Linking in Accessory Pathways
Functional Loss of Antegrade Preexcitation

Mario D. Gonzalez, MD; Arnold J. Greenspon, MD; and Gregory A. Kidwell, MD

Background. Concealed retrograde activation has been proposed as a mechanism for antegrade conduction block in the bundle branches and atrioventricular accessory pathways. We studied this hypothesis (linking) in 10 patients with the Wolff-Parkinson-White syndrome in whom antegrade preexcitation could be persistently blocked by overdrive atrial pacing.

Methods and Results. An atrial pacing protocol, with a decremental ramp followed by an incremental ramp, defined a range of atrial paced cycle lengths (linking window) associated with both persistent conduction and block in the accessory pathway. Within the limits of the linking window, the ability of an atrial impulse to conduct over the accessory pathway was dependent on the preceding state (i.e., conduction or block). The observed linking window ranged from 70 to 290 msec (mean, 185±68 msec) and closely approximated the measured delay in retrograde activation of the accessory pathway during persistent antegrade block. The mean antegrade effective refractory period of the accessory pathways was long (486±156 msec), and in each case, it exceeded the antegrade refractory period of the normal atrioventricular pathway. Critically timed premature ventricular extrastimuli, delivered while linking was maintained in the accessory pathway, were able to interrupt the linking and restore antegrade accessory pathway conduction.

Conclusions. These observations suggest that accessory pathway linking is associated with bidirectional block in the accessory pathway. The ability to initiate linking (and the stability of the phenomenon) depends on a critical relation between antegrade accessory pathway refractoriness and the magnitude of retrograde accessory pathway activation delay. (Circulation 1991;83:1221–1231)

Persistent bundle branch block can result from repeated concealed retrograde conduction of impulses propagated from the contralateral bundle branch. This was first postulated by Gouaux and Ashman1 in a clinical study of atrial fibrillation and later demonstrated by Wellens and Durrer2 in a patient with supraventricular tachycardia. Rosenbaum et al3 used the term “linking” to describe this phenomenon in patients with alternating bilateral bundle branch block. Similarly, persistent block in an accessory pathway during rapid atrial pacing has been attributed to repetitive, concealed retrograde conduction of impulses conducted over the normal atrioventricular (AV) pathway.4–6 This assumption was supported by restoration of conduction over the accessory pathway whenever the atrial impulse was blocked in the normal pathway.

The present study tested the hypothesis that delayed retrograde activation of the accessory pathway is responsible for the perpetuation of antegrade block during atrial pacing and, furthermore, that the magnitude of this delay is related to the conduction time from the site of pacing to the site of retrograde conduction block.

Methods

Among patients studied with overt Wolff-Parkinson-White syndrome, 10 were selected for this study on the basis of showing persistent antegrade block in the accessory pathway during decremental atrial pacing. There were five men and five women with a mean age of 40±12 years (mean±SD) (range, 22–58 years). All patients had recurrent episodes of spontaneous arrhythmia (atrial fibrillation in four and orthodromic tachycardia in six). The location of the bypass tract was left lateral in four, left anterior in four, left paraseptal in one, and right paraseptal in one. One patient had mitral valve prolapse, one had a congestive cardiomyopathy, and the remainder had no demonstrable organic heart disease. Seven of the patients were receiving antiarrhythmic drugs at the time of the study (amiodarone in four, procainamide in two, and flecainide in one). Persistent antegrade block in the accessory pathway could not be induced

From the Department of Medicine, Division of Cardiology, Jefferson Medical College, Thomas Jefferson University, Philadelphia.

Address for correspondence: Gregory A. Kidwell, MD, Suite 5611 NH, Thomas Jefferson University Hospital, 111 South 11th Street, Philadelphia, PA 19107.

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with atrial pacing during the drug-free study in any of these seven patients. Patients were studied in the fasting state after informed consent was obtained.

