His Bundle Recordings in Patients with Reciprocating Tachycardias and Wolff-Parkinson-White Syndrome

By Cesar A. Castillo, M.D., and Agustin Castellanos, Jr., M.D.

SUMMARY
The mechanisms of reciprocating tachycardias were studied in three patients with WPW syndrome using the catheter technic of His bundle recordings. In the first case it could not be determined with certainty whether the tachycardias involved two anatomically independent fascicles or a single longitudinally dissociated pathway. They were terminated by carotid sinus pressure, which caused A-V nodal block, or by properly timed atrial stimuli, which interrupted the circuit. Short-lived paroxysms of atrial fibrillation in cases 1 and 2 were most probably related to atrial vulnerability. In case 3 there were three types of QRS complexes in lead II representing (a) exclusive His bundle conduction, (b) simultaneous His and Kent bundle conduction, and (c) coexisting His and infra-nodal preferential (Mahaim fiber[?]) conduction. This patient also had three types of reciprocating tachycardias—two of ventricular, and one of atrial origin. The reciprocating circuit probably involved the three pathways.

Additional Indexing Words:
Mahaim fiber Retrograde conduction Kent bundle

SUPRAVENTRICULAR arrhythmias are common in patients with the WPW syndrome.1-3 However, there have been few published reports dealing with the mechanisms of reciprocating tachycardias in this entity.4-15 It appeared of interest, therefore, to present the tracings of three patients who developed ectopic tachyarrhythmias during the recording of the electrical activity of the specialized conduction system. A careful analysis of these rhythms extends the usefulness of the recently introduced technic of His bundle recordings16 by increasing our knowledge of basic electrophysiologic phenomena in the human heart.

From the Section of Cardiology, Department of Medicine, University of Miami School of Medicine, the Cardiopulmonary Laboratory, Veterans Administration Hospital, and the Division of Electrophysiology, Jackson Memorial Hospital, Miami, Florida.

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Figure 1

Case 1. Sinus rhythm and WPW syndrome type A.

the desired intracardiac leads at paper speeds of 50 or 100 mm/msec. All tracings were stored on magnetic tape for future playback at any desired speed. The pulses were delivered by a pacemaker which has been used for continuous coupled and paired electrical stimulation. Such a stimulator has uses similar to the one described by Schuilenburg and Durrer. The pulses were slightly underdamped, of twice diastolic threshold value and 2.5 msec in duration. When the atria were stimulated, the emission of the impulse was taken as the onset of the P wave. During sinus rhythm the beginning of the P wave in lead II was used to measure the corresponding intervals. However, in the presence of retrograde atrial conduction the beginning of atrial activation occurred in the HBE lead (low bipolar atrial lead) and was taken as the onset of the retrograde P wave (P'). During ventricular stimulation, the stimulus artifact in lead II was equated with the beginning of ventricular depolarization.

The following intervals were measured whenever feasible: (a) P-R, (b) P-H, (c) H-R, (d) R-H, (e) R-P', (f) H-P', P-H, and P-R. With the technique used in our department, retrograde activation of the atrial (P') and His bundle (H') did not produce deflections which were the mirror image of the ones seen during orthograde sinoventricular conduction.

Cases Studied

Case 1

This patient was a 40-year-old male with a 16-year history of palpitations. During the last year the attacks of rapid heart action had lasted longer and were accompanied by a sensation of substernal discomfort and weakness.

The ECG showed sinus rhythm with a rate of 66/min and a WPW syndrome type A (fig. 1). Cardiac catheterization was normal, but coronary angiography revealed 60% occlusion of the anterior descending artery.

Case 2

The tracings of this 62-year-old male with a long history of paroxysmal tachycardia were presented in a previous communication dealing with other aspects of the WPW syndrome. The ECG showed WPW syndrome type A.
Case 3

A 23-year-old veteran was referred for evaluation of a heart murmur. He had no previous history of paroxysmal tachycardia. The ECG showed a WPW syndrome type A (fig. 2). The delta wave was positive in V1 and V2, but the R to S ratio was less than 1 in these leads. Cardiac catheterization and coronary angiograms were normal.

