Electrical Stimulation of the Heart in Patients with Wolff-Parkinson-White Syndrome, Type A

By Hein J. J. Wellens, M.D., Reinder M. Schuilenberg, M.D., and Dirk Durrer, M.D.

SUMMARY
The initiation and termination of tachycardias were studied in five patients with Wolff-Parkinson-White syndrome, type A. In all patients, electrical stimulation was performed from the right side of the heart. In three patients the effect of induced left-sided premature beats was studied as well. In contrast to patients with Wolff-Parkinson-White syndrome, type B, there was difficulty in initiation and termination of tachycardias with premature beats applied to the right atrium or ventricle. This could easily be accomplished with left-sided premature stimuli. In two patients the simultaneous recording of right and left atrial activation during a tachycardia, showing atrioventricular conduction by way of the A-V nodal-His pathway and ventriculoatrial conduction via the anomalous connection, revealed that left atrial activation occurred far in advance of right atrial activation. During regular driving of the right and the left atrium at identical rates the most pronounced pre-excitation pattern was seen after left atrial stimulation.

Our results are in agreement with a circus movement or reciprocal mechanism by way of the A-V nodal-His pathway and the anomalous A-V connection as a causal mechanism for tachycardias in Wolff-Parkinson-White syndrome, type A. In contrast to the usual finding of atrioventricular conduction during the tachycardia via the A-V nodal-His pathway and ventriculoatrial conduction by way of the anomalous connection, one patient showed tachycardias running in the opposite direction. A location of the anomalous pathway on the left side of the heart is supported by our findings.

Additional Indexing Words:
Pre-excitation Accessory bundle Tachycardia Re-entry
Premature beat Ventricular echo Delayed capture Circus movement

In patients with Wolff-Parkinson-White syndrome, type B, who have a history of tachycardias, Durrer et al. found that tachycardias could reproducibly be initiated and terminated by a single electrically induced right atrial or right ventricular premature beat. Epicardial excitation mapping and the outcome of surgical interventions in patients with the Wolff-Parkinson-White syndrome, type B, indicate that the typical electrocardiogram in these patients can be the result of an accessory atrioventricular pathway inserting into the anterolateral aspect of the right ventricle. The results of electrical stimulation, epicardial excitation mapping, and surgery support the concept advanced in 1926 by de Boer that tachycardias in these patients may be caused by a circus movement consisting of atrium to A-V junction to His bundle to ventricle to anomalous A-V connection to atrium, in that order.

The site of the anomalous A-V connection is less clear in patients with the Wolff-Parkinson-

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White syndrome, type A. The suggestion that it is located on the posterior side of the heart, connecting the left atrium with the left ventricle as postulated by Oehnell \(^7\) has recently been challenged by Boineau and Moore. \(^5\)

In this article we would like to report the outcome of our stimulation studies in five patients with the Wolff-Parkinson-White syndrome, type A, and to compare these results with those previously obtained during similar studies in patients with Wolff-Parkinson-White syndrome, type B. As will be shown, our results enable us to comment upon the location of the anomalous A-V connection in patients with the Wolff-Parkinson-White syndrome, type A.

**Methods**

Sex, age, tachycardia frequency, incidence, and duration of the attacks of tachycardia of the five patients studied are given in table 1. The ECGs of all patients fulfilled the criteria for Wolff-Parkinson-White syndrome, type A, as given by Rosenbaum et al. \(^8\) During sinus rhythm none of our patients showed periods of exclusive conduction by way of the A-V node—His bundle only.

A description of our stimulator and methods of stimulation have been given previously. \(^1\) By use of the Seldinger technique, two or more electrode catheters were passed under local anesthesia through one or both femoral veins and positioned under fluoroscopic control at the desired intracavitary location.

In patients A and B, only the right side of the heart was studied. In patients C and E, the evaluation of coexistent mitral valve disease necessitated the performance of a trans-septal left atrial catheterization. Patient D surprised us with an open foramen ovale. In the latter three patients we could, therefore, also study the results of electrical stimulation of the left side of the heart.