In four patients, programmable transesophageal atrial pacing was used as previously described, and therefore, only antegrade properties of AV conduction were assessed. In the remaining six patients, a complete intracardiac electrophysiological study was performed. Quadripolar or hexapolar catheters were percutaneously introduced and positioned in the right ventricular apex, right atrium, and coronary sinus. A tripolar catheter was positioned across the tricuspid valve for recording the His bundle potential. Multiple surface electrocardiographic leads and intracardiac electrograms (filter frequencies, 50–500 Hz) were simultaneously recorded on a multichannel direct ink jet recorder (Mingograph 800, Siemens-Elema, Solna, Sweden) at paper speeds of 50–200 mm/sec. Programmed atrial and ventricular stimulation was performed with a stimulator (Bloom Associates, Reading, Pa.) delivering rectangular pulses of 2 msec in duration at a current intensity of two times diastolic threshold. Refractory periods were determined by delivering a single premature stimulus at progressively shorter coupling intervals after a train of eight regularly driven basic stimuli. In addition, conduction over both the normal and the accessory pathways was evaluated during decremental and incremental atrial pacing. Decremental pacing ramps were performed by slowly decreasing the paced cycle length until the transition from 1:1 accessory pathway conduction to persistent antegrade block was defined. Thereafter, incremental pacing was performed by increasing the paced cycle length until antegrade accessory pathway conduction was restored. While persistent antegrade block in the accessory pathway was maintained by constant atrial pacing (linking), single ventricular extrastimuli were introduced at progressively shorter coupling intervals in an attempt to “peel back” the refractory period of the accessory pathway. Induction of supraventricular tachycardia was performed by delivering programmed atrial or ventricular extrastimuli.

Definitions

The antegrade effective refractory period of the AV node was defined as the longest A1–A2 interval at which A2 was not followed by a His deflection (H2). The antegrade effective refractory period of the accessory pathway was defined as the longest A1–A2 interval at which A2 was not associated with ventricular preexcitation. During decremental atrial pacing, “linking onset” was defined as the longest atrial paced cycle length producing persistent block in the accessory pathway with preservation of 1:1 conduction over the normal AV pathway. During incremental atrial pacing, “linking offset” was defined as the shortest atrial paced cycle length at which conduction over the accessory pathway was restored. The “linking window” was defined as the range of atrial paced cycle lengths associated with persistent antegrade block in the accessory pathway (offset minus onset).

Results

Persistent Block in the Accessory Pathway During Decremental-Incremental Atrial Pacing

Figure 1 shows a typical response to decremental-incremental atrial pacing in a patient with a right parasetal accessory pathway (patient 4). Leads I, aVF, V1, and V6 were simultaneously recorded at a slow paper speed (10 mm/sec). During decremental atrial pacing, cycle lengths between 500 and 345 msec were associated with 1:1 antegrade conduction over the accessory pathway. When the cycle length was further reduced to 340 msec, there was a sudden transition to persistent antegrade block in the accessory pathway with maintenance of 1:1 conduction over the normal AV pathway (linking onset). Conduction block persisted in the accessory pathway even though the cycle length was subsequently increased to values previously associated with 1:1 conduction (incremental ramp). Conduction over the accessory pathway resumed only when the cycle length was increased to 450 msec (linking offset). During incremental atrial pacing, cycle lengths between 340 and 450 msec resulted in block in the accessory pathway. Figure 2 depicts the decremental and incremental ramps of atrial pacing. It is evident that the same cycle length can result in either conduction (○) or block (●) in the accessory pathway. Between these limits (340–450 msec), the ability of an atrial impulse to conduct over the accessory pathway was dependent on the preceding state (i.e., conduction or block).

Figure 3 is a diagram that illustrates the proposed activation patterns during antegrade bypass conduction (left panel) and during persistent antegrade block in the accessory pathway (right panel). After antegrade block, delayed retrograde activation of the accessory pathway occurs by the normal AV pathway. The delay in retrograde activation of the accessory pathway equals the combined AV and ventriculoatrial (VA) conduction times along the circuit. This delayed activation results in a temporal shift of accessory pathway refractoriness and maintenance of antegrade block, provided that the next atrial impulse arrives within a critical time frame (linking window).

Estimation of Linking Window

Figure 4 shows the comparison between the observed and the estimated linking windows (left and right panels, respectively) in a patient with a left anterior accessory pathway (patient 2). Linking onset occurred at an atrial paced cycle length of 510 msec (left upper panel), and linking offset occurred at a paced cycle length of 720 msec (left lower panel). This results in an observed linking window of 210 msec. The AV conduction time was measured during atrial pacing at the site of the accessory pathway and at the cycle length of linking offset (pacing cycle
length, 720 msec). The VA conduction time was obtained during orthodromic tachycardia with a recording electrode positioned at the site of atrial insertion of the accessory pathway. In this patient, the estimated linking window (AV+VA intervals) was 220 msec and was similar to the observed linking window. However, in most patients, the estimated value was longer than the observed value (see below).