Results

Mode of Onset and Possible Mechanisms of Reciprocating Tachycardias in the WPW Syndrome

Case 1

During spontaneous sinus rhythm and ventricular pre-excitation at a rate of 100/min this patient developed the arrhythmia shown in figure 3. The first two, slightly notched P waves, were sinus in origin. Therefore, the onset of atrial activity in the high bipolar right atrial lead (BAE) preceded that of the HBE (low right atrial lead). Both P-R and P-H intervals measured 120 msec. The inscription of the H deflection coincided with that of the delta wave in lead II indicating ventricular pre-excitation through a bypass of the “main” His bundle. The QRS complexes were therefore “fusion” beats resulting from ventricular activation through both pathways. Intracardiac recordings showed that the third P wave was inscribed first in the low right atrium and was suggestive of inferosuperior spread of activation. Usually the first negative P wave of a reciprocating tachycardia in the WPW syndrome is preceded by a long P-R interval. According to Wolff, and Schamroth the Kent bundle cannot conduct retrogradely after a beat with a short P-R interval and anomalous ventricular activation. Scherf and Cohen stated that a negative P after a WPW beat should be interpreted as an atrial extrasystole.
The premature atrial beat in our case was followed 160 msec later by an H deflection, which in turn, preceded the onset of a different QRS complex by 50 msec. A reciprocating tachycardia was thus initiated. Values for the corresponding intervals during the tachycardia were as follows: R-P = 100 msec; P-R = 200 msec; P-H = 150 msec; H-R = 50 msec, and QRS duration, 100 msec. During the tachycardia A-V conduction occurred exclusively through the His bundle. The resulting QRS complexes showed abnormal left axis deviation (Q waves in leads II and III) that probably indicates the presence of an old inferior wall myocardial infarction or block in the anterosuperior subdivision of the left branch, or both. Whether this arrhythmia resulted from a circus movement involving two anatomically separate communications (His and Kent bundles), or from a functional intranodal dissociation as conceived by Moe and Mendez could not be determined with certainty. Although we favor the first mechanism, both are represented in the diagram of figure 3.

On one occasion a single P wave appearing 180 msec after the preceding delta wave triggered a run of atrial flutter (fig. 4), which promptly converted to atrial fibrillation. The latter lasted only a few seconds. During atrial fibrillation A-V conduction occurred mainly through the His bundle. Unevenly spaced QRS complexes showed a pattern similar to the one presented on the right side of figure 3. The atrial extrasystole could have triggered atrial fibrillation when falling in the vulnerable period of the atria.

The Kent bundle in a retrograde fashion to trigger the tachycardia.

The bottom diagram offers another explanation. The atrial extrasystole, after being blocked through the Kent bundle, entered the A-V node through the pathway. Thereafter it returned to the atria via the pathway and at the same time proceeded into the ventricles through the His bundle. Retrograde (V-A) Kent bundle conduction was not possible because the atria had been rendered refractory by the atrial echoes. In this figure as well as in figures 6 and 13 paper speed was 50 mm/sec. In all other figures it was 100 mm/sec.
A reciprocating tachycardia could also be induced during atrial pacing at a rate of 90/min (fig. 5). The first two ventricular complexes showed increasing degrees of pre-excitation. The measurements (in msec) made in these two beats are given in table 1. The second H deflection was inscribed after the onset of the R wave in lead II. The corresponding QRS complex (with right axis deviation) probably represented "exclusive or predominant conduction through the Kent bundle." The third P wave was not conducted through the Kent bundle, which failed in "all or none fashion." A-V conduction occurred exclusively through the His bundle. The P-H and P-R intervals measured 210 and 260 msec, respectively. The corresponding (third) QRS complex again was like that in figures 3 and 4, showing left axis deviation. It appears that two distinct types of A-V block occurred more or less simultaneously; type I (Wenckebach) in the A-V node and type II (Mobitz) in the Kent bundle.

The fourth P wave was also propagated through the His bundle with a long A-V conduction time. The resulting (fourth) QRS complex was followed by a retrograde P wave (P-) which triggered a run of reciprocating tachycardia with similar characteristics as the one shown in figure 3. Pacing was stopped after the onset of the arrhythmia. The corresponding intervals during the tachycardia had the following values: R-P- = 120 msec; P-R = 235 msec, and P-H = 185 msec. Note specifically that the R-P- was significantly shorter than the P-R. Atrial vulnerability cannot be invoked in the genesis of this arrhythmia because the triggering P wave fell late in the atrial cycle. Thus a reciprocating (and not a "unifocal") A-V nodal tachycardia was probably present.

