Atypical Patterns of Retrograde Conduction Over Accessory Atrioventricular Pathways in the Wolff-Parkinson-White Syndrome

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SUMMARY Patterns of ventriculoatrial conduction have been used to distinguish retrograde conduction over an accessory atrioventricular pathway from that over the normal atrioventricular conduction system. Ventriculoatrial conduction at a constant interval during incremental ventricular pacing and during progressive prematurity of ventricular extrastimuli has been considered characteristic of conduction over an accessory pathway. We describe three patients with the Wolff-Parkinson-White syndrome who had progressive or sudden increments in ventriculoatrial conduction over an accessory pathway during fixed-rate ventricular pacing or during introduction of ventricular extrastimuli. Such properties have been considered characteristic of conduction over the normal atrioventricular conduction system. We conclude that retrograde conduction over accessory pathways may resemble conduction over the normal atrioventricular conduction system.

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA in the Wolff-Parkinson-White syndrome most frequently results from reentry using the normal atrioventricular (AV) conduction system in the antegrade direction and an accessory AV connection in the retrograde direction. Recognizing the participation of the accessory pathway in such tachycardias has become more important since the development of surgical techniques that permit ablation of accessory pathways in selected patients with incapacitating tachycardia poorly controlled by conservative measures. During reciprocating tachycardia, distinguishing between retrograde conduction over an accessory pathway and retrograde conduction over the normal AV conducting system is crucial. In addition to other criteria, patterns of ventriculoatrial conduction are considered helpful in recognizing accessory pathways. A constant ventriculoatrial conduction time during increasingly rapid ventricular stimulation and during progressive prematurity of a ventricular extrastimulus has been considered characteristic of conduction over an accessory pathway. A significant, progressive increment in ventriculoatrial conduction times during such procedures has been considered characteristic of conduction over the normal AV conduction system.

We describe three patients with accessory pathways who had patterns of retrograde conduction considered characteristic of conduction over the normal AV conducting system.

Methods

Patients

Thirty consecutive patients with the Wolff-Parkinson-White syndrome underwent electrophysiological studies at Duke University Medical Center between February 1978 and November 1978 for evaluation of tachyarrhythmias.

Stimulating and Recording Techniques

The method of study of patients with preexcitation syndromes used in our laboratory has been described in detail elsewhere. As part of this study, all patients underwent incremental right ventricular pacing to an end point of ventriculoatrial block or until a pacing cycle length of 240 msec was achieved. All patients had multiple refractory period determinations performed from the right ventricular apex.

Biopolar atrial electrograms (50–1 kHz) were recorded from the lateral right atrium and the atrial septum (His bundle electrogram) throughout the course of the study. In addition, unipolar atrial electrograms (0.15–1 kHz) were recorded with a multipolar catheter at four to eight coronary sinus sites 1 cm apart. The most proximal electrode was positioned near the orifice of the coronary sinus. Bipolar derivatives of adjacent electrode pairs were recorded. The right atrial endocardium was mapped during right ventricular pacing (cycle length 350–400 msec) and during reciprocating tachycardia with a semirigid mapping catheter to verify the location of accessory pathways.

Measurement Techniques

Incremental pacing and refractory period data were measured and plotted using an interactive computer.
program. Retrograde conduction time over the accessory pathway was assessed by measuring ventriculoatrial intervals. These were measured from the onset of earliest recorded ventricular activity to the rapid deflection of the atrial electrogram closest to the accessory pathway.

An increment in ventriculoatrial conduction may be secondary to intraventricular delay, delay in the accessory pathway or intra-atrial delay. Intraventricular conduction delay was assessed by measuring intervals between the right ventricular electrogram (near the pacing electrode) and left ventricular electrograms (measured at the coronary sinus). The right ventricular-to-left ventricular (RV-LV) interval was then plotted as a function of cycle length (for fixed-rate pacing) or as a function of coupling interval (for right ventricular refractory period determinations). Intra-atrial delay was assessed by recording from several atrial locations during stimulation studies and measuring intra-atrial conduction intervals. The possible contribution of intra-atrial delay to ventriculoatrial conduction time was minimized by recording from the atrial electrogram closest to the accessory pathway.

