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A FUNCTIONAL APPROACH TO THE PREEXCITATION SYNDROMES

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The preexcitation syndromes encompass a variety of electrocardiographic manifestations and arrhythmias, all of which are related to the presence of an anomalous connection (accessory pathway). Accessory pathways differ in the structures they connect and in their functional properties. These differences account for the variability in electrocardiographic patterns and arrhythmias. This article describes our approach to patients with preexcitation syndromes. We begin with an assessment of the location and functional properties of accessory pathways, followed by an evaluation of arrhythmias. This information provides the basis for selecting appropriate therapy. For a more detailed discussion of the anatomic and histologic features and of the historical evolution of the terminology, the reader is referred to several excellent reviews.[17, 40, 96]

FUNCTIONAL AND ELECTROPHYSIOLOGIC PROPERTIES OF ACCESSORY PATHWAYS

Accessory Atrioventricular Pathways Associated with Preexcitation

The most common type of accessory pathway is composed of working myocardium and connects the right or left atrium to the adjacent ventricle. Most of these accessory atrioventricular (AV) pathways are capable of conduction in both the antegrade (atrium to ventricle) and retrograde (ventricle to atrium) directions. The eponym "Kent bundle," frequently used to describe this type of accessory pathway, is probably a misnomer because Kent[65, 66] described a gnarl of specialized conduction fibers located in the right anterolateral region of the tricuspid anulus that may be the substrate for Mahaim fibers (right atriofasicular fibers) described later in this article.

Almost all left free-wall accessory pathways course through the subepicardial fat pad and connect the epicardial surface of the left atrium to the epicardial surface of the left ventricle (Fig. 1).[12, 13] Right-sided pathways may course subepicardially or subendocardially.[12, 13] Anatomic studies have shown that some accessory AV pathways are branching structures.[12, 13] The presence of multiple functioning components has been demonstrated in many patients (1) by recording multiple distinct accessory pathway activation potentials at different times from the same closely spaced bipolar electrode, (2) by recording simultaneous accessory pathway potentials at separate positions, or (3) by documenting intermittent loss of one potential with a small prolongation of the accessory
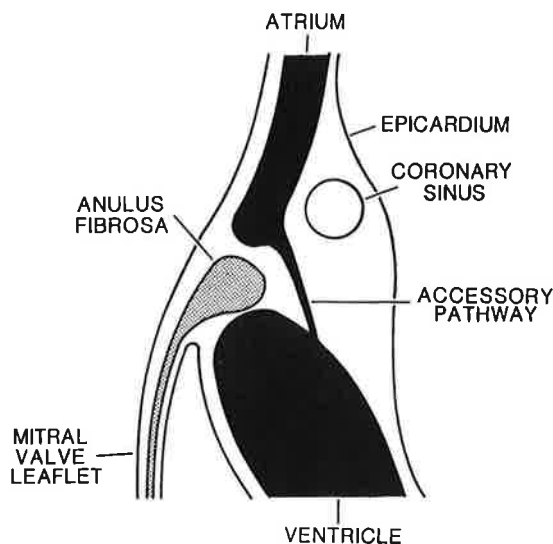
Figure 1. Diagrammatic cross-sectional view of the mitral anulus illustrating the typical course of left free-wall accessory AV pathways. The accessory pathway is located on the epicardial side of the mitral anulus, connecting the epicardial surfaces of the left atrium and ventricle. (*From* Jackman WM: New catheter techniques for recording accessory AV pathway activation. *In* Benditt DG, Benson DW (eds): Cardiac Preexcitation Syndromes. Origins, Evaluation, and Treatment. Boston, Martinus Nijhoff, 1986, p 413; with permission.)

pathway conduction time.[59, 60, 61, 64] In approximately 20% of patients undergoing radiofrequency catheter ablation, accessory pathway conduction is eliminated incrementally by applications of energy at multiple close sites.[63, 84] This further supports the concept that accessory pathways are composed of multiple functional components.

Accessory AV pathways can be located anywhere along the mitral or tricuspid anulus except the aortic-mitral continuity (the left anteroseptal region). In 439 patients undergoing electrophysiologic study at our institution, 55% of accessory AV pathways were located in the left free-wall, 25% in the posteroseptal region, 14% in the right free-wall, and 6% in the anteroseptal region. Multiple accessory pathways (at lease 3 cm apart) were present in 5% of patients. This distribution is similar to that in earlier reports.[9, 47, 105]

The true prevalence of accessory pathways in the total population is unknown. Studies involving screening electrocardiograms in large populations (i.e., military pilots) have found ventricular preexcitation in 0.1% to 0.3% of subjects, the majority of whom were asymptomatic.[51, 107, 110] For an overall preva-

lence, this value should be increased by approximately 25% to include individuals with accessory pathways that conduct only in the retrograde direction (no preexcitation) and individuals with negligible preexcitation during sinus rhythm due to short AV nodal conduction times with delayed ventricular activation via the accessory pathway due to left lateral location or long intrinsic accessory pathway conduction times.

Most patients with accessory pathways have no other structural heart disease. However, some congenital cardiac anomalies are associated with a relatively high prevalence of accessory pathways. Accessory pathways are found in 4% to 26%[46, 104] of patients with Ebstein's anomaly, and there is a higher than expected prevalence in patients with mitral valve prolapse and hypertrophic cardiomyopathy.

The conduction properties of the common form of accessory AV pathway are similar to those of normal atrial or ventricular myocardium. The conduction time is short (20 to 50 msec) and remains relatively constant during decremental atrial or ventricular pacing until the pacing cycle length is shortened to within 50 msec of the cycle length that produces accessory pathway conduction block. Close to the cycle length producing block, the accessory pathway conduction time lengthens somewhat, usually by less than 40 msec. The refractory period of the accessory pathway shortens as the heart rate increases.[119] This accounts for the rapid ventricular rate during atrial fibrillation observed in some patients.

The conduction properties of the common form of accessory AV pathway are usually minimally affected by changes in autonomic tone.[99] However, in some patients, the conduction capability of the accessory pathway increases significantly with the administration of catecholamines such as isoproterenol or is depressed by beta-adrenergic blocking agents.[44, 115, 125] Similarly, although calcium channel blockers and adenosine usually do not directly affect accessory pathway conduction,[92, 101, 116] depression of accessory pathway conduction may be substantial in individual patients.

An early study found that ouabain shortened the refractory period of the accessory pathway.[126] The principal effect of ouabain is a centrally mediated increase in vagal tone.[24] Because cholinergic stimulation shortens refractoriness in the atrium[97] and lengthens

refractory periods in all other cardiac tissues, this observation suggests that some accessory pathways may be composed of atrial myocardium.

The conduction capability of accessory pathways may differ significantly between patients. In addition, the conduction capability of an accessory pathway may be different in the antegrade and retrograde directions. The accessory pathway may be able to conduct impulses at rapid rates in the antegrade direction but to conduct impulses only at slow rates in the retrograde direction and vice versa. These properties determine the type and severity of arrhythmias that can occur in individual patients. Orthodromic AV reentrant tachycardia is likely in a patient in whom the pathway has a short refractory period in the retrograde direction and a long refractory period in the antegrade direction. Orthodromic AV reentrant tachycardia is unlikely if the refractory period in the retrograde direction is very long. Conversely, antidromic AV reentrant tachycardia may occur in a patient with a short antegrade accessory pathway refractory period and long retrograde refractory period and is unlikely to occur if the antegrade refractory period is long.

The variability in the conduction capability of accessory pathways is probably related to differences in the anatomic geometry of the pathways rather than to differences in cell type or function. Conduction may be limited at the site of an abrupt change in pathway diameter (impedance mismatch hypothesis)[33, 54] or at a change in the direction of fiber orientation (anisotropy).[111] Studies using catheter recordings of accessory pathway activation have shown that for a given accessory pathway, the conduction-limiting site (weakest link) is usually the same for conduction in the antegrade and retrograde directions.[73] In left free-wall accessory AV pathways, conduction is usually limited near the ventricular insertion of the accessory pathway (Fig. 2). Conduction over right-sided and septal pathways may be weakest at either the ventricular or atrial insertion site.

Electrocardiographic and Electrophysiologic Characteristics

Ventricular preexcitation is present if any part of the ventricles is activated earlier by the accessory pathway than it would have been if the atrial impulse reached the ventricles exclusively over the normal AV conduction system. During sinus rhythm in most
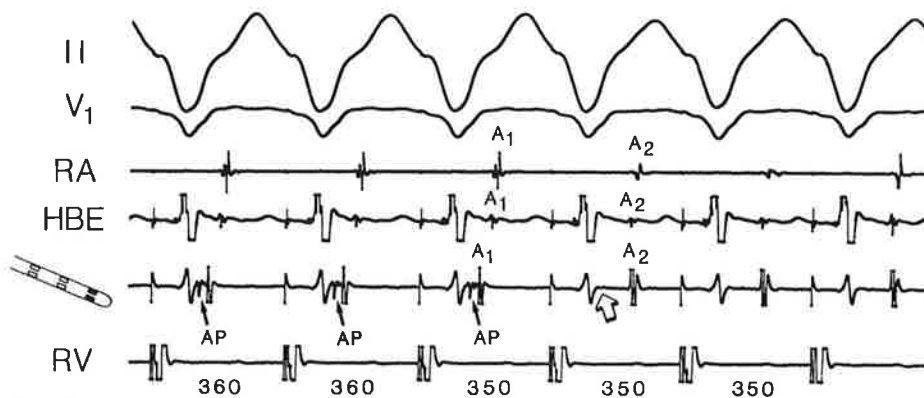
Figure 2. Use of catheter recordings to identify the site of retrograde conduction block in a left posterior accessory AV pathway. From the top, tracings are leads II and V$_1$, and electrograms from the right atrium (RA), His bundle region (HB), orthogonal electrodes on a catheter in the coronary sinus close to the accessory pathway (catheter symbol),[59] and right ventricle (RV). During right ventricular pacing at cycle length 360 msec (first three complexes), retrograde (VA) conduction occurred over the accessory pathway, with the earliest retrograde atrial potential recorded from the coronary sinus (A$_1$). An activation potential from the accessory pathway (AP) was recorded before atrial activation. As the ventricular pacing cycle length was shortened from 360 msec to 350 msec, retrograde block in the accessory pathway occurred and retrograde conduction occurred over the AV node, reflected by abrupt prolongation of the VA interval and a shift in the site recording the earliest atrial potential to the HB electrogram (A$_2$). The loss of retrograde accessory pathway conduction was associated with the loss of the retrograde accessory pathway potential (*open arrow*) indicating block occurred at or near the ventricular end of the accessory pathway. (*From* Jackman WM: New catheter techniques for recording accessory AV pathway activation. *In* Benditt DG, Benson DW (eds): Cardiac Preexcitation Syndromes. Origins, Evaluation, and Treatment. Boston, Martinus Nijhoff, 1986, p 428; with permission.)
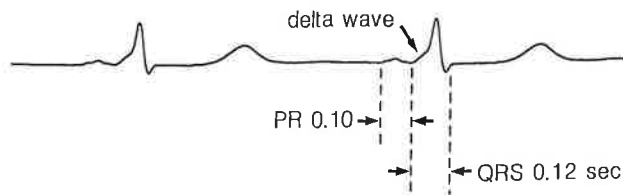
Figure 3. Electrocardiagram lead II demonstrating the features of ventricular preexcitation that include (1) a short PR interval, (2) a delta wave, and (3) prolonged QRS duration.

patients with preexcitation, the ventricle is activated by both the accessory pathway and the AV node, resulting in a fusion QRS complex. The delta wave, or initial slurred portion of the QRS complex, represents activation of the portion of the ventricles that is prematurely activated by the accessory pathway. As illustrated in Figure 3, the electrocardiographic description of ventricular preexcitation includes (1) a shortened PR interval (often less than 120 msec), (2) the presence of a delta wave, and (3) prolonged QRS duration (greater than 120 msec). The amount of the QRS complex resulting from preexcitation is variable and is greatest when (1) the accessory pathway is located close to

the sinus node, (2) the accessory pathway conduction time is short, and (3) AV nodal conduction time is prolonged. Preexcitation may be minimal or absent on a standard electrocardiogram during sinus rhythm, despite the presence of antegrade accessory pathway conduction, when the accessory pathway is located far from the sinus node (i.e., left lateral accessory pathway) and the AV nodal conduction time is short (Fig. 4).