Figures 3 to 5 show that three different types of ventricular complexes, each with a distinct AQRS, occurred in this patient: (a) beats conducted exclusively through the His bundle with abnormal left axis deviation, (b) beats presumably conducted exclusively or mainly through the Kent bundle with right axis deviation, and (c) fusion beats with a normal QRS axis.

| Table 1 |
|---|---|---|---|---|
|   | P-H | H-R | P-R | P-J | QRS |
| First beat | 120 | 0 | 120 | 250 | 130 |
| Second beat | 190 | - | 120 | 300 | 180 |
Figure 5

Case 1. Diagrams are like those in figure 3. The first QRS complex was a fusion beat resulting from ventricular activation through both Kent and His bundles. The second QRS complex was a pure WPW beat. The impulse transversing the His bundle, therefore, did not contribute to ventricular depolarization and is represented as being blocked above the ventricles. The third P wave was blocked through the Kent bundle. For diagrammatic purposes this block is represented as occurring within the Kent bundle, but the impulse could have failed to penetrate the Kent bundle. In this beat A-V conduction occurred exclusively through the His bundle. The corresponding (third) QRS complex did not have retrograde conduction to the atria through the Kent bundle probably because the atria were prematurely depolarized by the fourth (and last) stimulus artifact (St). Cessation of pacing at this moment allowed sufficient time for retrograde conduction through the Kent bundle, thus permitting the necessary conditions for the initiation and perpetuation of a reciprocating tachycardia.

Case 2

The first three ventricular complexes in figure 6 were interpreted as follows: The first was a pure sinus beat conducted exclusively through the His bundle (note absence of delta wave); the second was a spontaneous ventricular extrasystole; and the third, with a delta wave, was a WPW fusion beat resulting from ventricular activation via Kent and His bundles. The third QRS complex was followed after an interval of 120 msec by a negative P wave (Px) which initiated (probably because of atrial vulnerability as in figure 4) a run of atrial flutter with an atrial rate of around 300/min. During this arrhythmia there was an increasing degree of ventricular activation through the Kent bundle as evidenced by the progressive widening of the QRS complexes. The His bundle was activated simultaneously with, or slightly after, the delta wave in lead II.

At the same time a coexisting supra-Hisian Wenckebach phenomenon was manifested by a progressive prolongation of the P-H intervals. The patterns of atrioventricular conduction were most interesting and will be described in detail with the help of the diagram (fig. 6):
蔡 2. Atrial flutter triggered by a premature atrial contraction (Px) in a patient with WPW type A. Diagrams are like those in figures 3 and 5. The first beat shows normal A-V and intraventricular (I-V) conduction. A Wenckebach phenomenon in the A-H area was associated with a progressive widening of the QRS complexes. The last beat, which showed the greatest degree of distortion, probably resulted from predominant or exclusive Kent bundle conduction. Note that the orientation of the delta wave in this beat (inferiorly and to the right) was different from its orientation (inferiorly and to the left) in fusion beats.

Px was blocked in both His and Kent bundles.

P1 was blocked in the Kent bundle but conducted through the A-H region with a P-H interval of 240 msec.

P2 was conducted through the Kent bundle with a P-R interval of 110 msec but was blocked in the A-H region.

P3 was blocked in the Kent bundle but was conducted through the His bundle with a P-H interval of 340 msec.

P4 propagated through the Kent bundle with a P-R interval of 110 msec, but was blocked in the A-H region.

P5 was blocked in the Kent bundle but conducted through the A-H region with a P-H interval of 350 msec.

P6 propagated through the Kent bundle with a P-R interval of 110 msec but was blocked in the A-H region.

P7 was blocked in the Kent bundle. It could not be determined with certainty whether it ever reached the His bundle.

P8 was conducted through the Kent bundle producing a pure WPW beat (with right axis deviation). An H deflection could not be identified within it.

The orientation of the delta wave was not the same in “fusion” as in “pure” WPW beats.

Case 3

The second QRS complex in figure 7 showed normal atrioventricular and intraventricular conduction times. In contrast, the first
Case 3. Different types of QRS complexes: classical WPW beat (left), normal beat (center), and right ventricular apical extrasystole (right).

