THE UNIVERSITY OF TEXAS SOUTHWESTERN
MEDICAL CENTER

MEDICAL GROUND ROUNDS

"MANAGEMENT OF THE WOLFF-PARKINSON-WHITE SYNDROME"

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OUTLINE

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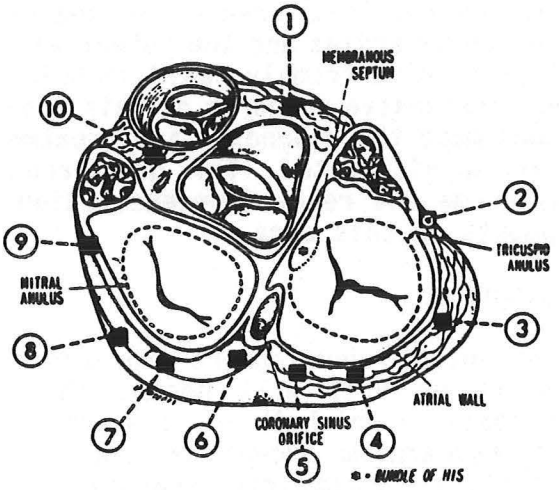
The Wolff-Parkinson-White (WPW) syndrome is a fairly common cardiovascular condition in which the presence of an anomalous atrioventricular pathway causes the early activation of a portion of the ventricular myocardium, resulting in a wide QRS complex and a short PR interval on the surface EKG. The anomalous atrioventricular connection can also create the anatomical substrate for the development of reentrant tachycardias and the potential for sudden cardiac death by virtue of the development of extremely rapid ventricular rates during atrial fibrillation or flutter. Extensive research on this disorder, especially within the last two decades, has made the diagnosis and treatment of patients with the WPW syndrome increasingly sophisticated. This grand rounds will serve as a brief overview of the WPW syndrome and related preexcitation syndromes, and examine several recent developments in this area.

I. Anatomy and Embryology

Normal cardiac embryologic development proceeds from a primitive tubular structure with continuity of what will become the atrial and ventricular myocardium to an adult heart in which there is electrical discontinuity of the atrial and ventricular myocardium except in the region of the penetrating atrioventricular bundle (1,2). Incomplete separation of atrial and ventricular myocardium during embryologic development can leave persistent strands of myocardial tissue that are felt to represent the anomalous atrioventricular connections responsible for the WPW syndrome. The fibrous tissues which separate the atrial and ventricular myocardium, referred to as the mitral and tricuspid annuli, are derived from an ingrowth of the atrioventricular sulcus tissue, and are continuous with the atrioventricular valves. The mitral annulus tends to be a well-formed collagenous band. As a result, when accessory atrioventricular connections occur on the left side of the heart, they tend to be located outside of an intact mitral annulus, usually on the epicardial side, within the epicardial fat. Defects in the fibrous portion of the tricuspid annulus are common (3). Occasionally, such annular defects result in the persistence of strands of myocardial tissue which connect the atrial and ventricular myocardium (2,3). A subendocardial location of the pathway may also occur with right sided pathways (4). Accessory atrioventricular connections are often fan-shaped with a single trunk on the atrial side, and multiple insertions on the ventricular side (3). They most commonly appear to be composed of myocardial tissue, but may in some cases be derived from specialized conducting tissue (3,5). The term Kent bundle is still commonly used to describe accessory atrio-ventricular connections occurring outside of the region of the normal specialized conduction tissues, although it has been argued that what Kent observed were islands of residual atrioventricular ring tissue which are a common incidental anatomical finding(5). Accessory atrioventricular connections (Kent bundles) can occur at any location along the mitral or tricuspid annulus, except in the area where the mitral annulus and the aortic valve are in close apposition. Several classification schemes have been devised to localize these tracts. The original classification of Rosenblum (6) divided accessory pathways into Type A and Type B, based upon the morphology of the QRS complex in lead V1. This classification scheme is of limited usefulness, because it is insufficiently precise. Gallagher, et al (7) identified 10 possible locations for atrioventricular connections based upon their extensive experience, and catalogued the typical delta wave polarity in each of the standard EKG leads for each type:



1. RIGHT ANTERIOR PARASEPTAL
2. RIGHT ANTERIOR
3. RIGHT LATERAL
4. RIGHT POSTERIOR
5. RIGHT PARASEPTAL
6. LEFT POSTERIOR PARASEPTAL
7. LEFT POSTERIOR
8. LEFT LATERAL
9. LEFT ANTERIOR
10. LEFT ANTERIOR PARASEPTAL

DELTA WAVE POLARITY

	I	II	III	AVR	AVL	AVF	V_1	V_2	V_3	V_4	V_5	V_6
①	+	+	+(±)	-	±(+)	+	±	±	+(±)	+	+	+
②	+	+	-(±)	-	+(±)	±(-)	±	+(±)	+(±)	+	+	+
③	+	±(-)	-	-	+	-(±)	±	±	±	+	+	+
④	+	-	-	-	+	-	±(+)	±	+	+	+	+
⑤	+	-	-	-(+)	+	-	±	+	+	+	+	+
⑥	+	-	-	-	+	-	+	+	+	+	+	+
⑦	+	-	-	±(+)	+	-	+	+	+	+	+	-(±)
⑧	-(±)	±	±	±(+)	-(±)	±	+	+	+	+	-(±)	-(±)
⑨	-(±)	+	+	-	-(±)	+	+	+	+	+	+	+
⑩	+	+	+(±)	-	±	+	±(+)	+	+	+	+	+

± = Initial 40 msec delta wave isoelectric
+ = Initial 40 msec delta wave positive
- = Initial 40 msec delta wave negative

Fig. 1 *Top*- Location of accessory pathways responsible for the WPW syndrome. *Bottom*- The polarity of the delta wave in the standard ECG leads for each of the above pathway locations is described. From Gallagher, et al (7).