**Electrophysiological Findings**

Table 1 summarizes the pertinent electrophysiological findings in the study population. When accessory pathway linking was observed, the antegrade effective refractory period of the AV node was always less than the antegrade effective refractory period of the accessory pathway (290±45 versus 486±156 msec, respectively). This was expected, in that 1:1 AV conduction over the normal AV pathway is a necessary prerequisite to establish the AV–accessory pathway link. In fact, in most patients (seven of 10), linking could not be established during the drug-free electrophysiological study. This was because the effective refractory period of the normal AV pathway exceeded or equaled that of the accessory pathway in the absence of antiarrhythmic drug therapy. The three patients with accessory pathway linking in the drug-free state had antegrade refractory periods of 530, 510, and 340 msec. In those patients studied on antiarrhythmic drugs (patients 3–9), drug administration extended the accessory pathway effective refractory period beyond that of the AV node (mean increase, 244±179 msec). In the six patients in whom both were measured, the antegrade effective refractory period of the accessory pathway was always greater than the retrograde effective refractory period (395±100 versus 270±31 msec). This relation

**Figure 1.** Tracings demonstrating linking onset and offset. Continuous recording at a slow paper speed (10 mm/sec) demonstrates the effect of decremental (upper panel) and incremental (lower panel) atrial pacing in a patient with a right parasetal accessory pathway (patient 4). Surface electrocardiographic leads I, aVF, V1, and V6 are simultaneously displayed. For clarity, intracardiac electrograms are purposely omitted. Linking onset (ON) and offset (OFF) occurred at a cycle length of 340 and 450 msec, respectively. See text for additional details.

**Figure 2.** Plot of atrial paced cycle lengths (PCL) during a continuous decremental-incremental ramp (patient 4). ◯, antegrade conduction over the accessory pathway; Ⓜ, conduction block in the accessory pathway. Paced cycle length was gradually decreased until conduction block occurred in the accessory pathway and then was increased until conduction resumed. Linking window is defined by the range of atrial paced cycle lengths associated with antegrade block during the incremental ramp.
between antegrade and retrograde refractory periods is one that favors a stable linking interaction (see below).

Linking onset generally occurred at a cycle length slightly longer than the effective refractory period of the accessory pathway; that is, accessory pathway refractoriness was longer during constant overdrive pacing compared with that of extrastimulus method. Nevertheless, a strong correlation existed between the observed paced cycle length of linking onset and the antegrade effective refractory period of the accessory pathway (r=0.97, see Figure 5). The observed linking window ranged from 70 to 290 msec. The retrograde activation delay could be estimated in eight of the 10 patients (orthodromic supraventricular tachycardia could not be induced at the time of the study in patients 8 and 9). Although the estimated retrograde activation delay provided a good approximation of the observed linking window, the estimate was longer than the observed linking window in most patients (220±64 versus 184±77 msec).

The effect of decremental and incremental pacing from different atrial sites was only tested in two patients. Theoretically, the linking window should increase if the time between antegrade accessory pathway block and retrograde activation increases. In patient 1 (left lateral accessory pathway), the linking window increased by 35 msec when coronary sinus pacing (at the site of the accessory pathway insertion) was compared with low right atrial pacing (near the AV node). This was expected because pacing at the site of the accessory pathway should maximize the relative retrograde activation delay (AV+VA, see Figure 3). However, in patient 4 (right paraseptal accessory pathway), distal coronary sinus pacing also increased the linking window by 40 msec compared with low right atrial pacing. This was unexpected because the change in pacing site did not result in an appreciable change in the relative antegrade and retrograde activation times. When possible (invasive studies), linking was initiated with atrial pacing at the site of accessory pathway insertion. This was done to

**Figure 3.** Schematic representation of accessory pathway (AP) linking. *, pacing from the distal coronary sinus (CS). Left panel: Each atrial complex is conducted over both pathways. Right panel: After the initiation of antegrade block in the accessory pathway, the stimulus is conducted sequentially from the left atrium to the low right atrium (LRA), atrioventricular node, His-Parkinje system, and then retrogradely to the ventricular insertion of the bypass tract. Numbers represent conduction times in milliseconds.