The presence or absence of ventricular preexcitation can be determined definitively by an electrophysiologic study in which transvenous electrode catheters are used to record activation of key regions of the heart during programmed electrical stimulation of
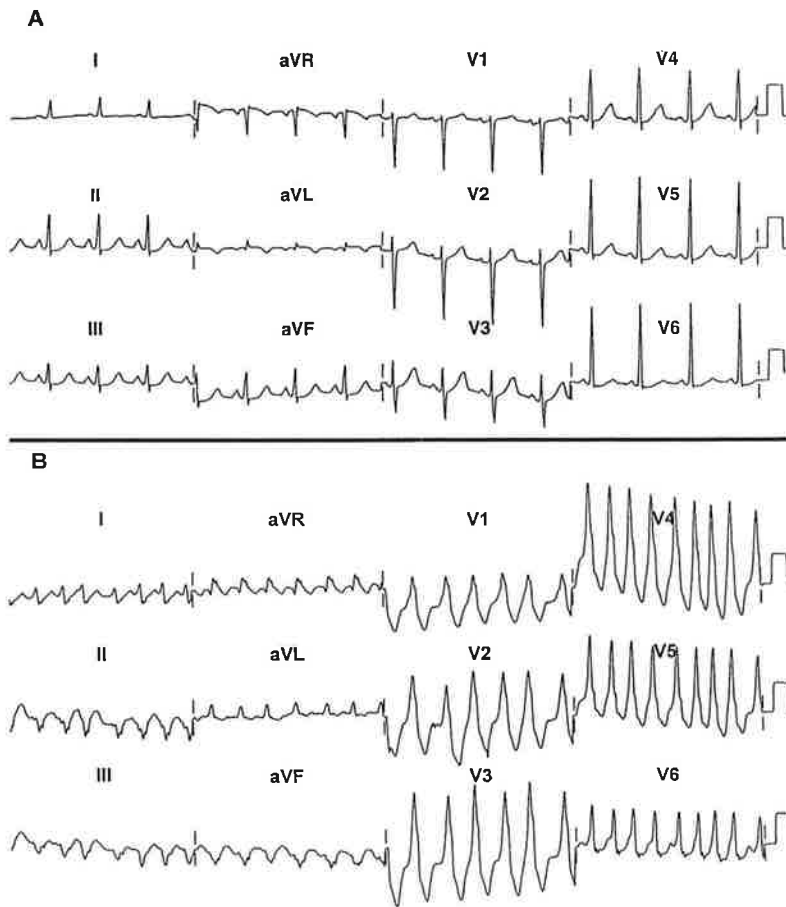
Figure 4. A, Ventricular preexcitation was not evident during sinus rhythm in this patient with a left free-wall accessory pathway, located far from the sinus node. B, However, during atrial fibrillation in this same patient, conduction over the accessory pathway resulted in a rapid ventricular response with preexcited QRS complexes.

the atria and ventricles. In patients without an accessory pathway, the His bundle must be activated (producing a His bundle potential) before activation of the ventricles. The interval from His bundle activation to activation of the ventricles (H to V interval) is at least 35 msec. In patients with an accessory pathway that bypasses the normal conduction delay in the AV node, the onset of ventricular activation is not dependent on His bundle activation. The resulting H to V interval is shortened. Ventricular activation can even occur before His bundle depolarization, resulting in a negative H to V interval. The hallmark of antegrade accessory pathway conduction is progressive shortening of the H to V interval with a progressive increase in preexcitation as AV nodal conduction time is lengthened by programmed atrial stimulation (Fig. 5). With shorter premature atrial coupling intervals or faster atrial pacing rates, AV nodal delay increases, which delays activation of the ventricles by the His-Purkinje system. Because the conduction
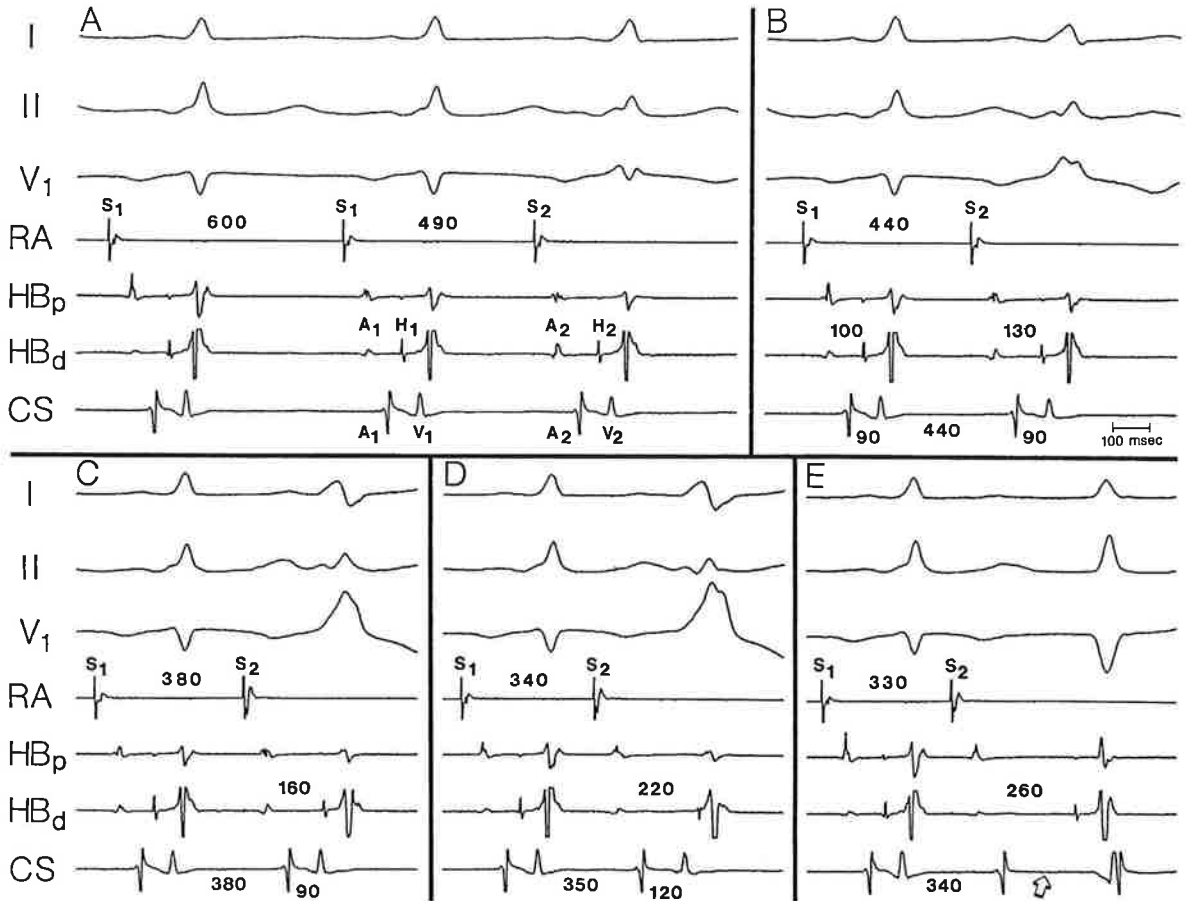
Figure 5. Characteristics of accessory pathway conduction in the antegrade direction (ventricular preexcitation) revealed by programmed atrial stimulation during electrophysiologic testing in a patient with a left free-wall accessory pathway. $A–E$, An atrial extrastimulus (premature atrial stimulus, S_2) at the end of a train of 8 atrial-paced complexes (pacing stimuli, S_1) at a cycle length of 600 msec. $A–C$, As the coupling interval for the extrastimulus ($S_1–S_2$ interval shown in RA electrogram) was shortened from 490 to 380 msec, the AV nodal conduction time ($A_2–H_2$ interval in the distal His bundle (HB_d) electrogram) increased to 160 msec, while the accessory pathway conduction time ($A_2–V_2$ interval in the coronary sinus (CS) electrogram) remained constant at 90 msec. This produced a progressive increase in the proportion of ventricular myocardium activated by the accessory pathway over that activated by the normal conduction system, reflected as an increase in the degree of ventricular preexcitation. D, As the atrial coupling interval ($A_1–A_2 = 350$ msec in CS electrogram) approached the refractory period of the accessory pathway, the accessory pathway conduction time ($A_2–V_2$ in CS electrogram) increased from 90 msec to 120 msec. E, At the effective refractory period of the accessory pathway ($A_1–A_2$ interval = 340 msec in CS electrogram), antegrade conduction over the accessory pathway is lost (unfilled arrow indicates absence of early activation in the CS electrogram), with an abrupt prolongation of the PR interval and normalization of the QRS complex.

time over the accessory pathway remains relatively constant at faster atrial pacing rates, the interval from the atrial pacing stimulus to the onset of the delta wave (stimulus to delta interval) remains constant. The fixed conduction time over the accessory pathway, coupled with the delay in activation of the His-Purkinje system, results in an increase in the amount of ventricular myocardium activated by the accessory pathway, i.e., an increase in the degree of preexcitation (Figs. 5 A to D). If conduction block occurs in the accessory pathway, there is a sudden loss of preexcitation manifested by prolongation of the P to R interval with normalization of the H to V interval and the QRS complex (Fig. 5E). In summary, the electrophysiologic findings of ventricular preexcitation during programmed atrial stimulation include (1) an abnormally short or negative H to V interval, (2) a relatively constant interval between the pacing stimulus and onset of the delta wave (stimulus to delta interval), (3) increasing preexcitation with increasing AV nodal delay, and (4) sudden normalization (prolongation) of the H to V interval and QRS complex with loss of accessory pathway conduction.

The capability of the accessory pathway to conduct atrial impulses to the ventricles determines the likelihood of a rapid ventricular response during atrial fibrillation. The antegrade conduction capability of the accessory pathway is measured during the electrophysiology study by (1) decremental atrial pacing (continuous atrial pacing with a progressive decrease in pacing cycle length, i.e., 10 msec decrements, after a steady state is reached at each pacing cycle length); (2) atrial extrastimulus testing; and (3) pacing-induced atrial fibrillation. Accessory pathway conduction is assessed during decremental atrial pacing by determining the shortest atrial pacing cycle length maintaining 1:1 antegrade accessory pathway conduction. The effective refractory period of the accessory pathway (the longest premature atrial coupling interval that fails to activate the ventricles via the accessory pathway) is measured during atrial extrastimulus testing. During pacing-induced atrial fibrillation, the shortest and mean coupling intervals (R to R intervals) between preexcited QRS complexes are measured.[68] Values of 250 msec or less for any of these variables identify an accessory pathway capable of generating rapid ventricular rates during atrial fibrillation. Occasionally patients may have values longer than 250 msec for some or all of these parameters and may still present with a rapid ventricular response during atrial fibrillation. In these patients, the difference in antegrade conduction capability of the accessory pathway during electrophysiologic study and during spontaneous atrial fibrillation may be related to differences in autonomic tone. The ventricular response via the accessory pathway during atrial fibrillation has been shown to increase significantly with isoproterenol administration in some patients.[44, 115, 125]

Infrequently, the accessory pathway does not conduct during sinus rhythm or during atrial pacing at slow rates but exhibits 1:1 conduction during rapid atrial pacing (Fig. 6).[25] The mechanism for this unusual behavior is unknown. It could be speculated that in a plexus of interconnected fibers, activation of one fiber might inhibit propagation along another fiber.[98] Failure to activate the first fiber at faster atrial pacing rates might release the inhibition on the second fiber, allowing propagation of the impulse to the ventricle over the second fiber (Fig. 7). Evidence that accessory pathways are frequently composed of an interconnected network of functioning fibers has been obtained by direct catheter recordings of accessory pathway activation that show multiple discrete accessory pathway activation potentials and, rarely, reentry within the network of fibers.[60, 64]

Retrograde conduction over the accessory pathway can be demonstrated at electrophysiologic study by programmed stimulation of the ventricles or by inducing orthodromic AV reentrant tachycardia. The hallmarks of retrograde accessory pathway conduction are (1) an eccentric retrograde atrial activation sequence and (2) retrograde conduction and atrial activation independent of His bundle activation. In the absence of an accessory pathway, retrograde (V to A) conduction can occur only via the His bundle and AV node, and the atrium is activated earliest in the anterior septum, close to the His bundle (except during retrograde conduction over the slow AV nodal pathway, which activates the atrium at the posterior septum, close to the coronary sinus).[56, 114] In the presence of a left free-wall or right free-wall accessory pathway, the earliest retrograde atrial activation is recorded at the left free-wall or right free-wall adjacent to the mitral or tricuspid anulus at the site of the accessory pathway (eccentric retrograde atrial

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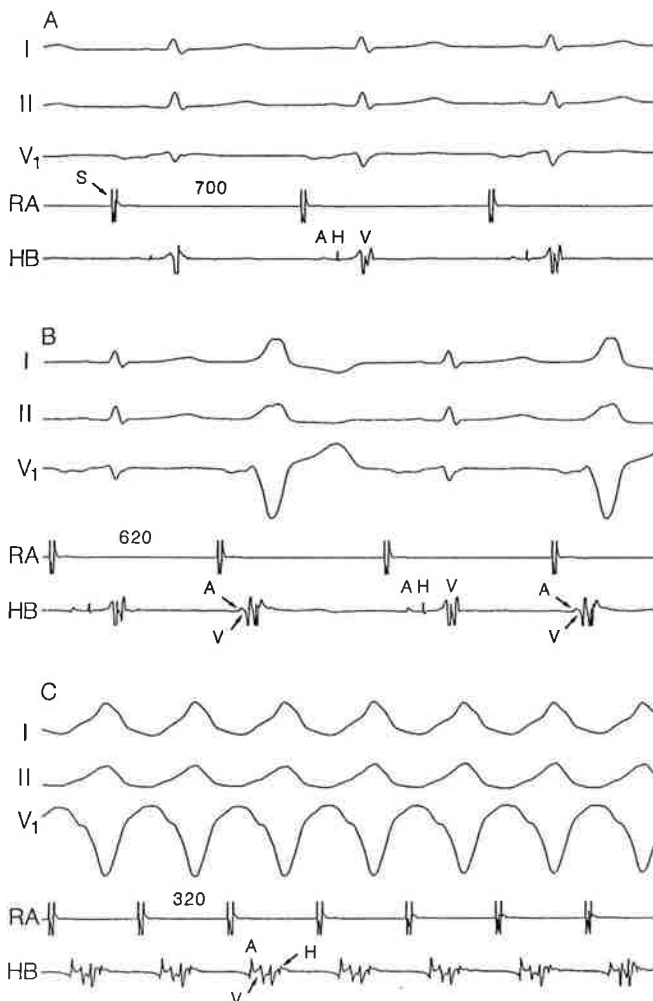
Figure 6. Paradoxic response to rapid atrial pacing in a patient with an anteroseptal accessory pathway. A, During right atrial pacing at a cycle length of 700 msec, antegrade accessory pathway conduction was absent. B, Shortening the atrial pacing cycle length to 620 msec resulted in 2:1 conduction over the accessory pathway. In preexcited complexes, ventricular activation is early in the His bundle (HB) electrogram, consistent with an anteroseptal accessory pathway. C, Further shortening of the atrial pacing cycle length to 320 msec resulted in 1:1 conduction over the accessory pathway. All QRS complexes were fully preexcited, with His bundle activation (H) occurring midway through the QRS complex. S = pacing stimulus artifact.