Classification schemes on intermediate complexity are now commonly utilized. For purposes of planning surgery and analyzing surgical results, it is useful to describe accessory pathways as being localized to one of four regions: left free wall, posterior septal, right free wall, and anterior septal.

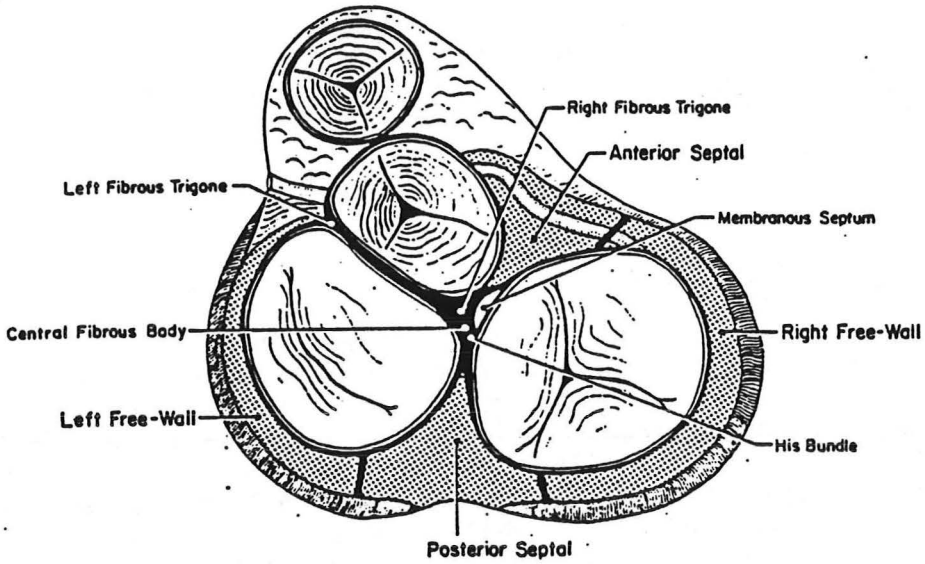


Fig. 2 Cut away view of the heart at the level of the mitral and tricuspid annuli demonstrating the boundaries of four anatomic areas where accessory pathways can occur. From Cox, et al (8).

Using the above classification scheme, the distribution of accessory atrioventricular pathways in a large series of surgical patients was found to be: left free wall-47%, posterior septal-27%, right free-wall-17%, anterior septal-9% (9). Multiple (two or three) accessory pathways were identified in 15% of patients in this series.

In addition to the more common accessory atrioventricular connections responsible for the Wolff-Parkinson-White syndrome described above, several other anomalous conduction pathways which involve portions of the normal conduction system have been described. These include Mahaim fibers (nodoventricular or fasciculoventricular [7,10]), atriofascicular connections (11,12) and intranodal or atrionodal connections (James fibers [13]). The latter are a common anatomic finding in normal hearts and their role in preexcitation syndromes is speculative (7). Most of these anomalous pathways can potentially result in ventricular preexcitation and a short PR interval. (The PR interval is usually normal, however, with fasciculoventricular Mahaim fibers). In the case of intranodal, atrionodal, and atriofascicular connnections, however, the QRS duration will be normal, since the anomalous pathway connects distally with the normal conduction system. The association of supraventricular tachycardias with a short PR interval and normal QRS duration has been referred to as the Lown-Ganong-Levine syndrome (14,15). Enhanced AV nodal conduction is a term which has been used to describe the PR interval shortening in this syndrome. This may be mediated by pathways which partially or completely bypass the AV node (James fibers or atriofascicular connections, respectively), by an anatomically compact AV node, or by rapid conduction through an anatomically normal AV node (7,16).

Of the several types of preexcitation syndrome, the WPW syndrome is by far the most common. In a large series of patients coming to electrophysiology study (9), the following distribution of accessory pathways was found: accessory atrioventricular pathways-456 (83%); Mahaim fibers-24 (4%); enhanced AV nodal conduction-69 (13%).

II. Incidence and Genetics

Numerous studies have utilized the surface ECG as a screening test to examine the frequency of occurence of ventricular preexcitation of the WPW type in various populations. A very large study of healthy applicants to U.S. Air Force flight training programs concluded the incidence of the WPW pattern to be 1.5 per thousand (17). A similar Canadian study estimated the incidence to be 3 per thousand (18). Studies in healthy children have yielded similar results (0.5 to 3.1 per thousand [19]), while studies in patients referred to heart clinics report higher incidences of approximately 5 per thousand (19). Most studies report a preponderance of males (approximately 65% [19]). These figures probably underestimate the frequency of the condition, since it is well known that preexcitation can be intermittent in many individuals. In addition, many individuals have concealed bypass tracts capable only of retrograde conduction (from the ventricle to the atrium), and which will therefore not manifest preexcitation on the surface ECG.