**Figure 4.** Tracings demonstrating comparison of the observed and the estimated linking windows. Left panels: Atrial pacing from the distal coronary sinus is performed while recording electrograms from the coronary sinus (CS), His bundle region (HBE), and surface electrocardiographic lead V1 (patient 2). Observed linking window is the difference between the offset and onset paced cycle lengths (720−510=210 msec). Estimated linking window is obtained by adding the atrioventricular (AV) interval (measured during persistent linking) to the ventriculoatrial (VA) interval (measured during orthodromic supraventricular tachycardia 120+100=220 msec). A, atrium; V, ventricle. See text for further details.
TABLE 1. Electrophysiological Findings in Patients With Linking in the Accessory Pathway

<table>
<thead>
<tr>
<th>Patient</th>
<th>Drug</th>
<th>ERP (msec)</th>
<th>Linking (msec)</th>
<th>Window (msec)</th>
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<tr>
<td></td>
<td></td>
<td>AP_{ant}</td>
<td>AVN</td>
<td>AP_{ret}</td>
</tr>
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<td>530</td>
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<td>310</td>
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<td>None</td>
<td>530</td>
<td>260</td>
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<td>Amiod</td>
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<td>8</td>
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<td>9</td>
<td>Amiod</td>
<td>245</td>
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</tr>
<tr>
<td>10</td>
<td>None</td>
<td>340</td>
<td>240</td>
<td>&lt;240</td>
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</table>

In patients 6–9, only atrial pacing (transesophageal) was performed. Atrial pacing in patient 10 was performed at a site distant (high right atrium) from the atrial insertion of the accessory pathway (left anterior). Consequently, the observed and estimated linking windows were significantly less than the group means (see text for discussion).

ERP, effective refractory period; AP_{ant}, antegrade accessory pathway during the drug-free state; AP_{ret}, antegrade accessory pathway during drug therapy; AVN, atrioventricular node; AP_{nos}, retrograde accessory pathway; Proc, procainamide; Flec, flecainide; Amiod, amiodarone.

provide the greatest possible retrograde activation delay (relative to the time of antegrade accessory pathway block). However, in patient 10 (left anterior accessory pathway) linking could not be established when pacing was performed from the distal coronary sinus. In this patient, the antegrade effective refractory period of the accessory pathway was 340 msec, whereas the retrograde effective refractory period of the accessory pathway was limited by ventricular refractoriness (<240 msec). The estimated retrograde activation delay (AV+VA) in this patient was 270 msec during coronary sinus pacing. Although antegrade accessory pathway block could be easily achieved with coronary sinus pacing, it was always associated with the initiation of orthodromic supraventricular tachycardia (i.e., the retrograde impulse arrived after the expiration of accessory pathway refractoriness). When decremental pacing was performed in the high right atrium, accessory pathway linking with a window of 70 msec was reproducibly observed. This shift in pacing site decreased the estimated retrograde activation delay to 105 msec (change in the relative antegrade and retrograde activation times of 165 msec). Consequently, the shift in pacing site caused the retrograde impulse to arrive before the expiration of accessory pathway refractoriness resulting in the bidirectional block necessary for stable accessory pathway linking.

Restoration of Antegrade Accessory Pathway Conduction After an Appropriately Timed Ventricular Extrastimulus

If persistence of antegrade block during constant atrial pacing is secondary to delayed retrograde activation of the accessory pathway, antegrade conduction should be restored by preexciting the region of retrograde block. Figure 6 shows the effect of single ventricular extrastimuli, delivered during constant atrial pacing from the coronary sinus, in a patient with a left lateral accessory pathway (patient 2). In this case, linking onset (panel A) and linking offset (panel B) occurred at atrial paced cycle lengths of 550 and 740 msec, respectively. Accessory pathway linking at an atrial paced cycle length of 600 msec produced persistent antegrade accessory pathway block (panels C and D). The introduction of a properly timed ventricular extrastimulus resulted in the resumption of antegrade conduction of previously blocked atrial impulses. In this patient, ventricular extrastimuli with coupling intervals between 600 and 420 msec failed to restore antegrade conduction over the accessory pathway. In contrast, ventricular extrastimuli with coupling intervals equal to or less than 410 msec were able to restore antegrade conduction. Simple collision of the retrograde ventricular impulse in the AV node (atrial electrogram not