A bipolar catheter was used for stimulation. The duration of the stimuli was 2 msec. Current strength was less than two times the diastolic threshold current. The protocol of the investigation in each patient consisted of:

1. **The study of initiation of a tachycardia:**
   a. One method was by the induction of one or sometimes two premature stimuli to the atrium (in all five patients to the right atrium; in patients C, D, and E also to the left atrium) once after eight beats of a regular driven atrial rhythm. The interval between the last beat of the regular driven rhythm and the induced premature beat (the premature beat interval) was thereby gradually shortened until either a tachycardia resulted or the atrium became refractory to stimulation.
   b. Another method was by the induction of one or two premature stimuli to the ventricle (in all five patients to the right ventricle; in patients C, D, and E also to the left ventricle) once after eight beats of a regular driven ventricular rhythm. The premature beat interval thereby was gradually shortened until either a tachycardia resulted or the ventricle became refractory to stimulation.

2. **The study of termination of the tachycardia:**
   Using the synchronizing circuit of our stimulator, we induced premature beats during the tachycardia after every eighth tachycardia complex. This was done until either the tachycardia was terminated or the atrial or ventricular tissue became refractory to the premature stimulus. In all five patients the influence of right-sided premature beats was studied, and in patients C, D, and E, the influence of left-sided ones also.

3. **The study of the influence of induced atrial and ventricular premature beats on the time relations of the tachycardia:**

<table>
<thead>
<tr>
<th>Pat.</th>
<th>Sex</th>
<th>Age (yr)</th>
<th>Tachycardia (beats/min)</th>
<th>Incidence</th>
<th>Duration (yr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Male</td>
<td>44</td>
<td>180</td>
<td>once a day</td>
<td>23</td>
</tr>
<tr>
<td>B</td>
<td>Male</td>
<td>54</td>
<td>140</td>
<td>twice a yr</td>
<td>1</td>
</tr>
<tr>
<td>C</td>
<td>Female</td>
<td>46</td>
<td>150</td>
<td>almost daily during the last 2 yr</td>
<td>28</td>
</tr>
<tr>
<td>D</td>
<td>Male</td>
<td>51</td>
<td>185</td>
<td>twice a week</td>
<td>4</td>
</tr>
<tr>
<td>E</td>
<td>Female</td>
<td>24</td>
<td>205</td>
<td>twice a week</td>
<td>10</td>
</tr>
</tbody>
</table>

*Table 1*
In patients C and E we also simultaneously recorded left and right atrial activation during the tachycardia from two fixed points, close to the A-V ring, using a unipolar electrode. Before electrical stimulation, the position of the catheters was always controlled fluoroscopically.

At the beginning of the recording of intracavitary activation and electrical stimulation, a drawing was made of the position of the catheters. The results were observed in the ECG of leads I, II, III, V₁, and V₆, and in that of multipolar intra-atrial leads. The electrocardiograms were registered on an 8-channel high-frequency direct-writing Elema recorder and stored on magnetic tape with an Ampex FR 1300 tape recorder.

**Results**

**Initiation of Tachycardias**

A. Right Atrial Stimulation

In patients A, B, C, and E it was impossible for us to induce a tachycardia with a single right atrial premature beat during regular driving of the right atrium. In these four cases, the right atrium became refractory to electrical stimulation before blockade of the impulse in either the Kent or the His pathway occurred.

In patient D, however, during driving of the right atrium with a basic cycle length of 660 msec, an induced right atrial premature beat after an interval of 260 msec resulted in a tachycardia with QRS complexes, measuring 135 msec in width and showing a configuration compatible with excitation of the ventricle by way of the Kent bundle only. The attacks of tachycardia were shortlived, measuring 8–16 beats, and could readily be induced by right atrial premature beats up to a premature beat interval of 184 msec. It is very likely that this represents the first reproducible demonstration of a circus move-

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**Figure 1**

Patient D. Initiation of a tachycardia, showing exclusive Kent conduction by way of a single right atrial premature beat. The configuration of the QRS complex after a right atrial premature beat (premature beat interval, 260 msec) during regular driving of the right atrium (BCL 660 msec) suggests conduction to the ventricle by way of the Kent bundle only. This QRS complex is followed by a tachycardia showing similar QRS complexes. As clarified in the diagram, this could be a tachycardia with atrioventricular conduction through the Kent bundle and ventriculoatrial conduction through the His pathway.
ment with atrioventricular conduction through the Kent bundle and retrograde ventricular atrial conduction by way of the His bundle (fig. 1). The tachycardia always ended by a ventricular complex not being followed by an atrial complex (fig. 1).