Several studies have demonstrated an association between the WPW syndrome and congenital heart disease. In children, the incidence of congenital heart disease is patients with the WPW syndrome is as high as 33 to 53% in a heart clinic population (20-22). The figure is probably somewhat lower for adults, due to the high mortality of the associated congenital heart disease (21). Numerous congenital heart defects have been reported to be associated with the WPW syndrome:

ASSOCIATION OF THE PREEXCITATION SYNDROMES WITH CONGENITAL HEART DISEASE

	Sherf and Neufeld	Gallagher, et al.
Ebstein's Anomaly	15	8
Mitral Valve Prolapse	--	11
Mitral Insufficiency ? Etiology	--	1
Ventricular Septal Defect	7	2
Atrial Septal Defect	2	1
Transposition	5	1
Patent Ductus	4	--
Tetralogy of Fallot	4	--
Dextroversion	1	--
Coarctation of the Aorta	1	--
Coarctation of the Aorta & Bicuspid Aortic Valve	--	1
Cardiomyopathy	5	8
Pulmonic Stenosis	3	--
Bicuspid Pulmonic & Aortic Valve	--	1
Aortic Insufficiency - Etiology?	--	1
Tricuspid Atresia	1	--
Mitral Atresia	1	--
Double Outlet	1	--
Anomalous Venous Return	--	1
Single Ventricle	--	1

TABLE I. Congenital heart defects associated with the WPW syndrome. Adapted from the review of Sherf and Neufeld (19) and the series of Gallagher, et al (7).

In addition to the findings of these large series, other reported associations with congenital heart disease include coronary sinus diverticulum with posterior septal pathways (23) endocardial cushion defect (24), complex congenital aortic stenosis (25), and subvalvular aortic stenosis (26). An association with Marfan's syndrome and accompanying cardiac lesions has been described (27), as has an association with pectus excavatum (28).

The association of the WPW pattern with Ebstein's anomaly of the tricuspid valve is the most striking, being present in 7% of 267 patients coming to surgery for preexcitation in the large series reported by Gallagher, et al (9). Approximately 5 to 25% of patients with Ebstein's anomaly have evidence of preexcitation of ECG, which is usually due to a right-sided accessory pathway (29,30). Patients with Ebstein's anomaly and preexcitation have an unusually high incidence of Mahaim fibers and multiple accessory pathways (21% and 53%, respectively [9]).

Interestingly, patients with mitral valve prolapse who are found to have accessory AV connections at electrophysiologic study typically have left-sided AV connections (31). The finding of accessory pathways on the same side of the heart as the atrioventricular valve abnormality in Ebstein's anomaly and mitral

valve prolapse is consistent with defective formation of the region of the atrioventricular annulus during cardiogenesis.

Familial occurance of the WPW syndrome, although rare, is well described, and may occur in the presence or absence of associated cardiomyopathy (32-37). Most studies demonstrating familial involvement suggest an autosomal dominant mode of inheritance. The propensity for sudden cardiac death to occur in some families is one indication for an aggressive diagnostic approach in affected members (32).

III. Clinical Manifestations

A broad spectrum of clinical presentations can occur in patients with preexcitation, ranging from totally asymptomatic (with preexcitation being discovered incidently on a routine ECG), through symptoms associated with tachyarrhythmias (palpitations, symptomatic tachycardia, dizziness, syncope, or angina), to sudden death. Estimates of the frequency of symptoms vary considerably between different series depending upon the population studied. Studies in military recruits give estimates of 4.3 to 13% (17,38) while studies from cardiac clinics report symptomatic rates as high as 84% (39).

Although little data on the incidence of sudden death in the WPW syndrome is available, it is considered a rare but well-documented complication that creates a number of important dilemmas in appropriate management of these patients. Sudden death is generally thought to occur as a result of deterioration of a very rapid ventricular response to atrial fibrillation into ventricular fibrillation (40). It usually occurs in patients with a previous history of tachycardias (atrial fibrillation or both atrial fibrillation and reciprocating tachycardias), but it can occur without previous symptoms (3 of 25 patients in the series of Klein, et al [40] presenting with ventricular fibrillation had no previous history of symptoms, and 5 had only slight symptoms). The strongest correlation between electrophysiologic findings and sudden death in these patients was the finding of a shortest RR interval between preexcited beats during atrial fibrillation of 250 msec or less.

Rinne, et al examined the relationship between clinical presentation and the presence of an RR interval between consecutive preexcited beats during atrial fibrillation of 250 msec or less in WPW patients (41). They found that 75% of patients presenting clinically with atrial fibrillation and 70% of patients presenting with both atrial fibrillation and AV reciprocating tachycardia fell into this group. 41% of patients presenting with AV reciprocating tachycardia and 51% of patients presenting only with palpitations fell into this higher-risk group.

The absence of symptoms in a patient with the WPW pattern on ECG appears to place the patient in a group less prone to the development of life-threatening arrhythmias. This is confirmed by a study by Milstein, et al that showed that, compared to symptomatic controls, asymptomatic patients have a slower average heart rate and longer minimum RR interval between consecutive preexcited beats in atrial fibrillation, although 17% of asymptomatic patients did have potentially lethal ventricular rates during atrial fibrillation (42). Other

longitudinal studies by this same group have recently shown that 31% of asymptomatic patients with the WPW pattern actually lose the capacity for antegrade conduction over their accessory pathway with time (43). The prognosis for patients with asymptomatic WPW pattern is therefore generally quite favorable, but the fact that a small number of patients present with sudden death as the initial manifestation of their WPW syndrome (40,44) has generated a great deal of interest in the development of methods to identify those patients who are at risk, as will be discussed below.

IV. Diagnosis and Risk Stratification

A) The surface electrocardiogram. This was the tool utilized by Wolff, Parkinson, and White in their original description of patients with a wide QRS complex and short PR interval who were prone to the development of paroxysmal tachycardias (45), and it continues to be of great value in the diagnosis and management of patients with the WPW syndrome.