FIGURE 5. Plot of relation between linking onset and the antegrade effective refractory period (ERP) of the accessory pathway. Although the paced cycle length of linking onset was generally greater than the antegrade effective refractory period of the accessory pathway, a good correlation was observed between the cycle length of antegrade accessory pathway block (linking onset) and the antegrade accessory pathway effective refractory period.
followed by a His deflection) was not sufficient to restore antegrade accessory pathway conduction. Successful restoration of antegrade accessory pathway conduction by ventricular extrastimuli required a critical temporal shift in accessory pathway activation.

Figure 7 is a ladder diagram that suggests a mechanism for initiation and interruption of linking in patient 2. The accessory pathway is represented with different antegrade and retrograde refractory periods (determined by the extrastimulus technique, see Table 1). No precise anatomic location is assumed because block may occur in the accessory pathway itself or in the surrounding atrial or ventricular muscle. When the atrial paced cycle length reaches the antegrade accessory pathway refractory period (500 msec, upper panel), block occurs in the accessory pathway, but conduction proceeds through the normal AV pathway (dotted line). The ventricular–accessory pathway junction is then activated retrogradely, 220 msec after the time of antegrade block. This temporally shifts the refractory period of the accessory pathway by 220 msec and perpetuates the antegrade block. At an atrial paced cycle length of 600 msec (lower panel), antegrade block persists because of a residual refractoriness of 120 msec in the accessory pathway. (Residual refractoriness of the accessory pathway is estimated by subtracting the atrial paced cycle length from the sum of the antegrade effective refractory period of the accessory pathway and the retrograde activation delay. Consequently, residual refractoriness will vary inversely with the atrial paced cycle length). There are two possible ways to restore antegrade conduction: 1) increasing the atrial paced cycle length (by an amount sufficient to exceed residual refractoriness of the accessory pathway) or 2) advancing retrograde activation of the accessory pathway with a premature ventricular stimulus. In the lower panel, a properly timed ventricular extrastimulus is shown to preexcite the region of block and restore antegrade conduction. Theoretically, the longest ventricular extrastimulus coupling interval (V−Vₖ) capable of restoring antegrade conduction can be estimated with the following equation:

\[ V - V_c \leq PCL - (AP_{RR} + CT_p) \]

where PCL is the atrial paced cycle length, AP_{RR} is the residual refractoriness in the accessory pathway after the arrival of the atrial impulse, and CT_p is the interventricular conduction time (estimated as the difference between the VA time during right ventricular pacing and reciprocating tachycardia).

The ability of ventricular extrastimuli to restore antegrade conduction through the accessory pathway was also dependent on the atrial pacing rate and the ventricular refractory period. Figure 8 compares the effects of ventricular extrastimuli applied during two
different atrial paced cycle lengths. During linking at an atrial paced cycle length of 600 msec (left panels), ventricular extrastimuli with coupling intervals of 410 msec or less were able to restore antegrade accessory pathway conduction. In contrast, when the atrial paced cycle length was reduced to 500 msec, even a ventricular extrastimulus with the shortest possible coupling interval was not able to restore conduction through the accessory pathway. Figure 9 is a schematic representation of the proposed mechanism by which the atrial cycle length can affect the ability of ventricular extrastimuli to restore antegrade accessory pathway conduction. As in the previous example, the antegrade refractory period and the AV+VA time are considered to be 500 and 220 msec, respectively (patient 2). At an atrial cycle length of 600 msec (upper panel), the residual refractoriness of the accessory pathway is 120 msec. However, when the atrial cycle length is decreased to 500 msec (lower panel), residual refractoriness increases to 220 msec. In this case, the interventricular conduction time was estimated to be 80 msec. Therefore, at an atrial paced cycle length of 600 msec, the ventricular extrastimulus must be applied at least 200 msec (120+80 msec) before the next expected ventricular depolarization to restore antegrade accessory pathway conduction, resulting in a coupling interval of 400 msec (600–200 msec). At an atrial paced cycle length of 500 msec, a ventricular extrastimulus with a coupling interval of 200 msec or less would be required [500–(220+80) msec] to sufficiently shift antegrade accessory pathway refractoriness and restore conduction. Ventricular extrastimuli of this coupling interval were prevented by ventricular refractoriness.