A Sinus Rhythm

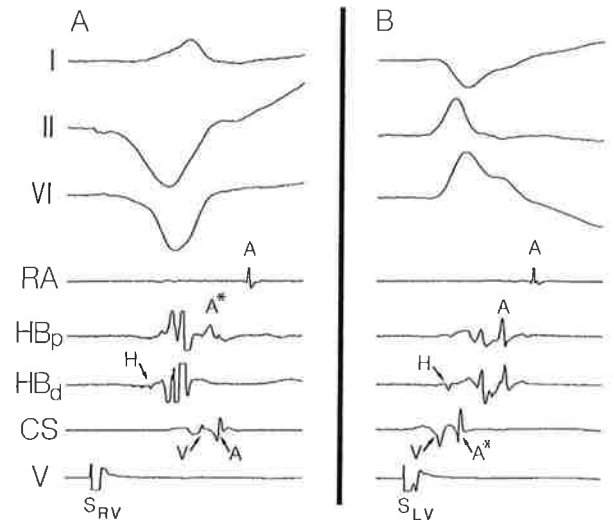B 1st Atrial Paced Complex

C Sustained Atrial Pacing
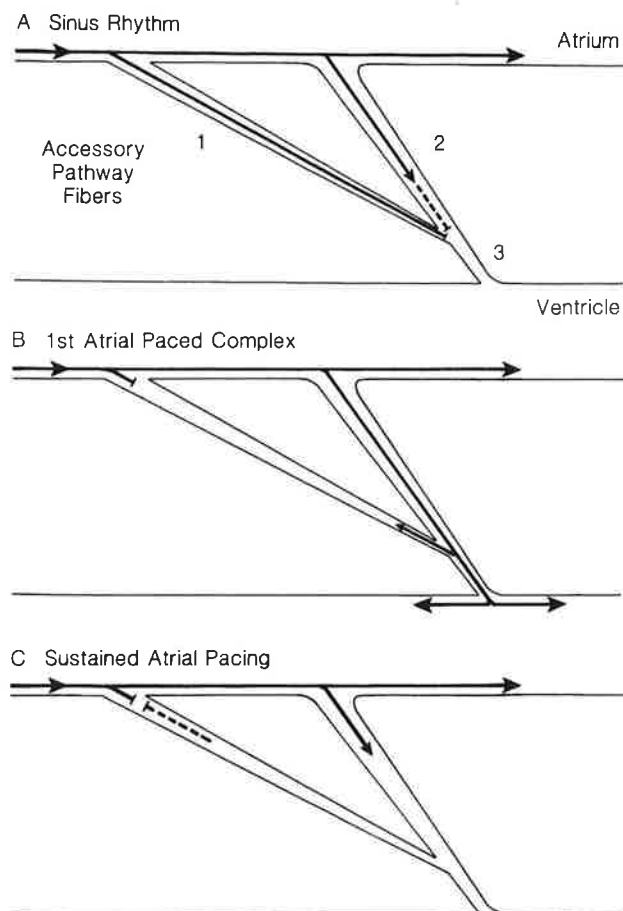
Figure 7. Subthreshold inhibition hypothesis for bradycardia-dependent accessory pathway conduction block. The figures schematically represent the AV sulcus with atrial myocardium at the top, two intersecting accessory pathway fibers in the middle, and ventricular myocardium at the bottom. *A*, The first complex of a train of atrial pacing at a rapid rate. The early atrial impulse is unable to excite pathway fiber 1, but successfully activates fiber 2. The activation wavefront of fiber 2 is sufficient to excite the common bundle (fiber 3), which also activates successfully the ventricular myocardium. The activation wavefront of fiber 2 retrogradely activates fiber 1. *B*, During sinus rhythm, atrial activation propagates along the AV sulcus, resulting in activation of accessory pathway fiber 1 before activation of fiber 2. The activation wavefront of fiber 1 is insufficient to activate the common bundle (fiber 3). The activation wavefront of fiber 2 arrives at the common bundle shortly thereafter, but is also unable to excite the common bundle because of subthreshold simulation from the nonpropagated impulse of fiber 1. *C*, Retrograde activation of fiber 1 prevents subsequent antegrade activation of that fiber during the period of atrial pacing, allowing uninhibited propagation along fiber 2 and 1:1 antegrade accessory pathway conduction. (*From* Kuck KH, Jackman WM, Friday KJ, et al: Sites of conduction block in accessory atrioventricular pathways: Basis for concealed accessory pathways. *In* Zipes DP, Jalife J (eds): Cardiac Electrophysiology: From Cell to Bedside, Philadelphia, WB Saunders, 1990, p 511.)

activation sequence). The earliest retrograde atrial activation recorded at the septum may reflect retrograde conduction over either a septal accessory pathway or the AV node.

During retrograde accessory pathway conduction, the timing of atrial activation is dependent on the timing of ventricular activation near the accessory pathway and is not related to retrograde His bundle activation. These relationships can be verified by ventricular pacing techniques that delay the timing of retrograde His bundle activation relative to the timing of ventricular activation close to the accessory pathway. One approach is to pace the ventricular myocardium close to the mitral or tricuspid anulus at the site of the accessory pathway (Fig. 8*A*). This results in retrograde activation of the accessory pathway before retrograde activation of the His bundle. The earliest atrial activation is recorded nearly simultaneous with activation of the His bundle, indicating that retrograde conduction must have occurred over an anomalous pathway. These relationships

are reversed when ventricular pacing is performed at the right ventricular apex or at other sites close to the peripheral inputs of the Purkinje system and far from the accessory pathway. During pacing at these sites, retrograde conduction frequently occurs earlier over the AV node than in the accessory pathway (Fig. 8*B*).

Selective retrograde accessory pathway conduction also can be demonstrated by delaying or eliminating retrograde AV nodal conduction. This can be accomplished (1) by initiating orthodromic AV reentrant tachycardia in which retrograde conduction occurs exclusively over the accessory pathway, (2) by ventricular pacing at faster rates or inducing closely coupled ventricular extrasystoles to delay retrograde AV nodal conduction, or (3) by the administration of pharmacologic agents (such as adenosine) during ventricular pacing that produce transient retrograde block in the AV node, unmasking retrograde accessory pathway conduction. Adenosine rarely produces retrograde block in accessory

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Figure 8. Effect of ventricular pacing site on the retrograde atrial activation sequence in a patient with a concealed left free-wall accessory pathway. *A,* During right ventricular pacing (S_{RV}), far from the accessory pathway, ventricular activation (V) near the left free-wall accessory pathway occurs late and earliest retrograde atrial activation (A*) occurs via the His bundle and AV node. Accessory pathway conduction is therefore not apparent. *B,* During left ventricular pacing (S_{LV}), ventricular activation (V) at the left free-wall accessory pathway occurs early, and earliest retrograde atrial activation (A*) clearly occurs via the accessory pathway. Therefore, pacing close to the accessory pathway may make accessory pathway conduction manifest.

pathways.[92, 101] Therefore, the occurrence of V to A block with adenosine during ventricular pacing does not absolutely exclude the presence of an accessory pathway.

Identification of Accessory Pathway Location

The mapping procedure used to localize an accessory pathway during an electrophysiologic study utilizes recordings of atrial and ventricular activation at multiple sites (at less than 5 mm intervals) around the mitral and tricuspid anuli. Recordings from the region of the mitral anulus are obtained by inserting a multi-electrode catheter into the coronary sinus and the great cardiac vein, which courses around the left AV groove (Fig. 9). This catheter is usually inserted into a subclavian or internal jugular vein. The tricuspid anulus can be mapped directly using a deflectable, multi-electrode catheter inserted into a femoral, subclavian, or internal jugular vein (Fig. 9). The approximate location of the accessory pathway is identified as the site recording the earliest ventricular potential during antegrade accessory pathway conduction (sinus rhythm or atrial pacing) and the site recording the earliest atrial potential during retrograde accessory pathway conduction (ventricular pacing or orthodromic AV reentrant tachycardia), as shown in Figures 10 and 11. These two sites often differ by 1 to 2 cm because either the accessory pathway crosses the AV groove obliquely[59] or different

fibers in the accessory pathway network are responsible for antegrade and retrograde conduction.[84] For finer localization (within 3 to 4 mm), activation potentials from the accessory pathway fibers can be recorded using closely spaced bipolar electrodes in approximately 90% of patients (Fig. 10).[59, 61]

Accessory Pathways Associated with Unidirectional Conduction

As described previously, the conduction properties of an accessory pathway may differ significantly in the antegrade and retrograde directions. The extreme situation, with rapid accessory pathway conduction in one direction and no conduction in the opposite direction, is not infrequent. In symptomatic patients undergoing electrophysiologic study at our institution, accessory pathways conducting only in the retrograde direction comprise 24% of all accessory pathways and account for 32% of left free-wall pathways, 13% of posteroseptal pathways, 11% of right free-wall pathways, and 23% of anteroseptal pathways. These patients present with orthodromic AV reentrant tachycardia identical to that seen in patients with bidirectionally conducting pathways. Because of the absence of antegrade accessory pathway conduction, the electrocardiogram is normal, without ventricular preexcitation. Consequently, these pathways are frequently referred to as "concealed" accessory pathways
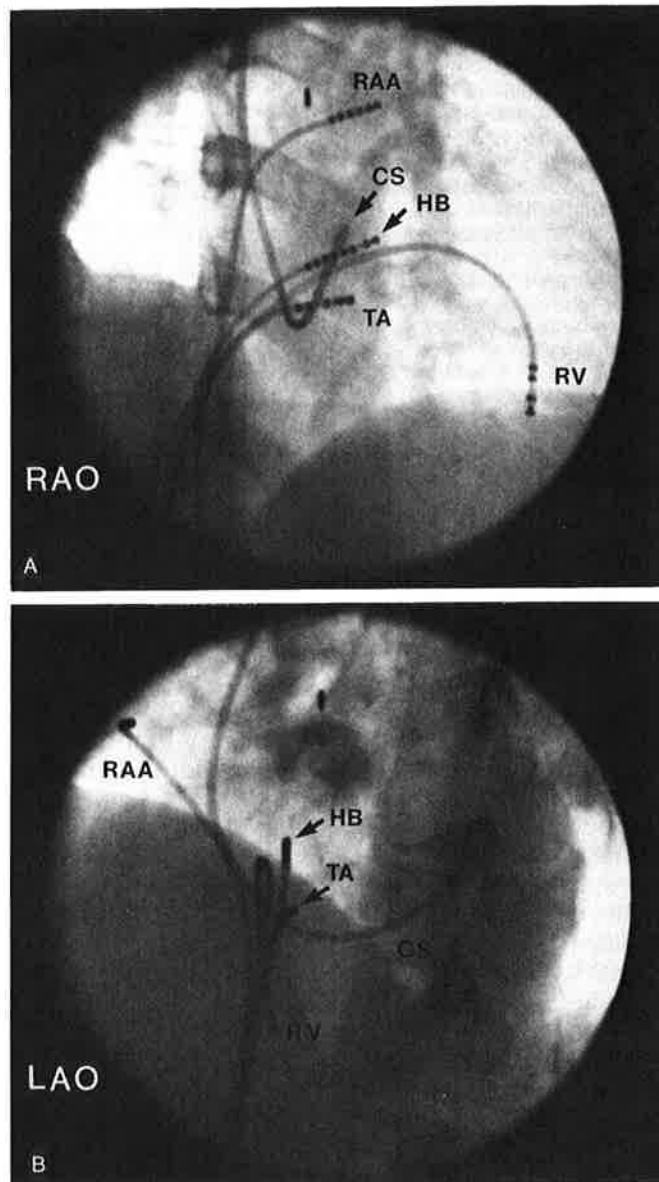
Figure 9. Catheters used to localize an accessory pathway. Radiographs show orthogonal views in the (A) 30-degree right anterior oblique projection and (B) 60-degree left anterior oblique projection. Left free-wall accessory pathways are localized using a catheter in the coronary sinus (CS) to record atrial and ventricular activity close to the mitral anulus as well as accessory pathway activation. Right-sided accessory pathways are localized using a catheter placed against the tricuspid anulus (TA). A catheter is positioned across the anteroseptal tricuspid anulus to record the His bundle activation (HB). In this patient, catheters are also positioned in the right atrial appendage (RAA) and right ventricular apex (RV) for programmed electrical stimulation.

