dissociated and occasionally resulted from retrograde conduction. Atrial extrastimuli delivered during tachycardia did not advance either the His bundle or the next QRS complex. However, premature stimulation of the right ventricle advanced the His potential. Termination of the tachycardia repeatedly occurred after a paced ventricular beat followed by a reentrant QRS that failed to activate the His bundle.

After this study, a cardiac demand pacemaker was implanted and the patient was given oral amiodarone (600 and then 400 mg/day). No tachycardia has occurred since.

Case 3

A 47-year-old man with aortic valve disease underwent cardiac pacemaker implantation in September 1979 because of syncope due to paroxysmal AV block. Four months later, the patient had repeated episodes of tachycardia. The ECG showed a regular tachycardia with a rate of 200 beats/min (fig. 6). The QRS complexes were wide (0.13 second) with a left bundle branch block pattern and an axis of −45°. Atrial activity appeared to be dissociated (confirmed by intracavity recordings). The tachycardia was suppressed by...
100 mg of i.v. lidocaine. In sinus rhythm, the ECG revealed complete right bundle branch block, with QRS complexes widened to 0.14 second and a frontal QRS axis of −70°. The PR interval was 0.22 second. The spontaneous episodes were always triggered in the same way: When the sinus rate slowed to less than 90 beats/min, the PR interval lengthened to 0.31 second and complete left bundle branch block appeared. The tachycardia then ensued with QRS complexes identical to the initiating beat.

The patient underwent electrophysiologic studies. The AH interval was 120 msec and the HV interval 85 msec. Atrial extrastimuli elicited during either atrial pacing (cycle length of 600 msec) or sinus rhythm produced slowing of AV nodal conduction. Atrial pacing at an increasing frequency was followed by an AV nodal Wenckebach phenomenon at 170 beats/min. Tachycardia episodes constantly interrupted normal sinus rhythm. The arrhythmia was invariably preceded by a sudden increase of the HV interval to 180 msec and by the appearance of a left bundle branch block (fig. 7). His bundle activity was identified 120 msec after each QRS complex and 180 msec before the following one, and AV dissociation was present. An additional catheter was inserted for distal recording of the AV conduction tissue. A new deflection (A-Vd), presumably originating from the right bundle branch, was detected 40 msec before the initiating QRS complexes and each subsequent tachycardia beat (fig. 7). Neither premature nor rapid atrial stimulation altered the course of tachycardia. Delivery of a ventricular extrastimulus at the same time of the AVd potential advanced the next His bundle potential. Cessation of the tachycardia was accompanied by the disappearance of the AVd potential. The arrhythmia could also be produced by paired ventricular stimulation. There was no retrograde conduction to the atria, but critical lengthening of S2H2 was followed by His-Purkinje reentry (V3). Using a basic cycle length of 500 msec, reentry always occurred at a V1V2 coupling interval of 280 msec such that S2H2 increased to 160 msec and H2V3 was 150 msec. This sequence was the prelude to a tachycardia in which the QRS complexes were identical both to the V3 reentry beat and to the ventricular complexes recorded during spontaneous attacks.

The patient was treated with oral quinidine (1.650 g/day of arabogalactane sulfate). The tachycardia attacks disappeared and the ECG showed a long PR interval (0.31 second) and a permanent left bundle branch block (fig. 6). Another electrophysiologic study after 3 days of treatment showed an HV interval of 180 msec.
No His potential was visible after the QRS complex and no spontaneous arrhythmias occurred. Premature stimulation of the right ventricle at two successive paced cycles (600 and 500 msec) failed to initiate the tachycardia.

On January 8, 1980, the patient underwent insertion of a Lillehei-Kaster aortic prosthesis. Amiodarone, 200 mg/day, was substituted for quinidine, and there has been no recurrence of arrhythmia.

**Discussion**

**Case 1**

In the first patient, the tachycardia was initiated after rapid atrial pacing. The identical appearance of the tachycardia QRS complexes and the initiating supraventricular beats argues against a mechanism originating within the ventricles. Atrial tachycardia can be excluded because atrial depolarization was not a necessary part of the tachycardia circuit. The same finding eliminates an atrio-His-AV node reentry due to the presence of bypass fibers.

AV nodal reciprocation is still possible. Failure to capture the His bundle electrogram with atrial premature depolarizations could represent lack of atrial requirement. However, the tachycardia could be reproducibly terminated with a ventricular premature depolarization that failed to reach the His bundle. This latter response also makes a His bundle tachycardia unlikely.

The occurrence of the right bundle branch block as a prerequisite for arrhythmia initiation provides support for bundle branch reentry. The following phenomena can be assumed. During right bundle branch block, excitation is conducted to the ventricles by the left bundle branch only. The impulse then crosses the septum, penetrates the now-recovered right bundle branch retrogradely, and rejoins the His bundle, from which it reenters the left bundle branch. The QRS left-axis deviation during tachycardia suggests that the left anterior fascicle is excluded from the circuit as a result of block. The mechanism therefore would be an excitation loop whose antegrade and retrograde limbs are, respectively, the left posterior fascicle and the right bundle branch, a loop closed by ventricular myocardium and the His bundle bifurcation.

A somewhat different circuit can also be imagined. Its retrograde part would include Mahaim fibers connecting the AV node or the His bundle to the ventricular myocardium.20 These accessory fibers would conduct the impulse in a retrograde fashion only.21,22 This latter hypothesis does not account for the QRS deformation preceding the onset of arrhythmia.