In patient C, in which one atrial premature beat did not elicit a tachycardia, two right atrial premature beats given in close succession resulted in a tachycardia. The shortening of the refractory period of the right atrium after the first premature beat made it possible for us to give such an early second premature beat, that this impulse found the anomalous pathway towards the ventricle refractory. In this way a tachycardia with antegrade His conduction and retrograde Kent conduction could reproducibly be induced.

B. Left Atrial Stimulation

In patient C a tachycardia could readily be induced by one premature left atrial beat given during regular left atrial beat driving (fig. 2). This tachycardia showed antegrade His conduction.

In patient D, when the left atrium was driven with a basic cycle length of 660 msec, a left atrial premature beat given after an interval of 260 msec initiated a tachycardia similar to the one that could be initiated from the right atrium in this patient (a tachycardia probably caused by a circus movement with antegrade Kent and retrograde His conduction; fig. 3). This tachycardia (measuring 8–17 beats) could reproducibly be initiated by one left atrial premature beat up to the refractory period of the left atrium. If one compares figures 3 and 1, it becomes clear that during driving of the right and left atria with identical frequencies (basic cycle length of 660 msec) the contribution to ventricular

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**Figure 2**

Patient C. Initiation of a tachycardia by way of a single left atrial premature beat (premature beat interval, 280 msec) during regular driving of the left atrium (BCL 660 msec).
excitation by way of the anomalous connection is greater if stimulation is performed from the left atrium.

In patient E one left atrial premature beat during regular left atrial stimulation repeatedly resulted in a tachycardia with antegrade His and retrograde Kent conduction.

C. Right Ventricular Stimulation

In patient A, shortening of the interval of an induced right ventricular premature beat during regular driving of the right ventricle (basic cycle length 650 msec) resulted in a gradual increase in QP time. The P waves were reproducibly followed by a tachycardia with antegrade His conduction at a premature beat interval of 330 msec.

In patient B shortening of the interval of the induced right ventricular premature beat resulted in only a slight increase in QP interval. The right ventricle became refractory to the premature stimulus without induction of a tachycardia. Two closely given right ventricular premature beats, however, resulted in a tachycardia. Depending upon the interval between these premature beats, there were two different mechanisms responsible for the initiation of the tachycardia.

Mechanism 1. Initiation of a tachycardia by way of the Kent bundle during blocked retrograde conduction through the His bundle. When the right ventricle was driven with a basic cycle length of 640 msec, two right ventricular premature beats were given after intervals of 280 and 570 msec, respectively, following the last beat of the regular driven right ventricular rhythm. The second ventricular premature beat was followed after 630 msec by a small QRS complex initiating a tachycardia, showing antegrade His conduction. From the work of Janse et al., a sudden increase in heart rate

![Figure 3](http://circ.ahajournals.org/)

*Figure 3*

Patient D. Initiation of a tachycardia, showing exclusive Kent conduction by way of a single left atrial premature beat. During regular driving of the left atrium (BCL 660 msec) a left atrial premature beat given after an interval of 260 msec initiates a tachycardia showing atrioventricular conduction by way of the Kent bundle only. Compare this figure with figure 1; note the difference in QRS complex configuration during regular driving of the right and left atrium (most marked in lead II).
results in marked changes in refractoriness of the heart. These changes take several beats before becoming stable and are not completely identical in the conduction system and the ventricular muscular tissue. This may augment the dissociation in functional properties of the Kent and A-V node–His bundle pathway, necessary for the initiation of the tachycardia, by giving two premature beats instead of one. The second ventricular premature beat can be looked upon as the third beat of a tachycardia. Apparently, as shown in the diagram (fig. 4), the first induced premature beat is followed by retrograde conduction towards the atrium through both Kent and His bundles. The second premature beat, however, is only retrogradely conducted through the Kent bundle, the His bundle or distal part of the A-V node now being refractory. This situation, however, creates the possibility for the impulse to return to the ventricle by way of the now recovered His bundle, thereby initiating a tachycardia. Since no His bundle recording was obtained in this patient, the exact site of blockade of the retrograde impulse is not known.

