CASE REPORT

Mechanism of Reciprocating Tachycardia Initiated During Sinus Rhythm in Concealed Wolff-Parkinson-White Syndrome

Report of a Case

RUEY J. SUNG, M.D., AGUSTIN CASTELLANOS, M.D., HENRY GELBAND, M.D., AND ROBERT J. MYERBURG, M.D.

SUMMARY Reciprocating tachycardia in a patient with a left-sided atrioventricular accessory pathway (AP) (Kent bundle, type A) capable only of ventriculo-atrial (V-A) transmission is described. The V-A AP is established as an essential link of the tachycardia circuit, as evidenced by: 1) retrograde atrial activation of the left atrium (LA) 60 msec or more before the low and high right atrium during reciprocating tachycardia and during V-A conduction; 2) the absence of refractory-dependent delay in V-A conduction time with progressively premature ventricular stimulation, characteristic of retrograde conduction through an AP; and 3) the absence of antegrade conduction through the Kent bundle during sinus rhythm, reciprocating tachycardia, pacing from either atrium, or during induced atrial flutter-fibrillation. The onset of the tachycardia was unique in that it could be initiated and perpetuated during sinus rhythm, without a triggering mechanism of an atrial or ventricular extrasystole. The interplay of the following two events seemed to favor the initiation of the tachycardia: 1) shortening of the atrial cycle length causing a decrease in the refractory period of the LA and/or the AP; and 2) the development of rate-dependent left bundle branch block, delaying impulse arrival at the ventricular end of the AP. These observations described an additional mechanism of reciprocating tachycardia in patients with the Wolff-Parkinson-White syndrome.

IN WOLFF-PARKINSON-WHITE (WPW) SYNDROME, the classic electrocardiographic features, as well as the electrophysiological response to atrial stimulation studies, will not be present when the anomalous Kent bundle is capable only of ventriculo-atrial (V-A) conduction (concealed WPW syndrome). Such patients may have reciprocating tachycardia, in which antegrade conduction proceeds across the atrioventricular (A-V) node and His bundle, producing QRS morphology similar to that seen in sinus rhythm. Using the intracardiac recording technique and extra-stimulus method, the analysis of the electrophysiological findings in a patient with recurrent tachycardia revealed that a left-sided accessory pathway (Kent bundle, type A) with antegrade unidirectional block was an essential link of the tachycardia circuit, and that the initiation of the tachycardia appeared to depend upon the relationship between the impulse arrival at the posterobasal left ventricle and the refractory period of the left atrium and/or the accessory pathway. Factors influencing the electrophysiological relationship between these tissues may, therefore, precipitate and perpetuate the onset of reciprocating tachycardia without the usual triggering mechanism provided by an atrial or ventricular extrasystole.

Methods

The patient was informed of and consented to the study. At the time of the study, the patient was not receiving digitalis or any antiarrhythmic drugs, and the study was performed in a postabsorptive, nonseated state.

High right atrial (HRA) activity was recorded through the proximal pair of electrodes of a hexapolar electrode catheter (Berkvits-Castellanos USCI #283-202) and left atrial (LA) activity was recorded with a quadripolar electrode catheter (USCI #5655) placed in the distal coronary sinus. Because of its proximity to the left ventricular wall, the quadripolar catheter electrode located in the distal coronary sinus also recorded a bipolar electrogram from the posterobasal portion of the left ventricular muscle (VP), coinciding with the terminal phase of the QRS complex. Using a conventional technique, His bundle electrograms (HBE) were obtained through a tripolar electrode catheter, from which low right atrial (LRA) activity was recorded as well. The intracardiac electrograms were then displayed simultaneously with standard surface ECG leads I, II, and V1 on a multichannel oscilloscopic photographic recorder (Electronics for Medicine, DR-16, White Plains, New York) at a paper speed of 100 or 150 mm/sec, using filter settings between 40 and 500 Hz and one second timelines. Bipolar atrial and ventricular stimulation was performed through the remaining pairs of electrodes of the hexapolar and quadripolar electrode catheters. The stimuli consisted of rectangular impulses of 2.0 msec duration and approximately twice diastolic threshold, and were applied both in the form of continuous pacing and premature stimulation after each eighth spontaneous or paced beat.