Figure 10. Orthodromic AV reentrant tachycardia (left) in a patient with a left free-wall accessory pathway. The earliest retrograde atrial potential was recorded in the coronary sinus electrogram (CS). Note that a potential resulting from retrograde activation of the accessory pathway is recorded before the earliest retrograde atrial potential.
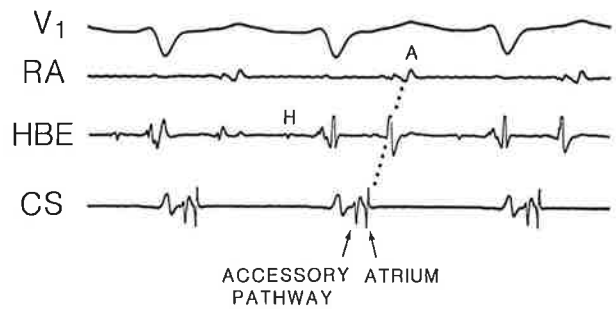
in that the presence of an accessory pathway is not evident or is concealed on the electrocardiogram.

In most (93%) concealed pathways, the sinus atrial impulse activates the accessory pathway, but the accessory pathway fails to activate the ventricle; i.e., the impulse blocks at the interface between the accessory pathway and the ventricle (Fig. 12).[73] In 7% of concealed accessory pathways, the atrial impulse is blocked at the atrial insertion of the accessory pathway.[73] Because the accessory pathway is not activated antegradely during sinus rhythm, the ventricular impulse regularly activates the accessory pathway in the retrograde direction. This results in incessant AV reentrant tachycardia in some patients.
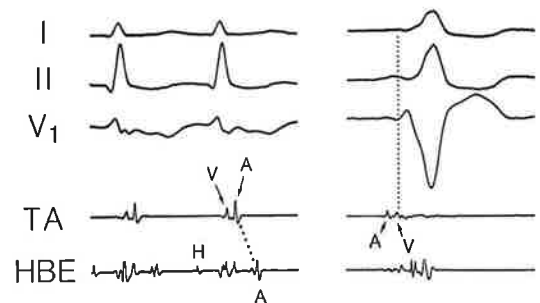
Accessory pathways that conduct only in the antegrade direction are less common, in our experience comprising 3% of accessory AV pathways and accounting for 2% of left free-wall pathways, 4% of posteroseptal pathways, 5% of right free-wall pathways, and 4% of anteroseptal accessory pathways. These patients usually present with atrial fibrillation with a rapid ventricular response over the accessory pathway but, occasionally, have antidromic AV reentrant tachycardia.

## Pathways with Decremental (Atrioventricular Node–Like) Conduction Properties

Mahaim Fibers (Right Atriofasicular Pathways)

In approximately 3% (12 of 439) of symptomatic patients, the accessory pathway exhibits unique characteristics. These fibers (1) conduct only in the antegrade direction; (2) have long conduction times that prolong further at faster atrial pacing rates and exhibit Wenckebach periodicity during second-degree block (decremental properties); and (3) are blocked by pharmacologic agents that block AV nodal conduction, such as adenosine and verapamil. These AV node–like properties led early investigators[42, 124] to believe these fibers were the physiologic correlate of the fiber described histologically by Mahaim,[80, 81] which originated in the AV node and inserted into the right ventricular septum, and were subsequently classified as nodoventricular fibers.[5] However, recent electrophysiologic evidence suggests that in most (if not all) of these patients, the fibers do not originate in the AV node.[45, 69, 94, 117]

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Figure 11. Orthodromic AV reentrant tachycardia (left) in a patient with a right anterolateral accessory pathway showing that earliest retrograde atrial activation was recorded from the anterolateral tricuspid anulus (TA). During sinus rhythm (right), earliest antegrade ventricular activation (V) was recorded from the same site along the tricuspid anulus and preceded the onset of the delta wave (dotted line).
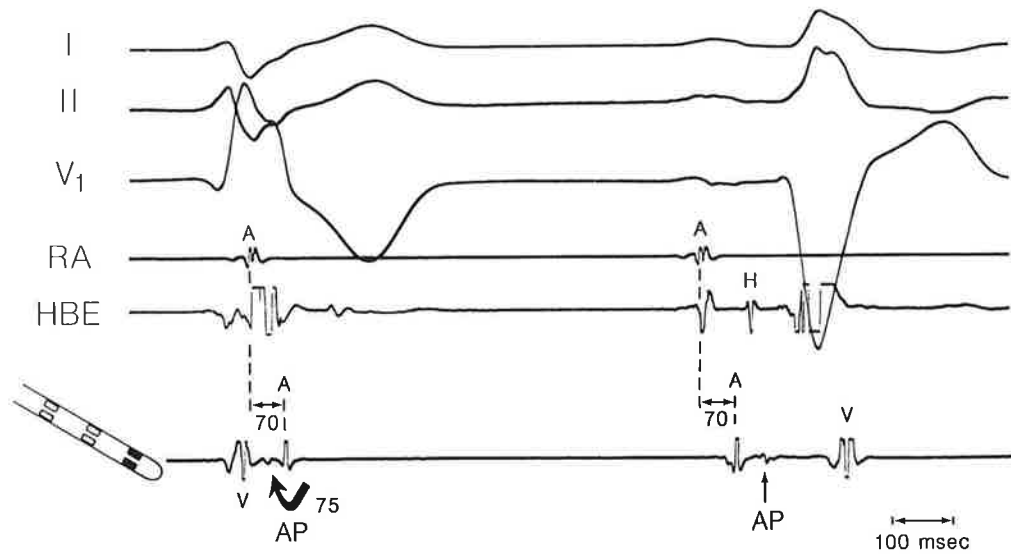
Figure 12. Catheter recording of activation of a concealed left lateral accessory AV pathway. Right complex: During sinus rhythm, the catheter electrode in the coronary sinus (*bottom tracing*) records atrial activation (A) followed by an accessory pathway activation potential (AP). The wide QRS complex results from left bundle branch block; not preexcitation. The absence of antegrade accessory pathway conduction is reflected by the long interval between the AP potential and the local ventricular potential (V). The presence of the AP potential indicates that the accessory pathway was activated and that the site of conduction block was located at or near the junction of the accessory pathway and left ventricle. Left complex: A spontaneous, late-diastolic ventricular extrasystole retrogradely activated the accessory pathway, advancing the timing of the AP potential by 75 msec (*curved arrow*). The local atrial potential (A) was recorded 70 msec after the right atrial potential (A in RA electrogram), just as in the right complex, and the morphology of the atrial potential was also the same, indicating that atrial activation was not affected by the ventricular extrasystole and that the AP potential does not represent a fragmented atrial potential. (*From* Kuck KH, Jackman WM, Friday KJ, et al: Sites of conduction block in accessory atrioventricular pathways: Basis for concealed accessory pathways. In Zipes DP, Jalife J (eds): Cardiac Electrophysiology: From Cell to Bedside. WB Saunders, 1990, p 511.)
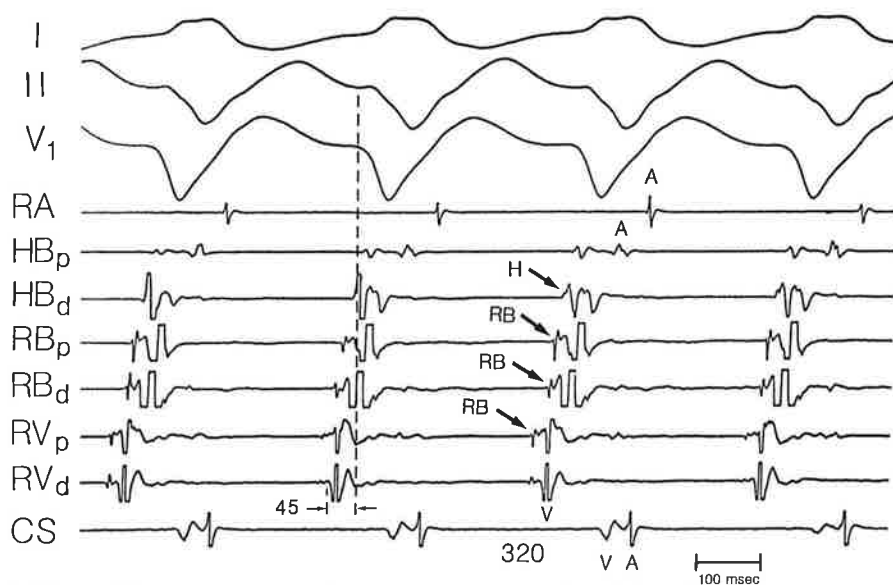
Figure 13. Antidromic AV reentrant tachycardia in a patient with a right atriofasicular ("Mahaim") pathway. Catheters were positioned in the right atrial appendage (RA), His bundle region (proximal [HB$_p$] and distal [HB$_d$] pairs of electrodes), middle right ventricular septum overlying the middle of the right bundle branch (RB$_p$ and RB$_d$), right ventricular apex close to the distal insertions of the right bundle branch (RV$_p$ and RV$_d$), and coronary sinus (CS). The accessory pathway inserts into the distal end of the right bundle branch along the right free-wall close to the right ventricular apex. Therefore, earliest ventricular activation (V) during antidromic AVRT was recorded from the right ventricular apex (RV electrograms) and was preceded by a potential resulting from activation of the right bundle branch (RB). Retrograde activation of the right bundle branch (RB potentials) was traced along the septum (RB electrograms), resulting in retrograde activation of the His bundle (H) and AV node, with earliest retrograde atrial activation recorded in the HB$_p$ electrogram. Note the long AV interval resulting from the long accessory pathway conduction time, typical of a right atriofasicular ("Mahaim") fiber.

The proximal end originates in the right atrium close to the lateral or anterolateral tricuspid anulus. The other end inserts into the right ventricular free-wall, close to the apex (rather than close to the anulus as is true in all other types of accessory pathways). Catheter recordings close to the ventricular insertion of these accessory pathways have shown activation potentials from the distal right bundle branch before ventricular activation, suggesting that the pathway may connect directly to the distal end of the right bundle branch (Fig. 13). This possible connection with the distal right bundle branch has led to the terminology *right atriofasicular pathway*.[41, 117] The embryology of these fibers is uncertain, but we speculate that they may represent a duplicated AV node and Purkinje system. While looking for the normal conduction system, Kent[65, 66] described a "node" of specialized cells at the right anterolateral tricuspid anulus. Although Kent is associated with describing the common form of AV accessory pathway, he may have actually described a right atriofasicular pathway.

Because right atriofasicular pathways con-

duct only in the antegrade direction, these patients present with antidromic AV reentrant tachycardia. Due to the long conduction time of these accessory pathways, the electrocardiogram during sinus rhythm usually does not exhibit ventricular preexcitation. Preexcitation is evident only when conduction in the AV node is delayed or blocked by atrial extrasystoles, atrial pacing, or pharmacologic agents (Fig. 14).

Permanent Form of Junctional Reciprocating Tachycardia

In approximately 2% (8 of 439) of symptomatic patients, the accessory pathway conducts only in the retrograde direction, has long conduction times, and exhibits decremental conduction properties during ventricular pacing. The conduction time changes with autonomic tone and is prolonged or blocked by pharmacologic agents such as adenosine, verapamil, or beta-adrenergic blocking agents. These pathways are most commonly located in the posterior septum but are at times located in other areas such
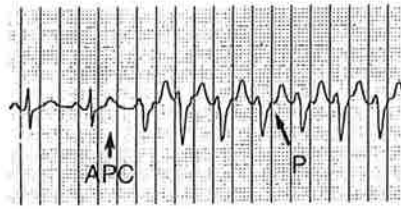
Figure 14. Holter monitor recording of spontaneous initiation of antidromic AVRT in a patient with a "Mahaim" pathway. The first two complexes were recorded during sinus rhythm and show no ventricular preexcitation. An atrial extrasystole (APC) is blocked in the AV node but is conducted by the "Mahaim" pathway. This results in a long PR interval with a fully preexcited QRS complex and the initiation of antidromic AVRT. Note the long PR interval during tachycardia, indicative of the long, antegrade-accessory pathway conduction time.

as the left anterolateral region. Histologic examination in one patient[31] revealed a posteroseptal accessory pathway composed of normal myocardium but following a long "sinuous, tortuous" path.