Definitions

$S_1$, $A_1$ and $V_1$ refer to the stimulus, atrial electrogram and ventricular electrogram, respectively, of a basic drive beat.

$S_2$, $A_2$ and $V_2$ refer to the stimulus, atrial electrogram and ventricular electrogram, respectively, of the induced premature beat.

The antegrade effective refractory period (ERP) of the accessory pathway is the longest $A_1A_2$ not conducted with preexcitation at a specified pacing site and drive rate. $A_1A_2$ is measured at the atrial electrogram closest to the accessory pathway.

The retrograde ERP of the accessory pathway is the longest $V_1V_2$ that does not conduct to the atrium over the accessory pathway at a specified pacing site and drive rate. $V_1V_2$ is measured at the ventricular electrogram closest to the accessory pathway.

Ventriculoatrial conduction time is the interval from the earliest recorded ventricular activation to the intrinsic deflection of the atrial electrogram recorded at a specified site. Ventriculoatrial, conduction time refers to this interval measured at the atrial electrogram closest to the accessory pathway.

Results

Three patients had atypical patterns of retrograde conduction. These patterns were:

1) Gradual prolongation of ventriculoatrial conduction time during fixed-rate pacing of the right ventricle at progressively shorter cycle lengths.

2) Gradual prolongation of ventriculoatrial conduction time in response to increasing prematurity of a ventricular extrastimulus.

3) Sudden prolongation of ventriculoatrial conduction time in response to increasing prematurity of a ventricular extrastimulus ("discontinuous" retrograde curve).

Gradual Prolongation of Ventriculoatrial, Conduction Time during Fixed-rate Pacing of the Right Ventricle at Progressively Shorter Cycle Lengths

Patient TG was a 54-year-old male referred for assessment of tachycardia associated with the Wolff-Parkinson-White syndrome. We found supraventricular tachycardia with normal QRS morphology during the electrophysiologic study. The sequence of retrograde atrial activation was eccentric (earliest recorded activity occurring at the distal coronary sinus) and demonstrated the participation of a left lateral AV pathway during tachycardia.

During ventricular refractory period determinations, the sequence of retrograde atrial activation demonstrated symmetrical spread from the septum, suggesting conduction over the normal AV conduction system.

Figure 1 illustrates fixed-rate pacing of the right ventricle at progressively shorter cycle lengths. Figures 2A and B show electrograms corresponding to data points on the curve in figure 1 at cycle lengths 300 and 250 msec.

Comment

The abrupt change in the sequence of retrograde atrial activation at cycle length 300 msec and less (fig.
1) must represent block in the AV conduction system, with atrial activation subsequently occurring exclusively over the accessory pathway. Conduction over the accessory pathway resulted in retrograde atrial activation recorded earliest at the distal coronary sinus, near the site of the pathway. Conduction at cycle lengths longer than 300 msec occurred over both the normal AV conduction system and the accessory pathway (atrial fusion). The morphology of the unipolar atrial electrogram at the distal coronary sinus was unchanged at all pacing cycle lengths, suggesting that this part of the atrium was always activated from the same direction; that is, by the accessory pathway. Looking exclusively at the ventriculoatrial conduction interval measured at the distal coronary sinus (VDCSA in fig. 1), it is evident that the ventriculoatrial conduction interval started to lengthen at cycle lengths shorter than 380 msec. At the same time, the ventriculoatrial conduction interval measured at the atrial deflection at the His bundle electrogram, which is activated via the normal AV conduction system, was relatively constant. This strongly suggests that the atrium activated at the distal coronary sinus was activated via the accessory pathway, at least at cycle lengths shorter than 380 msec.

The RV-LV interval (coronary sinus) activations remain constant for cycle length 300 and 250 msec (fig. 2) and for all other cycle lengths, suggesting that intramyocardial delay did not account for the prolongation of the ventriculoatrial_{AP} conduction interval.