Mechanism 2. Initiation of tachycardia by way of a ventricular echo beat

When, during right ventricular driving, two right ventricular premature beats were induced after intervals of 350 and 590 msec, respectively, following the last beat of the regularly driven right ventricular rhythm, a different mode of initiation of the tachycar-

![Figure 4](http://circ.ahajournals.org/)

Patient B. Initiation of a tachycardia by way of two right ventricular premature beats (E₁ and E₂) given in close succession.
WOLFF-PARKINSON-WHITE SYNDROME, TYPE A

dia was seen. As shown in figure 5, the second right ventricular premature beat was now followed after 400 msec by a smaller QRS complex measuring 90 msec in width. The latter complex is followed after 580 msec by a tachycardia with small QRS complexes. In view of the short distance (400 msec) between the second premature right ventricular beat and the first small QRS complex and considering the distance between the first and the second of the smaller QRS complexes, we feel that it is impossible to explain this sequence by mechanism 1. By use of the longitudinal dissociation concept of the A-V node advanced by Scherff and Moe and their coworkers, whereby the presence of two pathways (α and β) with different properties is assumed, it seems acceptable for us to consider the first small QRS complex as a ventricular echo. Following the second right ventricular premature beat, the A-V node is invaded from below. The impulse finds one pathway (α) refractory and travels by way of the other pathway (β). When the impulse arrives at the common pathway, it is propagated towards the atrium and also back towards the ventricle by way of the now recovered α pathway. This impulse activates the ventricles and fulfills the criteria for a ventricular reciprocal beat or ventricular echo. Atrial activation following the second ventricular premature beat and preceding the ventricular echo is probably the result of retrograde conduction by way of the Kent bundle only, the

Figure 5

Patient B. Initiation of a tachycardia by way of two right ventricular premature beats (E₁ and E₂) given in close succession. The mechanism is different from the one shown in figure 4. A ventricular echo beat as the initiating mechanism seems very likely. The first QRS complex is not conducted retrogradely towards the atrium, the atrium already being activated from the sinus node.

Circulation, Volume XLIII, January 1971
impulse being so delayed in the A-V node that it reaches the atrium when the atrium is refractory from the impulse resulting from retrograde Kent conduction. The configuration of the ventricular echo suggests that slightly aberrant conduction to the ventricles took place. The P wave following the echo beat is the result of retrograde conduction through the Kent bundle only, the His bundle being refractory. This impulse descends towards the ventricles by way of the His bundle, completing the first cycle of the ensuing tachycardia.

In patient C, for the same reasons as in patient B, it was not possible for us to initiate a tachycardia by way of one right ventricular premature beat. Two right ventricular premature beats, however, repeatedly resulted in a tachycardia, the mechanism being identical to mechanism 1 in patient B.

In patient D a single early induced right ventricular premature beat was frequently followed by a “spontaneous” second early right ventricular premature beat. This “spontaneous” ventricular beat after an early premature ventricular beat is, in our experience, not uncommon during electrical stimulation of the ventricle. This sequence was repeatedly followed by a tachycardia. The explanation for the mechanism for initiation of the tachycardia is essentially the same as

**Figure 6**

*Patient D. Initiation of a tachycardia by way of a single left ventricular premature beat. During regular driving of the left ventricle (BCL 515 msec) a premature beat was given after an interval of 260 msec. The possible mechanism of initiation of the tachycardia is shown in the diagram.*
mechanism 1 in patients B and C. It is important to note that in patient D it was possible for us to initiate tachycardias with antegrade Kent and retrograde His conduction during atrial stimulation, while during ventricular stimulation (fig. 6; left ventricular stimulation) a tachycardia could be initiated that ran in the reversed direction. In both situations the His bundle was the A-V connection that was blocked on shortening of the interval of the premature stimulus.