**Discussion**

This study demonstrates that during constant atrial pacing, persistent antegrade block in an accessory pathway can be maintained by concealed antegrade and retrograde activation. This phenomenon has been previously described by other investigators\(^6,^7\) and is now extended by our observations. Preservation of antegrade conduction through the normal AV pathway, at atrial cycle lengths that induce block in the accessory pathway, was a prerequisite for perpetuating conduction block in the accessory pathway (linking phenomenon). Our findings show that the extent of the conduction delay through the normal AV pathway is directly related to the stability of the phenomenon (linking window).
Persistence of Conduction Block at a Rate That Previously Resulted in 1:1 Atrioventricular Conduction: Linking Versus Fatigue Phenomenon

In a patient with atrial fibrillation, Gouaux and Ashman observed that prolonged periods of right bundle branch aberrancy occurred at a rate that previously resulted in normal intraventricular conduction. To explain this phenomenon, they postulated that aberrant conduction in one bundle branch was maintained by retrograde invasion of impulses conducted through the contralateral bundle branch. Moe et al. were able to show that this mechanism could operate long enough to perpetuate functional bundle branch block in the canine heart. After establishing linking in the accessory pathway, at an atrial paced cycle length near the refractory period of the accessory pathway, we were able to maintain linking throughout a wide range of paced cycle lengths. The increase in the paced cycle length necessary to restore antegrade accessory pathway conduction ranged from 70 to 290 msec (mean window, 185±68 msec). This range of paced cycle lengths was closely approximated by the estimated delay in retrograde accessory pathway activation (AV+VA time). In patients in whom the activation delay could be estimated (eight of 10 studies), the estimated linking window was 216±70 msec compared with an observed window of 184±77 msec. This finding strongly suggests that accessory pathway linking depends on a temporal shift of refractoriness at the ventricular insertion of the accessory pathway. Narula and Runge and Fisch et al. used the term “fatigue” to describe transient depression of conduction in the His-Purkinje system after a period of rapid pacing. They postulated that repetitive penetration of the affected site was responsible for the initiation of block. Their descriptions included overdrive suppression of conduction with both atrial and ventricular pacing. This mechanism was clearly not operative in our patients. Although conduction block in the accessory pathway was dependent on a critical rate of atrial pacing, conduction could be restored with a single premature ventricular extrastimulus (see below), and identical atrial rates were associated with both conduction and block in the accessory pathway.

Conduction Properties of the Accessory Pathway During Decremental-Incremental Atrial Pacing

During decremental atrial pacing, all our patients showed an abrupt transition from 1:1 antegrade conduction to complete and persistent block in the accessory pathway. It could be postulated that repeated antegrade activation would maintain the block. However, this type of response is not observed in experimental models. Inoue and Zipes showed that when a narrow isthmus of tissue with depressed conduction is stimulated, transient, not persistent, conduction block is observed. In contrast to the experimental models, the accessory pathway is activated asynchronously from both sides during the linking phenomenon. This bidirectional activation results in repetitive conduction failure in both directions. In our study population, the antegrade effective refractory periods of the accessory pathways were long (486±156...
msec). This is in part due to selection bias but is primarily due to antiarrhythmic drug therapy. The antegrade effective refractory period increased by 244±179 msec in the seven patients who received antiarrhythmic drugs. In all cases, drug administration extended accessory pathway refractoriness beyond that of the normal AV pathway and allowed us to induce and study the linking phenomenon.