Case 3. The second QRS complex had shorter P-R and H-R intervals than the normal one (fig. 7, center). This and the response to atrial pacing (see fig. 12) suggested that ventricular pre-excitation occurred through a preferential infra-nodal (Mahaim fiber?) bypass.

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beat in figures 7 and 8 displayed the typical pattern of WPW syndrome that this patient usually had (fig. 2). The P-R interval was short and the delta wave well delineated. The last beat in figures 7 and 8 (with abnormal left axis deviation) was a mechanically induced right ventricular apical extrasystole. Comparison of the normal beat in figure 7 (center) with the second beat in figure 8 showed a shorter P-R interval and initial slurring in the latter. Although the P-H interval did not change, the H-R interval was significantly shorter than in the normal complex (table 2). The fact that the P-H intervals were similar in this beat and in the normal beat, as well as the response to atrial pacing (see fig. 12) suggested that the ventricles were pre-excited by a preferential infra-nodal pathway. A para-specific (Mahaim) fiber could have been such a pathway.25, 26 This assumption is in keeping with James' postulates for Mahaim fiber conduction.27 He required for the diagnosis of Mahaim fiber conduction, the presence of early ventricular (septal) excitation, a slight degree of (initial) QRS distortion, and an isoelectric interval between the end of the P wave and the onset of ventricular depolarization.27

In summary, this patient had three distinct patterns of A-V and intraventricular conduction resulting from (a) exclusive propagation through the His bundle, (b) coexisting conduction through His and Kent bundles, and (c) no conduction through the Kent bundle with exclusive supra-Hisian propagation via the A-V node and infra-nodal propagation through the Mahaim fiber and His bundle. For the sake of simplicity, the latter beats will be referred to as having a Mahaim type of conduction.

The first reciprocating arrhythmia observed in this case was recorded before coronary angiography at a time that the regular electrocardiogram showed normal sinus rhythm (first and last complexes in figure 9). Three mechanical extrasystoles were induced after the first sinus beat. The last two ectopic contractions had retrograde conduction to the atria so that atrial activity was initially recorded in lower portions of the right atrium. The onset of the P wave in the HBE preceded that of the BAE. The third ectopic ventricular beat, with an R-P of 160 msec triggered a run of reciprocating tachycardia. When the arrhythmia became stabilized, the R-P, P-R, and P-H intervals measured 150, 335, and 280

**Table 2**

**Duration of Significant Intervals in Lead II During Different Types of A-V and I-V Conduction in Case 3**

<table>
<thead>
<tr>
<th></th>
<th>P-H</th>
<th>H-R</th>
<th>P-R</th>
<th>QRS</th>
<th>P-J</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal*</td>
<td>120</td>
<td>55†</td>
<td>175</td>
<td>80</td>
<td>255</td>
</tr>
<tr>
<td>WPW†</td>
<td>120</td>
<td>10</td>
<td>130</td>
<td>140</td>
<td>270</td>
</tr>
<tr>
<td>Mahaim§</td>
<td>120</td>
<td>40</td>
<td>160</td>
<td>100</td>
<td>260</td>
</tr>
</tbody>
</table>

*Second beat in figure 7.
†Upper limits of normal in our department.
‡First beat in figures 7 and 8 also shown in lead II of figure 2.
§Second beat in figure 8.
msec, respectively. The R-P interval was significantly shorter than the P-R. Apparently the third ventricular extrasystole was not conducted retrogradely to the atria through the normal A-V junction because the latter was refractory. It could, however, transverse the Kent bundle and reach the atria finally to activate the ventricles exclusively through the His bundle. The resulting QRS complex was of normal contour. A reciprocating tachycardia was thus initiated. The reciprocating circuit was as follows: V-K-A-H-V.*10

After coronary angiography, the patterns of pure His conduction (fig. 7, second beat) and His-Kent conduction (figs. 7 and 8, first beats) did not reappear.

After selective coronary arteriography (fig. 10) right ventricular pacing at a rate of 155/min also triggered a run of reciprocating tachycardia during which the QRS complex showed the pattern attributed to Mahaim fiber conduction (fig. 8, second beat). The first three pacemaker beats (measuring 180 msec) had an R-P interval of 230 msec. The third (retrograde) P wave was able (when pacing was discontinued) to capture the ventricles through the His bundle with prolonged P-H and P-R conduction times. A reciprocating tachycardia was thus initiated. When the latter became established, the R-P interval was shortened to 120 msec. The P-R and P-H intervals measured 350 and 310 msec, respectively. As in figures 3 and 5, the R-P was shorter than the P-R.