These patients frequently present in childhood with incessant orthodromic AV reentrant tachycardia. Because the retrograde conduction time over the accessory pathway is long, the P wave during tachycardia occurs long after the QRS complex ("long R to P tachycardia"). The long retrograde conduction time permits a relatively short antegrade AV nodal conduction time, resulting in a relatively short PR interval during tachycardia (Fig. 15). This pattern of incessant tachycardia with a long R to P interval was initially labeled the permanent form of junctional reciprocating tachycardia by Coumel et al.[29] Although less common, the permanent form of junctional reciprocating tachycardia can also result from the atypical form of AV nodal

reentrant tachycardia, which uses a slow AV nodal pathway for retrograde conduction.

The incessant nature of this form of AV reentrant tachycardia results from the ability of the accessory pathway to conduct retrogradely to the atrium during sinus rhythm without a preceding atrial extrasystole or AV nodal delay. This causes the tachycardia to reinitiate soon after it terminates (Fig. 15). The ability of the pathway to conduct retrogradely during sinus rhythm without preceding AV nodal delay (PR prolongation) suggests that the sinus atrial impulses do not penetrate the accessory pathway; i.e., the site of antegrade block is probably located at the atrial accessory–pathway junction.

ARRHYTHMIAS ASSOCIATED WITH ACCESSORY ATRIOVENTRICULAR PATHWAYS

Orthodromic Atrioventricular Reentrant Tachycardia

Orthodromic AV reentrant tachycardia is the most common tachyarrhythmia associated with an accessory AV pathway, occurring in approximately 70% of symptomatic patients.[90] As illustrated in Figure 16A, the reentrant impulse propagates from the atrium to the ventricle (antegrade direction) over the AV node and His-Purkinje system, and from the ventricle back to the atrium (retrograde direction) over the accessory pathway. The term *orthodromic* refers to activation of the normal conduction system (AV node and His-Purkinje system) in the normal (antegrade) direction. This tachycardia has also been referred to as "AV reentrant tachy-

Figure 15. Incessant orthodromic AVRT in a patient with a concealed posteroseptal accessory pathway which has a long conduction time and decremental conduction properties (permanent form of junctional reciprocating tachycardia). The tachycardia was terminated by a ventricular extrasystole. After two sinus complexes, the tachycardia started spontaneously without an extrasystole or significant PR prolongation (PR = 0.16 sec). During tachycardia, the P wave *(arrow)* in lead II is typically negative and occcurs long after the QRS complex (long R–P tachycardia), due to the long retrograde conduction time over the accessory pathway.
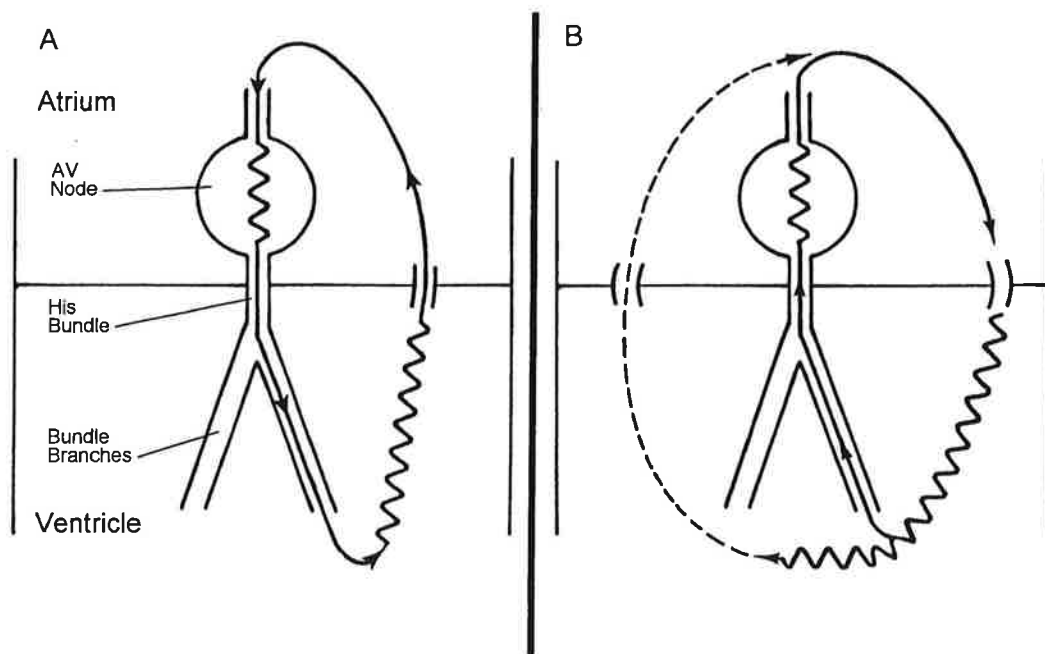
Figure 16. Schematic representation of orthodromic *(A)* and antidromic *(B)* AV reentrant tachycardia. During orthodromic tachycardia, the reentrant impulse propagates in the antegrade direction (from atrium to ventricle) over the AV node and His-Purkinje system, and in the retrograde direction (from ventricle to the atrium) over the accessory pathway. During antidromic tachycardia, the reentrant impulse propagates in the antegrade direction over the accessory pathway and in the retrograde direction over the normal conduction system (*solid line*) or a second accessory pathway (*dashed line*).

cardia using the accessory pathway for retrograde conduction," as "circus movement tachycardia,"[126] and as "orthodromic reciprocating tachycardia."[40]

Orthodromic AV reentrant tachycardia can be initiated by an atrial extrasystole or by a ventricular extrasystole. In a small number of patients, it may emerge from sinus rhythm without a preceding extrasystole. An atrial extrasystole can initiate orthodromic AV reentrant tachycardia by producing antegrade conduction block in the accessory pathway and sufficient delay over the normal conduction system to allow retrograde activation of the accessory pathway and atrium. A ventricular extrasystole produces orthodromic AV reentrant tachycardia when the ventricular impulse propagates retrogradely to the atrium over the accessory pathway while blocking in the normal conduction system. Retrograde block must occur either in the His-Purkinje system or very low in the AV node to allow the atrial impulse (originating from retrograde conduction over the accessory pathway) to be propagated through the AV node in the antegrade direction.[4] Orthodromic AV reentrant tachycardia can emerge from sinus rhythm without a

precipitating extrasystole in some patients with a concealed accessory pathway in which conduction block in the antegrade direction during sinus rhythm occurs at the atrial insertion of the accessory pathway. In some of these patients, the retrograde conduction time over the accessory pathway is long, permitting tachycardia to occur with almost every sinus impulse, resulting in incessant AV reentrant tachycardia or permanent junctional reciprocating tachycardia (see Fig. 15).

Orthodromic AV reentrant tachycardia results in a normal QRS complex or functional aberrant ventricular conduction but not a preexcited QRS complex, because antegrade conduction occurs exclusively over the AV node and His-Purkinje system. The retrograde atrial activation sequence is eccentric when the accessory pathway is located in the left free-wall (see Fig. 10), the right free-wall (see Fig. 11), or posteroseptal region. When the accessory pathway is located in the anteroseptal region, the retrograde atrial activation sequence is normal (similar to retrograde conduction over the AV node), with the earliest atrial activation occurring in the anterior septum near the His bundle (Fig. 17). Regardless of the retrograde atrial acti-
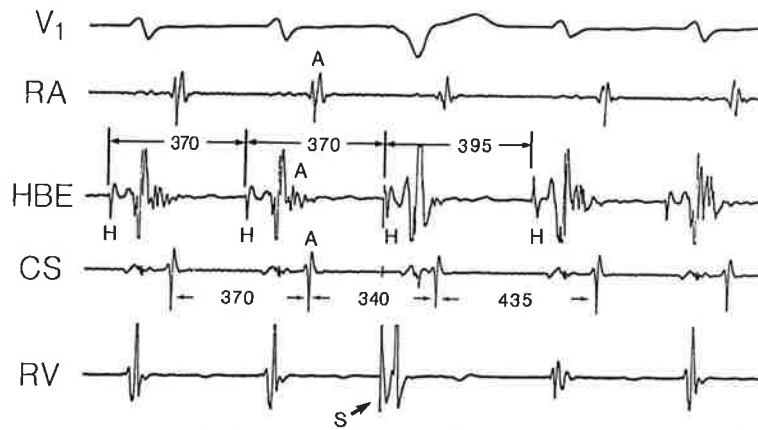
Figure 17. Orthodromic AVRT in a patient with a septal accessory AV pathway. The retrograde atrial activation sequence resulting from retrograde accessory pathway conduction is normal (similar to retrograde AV nodal conduction), with earliest atrial activation recorded in the His bundle electrogram (HBE). During tachycardia, a paced ventricular extrasystole is introduced (S-pacing stimulus) without affecting the timing or morphology of the antegrade His bundle potential. However, the timing of the next atrial impulse is advanced by 30 msec (A-A interval was shortened from 370 msec to 340 msec). Because the His bundle was refractory and unable to conduct an impulse to the atrium, retrograde conduction must have occurred over the accessory pathway (as seen in Figure 18). The subsequent H-H interval is prolonged (395 msec) because of the increased AV nodal delay resulting from the premature atrial activation.

vation sequence, the presence or absence of retrograde accessory pathway conduction during a supraventricular tachycardia can be determined at electrophysiologic study. This is accomplished by introducing a paced ventricular extrasystole at a time when the His bundle is refractory, i.e., after antegrade activation of the His bundle (Figs. 17 and 18).[28, 38, 89, 112, 118, 127, 130] When an accessory pathway is present, the premature ventricular impulse advances the timing of the next atrial impulse (see Fig. 17). In the absence of an accessory pathway, the ventricular extrasystole does not advance the next atrial impulse because the only route to the atrium (the His bundle) is refractory. This test is used to differentiate orthodromic AV reentrant tachycardia from AV nodal reentrant tachycardia.

Orthodromic AV reentrant tachycardia and AV nodal reentrant tachycardia can frequently be differentiated by examining the V to A interval. In orthodromic AV reentrant tachycardia, the reentrant impulse must reach the ventricles and then propagate to the base of the ventricles and conduct across the accessory pathway before reaching the atrium. Therefore, the V to A interval during tachycardia is 60 msec or longer, and the P wave is located at the end of the QRS complex or in the ST segment.[1, 19, 129] In AV nodal reentrant tachycardia, the ventricles do not form part of the reentrant circuit, and retrograde conduction begins before (or shortly