Results

Case Report

A 37-year-old white male related a history of frequent episodes of palpitations from early childhood. Recurrent
paroxysmal tachycardia had been documented for the past five years, and at no time had ventricular pre-excitation (an ECG delta wave) been recorded. On admission, he presented with profound weakness associated with rapid palpitations. Blood pressure was 110/70 mm Hg, and the physical findings were unremarkable except for borderline cardiomegaly. The electrocardiogram revealed a tachycardia at a rate of 150 beats/min. The QRS complex during both sinus rhythm and tachycardia had a complete left bundle branch block (LBBB) pattern (QRS duration 145 msec). Carotid massage and electrical countershock would temporarily interrupt the tachycardia.

Attempts at pharmacologic control of the rhythm disturbance were made initially with a 50 mg bolus of i.v. lidocaine, followed by continuous drips at a rate of 2–4 mg/min. Since this was unsuccessful in controlling the rhythm disturbance, intravenous procainamide boluses totaling 700 mg over a period of 70 min were administered without success. The patient was then digitalized intravenously with 1.75 mg of digoxin over 24 hours, and oral quinidine (1.2 g/day in divided doses) was given. This was also unsuccessful, and the patient was then given intravenous propranolol (a total of 10 mg in divided doses). When all of these forms of pharmacologic therapy proved unsuccessful, a transvenous right ventricular endocardial pacing catheter was inserted, and the arrhythmia finally controlled. An R-to-stimulus coupling interval of 330 msec terminated the tachycardia, followed by continuous right ventricular pacing at a rate slower than that of the tachycardia (110 beats/min compared to 150 beats/min).

Throughout the electrophysiologic study, no ventricular pre-excitation was evident during runs of sinus rhythm or tachycardia. Moreover, stimulation of either atrium did not induce ventricular pre-excitation. In sinus rhythm, at a cycle length of 790 msec, the P-R interval was 165 msec and the QRS duration 145 msec with a complete LBBB pattern. The P-A, A-H, and H-V intervals were normal (30, 90, and 45 msec respectively).

When a premature right ventricular stimulus was delivered during sinus rhythm, the LBBB was found to be rate-dependent (related to shortening of the cycle length). Figure 1 illustrates that the QRS complex of the sinus beat following a pause of 1,300 msec after an induced premature right ventricular depolarization narrowed from 145 to 90 msec with disappearance of the LBBB pattern. As the sinus cycle length shortened, the LBBB pattern reappeared. During this phase of the QRS complex changing between normal and complete LBBB pattern, the arrival of impulse at the posterobasal portion of the left ventricle (Vp), as measured from the onset of the QRS complex to the onset of Vp electrogram in LA lead was 60 msec without and 115 msec with LBBB pattern (the fourth and sixth QRS complexes respectively).

Figure 1. Rate-dependency of the LBBB pattern. The QRS complex narrowed its duration from 145 to 90 msec with simultaneous disappearance of LBBB pattern after a long pause of 1300 msec induced by a premature right ventricular depolarization. As the sinus cycle length shortened from 900 to 845 msec, the LBBB pattern reappeared. The dash lines demarcate the onset of the QRS complexes. The activation time of the posterobasal left ventricle measured from the onset of the QRS complex to the onset of Vp electrogram in LA lead was 60 msec without and 115 msec with LBBB pattern (the 4th and 6th QRS complexes respectively). More than 20 episodes of spontaneous reciprocating tachycardia were observed during the study, and these episodes all emerged from a sinus rhythm rather than being triggered by an atrial or ventricular extrasystole. However, premature stimulation of either atrium, and of the right ventricle, could also precipitate the onset of a sustained tachycardia. Of note was the observation that even late premature atrial depolarizations which induced only minimal A-V nodal conduction delay could trigger the onset of a run of tachycardia. Critical analysis of the onset of tachycardia revealed that the principal determinant for its initiation was