1. ECG Criteria for WPW Pattern

There are four conditions which are necessary for the diagnosis of the WPW pattern on the ECG (WHO criteria[46]): a. A PR interval of less than 0.12 seconds. b. A P-wave axis of >0 and $\leq$ 90 degrees. c. A QRS duration of >0.12 seconds. d. The presence of a delta wave. These findings are illustrated on the figure below:

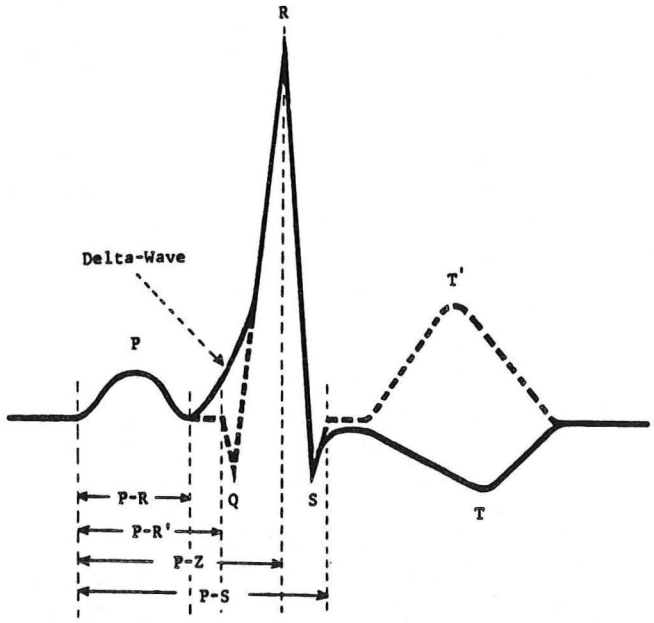


Fig. 3 Features of the classic WPW pattern on the surface ECG. From Chung (47).

The shortening of the PR interval in the WPW pattern is a manifestation of early activation of the ventricle via the accessory pathway. The PS interval is usually normal. The PR interval may, however be normal in conditions in which there is slow conduction through the normal pathway, or when only a slight degree of preexcitation is present (48). The characteristic QRS morphology of the WPW pattern reflects the fact that activation of the ventricles occurs over two different pathways - the accessory AV connection and the normal AV conduction pathway. The QRS pattern seen on the surface ECG therefore represents a fusion of patterns produced by activation of the ventricle from two different sites. The impulse typically travels most rapidly through the accessory pathway to activate a portion of the ventricle near the pathway's ventricular insertion. Since conduction proceeds relatively slowly through working myocardial tissues, the rate of change of the QRS complex on the surface ECG is also slow, resulting in the typical delta wave (7). Once the impulse traveling through the normal conduction pathways enters the His-Purkinje system, the rest of the ventricle depolarizes rapidly, and a more typical-appearing terminal QRS configuration may be seen. The pattern of repolarization is also altered so that an inverted T wave usually follows the QRS complex. The degree of preexcitation and the morphology of the QRS complex vary considerably with the location of the accessory pathway and the timing with which the impulses arriving via the two pathways excite the ventricles. Right-sided pathways tend to have more marked preexcitation than left-sided pathways, in part due to their closer proximity to the AV node (49). Changes in vagal tone primarily affect the AV node. Factors which increase vagal tone tend to slow conduction through the AV node and increase the degree of preexcitation seen on the ECG (48). Factors that accelerate conduction through the AV node (hyperadrenergic and vagolytic states, exercise) tend to diminish the amount of preexcitation. Catecholamines also have some effect on conduction through the accessory pathway (50), and can be used in combination with vagal maneuvers to unmask the presence of preexcitation (51).

Many patients are observed to have intermittent evidence of preexcitation on serial ECGs. This is a simple and useful predictor of accessory pathway conduction characteristics since, when preexcitation occurs only intermittently with slight alternations in vagal tone, it implies that the normal conduction pathway occasionally is completely responsible for ventricular depolarization, implying precarious conduction over the accessory pathway (52). Intermittent preexcitation is therefore a favorable prognostic indicator in patients with the WPW syndrome. Similarly, preexcitation which is present at rest but goes away with exercise is a second favorable non-invasive prognostic indicator in the WPW syndrome (53). (See section 5). The loss of preexcitation during exercise testing should be abrupt (implying block in the accessory pathway).

2. **Use of the ECG in Localizing Accessory Pathways.** The ECG can provide useful, but not absolute information about the location of accessory pathways. The classification system proposed by Roseblum (6) has been criticized as being insufficiently precise (7). However, the use complex criteria such as those of Gallagher, et al (Table 1) or the WHO criteria (46) to precisely localize accessory pathways has met with limited success (54). A classification scheme of intermediate complexity which attempts to localize the bypass tract to one of the four regions previously defined (Figure 2) has recently been described and correlated with accessory pathway localization by electrophysiologic study:

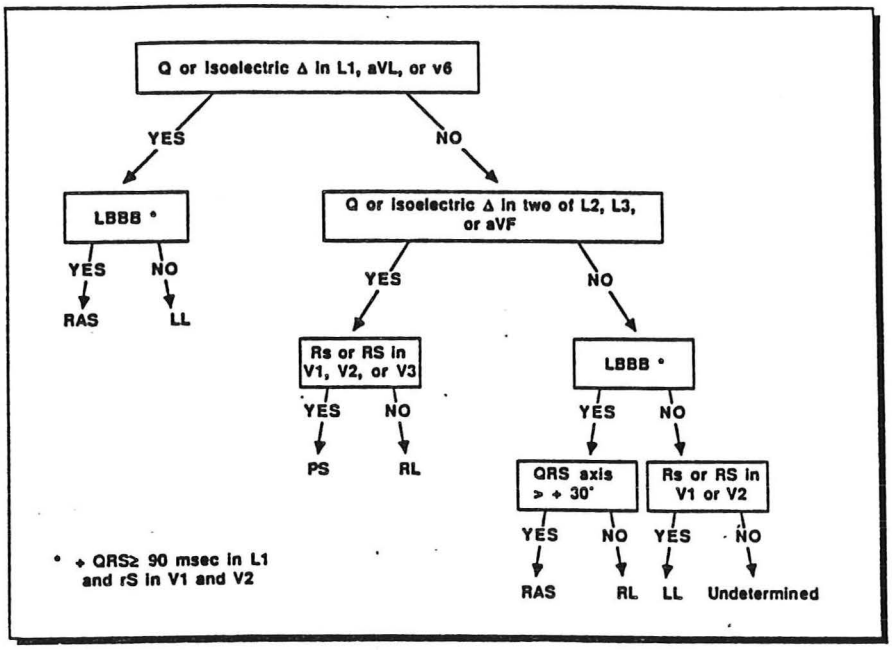


Fig. 4 Algorithm for predicting accessory pathway localization in the WPW syndrome (from Milstein, et al [55]).

