Reciprocating Tachycardia Using Bilateral Anomalous Pathways: Electrophysiologic and Clinical Implications

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SUMMARY A patient who had disabling supraventricular tachycardia showed electrophysiologic evidence of coexistent right and left Kent bundles. The supraventricular tachycardia was accelerated by sequential antegrade and retrograde conduction over the right and left Kent bundles, respectively. Spontaneous blocking of the conduction through the right bundle was associated with a slowing of the tachycardia and with the appearance of right bundle branch block (RBBB). Antegrade conduction was through the left bundle branch and was followed by an early retrograde atrial activation through the left Kent bundle, with consequent shortening of the ventriculoatrial (VA) interval. Thus, the coexistence of bilateral Kent bundles can be suspected whenever shortening of the VA interval in external electrocardiographic recordings occurs in the presence of a tachycardia with a RBBB pattern in a patient with right-sided preexcitation.

Our data also showed that bilateral accessory pathways may have different electrophysiologic properties and that unequal response to antiarrhythmic drugs may be expected.

IN MOST CASES of Wolff-Parkinson-White (WPW) syndrome, reciprocating tachycardias involve consecutively an anomalous pathway in one direction and the normal conduction system in the opposite direction. The coincidence of two anomalous pathways inserted at each atrioventricular (AV) ring — bilateral pathways — constitutes a very uncommon situation and has important electrophysiologic and clinical implications. If both accessory pathways become excited consecutively, a circuit obviating the slower conduction through the AV node may set the basis for a more rapid tachycardia. Experimental data suggest that these accessory pathways may have different electrophysiologic properties, and antiarrhythmic drugs may depress conduction through them to different degrees, resulting in unsatisfactory control of the tachycardias. We documented these characteristics in a patient who had electrophysiologic evidence of bilateral anomalous pathways.

Case Report

A 37-year-old female was referred for investigation of disabling paroxysmal supraventricular tachycardia refractory to oral administration of 10 mg/kg of amiodarone and 4 mg/kg of verapamil. The ECG recorded during sinus rhythm (fig. 1) met the criteria for lateral or posterior right-sided preexcitation. Physical examination and routine analysis were normal and the patient was studied in the postabsorptive, nonsedated state.

Four multipolar catheters were positioned fluoroscopically in the heart for recording and stimulating. Three of them were introduced percutaneously through the right femoral vein and positioned in the right atrium (quadripolar catheter), in the vicinity of the His bundle (bipolar) and at the apex of the right ventricle (bipolar). The fourth catheter (quadripolar) was introduced through an antecubital vein and positioned in the coronary sinus. Intracavitary bipolar or unipolar electrograms, as well as surface leads I, II, III and V1, were obtained simultaneously on an Elema Minograf 82 ink-jet recorder, at a paper speed of 100 mm/sec. Electrical stimulation was delivered by a Devices Ltd type 4279 isolated stimulator with square-wave, 2-msec pulses at twice the diastolic threshold.

After the introduction of the catheters, studies were performed to assess the presence of preexcitation, the location and mode of conduction of the anomalous pathway, the refractory periods, the mode of induction, termination and mechanism of the tachycardia, and the effects of intravenous procainamide. The antegrade and retrograde effective refractory periods of the normal and accessory pathways were determined by programmed atrial or ventricular premature beats (A2 or V2) delivered every eighth paced beat (A1 or V1). They were defined as the longest A1 A2 or V1 V2 that failed to conduct through the normal or the accessory pathways.

Electrophysiologic studies during sinus rhythm (fig. 2A) revealed an AH interval of 80 msec and a negative delta wave in lead V1, preceding the inscription of the His bundle electrogram by 5 msec. During atrial stimulation at various sites, the maximal degree of preexcitation was attained at the lateral margin of the right atrium (fig. 2B), indicating a close proximity of the right Kent bundle. Stimulation at different sites in the coronary sinus was unable to induce left-sided preexcitation, but instead nearly normalized the ventricular activation (fig. 2C).