![Figure 1](https:// Circulation.ahajournals.org/doi/fig/10.1161/01.CIR.60.3.339)
the relative time of impulse arrival at the posterobasal portion of the left ventricle (location of the ventricular end of the accessory pathway). For example, in figure 4, when HRA was prematurely stimulated, the prematurely conducted beat which precipitated the onset of tachycardia induced only a 10 msec prolongation of A-V nodal conduction time (the A-H interval increased from 90 to 100 msec). However, the interval between the LA and Vp electrograms of this prematurely conducted beat was 198 msec, which exceeded the LA-Vp interval (128 msec) of the preceding sinus beat by 70 msec.

A similar phenomenon was observed when the LA was prematurely stimulated. During the tachycardia, the LA-Vp intervals were maintained at 330–340 msec; and the reciprocal tachycardia circuit could easily be identified as the Vp electrogram of the prematurely conducted beat (the second QRS complex) was immediately followed by LA-LRA-HRA sequence of retrograde atrial activation.

**FIGURE 2.** Constant one-to-one ventriculo-atrial conduction during right ventricular pacing (cycle length 680 msec). Note LA activation preceded HRA and LRA activation by 60 and 70 msec, respectively.

**FIGURE 3.** The ventriculo-atrial conduction time remained unchanged despite progressively premature right ventricular stimulation (a coupling interval of 420 msec in the upper panel and of 265 msec in the lower panel). V-LA = 130 msec; V-LRA = 190 msec; and V-HRA = 200 msec.

**FIGURE 4.** Premature stimulation of the high right atrium (HRA) initiated the onset of a sustained tachycardia. The prematurely conducted beat (the second QRS complex) induced only slightly A-V nodal conduction delay (100 msec compared to 90 msec of the preceding sinus beat), but its corresponding A-Vp interval was 198 msec which exceeded that of the preceding sinus beat (the first QRS complex) (128 msec) by 70 msec. Note there was appearance of complete LBBB pattern with prolongation of the QRS duration in the prematurely conducted beat. In addition, the reciprocating tachycardia circuit could easily be identified as the Vp electrogram of the prematurely conducted beat (the second QRS complex) was immediately followed by LA-LRA-HRA sequence of retrograde atrial activation.
cating tachycardia, therefore, was sustained (at cycle length of 380 msec). The increase in the LA-Vp interval at the onset of tachycardia initiated by premature atrial stimulation was apparently due to a summed effect of 1) earlier activation of the LA (when the LA was prematurely stimulated), 2) with or without A-V nodal conduction delay (slight A-H interval prolongation), and 3) the development of rate-dependent complete LBBB. Consequently, the impulse arriving at the posterobasal portion of the left ventricle (Vp) was sufficiently late to exceed the refractory period of the accessory pathway and/or the LA, and enter these structures, establishing a conduction circuit for the reciprocating tachycardia. The sequence of the events described above could be easily identified at the onset of tachycardia (fig. 4), in which the Vp electrogram was immediately followed by LA-LRA-HRA retrograde sequence of atrial activation (note negative P waves in lead II during the tachycardia). Unfortunately, a sinus rhythm could not be maintained to allow measurement of the refractory period of the LA and the accessory pathway at various cycle lengths by the extra-stimulus method, 14-16 and intermittent right ventricular stimulation or fixed rate pacing was necessary to terminate the tachycardia.