Although algorithms of intermediate complexity have reasonable accuracy in predicting approximate accessory pathway localization (55-57), it is best to recognize the limitations of the surface ECG in localizing accessory AV connections, although it can serve as a useful guide in planning subsequent electrophysiologic study.

Tachycardia involving a nodofascicular Mahaim fiber has a characteristic left bundle configuration on ECG due to preexcitation of the right ventricle. Typical features are: a. cycle length 220-450 msec b. QRS axis 0 to -75 degrees c. QRS duration of 0.15 sec or less d. R wave in lead 1 e. rS in V-1 with precordial transition after V-4 (58).

3. Mimicry and Masking. The abnormal ventricular depolarization pattern seen in the WPW syndrome can mask other information which is usually obtainable from the ECG, such as the presence of myocardial infarction. Conversely, the abnormal QRS pattern can mimic myocardial infarction in some cases (such as pseudo-inferior infarction with posterior septal pathways). It is sometimes difficult to distinguish preexcitation from bundle branch block, especially when there is a large degree of preexcitation present. During exercise testing in patients with ventricular preexcitation, a very high incidence of false-positive ischemic-appearing ST segment depression is observed (59,60). A high percentage of abnormal thallium exercise tests has also been noted in patients with ventricular preexcitation (61), and may be analogous to the septal perfusion defects sometimes seen with left bundle branch block (62).

4. Tachycardia in the WPW Syndrome Clues from the ECG. The unique tachycardias associated with the WPW syndrome are those in which the accessory pathway participates. These include macroreentrant recriprocating tachycardias as well as atrial fibrillation and atrial flutter. It should be kept in mind that

virtually any other cardiac arrhythmia can also occur in these patients, in which case the accessory pathway would be said to play a "bystander" role (63).

The atrioventricular reciprocating tachycardias are classic examples of reentry. There are two basic types of recroprocating tachycardia seen in patients with the WPW syndrome, that are named after the direction of conduction across the accessory pathway during the tachycardia. Orthodromic recriprocating tachycardias conduct antegrade (from the atria to the ventricles) across the normal conduction pathway and retrograde via the accessory pathway. Antidromic recriprocating tachycardias conduct antegrade via the accessory pathway and retrograde through the normal conduction pathways. The possible reentrant circuits become much more complex in the setting of multiple accessory pathways:

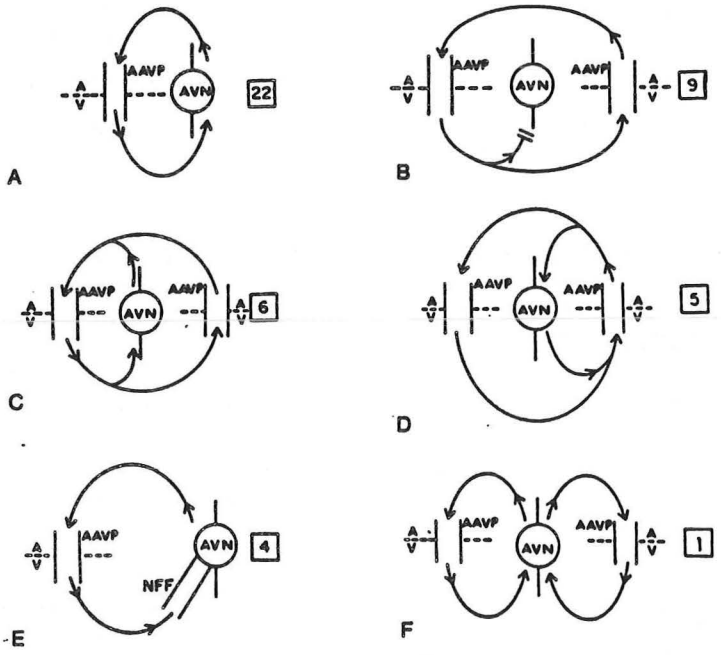


Figure 5. Possible reentry patterns producing preexcited recriprocating tachycardias in 42 patients. A. Antidromic reciprocating tachycardia with antegrade conduction over the accessory atrioventricular pathway (AAVP) and retrograde conduction through the AV node (AVN). B. Reciprocating tachycardia involving two AAVPs, not involving the AVN. C. Reciprocating tachycardia with antegrade conduction over one AAVP and retrograde conduction over both the AVN and a second AAVP. D. Reciprocating tachycardia with antegrade conduction over one or more AAVP and the AVN, and retrograde conduction over a second AAVP. E. Reciprocating tachycardia with antegrade conduction over one or more AAVPs and retrograde conduction via a nodofascicular Mahaim fiber (NFF). F. Reciprocating tachycardia with antegrade conduction over two AAVPs and retrograde activation via the AVN. From Bardy, et al (64) by permission of the American Heart Association.