Retrograde atrial activation during right ventricular stimulation (fig. 3A) was first recorded at the distal coronary sinus and reached consecutively the low
atrial septum and the low lateral right atrium. Such a left-to-right sequence indicates retrograde conduction through an accessory pathway.\textsuperscript{12} When ventricular pacing was accelerated, the ventriculoatrial (VA) conduction remained constant until a sudden 2:1 retrograde VA block occurred. These findings indicate that the right-sided pathway conducted only in antegrade direction, whereas the left-sided pathway conducted only in the retrograde direction.

The effective refractory periods at a basic cycle length of 380 msec were 300 msec for antegrade conduction over the right Kent bundle and 270 msec for retrograde conduction over the left Kent bundle.

Two alternating forms of regular paroxysmal supraventricular tachycardia were documented in this patient (fig. 4): a tachycardia with wide QRS complexes showing right-sided type of preexcitation at a rate of 180 beats/min and a tachycardia with right bundle branch block (RBBB) configuration at rates of 140 beats/min. Critically timed extrastimuli delivered to the atria could initiate or terminate both types of tachycardia. Circus movement of the excitation was the basis for these arrhythmias. In the case of wide QRS tachycardia (fig. 3B), antegrade activation took place over the right-sided accessory pathway and the atria were reached in a retrograde fashion through the left-sided Kent bundle (retrograde left-to-right atrial activation sequence). Antegrade conduction over the normal AV pathway cannot be asserted in view of the absence of a visible H potential preceding the ventricular electrograms. During tachycardia with RBBB configuration (fig. 3C), the ventricles were activated through the normal AV system and the atria were again reached by retrograde conduction through the left bypass tract.

The VA interval decreased during the RBBB tachycardia because the ventricular activation began with the left ventricle and then the left Kent bundle, and the atria were activated much earlier than in the case of wide QRS tachycardia, where ventricular ac-

**Figure 1.** ECG showing features of right-sided preexcitation.

**Figure 2.** Effects of atrial pacing site on the degree of preexcitation. Electrocardiographic leads I, II, III and V\textsubscript{1}, low right atrial (LRA) electrogram, and His bundle electrogram (HBE) are shown. S represents the stimulus artifact and H the His bundle potential. (A) In sinus rhythm the ventricles were activated 5 msec before the His bundle, indicating the existence of ventricular preexcitation. During atrial pacing at various sites, the shortest S–delta wave interval, which reflects the closest proximity of the bypass tract, was obtained at the lateral margin of the right atrium. At this level (B), the acceleration of the pacing produced progressive slowing of the intranodal conduction (H jumps progressively into the ventricular spike) with consequent increase of the ventricular preexcited area (progressive widening of the QRS complexes showing right-sided type of preexcitation). Stimulation at the distal coronary sinus nearly normalized the ventricular activation (C).
tivation first involved the right ventricle via the right-sided accessory pathway.

The interval between the three atrial signals was constant during right ventricular stimulation as well as during both types of tachycardia. The retrograde left-to-right atrial sequence was reflected on lead I by negative retrograde P waves (arrows, fig. 4).

Procainamide, 10 mg/kg i.v., produced blocking of the right-sided Kent bundle, and only the RBBB type of tachycardia could still occur. However, both types of tachycardia were successfully controlled with a daily oral maintenance dose of 50 mg/kg of procainamide.

Discussion

Bilateral accessory AV muscle bundles (Kent fibers) inserted at each AV ring have rarely been demonstrated in anatomic studies. In the few clinical reports available, this abnormality was suspected

Figure 3. Mechanism of the tachycardias. Similar leads and ECGs are shown together with the unipolar distal coronary sinus (DCS) electrogram. Arrows indicate the intrinsic deflections of the atrial electrograms. (A) Right ventricular stimulation revealed an abnormal retrograde left-to-right atrial activation sequence (DCS-low atrial septum (HBE)-LRA), indicating retrograde conduction through a left Kent bundle. (B) Tachycardia with right-sided Wolff-Parkinson-White configuration at a rate of 180 beats/min involving the right-sided bypass tract as antegrade conduction and the left accessory pathway as retrograde conduction. (C) Tachycardia with right bundle branch block (RBBB) pattern at a rate of 140 beats/min with slow antegrade conduction over the normal atrioventricular system (AH interval 250 msec) and retrograde conduction again through the left bypass tract. The ventriculoatrial intervals (V-DCS intervals, below) are shorter during this type of tachycardia. LRA = low right atrial electrogram; HBE = His bundle electrogram.