In order to substantiate the postulated mechanism for the initiation of the reciprocating tachycardia, those episodes of tachycardia emerging spontaneously from a sinus rhythm were analyzed in detail (fig. 5). Since the morphology of the QRS complex and the sequence of activation between the atria and the ventricles during tachycardia were similar whether the tachycardia was initiated by premature atrial or ventricular depolarizations or during sinus rhythm, two forms of reciprocating tachycardia occurring in the same patient seems unlikely. As shown in figure 5, the LA-Vp interval of the sinus beat initiating the onset of reciprocating tachycardia (the fifth QRS complex) had the same value (180 msec) as those of the preceding sinus beats. Nevertheless, without exception, a gradual decrease in the sinus cycle length (from 790 to 750 msec in figure 5) was a requisite condition for initiating tachycardia in each instance.

Although changes in the refractory period of the atrium and the accessory pathway in relation to the variations of the

![Figure 5](http://circ.ahajournals.org/)

**Figure 5.** Reciprocating tachycardia initiated during sinus rhythm without a triggering premature atrial or ventricular extrasystole. The A-Vp interval of the sinus beat (the 5th QRS complex) was 180 msec, the same as those of the preceding sinus beats; however, it was noted that gradual shortening of the sinus cycle length (from 790 to 750 msec) was a requisite condition for spontaneous onset of reciprocating tachycardia.

![Figure 6](http://circ.ahajournals.org/)

**Figure 6.** Sudden cessation of an atrial flutter-fibrillation initiated the onset of reciprocating tachycardia. The atrial cycle length during atrial flutter-fibrillation was 188 msec. Note that the last QRS complex of the atrial flutter-fibrillation conducted beat (the 4th complex in the figure) narrowed its duration with disappearance of complete LBBB pattern because of the preceding long pause; however, it initiated the onset of reciprocal tachycardia as its corresponding Vp electrogram was immediately followed by LA-LRA-HRA sequence of retrograde atrial activation.
sinus cycle length remain speculative in this case, it has been demonstrated that shortening of the atrial cycle length decreases the refractory period of the atrium and the refractory period of the accessory pathway in both A-V and V-A conduction.\textsuperscript{14} It could thus be reasonably postulated that a decrease in the refractory period of the LA and the accessory pathway would allow a sinus impulse arriving at Vp to propagate retrogradely through the accessory pathway and the atrium without interruption. The development of LBBB also favors the proposed mechanism by delaying the impulse arrival at the ventricular end of the accessory pathway. It may be argued that the sixth QRS complex in figure 5 resulted from a premature atrial depolarization, and triggered the onset of the tachycardia. However, the intimate relationship between the Vp and the LA electrograms during the reciprocating tachycardia was also present in the fifth QRS complex (i.e., the last “sinus” complex in figure 5), and whenever reciprocating tachycardia developed during a sinus rhythm with a gradual decrease in cycle length, this relationship between Vp and LA electrograms was observed. Therefore, we proposed that the reciprocating tachycardia was triggered by the effects of shortening of the sinus cycle length rather than a premature atrial depolarization.

Atrial flutter-fibrillation was deliberately induced several times during atrial pacing.\textsuperscript{15} The average ventricular response during atrial flutter-fibrillation was 130 beats/min. Invariably, a sustained reciprocating tachycardia emerged following the abrupt cessation of atrial flutter-fibrillation (fig. 6). The atrial cycle length during flutter-fibrillation ranged from 180 to 200 msec. A marked decrease of the refractory period of the LA and the accessory pathway as a result of the very short atrial cycle length during atrial flutter-fibrillation was likely to be responsible for the initiation of reciprocating tachycardia under these circumstances. This additional observation provides further evidence that shortening of the atrial cycle length, and hence, conceivably, a decrease in the refractory period of the LA and/or the accessory pathway, may precipitate the onset of reciprocating tachycardia.

In summary, the electrophysiological data recorded in this patient indicated the presence of a left-sided accessory pathway (Kent bundle, type A) which conducted only in the ventriculo-atrial direction. The initiation of reciprocating tachycardia was unique in that it appeared to depend upon the relative time of impulse arrival at the ventricular end of the accessory pathway in relation to the refractory period of the LA and the accessory pathway. In most instances, the tachycardia used the right bundle branch as a part of the conduction circuit because of the rate-dependent LBBB (fig. 7A, B). Right ventricular stimulation, by virtue of its concealed depolarization of the right bundle branch, could easily terminate the tachycardia.