a) Orthodromic recriprocating tachycardia- This is a narrow-complex tachycardia, since antegrade conduction to the ventricles occurs aver the normal conduction pathways, and represents the most common tachycardia seen in patients with anomalous AV connections. Heart rates are typically between 150 and 240 beats per minute (65). The heart rate during the tachycardia can be quite variable in the same individual at different times, depending on the autonomic influences on the AV node (66). The finding of a heart rate over 200 with a narrow-complex tachycardia is said to be suggestive of a tachycardia involving an anomalous AV connection (67). The retrograde activation of the atria via the accessory pathway results in a P-wave which follows shortly after the preceding QRS complex (R-P< P-R) (68). The R-P interval is approximately 70-135 msec (based on electrophysiologic study). Shorter R-P intervals (less than 61 msec) are more suggestive of an AV nodal reentry as the mechanism of the tachycardia (69). Other tachycardias such as ectopic atrial tachycardia with first degree block can simulate the R-P< P-R pattern seen with orthodromic reciprocating tachycardia, so that electrophysiology study may be necessary to definitively determine the tachycardia mechanism (65). A R-P> P-R pattern can occasionally be seen with orthodromic reciprocating tachycardia if there is slow retrograde conduction across the accessory pathway, as can occur in the incessant form of AV recriprocating tachycardia and patients with Ebsteins anomaly (48).

Another useful ECG finding in AV recriprocating tachycardia is that of QRS alternans. This finding has a sensitivity of 37% and a specificity of 96% in distinguishing orthodromic recriprocating tachycardia from other types of narrow complex tachycardia (70).

The development of functional bundle branch block during supraventricular tachycardia can also be a useful clue to the presence of orthodromic recriprocating tachycardia. Slowing of the tachycardia with the development of bundle branch block or fascicular block suggests the involvement of an accessory pathway near the site of the block (due to lengthening of the reentry pathway as a result of the bundle branch block or fascicular block [71,72]). Inferences about the location of the anomalous pathway can also therefore be made from this observation. The development of functional left bundle branch block is felt by some authors to suggest of orthodromic recriprocating tachycardia, even in the absence of tachycardia slowing (73).

If orthodromic AV recriprocating tachycardia is initiated by a premature atrial contraction in a patient with preexcitation during normal sinus rhythm, the QRS complex following the PAC will suddenly narrow due to the development of conduction block in accessory pathway. This finding provides useful information regarding the mechanism of the tachycardia (48). In patients without evident preexcitation during normal sinus rhythm, the development of tachycardia during a gradual increase in sinus rate, without the development of a prolonged PR interval, suggests the possibility of a concealed anomalous AV connection (74).

b) Antidromic recriprocating tachycardia utilizes the anomalous AV connection as the antegrade limb of the reentry circuit and is therefore characterized by a short PR interval and maximal ventricular preexcitation on the ECG. It occurs in approximately 10% of patients with the WPW syndrome (64). Heart rates are typically in the range of 150 - 240 beats per minute. Antidromic recriprocating

tachycardia can be difficult to distinguish from other types of wide-complex tachycardias such as supraventricular tachycardias with aberrant ventricular conduction and ventricular tachycardia (see discussion of wide complex tachycardias in section c below). P waves can be difficult to discern during antidromic recriprocating tachycardia, rendering this finding less helpful. Accessory pathways in patients with antidromic recriprocating tachycardia are usually located on the left free wall or the right free wall. Septal locations are rare, except in the setting of multiple accessory AV connections (63). This suggests that a critical distance between the normal conduction pathways and the accessory AV connection is usually necessary for the development on this arrhythmia.

c) Atrial fibrillation and atrial flutter are potentially life-threatening arrhythmias in subsets of patients with accessory AV pathways due to the potential for very rapid conduction over the accessory pathway to deteriorate into ventricular fibrillation. Atrial fibrillation and flutter occur in approximately 32% of patients with symptomatic WPW syndrome (75). Ventricular response rates during atrial fibrillation and flutter depend on the conduction properties of the accessory pathways and the normal conduction system, as well as the ventricular refractory period, and may be as high as 350 beats per minute (65). The presence of a regular wide complex tachycardia at a rate of >240 beats per minute suggests atrial flutter (76). The QRS complexes observed on the ECG may show intermittent preexcitation, or may be totally narrow-complex or totally preexcited, depending on the properties of the different conduction system components. The interval between two consecutive preexcited QRS complexes can be useful in distinguishing patients who are at risk for ventricular fibrillation. Retrospective analysis of patients presenting with ventricular fibrillation suggests that these risks are highest when this interval is less than 250 msec (40). The shortest RR interval during atrial fibrillation correlates reasonably well with the effective antegrade refractory period of the accessory pathway (77), however, atrial refractoriness may limit the determination of the antegrade refractory period in patients with rapid antegrade conduction, rendering this value less helpful (40). Assessment of accessory pathway characteristics during isoproterenol infusion or exercise testing may disclose other patients who are at risk for ventricular fibrillation by virtue of rapid rates during atrial fibrillation or short antegrade accessory pathway refractory periods, which may not have been apparent at rest (50,78,79).