**Gradual Prolongation of Ventriculoatrial_{AP} Conduction Time in Response to Increasing Prematurity of a Ventricular Extrastimulus**

Patient JMS was a 24-year-old male who underwent electrophysiologic studies for assessment of recurrent supraventricular tachycardia associated with the Wolff-Parkinson-White syndrome. Reciprocating tachycardia of cycle length 305 msec was readily induced and terminated by rapid atrial pacing. During reciprocating tachycardia, the QRS morphology showed no preexcitation and each QRS complex was preceded by a His deflection with an HV interval of 45 msec. Earliest atrial activation during reciprocating tachycardia occurred at the anteromedial right atrial annulus and at the His bundle electrogram (ventriculoatrial conduction time, 105 msec).

This atrial activation sequence was compatible with reentry in the AV node or reentry using an anterosetal accessory pathway. The participation of an accessory pathway in reciprocating tachycardia was demonstrated by: 1) a premature ventricular depolarization introduced into diastole during reciprocating tachycardia, at a time when the His bundle was refractory, that was able to preexcite the atrium by 25 msec; 2) right bundle branch block aberration observed during reciprocating tachycardia that prolonged the ventriculoatrial conduction time by 20 msec.

Figure 3A is a retrograde refractory period curve (right ventricular pacing, cycle length 500 msec) for this patient. The sequence of retrograde atrial activation was compatible with conduction over the AV node or a septal accessory pathway (i.e., activation spreading from the septal region).

Figure 3B shows the coupling interval, measured at the left ventricular (coronary sinus) electrogram, plotted as a function of coupling interval, measured at the right ventricular electrogram, during refractory period determinations from the right ventricle. The curve followed the line of identity until a cycle length of 240 msec was reached. This suggested that intramyocardial delay from the right ventricular pacing site to the base of the left ventricle did not account for the observed increments in ventriculoatrial_{AP} conduction time. Measurement of a right ventricular basal electrogram near the accessory pathway terminus would have been preferable to a left ventricular basal electrogram in assessing the contribution of intramyocardial delay. However, further evidence for the absence of significant intramyocardial delay was provided by the fact that the QRS duration and morphology (leads 1, 2, 3, V_{III}, V_{I}) of the premature beat was identical to that of the drive beats at a time when significant ventriculoatrial delay was observed.

Incremental ventricular pacing resulted in no significant increment in ventriculoatrial_{AP} conduction time to cycle lengths of 293 msec.

The functional properties of this patient's accessory pathway are listed in table 1.

**Comment**

Both the atrial excitation sequence and the gradual increment of ventriculoatrial conduction time shown in figure 3A could suggest exclusive conduction over the normal AV conduction system. However, preexcitation of the atria by an appropriately timed right ventricular extrastimulus during reciprocating tachycardia, at a time when the His bundle was refractory, verified the presence and participation of an accessory pathway in this patient's tachycardia. The atria were depolarized by this extrastimulus in a sequence identical to that observed during right ventricular pacing. Therefore, retrograde conduction during refractory period determinations from the right ventricle must have occurred at least in part over the accessory pathway.

**Operative Results**

This patient underwent surgery for ablation of his accessory pathway. Intraoperative mapping verified its location in the right anterosetal region, anterior to the recorded His bundle electrogram. An incision was made in the right anterosetal region approximately 1 cm above the atrial annulus. The incision stopped short of the atrial extension of the membranous septum.

One week postoperatively, the patient underwent a second electrophysiologic study using right atrial (unipolar) and right ventricular (bipolar) electrodes temporarily implanted at the time of surgery. Ventriculoatrial conduction was absent at any ventricular cycle length. Reciprocating tachycardia could no
longer be elicited. Incremental pacing from the high right atrium showed 1:1 AV conduction to cycle length 300 msec. The ERP of the atrioventricular conducting system was 245 msec (right atrial pacing site, cycle length 500 msec).