Figure 4. Spontaneous conversion of a tachycardia with right-sided Wolff-Parkinson-White configuration at 180 beats/min to a slower tachycardia at 140 beats/min with right bundle branch block (RBBB) configuration. Electrocardiographic leads are shown together with a diagram. The sinus rhythm at 100 beats/min (first three beats) was interrupted by an atrial premature beat (first asterisk), which initiated the rapid form of the tachycardia (beats 4-11). The ventricles were activated through the right-sided Kent bundle (RK) and the atria were reached by retrograde conduction over the left-sided Kent bundle (LK). The normal His-Purkinje pathway (H) was probably refractory because of the rapid rates. A ventricular premature depolarization (twelfth QRS complex) was followed by a slowing of the tachycardia. In this case, conduction to the ventricles occurred over the H pathway with RBBB configuration (last four beats) and the atria were again activated through the LK. Retrograde concealment into the RK beginning with the twelfth beat could have been responsible for antegrade blocking of this pathway during the slower form of the tachycardia.
Electrophysiologic Implications

It is generally accepted that slowing of a tachycardia with right-sided preexcitation morphology when RBBB occurs indicates late retrograde participation of a right-sided Kent bundle in the circuit of the tachycardia, because the activation of the right ventricle becomes actually delayed by the RBBB. However, when two accessory pathways are involved in the mechanism of a right-sided type of preexcitation tachycardia, as in the present case, the slowing of the rate coinciding with the appearance of RBBB may have a different mechanism. Indeed, as shown in figure 3, retrograde conduction took place over a left-sided accessory pathway, and slowing of the tachycardia was due to an abnormal intranodal slowing of the antegrade conduction (AH interval 250 msec) now traveling exclusively through the normal AV system, in view of the disappearance of the right-sided preexcitation (normal HV interval).

The finding that retrograde atrial activation during tachycardia with RBBB configuration occurs earlier than that during tachycardia with right-sided preexcitation has not been previously documented as being indicative of bilateral pathways. In this situation, ventricular activation was initiated within the left chamber, and hence the left-sided Kent bundle was promptly reached and the atria were rapidly activated (fig. 5).

Noninvasive techniques for recording atrial electrograms, such as transesophageal recordings performed during tachycardia, would probably have revealed such differences in VA conduction and would then have proved useful in detecting the presence of bilateral anomalous pathways. Conventional ECG might also contribute to the diagnosis, because retrograde P waves in lead I during tachycardia suggest retrograde participation of a left-sided bypass tract.

Clinical Implications

This study further shows that patients with bilateral anomalous pathways may develop a circus movement

![Diagram of Heart with Tachycardia](image-url)
tachycardia involving both anomalous connections. Such a situation deserves particular attention because as the normally delaying AV nodal structure is obviated, the frequency of the tachycardia may reach very high rates. On the other hand, only anti-arrhythmic drugs that would block the accessory pathways more than the normal AV conduction should be used to prevent the possibility of tachycardias involving the normal conduction system together with either one of the accessory connections. This could have accounted for the beneficial effects obtained with procainamide. Amiodarone and verapamil failed to prevent the tachycardias in this patient. The lack of consistent depressant effects of amiodarone on retrograde conduction through the accessory pathways and the more pronounced blocking effects of verapamil on the AV node rather than on the accessory connections could not possibly have prevented the circus movement tachycardia involving the two accessory pathways. The unequal mode of conduction of both Kent bundles further supports the hypothesis that the two accessory connections may have different electrophysiologic properties.

The demonstration of bilateral Kent bundles in a patient has important technical implications whenever surgical ablation of the accessory connections must be considered. In this regard, Denes et al. have already pointed out the relative contraindication to surgical ablation of only one accessory connection in these patients.

References

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Circulation. 1980;62:657-661
doi: 10.1161/01.CIR.62.3.657

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