after the onset) of His bundle activation. The V to A interval is frequently (but not always) shorter than 60 msec, with the P wave superimposed on the QRS complex. Although a V to A interval greater than 60 msec may occur with either arrhythmia, a V to A interval of less than 60 msec excludes orthodromic AV reentrant tachycardia.
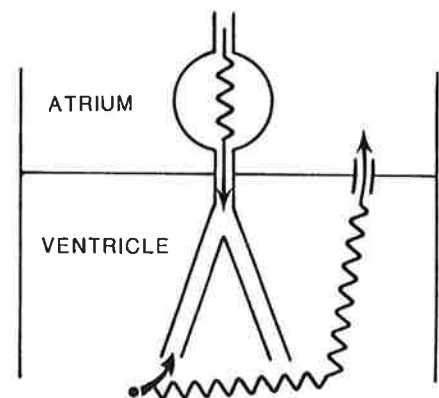
Figure 18. Schematic representation of the use of a paced ventricular extrastimulus during supraventricular tachycardia to identify the presence of retrograde accessory pathway conduction (see Figure 17). The ventricular extrasystole (dot) is introduced simultaneously with antegrade activation of the His bundle. If the timing of the next atrial impulse is advanced, this indicates that the ventricular extrasystole was conducted to the atrium. Because the His bundle was refractory, the ventricular extrasystole must have used an accessory AV pathway for conduction to the atrium.
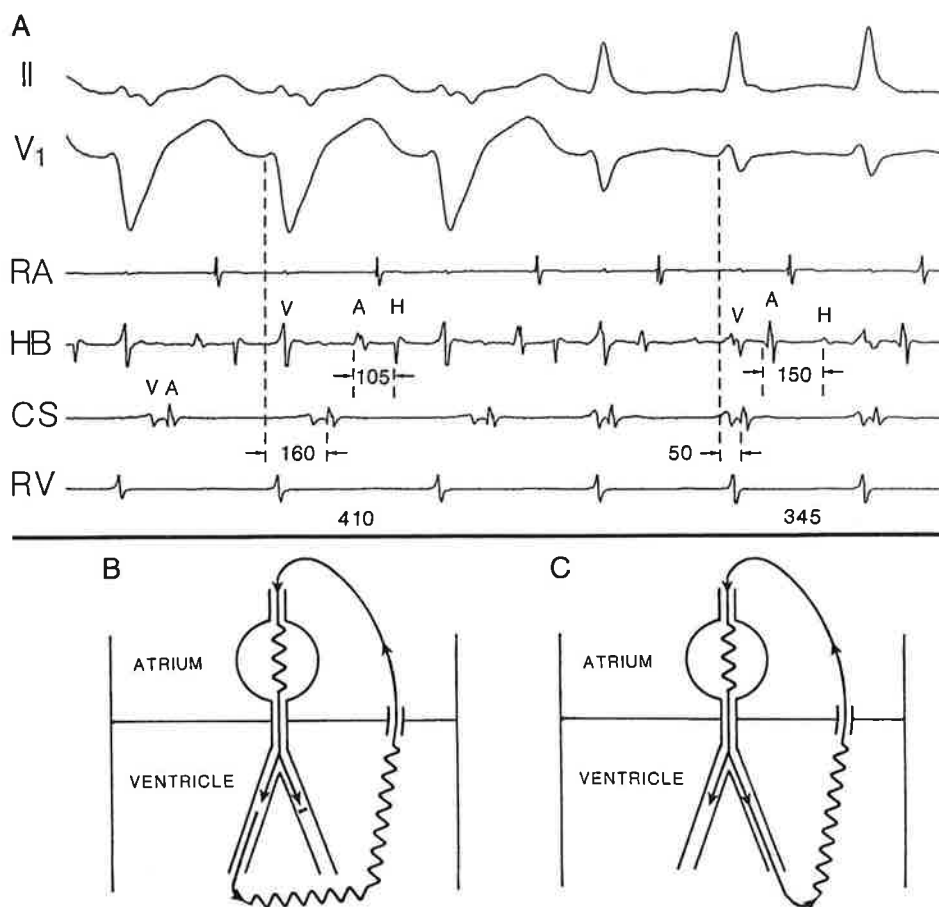
Figure 19. Effect of transient left bundle branch block on the cycle length of orthodromic AVRT using a left free-wall accessory AV pathway for retrograde conduction. *A,* Left bundle branch block (first 3 complexes) was associated with a long VA interval (160 msec). Resolution of left bundle branch block with normalization of the QRS complex (right) shortened the VA interval by 110 msec to 50 msec. The tachycardia cycle length was shortened by only 65 msec (410 to 345 msec) due to a 45-msec increase in the AV nodal conduction time (A-H interval) from 105 msec to 150 msec as a result of the faster rate. *B,* Schematic of the reentrant circuit in the presence of left bundle branch block. The ventricles are activated by the right bundle branch and the impulse must travel across the intraventricular septum and along the left free-wall to the accessory pathway. The time for intraventricular conduction from the distal right bundle branch to the site of the accessory pathway is long and results in an increase in the VA interval. *C,* When conduction over the left bundle branch is present, the intraventricular conduction time to the accessory pathway is shorter. Prolongation of the VA interval (≤35 msec) and tachycardia cycle length by left bundle branch block is diagnostic of AVRT using a left free-wall accessory pathway for retrograde conduction. Straight lines = rapid conduction; zigzag lines = slow conduction.

The effect of transient bundle branch block on the tachycardia cycle length, and more specifically on the V to A conduction time, can establish the participation of an accessory pathway in a supraventricular tachycardia. Because the ventricles are a part of the reentrant circuit in orthodromic AV reentrant tachycardia, bundle branch block on the same side as the accessory pathway lengthens the conduction time to the ventricular insertion of the accessory pathway. In left free-wall accessory pathways and right free-wall accessory pathways, the occurrence of left bundle branch block and right bundle branch block, respectively, during orthodromic AV reentrant tachycardia lengthens the V to A interval 35 to 120 msec (Fig. 19).[28, 57, 67, 95, 112] Contralateral bundle branch block (i.e., right bundle branch block in the presence of a left free-wall accessory pathway) does not effect the V to A interval. In patients with an anteroseptal accessory

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pathway, right bundle branch block lengthens the V to A interval during orthodromic AV reentrant tachycardia by 0 to 25 msec, and in patients with a posteroseptal accessory pathway, left bundle branch block during AV reentrant tachycardia lengthens the V to A interval by 0 to 25 msec.[67] Lengthening the V to A interval lengthens the tachycardia cycle length (slower rate), but the increase in tachycardia cycle length is frequently less than the increase in the V to A interval. This is due to shortening of the conduction time through the AV node as a result of the longer cycle length (Fig. 19). Importantly, an immediate lengthening of the tachycardia cycle length (V to A interval) with the occurrence of bundle branch block is pathognomonic for orthodromic AV reentrant tachycardia.

Antidromic Atrioventricular Reentrant Tachycardia

In antidromic AV reentrant tachycardia, the reentrant impulse travels in the opposite direction than during orthodromic AV reentrant tachycardia (Figs. 16 and 20). The reentrant impulse propagates in the antegrade direction over an accessory pathway and in the retrograde direction over the His-Purkinje system and AV node (see Fig. 16B). The term *antidromic* refers to activation of the normal conduction system in the abnormal (retrograde) direction. Unlike orthodromic AV reentrant tachycardia, antidromic AV reentrant tachycardia is uncommon, occurring in only 4% to 5% of patients with Wolff-Parkinson-White syndrome.[6, 9, 40] The low occurrence of this arrhythmia may be related to at least two factors. The first factor is that, in most patients, the AV node is unable to maintain 1:1 retrograde conduction at the rate of the tachycardia.[91] This helps explain the observation that a second accessory pathway forms the retrograde limb of the reentrant circuit in approximately 33% to 48% of patients with antidromic AV reentrant tachycardia (*dashed lines* in Fig. 16B).[6, 9, 18] The second factor relates to the requirements for initiation of the tachycardia. For an atrial extrasystole to initiate antidromic AV reentrant tachycardia, antegrade conduction must occur over the accessory pathway while antegrade conduction must be blocked in the normal conduction system. The block must
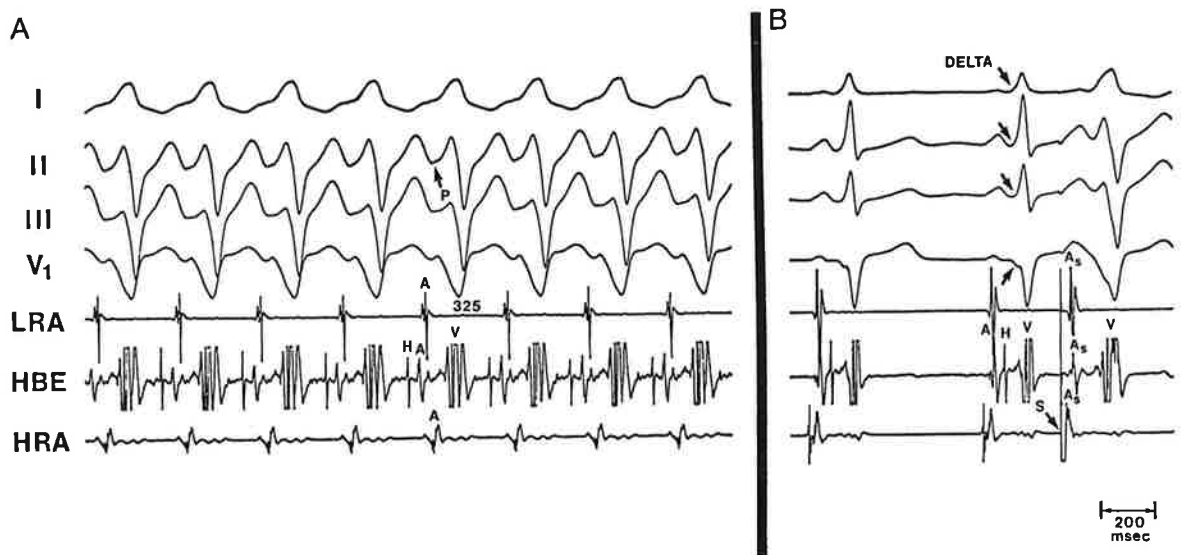
Figure 20. *A*, Antidromic AVRT using an anteroseptal accessory AV pathway for antegrade conduction and the His-Purkinje system and AV node for retrograde conduction. The P wave *(arrow)* immediately precedes and fuses with the QRS complex. The QRS complex is fully preexcited with initial forces identical to the delta wave during sinus rhythm shown in *B*. *B*, Pacing stimulus (S) delivered to the high right atrium (HRA) during sinus rhythm produces a premature atrial complex (A_s) that is conducted to the ventricles exclusively via the accessory pathway (fully preexcited QRS complex) due to conduction delay or block in the AV node (absence of His potential). The resulting QRS complex is identical to the QRS complex during tachycardia. LRA = lateral right atrium. (*From* Jackman WM, Friday KJ, Naccarelli GV: VT or not VT? An approach to the diagnosis and management of wide QRS complex tachycardia. Clin Prog Pacing Electrophysiol 1:239, 1983; with permission.)

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occur high in the AV node to allow the reentering ventricular impulse (resulting from antegrade conduction over the accessory pathway) to be conducted retrogradely through the AV node. Perhaps most atrial extrasystoles penetrate deep into the AV node, preventing early retrograde conduction through the AV node. For the tachycardia to be initiated by a ventricular extrasystole, the ventricular impulse must block retrogradely in the accessory pathway and be conducted retrogradely through the AV node. In most patients, the retrograde refractory period of the AV node is longer than that of the accessory pathway, causing retrograde block in the AV node more readily than in the accessory pathway. This limitation is not present in patients with two or more accessory pathways, but the occurrence of two accessory pathways (at least 3 cm apart) is present in only 5% of patients.
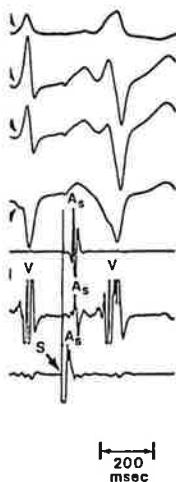
During antidromic AV reentrant tachycardia, a fully preexcited QRS complex emerges out of the P wave (see Fig. 20). However, in patients with atriofascicular (Mahaim) pathways, there is a long interval from the P wave to the onset of the QRS complex due to the long antegrade conduction time over the accessory pathway (see Figs. 13 and 14). The initial forces in the QRS complex during antidromic AV reentrant tachycardia are usually identical to the initial forces of the delta wave during sinus rhythm (see Fig. 20). The exception is in patients with multiple accessory pathways in which the delta wave during sinus rhythm is generated predominantly by the accessory pathway used for retrograde conduction during antidromic AV reentrant tachycardia. When the tachycardia utilizes the His-Purkinje system and AV node for retrograde conduction, the earliest atrial activation is recorded in the anteroseptal region, close to the His bundle, and, frequently, a retrograde His bundle potential can be recorded preceding atrial activation. In patients in whom a second accessory pathway is used for retrograde conduction, the retrograde atrial activation sequence will be eccentric, with earliest atrial activation being recorded at the site of the retrogradely conducting accessory pathway.

From the electrocardiogram, antidromic AV reentrant tachycardia may be difficult to distinguish from other tachycardias with preexcited QRS complexes in which the accessory pathway is not a component of the reentrant circuit, such as AV nodal reentrant

tachycardia, ectopic atrial tachycardia, or atrial flutter. The accessory pathway conducts atrial impulses during the tachycardia producing preexcited QRS complexes, but the presence or absence of accessory pathway conduction has no effect on maintenance of the tachycardia. Antidromic AV reentrant tachycardia requires 1:1 conduction in both the antegrade and retrograde directions (see Fig. 16*B*). Transient antegrade block in the accessory pathway with perpetuation of the arrhythmia eliminates AV reentrant tachycardia from the differential diagnosis. A 2:1 AV ratio also eliminates AV reentrant tachycardia and suggests atrial flutter with 2:1 conduction over the accessory pathway.