In patient E, no tachycardias could be initiated by one right ventricular premature beat, for reasons already given for patient B. Two right ventricular premature beats were not given.

D. Left Ventricular Stimulation

Left ventricular stimulation was done only in patients C, D, and E. In patients C and D, one early left ventricular premature beat resulted in retrograde conduction through the Kent bundle only. This was followed by antegrade A-V conduction through the His bundle, resulting in the first ventricular complex of the tachycardia. An example is given in figure 6. In patient E a tachycardia could not reproducibly be initiated by a single left ventricular premature beat.

**Termination of Tachycardias**

A. Right Atrial Premature Beats

In patients B and D, the tachycardia showing antegrade His conduction could readily be terminated by a single early right atrial premature beat. In patient A this could be done only by way of two right atrial premature beats given in close succession. In patients C and E, neither one nor two right atrial premature beats could terminate the tachycardia. This could only be done from the right atrium by driving at rates above the tachycardia frequency (“overdriving”).

In patient D the influence of premature beats on the tachycardias showing antegrade Kent conduction could not be studied. These

**Figure 7**

*Patient C. Termination of a tachycardia by way of a single left ventricular and left atrial premature beat (lower half of the figure). These observations were made during catheterizations on two different days. As shown in this figure, the tachycardia frequency was not the same. The pause following the termination of the tachycardia was ended by a sinus escape (upper half of the figure) and a nodal escape (lower half).*

*Circulation, Volume XLIII, January 1971*
tachycardias were too shortlived (8–17 beats) for accurate synchronization of the premature stimulus.

B. Left Atrial Premature Beats

In the three patients (C, D, and E) where termination by this method was studied, a single early left atrial premature beat could always terminate the tachycardia. An example is given in figures 7 and 9.

Influence of Induced Atrial and Ventricular Premature Beats on the Time Relations of the Tachycardia

A. Right Atrial Premature Beats

No changes in the time relations were seen after the induction of a single right atrial premature beat. In other words, the distance between the tachycardia QRS complex to the induced premature beat (the premature beat interval) and the distance from the induced premature beat to the next QRS complex of the tachycardia (the post-premature beat interval) was twice the cycle length of the tachycardia.

B. Left Atrial Premature Beats

In patients C and D, an early left atrial premature beat was followed by ventricular activation at a later time than expected during the tachycardia. An example is given in figure 10. For an explanation of this finding, see the discussion section. In both patients the tachycardia could be terminated by a single left atrial premature beat given after a shorter interval (fig. 7). In patient E, when a premature beat was induced during the tachycardia 90 to 75 msec after atrial activation, ventricular activation followed at an earlier time than expected during the tachycardia (fig. 11). A premature beat given after an interval of 75 msec resulted in ventricular activation after an interval slightly longer than the tachycardia cycle (fig. 11). Premature beats given after an interval of 70 msec resulted in termination of the tachycardia (fig. 11).

The results in patient E suggest that, at a premature beat interval from 90 to 75 msec, a part of the tachycardia cycle can be pre-excited, resulting in shortening of one tachy-
cardia cycle. Apparently, however, at an interval of 75 to 70 msec the atrial premature beat enters the A-V junction when this is still partly refractory from the foregoing impulse, resulting in slowing of transmission through the A-V junction (this becomes manifest by the interventricular interval after this premature beat being longer than the length of the cycle of the tachycardia).

C. Right Ventricular Premature Beats
No changes in the time relations of the tachycardia were seen after the induction of right ventricular premature beats.

D. Left Ventricular Premature Beats
In the three patients studied (C, D, and E), a single left ventricular premature beat induced during the tachycardia did not influence the time relation of the tachycardia.

Simultaneous Registration of Left and Right Atrial Activation during the Tachycardia

Simultaneous registration was done in patients C and E during a tachycardia showing antegrade conduction by way of the His bundle. We took care to position the recording electrodes close to the A-V ring. In both patients activation of the left atrium occurred much earlier than that of the right atrium. In patient C the beginning of left atrial activation preceded the beginning of the right atrial activation.