Comment

The absence of retrograde conduction over the normal AV conduction system after ablation of the accessory pathway strongly suggests that the curve in figure 3 represents exclusive conduction over the accessory pathway. Alternatively, it may be argued that injury to the AV node at the time of surgery resulted in complete retrograde conduction block over this structure. We feel that this is unlikely for the following reasons:

1) Intraoperative mapping of the His bundle region insured that the operative dissection did not extend to the area of the recorded His bundle electrogram or the atrium posterior to this region; 2) antegrade conduction disturbance over the AV node was not observed at any time during surgery; 3) antegrade conduction and refractoriness determined at the postoperative study were well within normal limits.

Sudden Prolongation of Ventriculoatrial Conduction Time in Response to Increasing Prematurity of a Ventricular Extrastimulus ("Discontinuous" Retrograde Curve)

Patient LR was a 32-year-old male investigated for Wolff-Parkinson-White syndrome associated with a rapid ventricular response during atrial fibrillation. Sustained reciprocating tachycardia was not observed during the control study prior to drug testing. Isolated ventricular echo beats of normal, nonpreexcited QRS morphology were observed after single ventricular extrastimuli. These were preceded by a His deflection (HV interval 45 msec) and terminated after retrograde atrial depolarization.

During incremental pacing of the right ventricle, no significant increase in ventriculoatrial conduction time was noted to cycle lengths as short as 300 msec (table 1).
A retrograde refractory curve (right ventricular pacing, cycle length 600 msec) for this patient is shown in figure 4. The pattern of retrograde atrial activation was slightly eccentric, with atrial activation at the proximal coronary sinus preceding atrial activation at the His bundle electrogram by 15-18 msec. A gradual increase in ventriculoatrial conduction time, followed by an abrupt increase, was observed with increasing prematurity of the ventricular extrastimulus. The sequence of atrial activation remained essentially unaltered at all coupling intervals. No intramycocardial conduction delay was observed, as assessed by measurement of RV-LV intervals.

**Comment**

Although the atrial excitation sequence during the retrograde refractory determination suggested conduction over a left posteroseptal accessory pathway, we have observed retrograde atrial activation over the normal AV conduction system during which atrial activation at the orifice of the coronary sinus preceded atrial activation at the His bundle electrogram by up to 15 msec. The following data provide additional evidence that retrograde conduction occurred over the accessory pathway for the curve shown in figure 4 and that the curve reflects properties of an accessory pathway.

Figures 5A and B show the records before (panel A) and after (panel B) the sudden increment in ventriculoatrial conduction times. The sequence of atrial activation after the premature beat in panel A (coupling interval 300 msec) was identical to that after the premature beat in panel B (coupling interval 290 msec). In addition, a ventricular echo beat was of normal QRS morphology and preceded by a His deflection (HV 45 msec). The presence of these identical echo beats before and after the increment in ventriculoatrial intervals suggested that retrograde atrial activation preceding the ventricular echo beats occurred by the same route before and after the increment. This was shown to be an accessory pathway when reciprocating tachycardia was observed in this patient after the administration of disopyramide, 150 mg, intravenously. This tachycardia (fig. 6) was associated with a narrow QRS complex and a sequence of retrograde atrial excitation identical to that observed after the single ventricular echo beats and after the premature beats in figures 5A and B. A ventricular depolarization programmed into diastole at a time when the His bundle was refractory was able to

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**Table 1. Functional Properties of Accessory Pathways**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/sex (years)</th>
<th>Location of accessory pathway</th>
<th>Conduction</th>
<th>Antegrade</th>
<th>Refractoriness</th>
</tr>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Pacing site</td>
<td>Shortest CL (msec) with 1:1 conduction</td>
<td>Pacing site</td>
</tr>
<tr>
<td>JMS</td>
<td>24/M</td>
<td>Right anteroseptal</td>
<td>LRA</td>
<td>298</td>
<td>LRA</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>CS</td>
<td>&lt;302</td>
<td>CS</td>
</tr>
<tr>
<td>LR</td>
<td>32/M</td>
<td>Left posteroseptal</td>
<td>LRA</td>
<td>220</td>
<td>LRA</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CS</td>
<td>&lt;200</td>
<td>CS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Left lateral</td>
<td>LRA</td>
<td>450</td>
<td>LRA</td>
</tr>
<tr>
<td>TG</td>
<td>54/M</td>
<td></td>
<td>CS</td>
<td>455</td>
<td>CS</td>
</tr>
</tbody>
</table>