As previously noted, the range of atrial paced cycle lengths associated with accessory pathway linking was closely related to the estimated delay in retrograde activation of the pathway. However, in all but one patient, the observed linking window was less than the estimated window. This finding is not readily explained. Fuente et al16 studied conduction through a surgically defined narrow isthmus of canine atrial tissue. They suggested that conduction in this preparation was similar to that observed in accessory pathways. Unidirectional block could be easily demonstrated to occur at the junction of the narrow band with the larger tissue mass. They showed that electrotonic interactions at the site of block could accelerate the repolarization of the cells proximal to the block. This electrotonic effect has also been reported in other in vitro models.17,18 Similarly, electrotonic influences at the site of block may be responsible for the difference between the observed and estimated linking windows. The estimated value assumes that

the duration of the antegrade effective refractory period of the accessory pathway is constant and similar to that determined with the extrastimulus technique. During the linking phenomenon, distal repolarized tissue could electronically abbreviate repolarization of the proximal elements. During bidirectional block, this mechanism could serve to decrease both the antegrade and retrograde refractory periods of the accessory pathway, thereby producing linking windows that are shorter than the estimated duration. In addition, failure to pace the atrium at a site close to the atrial insertion of the accessory pathway could potentially result in an observed linking window less than the estimated value.

Restoration of Antegrade Accessory Pathway Conduction by a Ventricular Extrastimulus

Wellens and Durrer2 postulated the mechanism of linking to explain left bundle branch aberrant conduction during supraventricular tachycardia. They also observed that after a premature ventricular complex the QRS normalized even though the atrial rate remained unchanged. We delivered single ventricular extrastimuli to advance the retrograde activation of the accessory pathway. Our results show that there is a minimal degree of prematurity necessary to facilitate antegrade conduction. This critical value is in accordance with the concept of “peeling
back” a refractory barrier as previously described in experimental studies of the normal AV pathway. Normalization of antegrade conduction in the accessory pathway after block in the normal AV pathway supports the theory that concealed retrograde activation of the bypass tract prevents antegrade conduction. However, the delayed nature of this retrograde activation has not been previously tested. In our study, ventricular extrastimuli that were insufficiently premature were not capable of restoring antegrade accessory pathway conduction even though conduction through the normal AV pathway was prevented.

Facilitation is another potential mechanism for the restoration of antegrade conduction after a premature ventricular extrastimulus. In a model of anisotropic conduction in depolarized Purkinje fibers, Gilmour et al showed that pacing from one side of the preparation could transiently improve conduction from the opposite side. However, this improvement in conduction was assessed only after several paced beats (overdrive facilitation) and not after a single extrastimulus. More importantly, they studied advanced degrees of conduction block and not persistent block, as observed in this study. In our patients, persistent block was changed to persistent conduction by a single ventricular extrastimulus, suggesting that preexitation of the refractory barrier is the operative mechanism. In patients with advanced AV block, AV conduction can be temporarily restored after a ventricular escape beat. A supernormal phase of conduction and Wedensky facilitation have been postulated to account for this restoration of conduction. Again, however, the effect on conduction is transient and not persistent as in our patients.

Limitations

The accessory pathway potential was not recorded, and therefore, the exact site of antegrade and retrograde block cannot be stated with certainty. In addition, the interactions between conducted and nonconducted impulses at the site of block are likely to be complex and not fully described by these results. Nevertheless, the observations on the conduction patterns during persistent linking of the accessory pathway remain valid. Regardless of the site of block, a temporal alternation in refractoriness, secondary to an activation delay, is consistent with our observations. Although other factors may modulate the response, the measured retrograde activation delay of the accessory pathway (during linking) was a good estimate of the linking window in our patients. Further study will be required to fully understand the mechanisms responsible for variance of the observed linking window from this estimated value. Although antiarrhythmic drugs were administered in most of our patients, linking can clearly occur in the drug-free state (patients 1, 2, and 10), and we do not believe that this is a significant limitation of the study. The linking phenomenon requires that the antegrade refractory period of the accessory pathway exceed that of the normal AV pathway, and in this study, antiarrhythmic drugs were used as a tool to study this electrophysiological behavior.

Electrophysiological Implications

Linking of an accessory pathway and the normal AV pathway can be readily demonstrated. This phenomenon can account for a functional loss of antegrade preexcitation at an atrial cycle length that is greater than the antegrade refractory period of the accessory pathway. To establish accessory pathway linking, the antegrade effective refractory period of the accessory pathway must exceed that of the normal AV pathway. Consequently, this phenomenon is expected in accessory pathways with long antegrade refractory periods. Although not observed in our study population, linking may be partly responsible for intermittent preexcitation during sinus rhythm in patients with long antegrade accessory pathway refractory periods.

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M D Gonzalez, A J Greenspon and G A Kidwell

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