Wide complex tachycardias with antegrade conduction over an accessory pathway can be difficult to distinguish from ventricular tachycardia or supraventricular tachycardias with aberrant ventricular conduction. Miles and Zipes (80) reported the findings of 143 patients presenting with wide complex tachycardias characterized by electrophysiologic study. 12 of the 143 patients had preexcitation as the cause of the wide QRS complex (9 of the 12 had atrial fibrillation or flutter). The characteristics of these tachycardias on the ECG are summarized in the table below:

TABLE 2: ECG CHARACTERISTICS OF WIDE COMPLEX TACHYCARDIAS

	V. Tach (%)	Aberrancy (%)	A. P. (%)
Number of Patients	113	18	12
QRS Duration (msec)	165 ± 35	138 ± 16	162 ± 25
Range (msec)	120-300	120-160	120-200
Cycle Length (msec)	368 ± 63	351 ± 90	303 ± 71
Range (msec)	240-560	290-450	200-400
RBBB pattern	63	9	9
Triphasic QRS	8	7	0
Biphasic QRS	34	0	1
Monophasic QRS	21	2	8
LBBB pattern	50	9	3
Triphasic QRS	7	2	0
Biphasic QRS	21	0	0
Monophasic QRS	22	7	3
QRS Concordance			
Negative	9	3	0
Positive	12	0	0
Normal Axis (-30° to +90°)	15 (13)	7 (39)	2 (17)
Left Axis (< -30° to -90°)	33 (29)	7 (39)	4 (33)
Extreme Axis (< -90° to ±180°)	35 (31)	0 (0)	1 (8)
Right Axis (> +90° to +170°)	30 (27)	4 (22)	5 (421)

(Abbreviations: A.P.= accessory pathway; RBBB = right bundle branch block; LBBB = left bundle branch block. From this figure, it can be appreciated that, in the absence of discernable P wave activity, there are no features of the ECG that are diagnostic of accessory pathway conduction. However, ECG findings may be useful in helping to rule out accessory pathway conduction. The finding of triphasic QRS complexes, QRS concordance, or QRS duration less than 140 msec suggest that the mechanism of the tachycardia does not involve an accessory pathway.

The electrical alternans is a frequent finding in wide-complex tachycardias incorporating a bypass tract, although it is also found frequently in ventricular tachycardia. If the mechanism of the tachycardia is known to be supraventricular, the finding of electrical alternans strongly suggests the incorporation of a bypass tract in the tachycardia circuit (81).

5. Non-invasive assessment of risk of sudden death. As mentioned above, the finding of intermittent preexcitation on serial ECGs during normal sinus rhythm and the disappearance of preexcitation with exercise have been correlated with long effective refractory periods in the accessory pathway, thereby implying a low risk of sudden death. In addition, pharmacologic testing with procainamide, disopyramide, or ajmaline has been advocated as a means of predicting the risk of sudden death (82,83,84). Disappearance of preexcitation with drug infusion has been correlated with long antegrade refractory periods in the accessory pathway.

Sharma, et al (84) evaluated 67 patients with the WPW syndrome, 9 of whom had a history of sudden cardiac death. Exercise tolerance testing and disopyramide infusion were performed in these patients (using persistence of preexcitation during exercise or following disopyramide infusion to define those patients at risk), and compared to the results of invasive electrophysiology testing (using an RR interval of $\leq$250 msec between consecutive preexcited beats during induced atrial fibrillation to define those patients at risk of sudden death). The ability of these tests to predict sudden death were:

	Sensitivity	Specificity	Predictive Accuracy
Exercise testing	80.0%	28.6%	11.8%
Disopyramide infusion	71.4%	26.1%	12.8%
Electrophysiologic testing	77.8%	48.3%	18.9%

Both exercise testing and disopyramide testing were able to predict the results of electrophysiologic testing with good sensitivity (100% and 90.6%, respectively), but only moderate specificity (54.1% and 52.3%, respectively). Thus, both non-invasive and invasive testing are fairly sensitive predictors of sudden death, but fail to identify approximately 20-30% of patients with this presentation. The specificity and predictive accuracy of all these tests are low, although invasive testing is somewhat more specific and has a higher predictive accuracy than non-invasive testing. Other groups have recently had less favorable results with non-invasive testing (85,86).

Some authors have advocated the use of esophageal electrodes to obtain electrophysiologic data on patients with the WPW syndrome, while avoiding the use of intracardiac electrodes (87). The esophageal electrode can be used to induce atrial fibrillation to evaluate antegrade conduction qualities of the accessory pathway, and to record atrial activity during tachycardias. In comparison with detailed electrophysiologic testing, this technique does not allow detailed mapping of accessory pathway location, and is of limited value in the study of recriprocating tachycardias, many of which require ventricular stimulation to elicit (66,88). The technique may be useful to avoid multiple invasive procedures in patients for whom serial electrophysiologic testing is required. It can also be combined with exercise testing to evaluate a patients risk for sudden death (89).

B) Electrophysiology study in patients with the WPW syndrome. Electrophysiologic testing provides valuable data in the diagnosis and management of patients with the WPW syndrome, and is the standard by which other tests are usually judged. Information which can be obtained from electrophysiologic testing includes: confirmation of the presence of preexcitation, delineation of the mechanism of tachycardias, determination of the conduction characteristics of the accessory pathway and the AV node-His-Purkinje system, determination the location and number of accessory pathways, estimation of the risk of sudden cardiac death and assessment of the effects of pharmacologic and other interventions. Some general principles of electrophysiologic testing in the WPW syndrome are discussed below.

1) Confirmation of the presence of preexcitation. This can be done by demonstrating that the onset of the delta wave on the ECG occurs simultaneous with or before the His bundle deflection on the His bundle electrogram (Normally, ventricular activation occurs 30-55 msec after His bundle activation [7]).