It seems that the last ventricular ectopic beat (third QRS complex) was conducted retrogradely to the atria via the Kent bundle. The impulse then traversed the A-V node in a forward direction (with considerable delay)

*Abbreviations: Ventricle to Kent bundle to atrium to His bundle to ventricle.
finally reaching the ventricles through the His bundle and Mahaim fiber. Therefore, the corresponding reciprocating circuit was represented as follows:

\[ V-K-A \ (H) \ V \]

\[ M \]

Retrograde P waves following both pacemaker and natural beats had a similar morphology in the intra-atrial leads. Hence, they must have had a similar point of entry in the atria. A spike-to-P interval of 230 msec appears to be too long to postulate retrograde conduction through the Kent bundle. Yet this interval represented the time that elapsed between the delivery of the impulse to the apex of the right ventricle and the onset of atrial depolarization. It included the conduction time from the stimulated site to the ventricular end of the accessory bundle as well as the duration of retrograde propagation through the Kent bundle. Assuming that the former (that is, pure intraventricular conduction) had a value of 110 msec, the retrograde conduction time through the accessory communication would be 230 – 110 or 120 msec. This value is similar to the R-P\(^{\text{c}}\) interval of natural beats. If retrograde conduction of pacemaker beats through the Kent bundle is accepted, it can be postulated, but not conclusively proved, that the first three QRS complexes also activated the His bundle in a retrograde fashion, the impulses being blocked above the bundle. The corresponding, presumably small, H deflections were then buried inside the ventricular complexes.

Transient ventricular pacing at a rate of 140/min resulted in advancing the subsequent (reciprocating) atrial and ventricular complexes to an earlier moment (fig. 11). The first QRS complex after cessation of pacing appeared 160 msec before the time that it would have been due if ventricular stimulation had not been performed. This is further evidence of a reciprocating arrhythmia using anatomically separate pathways.\(^{10,28}\)

Atrial pacing in this patient produced different effects from those in other patients with WPW syndrome studied in our department.\(^{17}\)

In figure 12 the first P-R and P-H intervals measured 240 and 200 msec, respectively. However, the H-R interval was the same as it was during sinus rhythm and Mahaim fiber conduction (second beat in fig. 8 and table 2). The second P-R interval was longer than the first (300 msec). This was due exclusively to a prolongation of the P-H interval to 260 msec. There was no change in the H-R interval or in QRS duration. This is the usual response to atrial pacing at critical rates seen in patients without WPW syndrome. These findings are in keeping with the assumptions made earlier that supra-Hisian propagation occurred only through the A-V node and that the bypass did not involve the latter.
Case 3. Atrial pacing produced different effects from those in other patients with WPW syndrome studied in our department.17 A reciprocating tachycardia was initiated after the second QRS complex. During the arrhythmia the artificial stimuli (St) failed to activate the atria.

The third P wave was retrograde and appeared 120 msec after the second QRS complex. Apparently, the latter had retrograde conduction through the Kent bundle. A reciprocating tachycardia was then initiated. During the latter, the R-P⁻ was significantly shorter than the P-R. The corresponding circuit was represented as follows:

\[
A \{ H \} \rightarrow M \rightarrow V \rightarrow K \rightarrow A.
\]

Mode of Termination of the Arrhythmias

Cases 1 and 2

The reciprocating tachycardias occurring in these patients (either spontaneously or induced by atrial stimulation) were terminated in two ways. Figure 13 shows the effects of carotid sinus pressure. This vagal maneuver produced progressive lengthening of the P-R and P-H intervals until a P⁻ wave failed to reach the His bundle (A-V nodal block). In addition, atrial stimuli were delivered at a time early enough to stimulate the atria before the oncoming retrograde P wave captured the ventricles with a long P-R interval and terminated the tachycardia (fig. 14).

Atrial fibrillation occurring in cases 1 and 2 was short-lived and disappeared spontaneously with prompt resumption of sinus rhythm.