Atrial Fibrillation

Atrial fibrillation is the second most common tachyarrhythmia in Wolff-Parkinson-White syndrome, occurring in 10% to 38% of patients.[23, 90, 109] Spontaneous sustained atrial fibrillation is most common in patients with antegrade accessory pathway conduction (overt preexcitation) and is uncommon in patients with concealed accessory pathways.[26, 34] Recent studies[34, 109] suggest that patients with accessory pathways having a short antegrade refractory period (and therefore a faster ventricular response) are more prone to the occurrence of atrial fibrillation. The high incidence of atrial fibrillation in patients with Wolff-Parkinson-White syndrome is not fully understood. Most patients with atrial fibrillation have no detectable structural heart disease and no evidence of electrophysiologic abnormalities in the atria.[23, 26, 109] At least two mechanisms have been proposed to explain the relationship between the presence of an accessory pathway and atrial fibrillation. First, atrial fibrillation often develops during orthodromic AV reentrant tachycardia.[23, 113] The rapid atrial rate and the eccentric pattern of atrial activation during AV reentrant tachycardia may lead to disorganization of the atrial wavefront and the initiation of atrial fibrillation. AV reentrant tachycardia cannot explain the occurrence of atrial fibrillation in all patients. Patients with accessory pathways that conduct only in the antegrade direction have an increased incidence of atrial fibrillation despite the absence of AV reentrant tachycardia, and patients with concealed accessory pathways have a low inci-

dence of atrial fibrillation despite the presence of AV reentrant tachycardia.[26, 34]

A second proposed mechanism invokes the complex anatomic structure of the accessory pathway as a factor in atrial fibrillation. As described previously, it appears that many accessory pathways are composed of multiple interconnecting fibers.[12, 13, 59, 60, 61, 64] Atrial impulses may enter these networks of fibers and, after some delay (due to circuitous propagation through the fibers), return to the atrium to help perpetuate atrial fibrillation. Localized reentry within the network of fibers (simulating atrial fibrillation and atrial flutter) has been demonstrated in a small number of patients using direct recordings of accessory pathway activation.[64] In support of both hypotheses, surgical ablation of the accessory pathway prevents the recurrence of atrial fibrillation in the majority of patients.[26, 109]
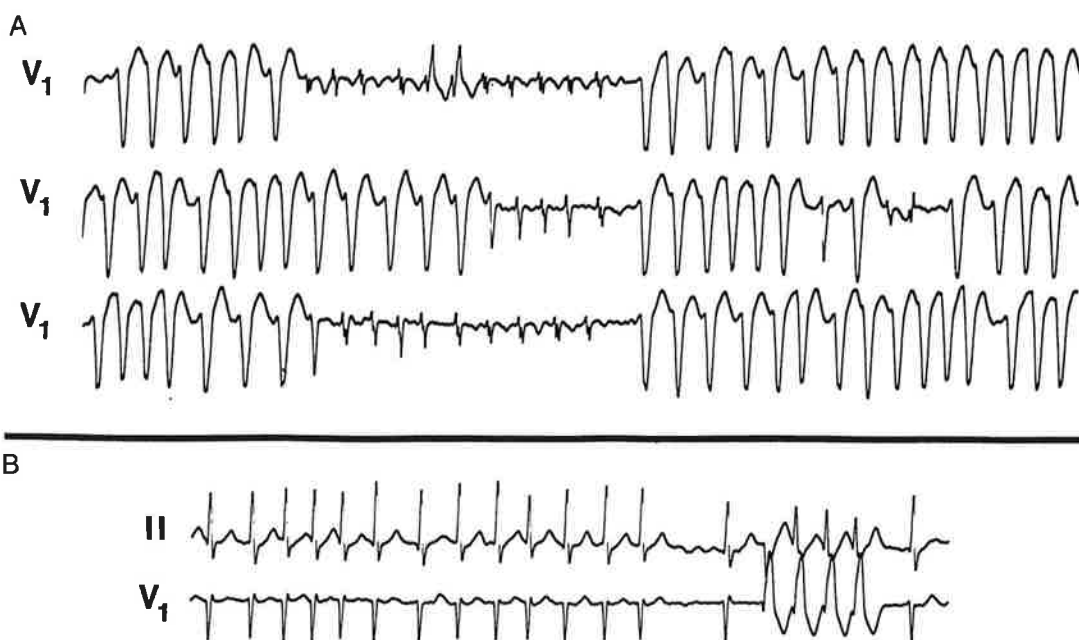
The ventricular response during atrial fibrillation is dependent upon the conduction properties of the accessory pathway and the AV node. When the refractory period of the accessory pathway in the antegrade direction is short, impulses are conducted to the ventricles over the accessory pathway at a rapid rate (Fig. 21). The QRS complexes are fully preexcited, and the ventricular cycle length is irregularly irregular. All of the QRS complexes have a similar morphology in comparison with atrial fibrillation with aberrant ventricular conduction in which QRS complexes

after long cycles often normalize to some degree.

When the antegrade refractory period of the accessory pathway is longer, atrial fibrillation is frequently associated with both preexcited QRS complexes and normally conducted QRS complexes. The electrocardiogram reveals groups of preexcited complexes separated from groups of normally conducted QRS complexes by relatively long intervals (Fig. 22, top panel).[15, 27, 71, 100, 118, 128] The clumping of preexcited complexes and normally conducted complexes has been attributed to concealed retrograde penetration into the AV node or accessory pathway, respectively. During a group of complexes conducted over the accessory pathway, the ventricular impulses penetrate the AV node in the retrograde direction, preventing antegrade conduction over the normal conduction system (Fig. 23). Sudden antegrade block in the accessory pathway eliminates the concealed retrograde penetration into the AV node. This permits the AV node to recover excitability, allowing antegrade conduction to proceed over the normal conduction system. These normally conducted complexes penetrate the accessory pathway in the retrograde direction, preventing any preexcited complexes. After a pause produced by block in the AV node, concealed retrograde penetration into the accessory pathway is eliminated. This permits the accessory pathway to recover excitability, and preexcited QRS
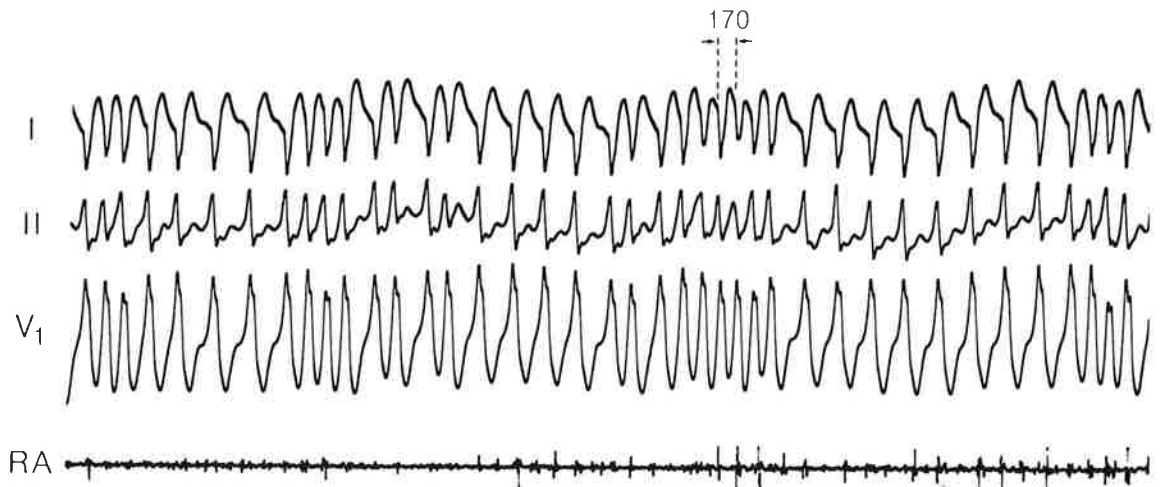
Figure 21. Atrial fibrillation induced at electrophysiologic study in a patient with a left lateral accessory pathway. All QRS complexes resulted from antegrade conduction over the accessory pathway with fully preexcited QRS complexes. The ventricular rate was rapid with a shortest R-R interval of only 170 msec. Despite the beat-to-beat variation in cycle length, all QRS complexes have a similar morphology. The right atrial electrogram (RA) shows the continuous, irregular potentials indicative of atrial fibrillation.
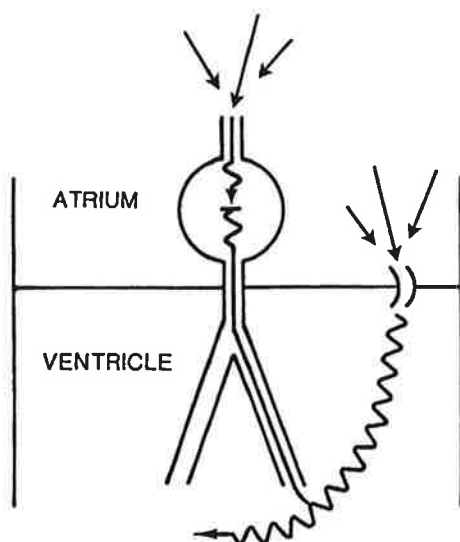
A

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Figure 22. Comparison of atrial fibrillation with intermittent ventricular preexcitation and intermittent aberrant ventricular conduction. *A,* During atrial fibrillation with preexcitation, groups of preexcited QRS complexes are separated by long cycle lengths from groups of normally conducted QRS complexes due to concealed retrograde penetration of the AV node (see Figure 23). *B,* During atrial fibrillation with intermittent aberrant ventricular conduction in a patient without an accessory pathway, a group of aberrant QRS complexes begin after an abrupt shortening of the cycle length (short cycle following a long cycle).

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complexes emerge. Therefore, the first preexcited complex occurs after a relatively long interval (see Fig. 22, *top panel*). This is easily distinguished from atrial fibrillation with in-

Figure 23. Schematic representation of atrial fibrillation resulting in perpetuation of preexcited QRS complexes due to retrograde penetration of the AV node, inhibiting antegrade conduction over the normal conduction system.

termittent aberrant ventricular conduction, in which the first wide QRS complex occurs after a short interval (see Fig. 22, *bottom panel*).

The morphology of the preexcited QRS complexes during atrial fibrillation should be examined carefully. Two or more distinct preexcited morphologies suggest the presence of multiple accessory pathways. This may be the only clue that a second accessory pathway is present.
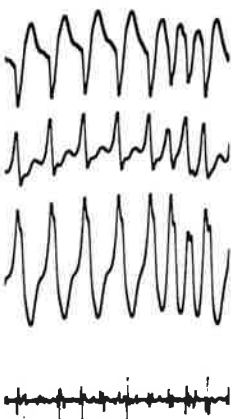
Atrial fibrillation with a rapid ventricular response via the accessory pathway may degenerate to ventricular fibrillation (Fig. 24), resulting in sudden death. The actual incidence of sudden death in patients with preexcitation is unknown but has been estimated to be 1 per 1000 patient years of follow-up. Factors that have been associated with the occurrence of ventricular fibrillation or sudden death include (1) a clinical history of both atrial fibrillation and AV reentrant tachycardia; (2) a rapid ventricular response via the accessory pathway during atrial fibrillation induced at electrophysiologic study, defined as a shortest R to R interval between preexcited QRS complexes of 250 msec or less; (3) multiple accessory pathways; and (4) the use of digitalis preparations.[68, 86]
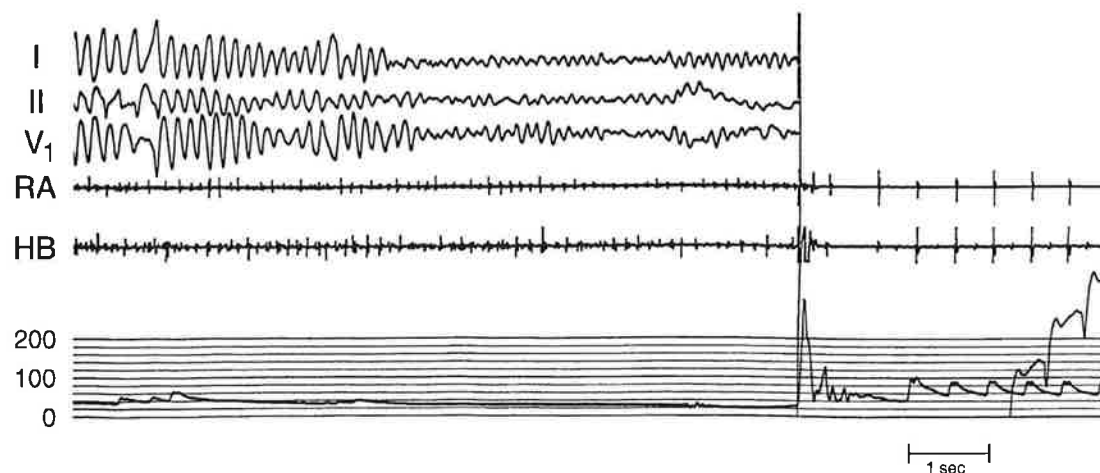
Figure 24. Degeneration of atrial fibrillation to ventricular fibrillation during an electrophysiologic study. *Left,* Atrial fibrillation with a rapid ventricular response due to conduction over a posteroseptal accessory pathway. All QRS complexes were fully preexcited. Atrial fibrillation produced marked hypotension (femoral arterial pressure is shown at the bottom), followed by the spontaneous occurrence of ventricular fibrillation. A direct current (DC) shock of 200 Joules terminated ventricular fibrillation and atrial fibrillation, restoring sinus rhythm and an adequate arterial pressure.

Conversely, the characteristics of a group at low risk for ventricular fibrillation include (1) intermittent ventricular preexcitation during sinus rhythm; (2) a shortest preexcited R to R interval greater than 280 msec during atrial fibrillation induced at electrophysiologic study; (3) no inducible AV reentrant tachycardia; and (4) a ventricular refractory period of 190 msec or less.[86]

Sudden death may be the first clinical manifestation of the Wolff-Parkinson-White syndrome. In two reports, preexcitation and arrhythmias were previously undiagnosed in 12% to 26% of patients resuscitated from ventricular fibrillation.[68, 86] Most of the patients in this group had structurally normal hearts and would otherwise have had a normal life expectancy.

The acute intravenous administration of verapamil during atrial fibrillation has resulted in acceleration of the ventricular response and ventricular fibrillation in a significant number of patients.[48, 49, 93, 100] The vasodilatation produced by verapamil results in hypotension, which is followed by an increase in sympathetic tone. This may shorten the refractory period of the accessory pathway, resulting in an increased ventricular response. Verapamil may also increase the ventricular response via the accessory pathway by producing antegrade block in the AV node, eliminating concealed retrograde penetration of the accessory pathway

by normally conducted complexes. The combination of a rapid irregular rate, an abnormal ventricular activation sequence (activation from one site in the base of the ventricles instead of the symmetric, nearly simultaneous activation produced by the His-Purkinje system), hypotension (poor myocardial perfusion), and increased sympathetic activity may result in a disorganized ventricular wavefront and ventricular fibrillation.