**Case 2**

The present discussion concerns a regular tachycardia with wide QRS complexes that show a left bundle branch block pattern. The AV dissociation excludes both atrial tachycardia and apart from exceptional cases,23 reciprocating junctional tachycardia. Recording sinus beats similar to the tachycardia beats sheds doubt on a true ventricular tachycardia with retrograde activation of the His bundle. The presence of a His potential associated with each ventricular complex is compatible with either His bundle tachycardia or reentry using Mahaim fibers retrogradely. The mode of arrhythmia initiation, however, favors bundle branch reentry. Premature ventricular stimulation delivered during regular right ventricle pacing caused tachycardia if it first gave rise to the V3 phenomenon.24 That V3 and the succeeding beats are identical suggests a common mechanism. Recent studies have provided definite evidence in support of bundle branch reentry as the mechanism of the V3 phenomenon.25,26 Furthermore, the cessation of tachycardia was associated with the blockade of ventricular premature complexes within the bundle branch system.

In our patient two kinds of left bundle branch block were present in sinus rhythm. In the one with the normal axis, conduction is considered to be symmetrically slowed over both left bundle branch subdivisions. The left bundle branch block with a left axis was tachycardia-dependent and block is thought to occur within the anterior fascicle.27 Conduction to the ventricles continues by the right bundle branch, and more
slowly by the left posterior fascicle. The transseptal wave coming from the right ventricle can spread through the nonexcited anterior wall and enter the terminals of the left anterior fascicle. The development of this process leads to further increase in QRS duration, probably in combination with some parietal conduction disorder. The impulse traverses retrogradely the anterior fascicle and blocks in a proximal refractory zone. During tachycardia, a similar circuit can be invoked. The impulse continues its retrograde course in the left anterior fascicle (unidirectional block) and rejoinsthe His-bundle bifurcation to reexcite the ventricles. The HV interval exceeds the sinus HV interval, suggesting that conduction in the antegrade limb of the circuit occurs in the relative refractory period of the bundle branches.

A variant mechanism implies the presence of a slow conduction area within the predivisional segment of the left bundle branch. The development of block at this site accounts for the widening of the QRS complexes and would be a determinant in the genesis of bundle branch reentry.

Case 3

In the third patient, several facts suggest bundle branch reentry as the tachycardia mechanism. First, the patient presented with an alternating bundle branch block. As soon as a sinus beat exhibited a left bundle branch block pattern, tachycardia was initiated, with identical QRS complexes and AV dissociation. Second, during paired ventricular stimulation, tachycardia followed the V3 phenomenon and had identical beats to V3. During tachycardia, premature ventricular depolarization synchronous with the Avd deflection advanced the subsequent His bundle potential. The premature beat found a pathway to the His bundle after the abnormal impulse had entered or as it was just entering the right bundle branch. One can assume that, quite apart from the possible existence of Mahaim fibers, the left bundle branch constitutes that return pathway and that there is free access to it at the time of premature stimulation. Finally, block between His bundle and right bundle branch was required to terminate the tachycardia after ventricular premature stimulation.

In sinus rhythm, excitation reaches the ventricles by the left posterior fascicle only. Despite a right bundle branch block pattern, slow conduction must be assumed in the right bundle branch. The impulse is blocked in the left anterior fascicle. When the QRS pattern changes from right to left bundle branch block, persistent conduction in the right bundle branch is unmasked and becomes operative. The left-axis deviation might mean that the posterior fibers participate in left ventricular activation. A circus movement is initiated by the transseptal wave entering the left anterior fibers retrogradely. Slow conduction in the right bundle branch had enabled these fibers to recover. The impulse is free to continue on its way up to the His bundle bifurcation and return to the ventricles. The right bundle branch appears to be the weak point in the circuit, as shown by the termination of tachycardia episodes, which always followed recording of a His bundle potential.

All our patients presented with advanced infra-Hisian conduction disturbances, as proved by the long HV interval in sinus rhythm, combined in two cases with unilateral or bilateral bundle branch block pattern. Two patients had aortic valve disease with calcification, which is often associated with damage to the His bundle and the proximal bundle branches. Because of an electrocardiographic pattern suggesting a supraventricular mechanism, two of our patients were initially and unsuccessfully treated with drugs like digitalis and verapamil. Lidocaine terminated the arrhythmias in cases 2 and 3. Episodes were prevented by quinidine in case 3, in whom a subsequent electrophysiologic study showed the formation of a bidirectional block in the left bundle branch, i.e., in the retrograde limb of the circuit. As demonstrated by Reddy and Lynch, retrograde blockade of the impulse is one of the mechanisms by which procainamide can abolish reentry within the His-Purkinje system; the other, less frequent effect is antegrade block of the circulating wave. The same authors showed that in certain cases, procainamide only made initiation of reentry by premature ventricular stimulation more difficult, either by lengthening the required SH delay or by increasing the effective refractory period of the ventricle. The effectiveness of amiodarone in cases 2 and 3 may have been due to an increase in the refractory periods of the His-Purkinje system and ventricular myocardium. However, little information is available about the effects of amiodarone on bundle branch reentry. Conversely, drug-induced shortening of the refractory period within the bundle branches may help to abolish a macrocircuit by improving conduction, as has been shown with diphenylhydantoin and lidocaine. Finally, antiarhythmic agents may be effective by abolishing extrastoles initiating reentry. Surgical section of the bundle branches has been suggested as a treatment for refractory reentrant tachycardia, but its effectiveness has not been demonstrated.

References

Bundle branch reentry: a possible mechanism of ventricular tachycardia.
P Touboul, G Kirkorian, G Atallah and P Moleur

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