Abbreviations: CL = cycle length; LRA = lateral right atrium; CS = coronary sinus; RVA = right ventricular apex; ERP = effective refractory period; VA = ventriculoatrial.
* Atrial electrogram measured at the site closest to accessory pathway.
† Did not preexcite at this CL.
‡ Atrial excitation via atrioventricular node for all coupling intervals.

**Figure 4.** Ventriculoatrial (VA) conduction time as a function of coupling interval (RV₁-RV₂) of the ventricular extrastimulus in patient LR. An abrupt increase in VA time occurs at coupling interval 290 msec. The atrial excitation sequence remains the same.
TABLE 1. (Continued)

<table>
<thead>
<tr>
<th>Conduction</th>
<th>Retrograde</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pacing site</td>
<td>Shortest CL (msec) with 1:1 conduction</td>
</tr>
<tr>
<td>RVA</td>
<td>293</td>
</tr>
<tr>
<td>RVA</td>
<td>&lt;300</td>
</tr>
<tr>
<td>RVA</td>
<td>250</td>
</tr>
</tbody>
</table>

preexcite the atria by 38 msec with the identical atrial activation sequence observed during reciprocating tachycardia (fig. 6). This verified use of the accessory pathway in the observed tachycardia.

Discussion

The presence of retrograde conduction over an accessory AV connection during reciprocating tachycardia is indicated by 1) eccentric atrial activation sequence;4, 9, 10, 19, 20 2) preexcitation of the atria during reciprocating premature ventricular depolarization introduced when the His bundle is refractory without altering the sequence of atrial excitation observed during tachycardia;6 and 3) the observation of lengthening of the ventriculoatrial conduction interval when bundle branch block occurs during reciprocating tachycardia.5 During ventricular pacing at progressively shorter cycle lengths, conduction over accessory pathways has been shown to exhibit little or no increment in ventriculoatrial conduction time. Conduction over the normal AV conduction system usually, but not invariably,4, 9, 10, 11, 12 shows significant increments in ventriculoatrial conduction time as cycle length decreases. In a selected population of patients with incapacitating tachycardias associated with the Wolff-Parkinson-White syndrome, ventriculoatrial conduction over the normal conducting system was frequently associated with little or no increment in ventriculoatrial conduction time.21

In response to progressive prematurity of a programmed ventricular extrastimulus, ventriculoatrial conduction time over the normal pathway generally shows gradual lengthening, but it remains relatively constant over an accessory pathway.4, 9, 10, 11, 22-24 Discontinuous retrograde refractory curves have been associated with conduction exclusively over the normal AV conduction system.25 They have also been observed in the presence of retrograde conduction over both the normal and an accessory pathway when conduction in one of these suddenly fails.4, 9, 10, 25

An increase in ventriculoatrial conduction time may reflect: 1) intramyocardial delay between the right ventricle (near the pacing site) and the ventricle at the base of the heart (near the accessory pathway); 2) delayed conduction over the accessory pathway; or 3) delayed conduction between the atrium at the terminus of the accessory pathway and the recorded atrial electrogram.2, 4 Demonstration of prolonged conduction over the accessory pathway must exclude intramyocardial and/or intra-atrial delay as the source of any observed ventriculoatrial prolongation. Intra-atrial delay in our patients was excluded by measuring the ventriculoatrial interval at the atrial electrogram closest to the accessory pathway.4 Intramyocardial delay was excluded by demonstrating absence of delay between the right ventricular electrogram near the pacing electrode and the ventricular electrogram nearest the accessory pathway.