Case 3

In this patient carotid sinus massage could not be applied successfully. Hence, its effects were not determined. The tachycardias of
ventricular origin with exclusive (forward) His bundle conduction were terminated by mechanical premature ventricular beats with retrograde conduction (fig. 9). The retrograde P wave appeared too early in the atrial cycle to be conducted through the A-V node so that it failed to activate the His bundle in a forward direction. In consequence, the circuit was interrupted, and the arrhythmia ended. The tachycardias that used the His bundle and Mahaim fiber in a forward direction were reset but could not be terminated by atrial or ventricular stimuli (fig. 11). They disappeared spontaneously when an early retrograde P wave failed to reach the His bundle in its journey toward the ventricles.

Discussion

In our department, two possible electrophysiologic mechanisms have been identified from the analysis of His bundle recordings in patients with classical WPW syndrome. One, discussed in a previous article, presupposes the existence of an “electrophysiologic” Kent bundle, hereby defined as a total bypass of the normal A-V junction irrespective of its anatomic location. In these cases the H deflection was inscribed after the onset of the delta wave in the peripheral leads (figs. 5 and 6) suggesting ventricular pre-excitation ahead of the His bundle. One patient (case 3 in our present report) appeared to have an infranodal preferential pathway (Mahaim fiber). Its presence was suggested by the fact that the H-R interval of the anomalous complexes under consideration was shorter than that of the normal beats (table 2; figs. 7 and 8). In addition, during atrial pacing at progressively higher rates the P-R interval increased (as in normal patients) exclusively at the expense of the P-H interval, while the H-R interval remained shorter than normal (fig. 12).

It appears that His bundle recordings have corroborated two of the previously postulated mechanisms for the WPW syndrome. These considerations are not only of academic importance but of clinical significance as well, since symptomatic WPW syndrome is a potential surgical entity.

No attempt was made in this communication to identify the anatomic location of the accessory communications. Yet, it appears that at least in some cases (figs. 1 to 4 of reference 17 and fig. 6 of this communication) the spatial orientation of the delta wave can be different in beats showing predominant or exclusive Kent bundle conduction from those.
resulting from the more or less simultaneous activation of the ventricles through both communications.

The tachycardias shown in this communication had R-P intervals which were consistently shorter than the P-R intervals. On the other hand, in patients without WPW syndrome, reciprocating tachycardias of ventricular origin in which retrograde conduction occurs through the His bundle (due to a functional longitudinal dissociation of the A-V junction) usually show R-P intervals which are definitely longer than the P-R intervals.2, 23, 29

During the tachycardias A-V conduction consistently occurred through the His bundle. In case 1 the corresponding QRS complexes were not normal but had the morphology attributed to block in the anterosuperior division of the left branch.21, 22 Laham3 and Giraud and associates80 have stressed that (especially in patients with atrial fibrillation) disappearance of WPW type of conduction can be followed by abnormal QRS complexes. These abnormal complexes have been attributed to (true) aberrant ventricular conduction or to pre-existing bundle branch or divisional block. The reciprocating tachycardias reported by Durrer and associates10 (presumably with exclusive A-V conduction through the A-V junction) in patients with WPW syndrome also showed aberrant ventricular conduction.

In patients with WPW syndrome the reciprocating tachycardias probably involve two separate anatomic communications.19 Other circuits, although less probable, may be present. For instance, a functional longitudinal intra-nodal dissociation28 with a final common pathway could not be definitely excluded in case 1. Moreover, Schamroth and Yoshonis31 postulated the existence of two independent, functionally and longitudinally dissociated, intra-nodal pathways. They did not imply the presence of a final common pathway, thus departing from the classical concepts of Moe and associates.23, 32 If their assumptions are not limited to the A-V nodal area but extend to the total A-V conduction system, it is conceivable that an impulse could propagate through one pathway at a faster speed and activate the atria ahead of the one ascending through the slower pathways. In fact, Kaufman and Rothberger,33 Sciacca and Sangiorgi,34 and Rossi35 have described independent muscle strands within the specialized atrioventricular and intraventricular systems. Preferential use of these fascicles has been considered possible by those who assumed that some forms of WPW are due to a bypass within the A-V system.36-38

Termination of WPW tachycardias by atrial and ventricular stimulation was previously described by Massumi9 and Durrer10 and their co-workers. This is further evidence in favor of a reciprocating arrhythmia. It is possible, however, that several mechanisms can operate in the same patient in different moments; for instance, in figures 4 and 6 the atrial arrhythmias were probably induced by atrial vulnerability.

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CESAR A. CASTILLO and AGUSTIN CASTELLANOS, JR.

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