Since the reciprocating tachycardia was refractory to medical therapy, and surgical intervention to disrupt the A-V node-His bundle structure followed by a permanent pacemaker implantation would not be considered by the patient, an artificial pacemaker at a fixed rate of 90 beats/min was implanted transvenously in the right ventricular endocardium. The patient has subsequently remained asymptomatic and free of tachyarrhythmia since discharge (seven months).

Discussion

For the purpose of localizing an accessory pathway, it is essential to record the sequence of retrograde atrial activation during reciprocating tachycardia and/or during ventriculo-atrial conduction induced by ventricular stimulation.\textsuperscript{6, 9, 10, 12} An electrode catheter in the coronary sinus position registers the LA electrogram and the postero-basal activity of the left ventricle (Vp) as well,\textsuperscript{7-10} and thus is useful in delineating the presence of a left-sided accessory pathway. The following observations suggest that a left-sided Kent bundle (type A) with antegrade unidirectional block was an essential link of the reciprocating tachycardia circuit in this patient:

1) Abnormal sequence of retrograde atrial activation\textsuperscript{6, 9, 10, 12} during reciprocating tachycardia and during ventriculo-atrial conduction induced by ventricular stimulation, in which the LA was activated 60 to 70 msec before the LRA in the vicinity of the A-V node and HRA.

2) Constant ventriculo-atrial conduction time without refractory-dependent delay despite progressively premature ventricular stimulation and fast ventricular response (150/min) during tachycardia (V-LA = 130 msec; V-LRA = 190 msec; V-HRA = 200 msec) is characteristic of retrograde conduction through an accessory pathway.\textsuperscript{16}

3) Presence of antegrade conduction block of the accessory pathway, since ventricular pre-excitation was not evident during sinus rhythm, reciprocating tachycardia, or atrial flutter-fibrillation, and could not be induced by pacing from either atrium.

Unidirectional block is a well established electrophysiological phenomenon. The experimental work of Fuente et al.\textsuperscript{15} provides an electrophysiological background for such a phenomenon to occur in the accessory pathway of the WPW syndrome. Clinical studies\textsuperscript{6-8} have confirmed this observa-

![Figure 7. Schematic drawing of the conduction pathways during A) sinus rhythm (SR) and B) reciprocating tachycardia (RT). Because of antegrade conduction block in the left-sided Kent bundle (K), the impulse proceeds across the atrioventricular node (AVN) and His bundle (HB). In the presence of rate-dependent conduction block occurring in the left bundle branch (LBB), the impulse propagates through the right bundle branch (RBB) and traverses the interventricular septum (IVS) to activate the left ventricle (LV). An appropriate interplay of impulse arrival time at the postero-basal LV (Vp) and the refractory period of the Kent bundle and the left atrium (LA) would allow the impulse to retrogradely enter those structures, establishing a reciprocating tachycardia (RT) circuit.](http://circ.ahajournals.org/doi/abs/10.1161/01.CIR.54.2.342)
tion and suggested that its occurrence may be more frequent than is generally realized. Without recording atrial activity close to the atrial end of the accessory pathway (LA and lateral RA electrograms in WPW syndrome, types A and B respectively), the electrophysiological findings in patients with an anomalous Kent bundle which conducts only retrogradely would be similar to those of A-V nodal re-entry. Differentiation between these two different mechanisms of reciprocating tachycardia is of clinical importance, since therapeutic approaches may differ. The conduction circuit observed during reciprocating tachycardia in this patient involved the A-V node-His bundle, the right bundle branch, the left ventricular muscle, the Kent bundle, and the LA (fig. 7). The likelihood of the tachycardia resulting from A-V nodal or bundle branch re-entry seems unlikely. In both of these situations, the sequence of retrograde atrial activation via the A-V node would result in the LRA being activated before or simultaneously with the LA, and then followed by HRA.