Digitalis preparations also have been associated with the degeneration of atrial fibrillation to ventricular fibrillation and sudden death in patients with preexcitation.[68, 108] In one report,[68] ventricular fibrillation developed in six patients during the course of atrial fibrillation shortly after the administration of intravenous digoxin. Most of these patients had had previous episodes of atrial fibrillation that had not degenerated into ventricular fibrillation in the absence of digoxin. Unlike the effects associated with verapamil, ventricular fibrillation can occur with an oral dose as well as an intravenous dose of digoxin. Although class I antiarrhythmic agents, such as procainamide, may reduce the ventricular response and convert atrial fibrillation to sinus rhythm, the administration of lidocaine has been associated with the degeneration of atrial fibrillation to ventricular fibrillation.[2] The mechanism for this response is unknown.

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TREATMENT OF THE PREEXCITATION SYNDROMES

Therapy for preexcitation syndromes can be directed at cure by eliminating the accessory pathway using ablative techniques or palliation of arrhythmias by pharmacologic means.

Ablative Therapy

Ablative therapy refers to the interruption of the accessory AV connection, which eliminates AV reentrant tachycardia and prevents a rapid ventricular response via the accessory pathway during atrial fibrillation. Ablation of the accessory pathway may decrease the incidence of atrial fibrillation.[26, 109] In the first ablative procedure a surgical approach was used whereby the heart was exposed and the accessory pathway localized and transected.[106] Two surgical techniques evolved, an endocardial approach[30, 55, 105] and an epicardial approach.[47, 82] Both approaches are highly successful but expose the patient to the inherent risks of operation.

As an alternative to surgical treatment, catheter ablation techniques using high-energy direct current (defibrillator) shocks, initially devised for ablation of the AV junction,[43, 103] were applied to ablation of accessory pathways.[10, 58, 87, 120, 123] Although a high rate of clinical success (67% to 97%) was achieved,[8, 88, 121, 122] barotrauma from high-energy shocks caused infrequent but serious complications, including cardiac perforation and death. These limitations led to the evaluation of radiofrequency current as an alternative energy source.[52, 53, 62, 75, 76] Radiofrequency current causes necrosis by heating the tissue without barotrauma or effects to other regions of the heart.

Before ablation, the accessory pathway is localized by mapping the mitral and tricuspid anuli as described previously. The ablation catheter is positioned against the mitral or tricuspid anulus at the site of the accessory pathway such that the ablation electrode records activation potentials from the accessory pathway (Fig. 25*A*). Radiofrequency energy is applied at 30 to 40 W to the ablation electrode for 20 to 60 sec (Fig. 25*B*).

The results of radiofrequency catheter ablation have been excellent. The technique eliminates accessory pathway conduction in more than 90% of patients at experienced centers.[21, 63, 74] Accessory pathway conduction returns in 3% to 9% of patients,[21, 63, 74] and most of these patients undergo a successful second ablation procedure. In four large series,[21, 63, 74, 77] with a combined experience of 596 patients, there was no mortality associated with the procedure. Complications occurred in 21 (3.5%) of the patients, including complete AV nodal block in four patients,
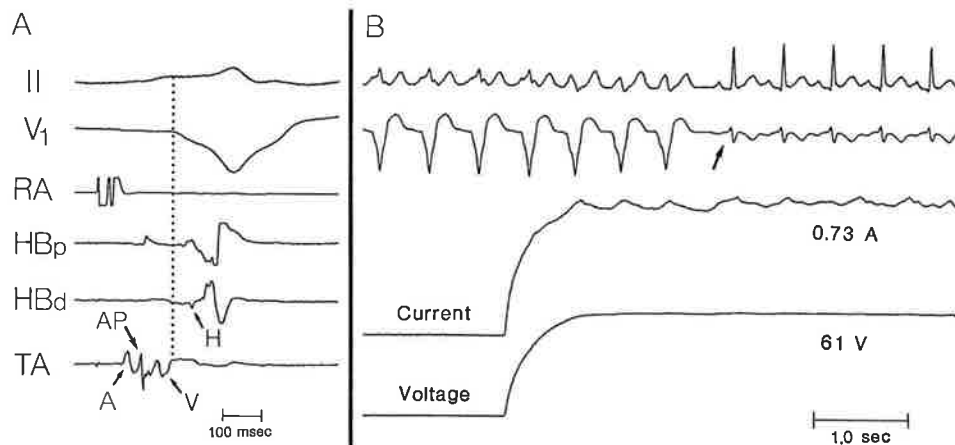
Figure 25. Radiofrequency catheter ablation of a right free-wall accessory pathway. *A,* Recorded during right atrial pacing to produce maximal preexcitation. The ablation catheter was maneuvered along the tricuspid anulus (TA electrogram) until the ablation electrode recorded accessory pathway activation (AP potential) indicating close proximity to the accessory pathway. Right ventricular activation (V) began 20 msec before the onset of the delta wave (*dotted vertical line*). *B,* Recorded as radiofrequency current was applied to the ablation electrode catheter at 61 V and 0.73 A (calculated power = 45 W). Accessory pathway conduction ceased 2.3 sec after the onset of the application of radiofrequency current (*arrow*), reflected by lengthening of the PR interval and normalization of the QRS complex.
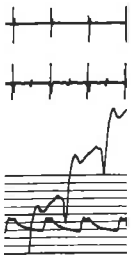
myocardial infarction in one patient, and cardiac perforation and tamponade in one patient. These results suggest that radiofrequency catheter ablation should be considered as primary therapy for symptomatic patients with AV reentrant tachycardia or atrial fibrillation with a rapid ventricular response via the accessory pathway. Recent analysis has suggested that radiofrequency catheter ablation is cost effective when compared with operation or chronic drug therapy.[32] Surgical ablation is currently reserved for the few patients in whom catheter ablation is unsuccessful.

Acute Management

Acute management of arrhythmias associated with preexcitation depends on the type of arrhythmia and the hemodynamic stability of the patient. Any arrhythmia associated with hemodynamic instability should be terminated immediately with direct current cardioversion under heavy sedation or general anesthesia. If the patient is hemodynamically stable, therapy is guided based on the diagnosis of arrhythmia from the electrocardiogram.

In patients who present with orthodromic AV reentrant tachycardia, interventions causing transient block in the AV node terminate the tachycardia. This can be accomplished by increasing vagal tone by carotid sinus massage or the Valsalva maneuver, or by intravenous administration of adenosine, calcium channel blockers (verapamil and diltiazem), or beta-adrenergic blockers (propranolol, metoprolol, and esmolol).

Adenosine, an endogenous nucleoside, is the drug of choice due to its high efficacy (greater than 95%) and extremely short half-life (less than 1.5 sec).[16, 22, 35, 79] The principal advantage of adenosine over other drugs, such as verapamil, is the very short period of AV nodal block (2 to 3 sec) and the minimal hemodynamic effects it produces. Therefore, if adenosine fails to terminate a tachyarrhythmia, the agent does not result in significant hemodynamic compromise. In contrast, if verapamil fails to terminate or slow a tachyarrhythmia, the vasodilatation and negative inotropic effects may result in acute hemodynamic decompensation.

Adenosine is effective only if given as a rapid intravenous bolus. The recommended starting dose is 6 mg, but if this is unsuccessful, doses of up to 18 mg can be used. The principal side effects are flushing and dyspnea, which last only a few seconds; transient arrhythmias are common (especially sinus bradycardia and nonsustained atrial fibrillation). Adenosine is ineffective in patients receiving phosphodiesterase inhibitors (i.e., theophylline) and is contraindicated in patients receiving dipyridamole, an agent that prolongs adenosine metabolism.[36]

Adenosine usually does not affect conduction in the accessory pathway; however, in some patients, accessory pathway conduction block can occur.[92, 101] Accessory pathways with decremental properties or long refractory periods are more likely to be affected.

In patients with atrial flutter or fibrillation with preexcited QRS complexes, the use of AV nodal blocking agents is contraindicated. The administration of intravenous verapamil and either intravenous or oral digitalis preparations may accelerate the ventricular response over the accessory pathway and produce ventricular fibrillation.[48, 49, 68, 93, 100, 108] Electrical cardioversion is usually the preferred option in this setting. If the tachycardia is hemodynamically stable, intravenous procainamide can be used to terminate the arrhythmia or slow the ventricular rate by depressing conduction through the accessory pathway. Lidocaine should be avoided in the treatment of atrial fibrillation with preexcited QRS complexes because it is usually ineffective[2, 11] and may accelerate the ventricular response and produce ventricular fibrillation.[2]

In patients with antidromic AV reentrant tachycardia, adenosine may be effective if the AV node is the retrograde limb of the reentrant circuit. However, if a second accessory pathway is being used for the retrograde limb, adenosine is not effective. In addition, antidromic AV reentrant tachycardia may be difficult to distinguish from atrial flutter with 2:1 conduction and other preexcited tachyarrhythmias. For these reasons, cardioversion should be considered in all patients with preexcited tachycardias.

Chronic Pharmacologic Therapy

Chronic antiarrhythmic drug therapy, once the only therapeutic option, is now considered primarily in patients who are not candidates for radiofrequency catheter ablation. Orthodromic AV reentrant tachycardia may be suppressed by drugs that either depress

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conduction through the AV node, such as beta-adrenergic blockers or calcium channel blockers, or drugs that depress conduction in the accessory pathway, such as quinidine, disopyramide, procainamide, flecainide, propafenone, amiodarone, and sotalol. Drugs that depress conduction in the accessory pathway are generally more effective (especially flecainide, propafenone, and amiodarone) and have the added advantage of reducing the incidence of atrial fibrillation and reducing the ventricular response if atrial fibrillation does occur. Catecholamines can decrease or counteract the antiarrhythmic effects of these agents,[3, 50, 83] suggesting a role for the addition of beta-adrenergic blocking agents. Even optimal antiarrhythmic therapy rarely prevents all episodes of AV reentrant tachycardia and may be associated with significant side effects.

Agents that depress accessory pathway conduction can produce severe ventricular arrhythmias in patients with structural heart disease, especially in those with depressed ventricular function or preexisting ventricular arrhythmias.[37] Antiarrhythmic agents can also exacerbate AV reentrant tachycardia. In patients in whom accessory pathways are marginally able to maintain conduction at the tachycardia rate, most episodes of tachycardia are aborted due to spontaneous retrograde block of the reentrant impulse in the accessory pathway. By using antiarrhythmic agents that prolong the AV nodal conduction time, the accessory pathway has more time to recover excitability, making the tachycardia more likely to be sustained or become incessant, albeit at a slow rate. Conversely, drugs that depress conduction in the accessory pathway may exacerbate AV reentrant tachycardia in patients in whom AV nodal conduction is marginal at the drug-free tachycardia cycle length.

Digoxin is contraindicated in patients who have preexcitation because of the risk of atrial fibrillation degenerating into ventricular fibrillation.[68, 108] Because ventricular preexcitation may not be apparent during sinus rhythm (see Fig. 4), digoxin should be used with caution in patients with paroxysmal supraventricular tachycardia.

The Asymptomatic Patient with Preexcitation

The principal concern regarding asymptomatic patients found to have ventricular

preexcitation on a routine electrocardiogram is that the first symptomatic event will be sudden cardiac death. However, the incidence of sudden death in this group is low,[7, 14, 20, 110] estimated to be at most 1 per 1000 patient years of follow-up.[70] At electrophysiologic study, these asymptomatic patients frequently lack retrograde accessory pathway conduction, usually do not have inducible sustained AV reentrant tachycardia, and tend to have a longer effective refractory period of the accessory pathway in the antegrade direction.[78, 85, 102] In several series with long-term follow-up,[14, 78, 85] most of these patients remained asymptomatic; none died suddenly; and in one study, 31% of patients lost preexcitation.[72]

Catheter ablation of the accessory pathway would presumably eliminate the risk of sudden death in the event of atrial fibrillation. The question remains whether the risk of sudden death outweighs the risks of procedure-related complications. Unfortunately, there is no specific marker to identify the asymptomatic patient at risk for sudden death. Loss of preexcitation with increasing heart rate during exercise or with the administration of procainamide has been shown to correlate with a long antegrade refractory period of the accessory pathway (and, thus, a slow ventricular response during atrial fibrillation); however, preexcitation is not lost during these tests in many patients at low risk. Invasive electrophysiologic testing permits measurement of the shortest preexcited R to R interval during induced atrial fibrillation. A value of less than 250 msec is sensitive but not specific or predictive.[14, 78, 85] Therefore, we and others[14, 39, 70, 78] do not recommend routine invasive electrophysiologic testing or therapy in the majority of asymptomatic patients with preexcitation. Exceptions to these recommendations might include patients with high-risk professions (i.e., pilots, bus drivers).

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