![Figure 9](http://circ.ahajournals.org/)

*Figure 9*

*Patient D. Termination of a tachycardia by one single left ventricular premature beat.*

*Circulation, Volume XLIII, January 1971*
activation by 80 msec; in patient E, by about 100 msec (fig. 12).

**Discussion**

The use of systematic electrical stimulation of the hearts of patients who have the Wolff-Parkinson-White syndrome and suffer from tachycardias have enabled us to study the mechanism of initiation and termination of supraventricular tachycardias in these patients. The results of our stimulation studies done in patients with Wolff-Parkinson-White syndrome, type A, reveal that:

1. In Wolff-Parkinson-White syndrome, type A, we found it easy to initiate and terminate a tachycardia by way of a single premature beat from the left side of the heart, while (in contrast to patients with Wolff-Parkinson-White syndrome, type B) this could frequently not be accomplished from the right side of the heart.

2. In two patients, simultaneous registration of left and right atrial activation during a tachycardia with antegrade A-V conduction by way of the His bundle showed that left atrial activation preceded right atrial activation by a considerable amount of time (80 and 100 msec, respectively).

3. In those patients where regular driving was performed at the same rate from the right and the left atrium, the greatest amount of pre-excitation was seen during left atrial stimulation.

These three findings all speak in favor of a left-sided location of the anomalous A-V connection in patients with Wolff-Parkinson-White syndrome, type A. Our results indicate

![Figure 10](http://circ.ahajournals.org/)

*Figure 10*

*Patient C. An induced early left atrial premature beat is followed by ventricular activation at a later time than expected during the tachycardia. The left ventricular premature beat does not influence the time relations of the tachycardia. As shown in figure 7, shortening the premature beat interval by 5 msec results in termination of the tachycardia.*
that Boineau and Moore's finding that Wolff-Parkinson-White syndrome, type A, in the dog heart is the result of an accessory bundle connecting the right atrium and right ventricle at the posterior side of the heart, cannot be applied to the cases we studied.

Recently Sherf and James postulated that the direction of input of the impulse from the atrium into the A-V conduction system determines the spread of excitation through the ventricular conduction system. In this way the difference in QRS configuration after left versus right atrial stimulation could be explained without the assumption of the presence of an anomalous atrioventricular connection. The validity of this concept, however, needs to be proven functionally by comparison of the configuration of the QRS complexes after right and left atrial stimulation at identical rates at different stimulation sites in patients who do not show signs of preexcitation.

Electrocardiograms appearing in articles reporting the outcome of right atrial stimulation at different stimulation sites and of left atrial stimulation failed to show such differences in QRS complex configuration. This has also been our own experience in six patients, in which the presence of an atrial septal defect enabled us to perform right and left atrial stimulation at identical driving rates.

From clinical electrocardiograms, we know that changes in the QRS complex after markedly different P waves are extremely unusual. Durrer et al. showed, in the perfused isolated human heart, that during ventricular stimulation followed by 1 to 1 ventriculoatrial conduction, activation of the left and the right atrium occurred almost simultaneously. In two of our patients left atrial activation preceded right atrial activation by a considerable amount of time during their tachycardia with ventriculoatrial conduction by way of the anomalous A-V connection. This points to a left atrial location of the atrial end of the anomalous A-V connection.

As shown in patient B, apart from the previously reported initiating mechanisms (atrial premature beat, ventricular premature beat, and atrial echo beat), a tachycardia can be initiated by a ventricular echo beat. In the absence of recordings of the electrical activity of the specific conduction system, we were not able to locate the exact site in the A-V junction where the mechanisms leading to the echo beat and ensuing tachycardia took place.

In patient D an early atrial premature beat induced during right and left atrial driving repeatedly resulted in a tachycardia with QRS complexes showing a maximal amount of preexcitation. As shown in the intra-atrial leads, there was a 1 to 1 relation between atrial and ventricular activation. The frequency of this tachycardia was 200 beats/min. Unfortunately the period of tachycardia lasted only 8 to 17 QRS complexes. This made it impossible for us to study the influence of atrial and ventricular premature beats on the tachycardia. Therefore, although it seems likely,
definite proof that this tachycardia represents a circus movement tachycardia with antegrade A-V conduction by way of the Kent bundle and ventriculoatrial conduction via the His bundle-A-V junction is lacking.