In most patients with the WPW syndrome, the antegrade effective refractory period of the accessory pathway is long relative to that of the A-V node. Therefore, the initiation of reciprocating tachycardia is usually related to antegrade conduction block of a premature beat in the accessory pathway. In contrast, in the presence of pre-existing antegrade unidirectional block in the accessory pathway, all the impulses will be propagated through the A-V node-His bundle route to reach the ventricular end of the accessory pathway (fig. 7). Factors influencing the refractory period of the atrium and the accessory pathway, as well as the relative time of impulse arrival at the ventricular end of the accessory pathway, are therefore critical in the initiation of tachycardia. Identifiable factors recorded in this study include: 1) shortening of the atrial cycle length following a spontaneous increase in sinus rate and induced atrial flutter-fibrillation, possibly thereby decreasing the refractory period of the atrium and the accessory pathway; 2) early activation by premature stimulation of the atrium ipsilateral to the accessory pathway; and 3) antegrade conduction delay such as prolongation of A-V nodal conduction time and the development of bundle branch block ipsilateral to the accessory pathway, thereby postponing impulse arrival at the ventricular end of the accessory pathway. Appropriate interplay of these factors may initiate and perpetuate the onset of reciprocating tachycardia without the usual triggering mechanism provided by an atrial or ventricular extrasystole. In contrast to the usual type of paroxysmal A-V nodal reciprocating tachycardia, Coumel et al. have recently demonstrated a “permanent” form of supraventricular tachycardia in which the tachycardia is usually precipitated by spontaneous acceleration of the sinus rate and the P-R interval preceding the tachycardia is always identical to those of basic sinus beats. The case presented herein fulfills the criteria of this entity both clinically and electrocardiographically (fig. 5). Coumel et al. postulate that this form of tachycardia can be attributed to a re-entrant mechanism occurring within the A-V node; one of the two pathways develops antegrade unidirectional block when the sinus cycle length reaches a critical value. However, study on the sequence of retrograde atrial activation, which is critical for differentiating A-V nodal re-entry from concealed WPW syndrome, was not performed in the patient series published by Coumel et al.

The property of antegrade unidirectional block with intact retrograde conduction in the accessory pathway clinically connotes certain prognostic implications. These patients are not likely to develop life-threatening, rapid ventricular responses during atrial flutter-fibrillation such as described by Castellanos et al., and a supraventricular premature beat will not be able to activate the ventricle early enough to initiate ventricular tachycardia or ventricular fibrillation (the R-on-T phenomenon) unless the A-V nodal conduction time is greatly shortened or there is a coexistent A-V nodal/septal bypass tract. However, recurrent reciprocating tachycardia in these patients may pose a therapeutic dilemma. Digoxin tends to prolong A-V nodal conduction time and shorten the refractory period of the atrium and the accessory pathway as well. Therefore, it may be contraindicated in such patients. Procaainamide, quinidine, and ajmaline lengthen the refractory period of the accessory pathway; however, these drugs may prolong His-Purkinje conduction time (H-V interval) and their effects on the electrophysiological properties of the accessory pathway during ventriculo-atrial conduction is not entirely clear. Adjustment of the impulse arrival time at the ventricular end of the accessory pathway and the refractory period of the corresponding atrium and the accessory pathway is a crucial factor in achieving an effective therapeutic result. An appropriate regimen, and/or a pacemaker, and/or surgical interruption of the reciprocating tachycardia circuit may be necessary in selected patients.

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Mechanism of reciprocating tachycardia initiated during sinus rhythm in concealed Wolff-Parkinson-White syndrome: report of a case.

R J Sung, A Castellanos, H Gelband and R J Myerburg

_Circulation._ 1976;54:338-344
doi: 10.1161/01.CIR.54.2.338

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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