These conclusions must be made with consideration to the limitations of clinical electrophysiologic techniques. It is clearly not possible to position catheters precisely at the atrial and ventricular termini of the accessory pathway. The measurement of the RV-LV interval is a reasonable, clinically attainable measure of intramyocardial delay. Similarly, the recording of the atrial electrogram at a site as close as possible to the accessory pathway minimizes the contribution of intra-atrial delay. Intra-atrial delay can also be estimated by recording at several atrial sites.

The patients described in this report showed patterns of decremental retrograde conduction over accessory pathways that have previously been considered to be peculiar to conduction over the AV node. These patterns include:

1) Both gradual and sudden increments in ventriculoatrialAP conduction time in response to a programmed ventricular extrastimulus of increasing prematurity; and

2) Gradual increment in ventriculoatrialAP conduction time during fixed-rate ventricular pacing at progressively shorter cycle lengths.
This decremental behavior of retrograde conduction over accessory pathways was observed in three of 30 consecutive patients with Wolff-Parkinson-White syndrome studied in our laboratory. Patterns of retrograde conduction cannot be used alone to distinguish between conduction over an accessory pathway and that over the normal AV conduction system. This is especially important when distinguishing between reentry confined to the AV node and reentry using a septal accessory pathway, since the sequence of atrial excitation during reciprocating tachycardia may not be helpful in distinguishing them.

The demonstration of these properties raises questions about the anatomic substrate of accessory pathways. These pathways are generally felt to consist of strands of working myocardium joining atria and ventricle. It is possible that individual muscle strands may have nonhomogeneous physiologic properties or that several strands making up an accessory pathway may be capable of longitudinal dissociation. It is also possible that some accessory pathways are composed of tissue other than working myocardium. A recent pathologic study described an accessory pathway that was composed in part of cells resembling those of AV junctional specialized tissue. Such structures might have physiologic properties similar to the normal AV node. In two of the three patients described in this report, the accessory pathway was shown to be located in the atrial septal region. This may be fortuitous, but it is possible that the anatomic substrate of accessory pathways in this region differs from that of parietal accessory pathways. We recently described a case of the "permanent" form of junctional reciprocating tachycardia in which an accessory ventriculoatrial pathway to the posterior atrial septum was demonstrated. This pathway conducted only in the retrograde direction and also had decremental properties. The accessory pathway may have actually been an accessory AV node.

Although the anatomic substrate of these accessory pathways must remain speculative, the clinical implication is clear. Patterns of retrograde conduction with decremental properties can occur over accessory pathways. Such findings can not be used to rule out conduction over an accessory pathway.

**Figure 5.** A) Analog data (patient LR) during addition of right ventricular extrastimulus at coupling interval 300 msec (third beat). B) Analog data (patient LR) during addition of ventricular extrastimulus at coupling interval 290 msec (third complex). There is an abrupt prolongation of ventriculoatrial (VA) conduction at this coupling interval (compare to SA) without change in sequence of atrial excitation sequence. A ventricular echo beat is produced, identical to that shown in panel A. This echo beat is also identical to that seen during reciprocating tachycardia in this patient. The VA intervals and atrial activation sequence after this echo beat are also identical to that seen in reciprocating tachycardia.

**Figure 6.** Reciprocating tachycardia in patient LR. A ventricular depolarization (arrow) programmed into the cardiac cycle at a time when this His bundle is refractory is able to preexcite the atrium by 38 msec without altering the sequence of atrial excitation observed in tachycardia. The AA interval during tachycardia (318 msec) is shortened to 280 msec. The HH interval during tachycardia (318 msec) is unaltered after the induced ventricular extrastimulus (320 msec).
Acknowledgment

The authors express their gratitude to Laura Cook, R.N., and to Donald Kopp, L.P.N., the staff of the Clinical Electrophysiology Laboratory; to Jackie Kasell, electronics consultant; to Don Powell and David Hugett, who prepared the illustrations; and to Ann Clayton, who prepared the manuscript.

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Atypical patterns of retrograde conduction over accessory atrioventricular pathways in the Wolff-Parkinson-White syndrome.
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Circulation. 1979;60:1477-1486
doi: 10.1161/01.CIR.60.7.1477

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

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