In patient D the induction of a ventricular premature beat during right or left ventricular driving repeatedly resulted in a tachycardia with antegrade A-V conduction by way of the A-V node-His bundle pathway. The way in which this type of tachycardia could be initiated and terminated demonstrated that this was a true circus movement tachycardia.

In a patient with Wolff-Parkinson-White syndrome, type B, we found\(^1\) that during a tachycardia a properly timed atrial and ventricular premature beat could shorten one cycle length of the tachycardia. All following cycles were shifted to an earlier time than would be expected without shortening of one cycle by the premature beat. According to Wenckebach and Winterberg,\(^{26}\) no circus movement can be present when an induced premature beat is followed by a compensatory pause. If one wants to use this criterion in a fair way, however, one has to realize that even if one induces atrial and premature beats up to the refractory period of the atrium and the ventricle, it will not always be possible to reach the tachycardia circuit before the next tachycardia impulse passes. Whether one reaches the tachycardia circuit or not depends on:

1. the distance between the site of stimulation and the tachycardia pathway,
2. the conduction properties of the tissue between the site of stimulation and the tachycardia pathway,
3. the frequency of the tachycardia and its resulting refractory period,
4. the spatial dimensions of the tachycardia pathway.

\[\text{Figure 12}\]

*Patient E. Simultaneous registration of left and right atrial activation during the tachycardia. Left atrial activation precedes the right atrial one by about 100 msec. Atrial activation is indicated by P.*
Electrical stimulation from the right side of the heart and the left ventricle did not influence the time relations during the tachycardia in any of our patients. A left atrial premature beat shortened one cycle of the tachycardia with a shift of the following tachycardia cycles to an earlier time in one of our patients (patient E). Further shortening of the premature beat interval, however, resulted in an interventricular interval slightly longer than the tachycardia cycle. Termination of the tachycardia in this patient occurred at a premature beat interval of 70 msec.

Lengthening of the interventricular interval after an early left atrial premature beat, making it longer than the tachycardia cycle, was also seen in patients C and D. This finding can be explained by the assumption that the atrial premature beat enters the A-V junction while this is still partly refractory from the foregoing impulse, resulting in marked slowing of transmission through the A-V junction. This explanation was given by Pick and Dominguez for a similar finding in A-V junctional tachycardias. They noted in a patient with A-V junctional tachycardia and interference dissociation that an atrial complex during the tachycardia was sometimes followed by a ventricular complex, appearing after an interventricular interval longer than the tachycardia interval. They considered this ventricular complex the result of a "captured" atrial beat. In view of the fact that, contrary to the usual shortening of the interventricular interval after an atrial capture, the interventricular interval now measured longer than the tachycardia interval, they introduced for this finding the name "delayed capture."

On further shortening of the premature beat interval, the prematurely induced atrial impulse finds the A-V junctional tissue refractory and is, therefore, not transmitted through the A-V junction but makes the A-V junction refractory for the next impulse of the tachycardia. This results in termination of the tachycardia.

The finding that only left atrial premature beats influenced the time relations of the tachycardia are another argument that the anomalous A-V connection is situated on the left side of the heart.

As pointed out above, several factors influence the outcome of premature beats on the time relations of a circus movement tachycardia. It remains to be demonstrated whether the fact that only left atrial premature beats could influence the time relations of the tachycardias in our patients means that the spatial dimensions of the circus movement are rather small and that the anomalous A-V connection in patients with Wolff-Parkinson-White syndrome, type A, is situated not far from the A-V junction.

References


11. MOE GK, MENDEZ C: The physiologic basis of reciprocal rhythm. Progr Cardiovasc Dis 8: 461, 1966
20. MIROWSKI M: Left atrial rhythm. Diagnostic criteria and differentiation from nodal arrhythmias. Amer J Cardiol 17: 203, 1966

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