2) Localization of accessory pathways. Several techniques provide information as to accessory pathway localization:

a. Clues to the presence and location of accessory pathways from the surface ECG have already been discussed.

b. Atrial pacing from multiple sites around the mitral and tricuspid annuli can be utilized to localize the site of an accessory pathway, using the criteria that, the closer the pacing catheter is to the accessory pathway, the shorter the resulting PR interval will be, and the greater the degree of ventricular preexcitation (90,91).

c. The site of earliest retrograde atrial activation during orthodromic recriprocating tachycardia or ventricular pacing similarly provides useful information about the atrial insertion of the accessory pathway (7,92).

d. A "preexcitation index" (93) can be a useful calculation in discriminating accessory pathway location. During recriprocating tachycardia, the interval is determined from the intrinsic QRS complex of the tachycardia to the latest premature ventricular stimulus which will cause result in early activation of the atrium. This value is then subtracted from the intrinsic cycle length of the tachycardia to yield the preexcitation index. The farther the accessory pathway is from the ventricular activation site (the RV apex), the more premature the ventricular stimulus has to be, and the larger the preexcitation index. This technique is particularly useful in discriminating septal and right free wall accessory pathways from left free wall accessory pathways. Septal and right free wall accessory pathways have a preexcitation index of < 45 msec, while the index is typically ≥ 75 msec with left free wall accessory pathways.

e. As previously noted, the prolongation of the cycle length of orthodromic recriprocating tachycardia with the development of bundle branch block (due to prolongation of VA conduction time) indicates an accessory pathway ipsilateral to the block. An increase in VA conduction time of 25 msec or more with the development of a bundle branch block indicates a free wall rather than a septal location of the accessory pathway (94,95).

f. The phenomenon of "paradoxical capture" can be used to localize the accessory pathway. This refers to the fact that, during recriprocating tachycardia, the VA conduction time of a premature stimulus delivered at the right ventricular apex varies with the location of the accessory pathway. For septal pathways, the VA conduction time is the same as that following the QRS complexes of the tachycardia. Left free wall accessory pathways typically have a VA conduction time that is 25 msec longer than that of the tachycardia. With right free wall accessory pathways, the VA conduction time is less than that of the tachycardia (96,97). The addition of double premature stimuli can further help to localize left-sided accessory pathways (98).

g. It is sometimes possible to record a small activation potential from the accessory pathway with a catheter positioned directly adjacent to the pathway. This small activation potential needs to be correlated with other data on accessory pathway location to be confident of its origin (99-101).

Clues to the presence of multiple accessory pathways (including "enhanced AV conduction") at electrophysiology study include persistently short AH or HV intervals during recroprocating tachycardia, change in the surface preexcitation configuration with pacing at faster rates or from different sites, varying sequences of retrograde atrial activation during recriprocating tachycardia or ventricular pacing, short VA intervals recorded at more than one site, and a fluctuating delta wave morphology during atrial fibrillation (102-104).

3) Determination of the functional properties of the accessory pathway and the normal conduction pathways is usually determined in both the antegrade and retrograde directions. This typically involves incremental pacing from the right atrium, left atrium (via a catheter in the coronary sinus), and the right ventricle. Effective refractory periods are determined by the shortest coupling interval at which a premature beat can be conducted by a given pathway at a specified baseline pacing rate (7,105). Accessory AV connections capable only of antegrade conduction ("concealed" pathways) are a fairly frequent electrophysiologic finding (106,107). Accessory pathways capable only of antegrade conduction are uncommon (66).

4) Induction of recriprocating tachycardias is often an important goal of electrophysiology testing, both to determine tachycardia mechanism definitively and to localize accessory pathways. This is usually achieved during incremental pacing from atrial or ventricular sites, or through the use of extrastimuli from atrial or ventricular sites to create unidirectional block in one arm of the reentry circuit (a necessary condition for reentry) (105,108). Orthodromic recriprocating tachycardia is the most common tachycardia produced. Confirmation of the participation of an accessory pathway in the reentry circuit can be obtained by demonstrating that introduction of a PVC during recriprocating tachycardia can cause retrograde atrial preexcitation at a time when the His bundle is refractory (66).

5. Induction of atrial fibrillation is an important aspect of the electrophysiologic study in patients capable of antegrade conduction across an accessory pathway. As previously discussed, the shortest interval between consecutive preexcited beats in atrial fibrillation is felt to be the most useful predictor of the risk of sudden cardiac death in patients with the WPW syndrome. Atrial fibrillation can be induced by in the electrophysiology lab by rapid atrial pacing at cycle lengths of 50 to 200 msec (40).

6) Assessment of the response to pharmacologic and other interventions. Once the baseline characteristics of the conduction pathways and tachycardia circuits are defined, the changes induced by pharmacologic intervention, surgery, catheter ablation or pacing techniques can be evaluated.

7) Risks of electrophysiology study. Major catheter-related complications (infection, thromboembolism, pneumothorax, cardiac perforation) occur in less than 2.0% of patients undergoing electrophysiologic study (109,110). The

mortality of the procedure is 0.1% or less (109,110). The induction of symptomatic arrhythmias is a frequent "complication" of electrophysiologic study, and is in fact frequently a goal of the study (105,111). The unintentional induction of atrial fibrillation during catheter manipulation can hinder the performance of subsequent portions of the electrophysiologic study. A signifigant percentage of patients may require electrical cardioversion or defibrillation if arrhythmias with hemodynamic compromise are induced.

C. Phase image analysis of multi-gated blood pool scintigrams has recently been utilized to localize accessory pathways by identifying the site of earliest detectable ventricular contraction (112-115). The addition of tomographic analysis improves the sensitivity of the technique (114).

D. Echocardiography has also been used to document abnormal patterns of ventricular activation in patients with anomalous AV pathways (116,117). Digitally processed two dimensional echocardiography correctly identified the location of 18 out of 22 accessory pathways verified by electrophysiologic study (117).

V. Therapy

For many patients with asymptomatic accessory AV connections or occasional episodes of tachycardia, no therapy is necessary, or vagal maneuvers may suffice (for recriprocating tachycardias) (118). For symptomatic patients or patients at increased risk of sudden death, a number of other options can be considered.

A. Pharmacologic therapy. The large reentry circuits which characterize the recriprocating tachycardias of the WPW syndrome typically incorporate the AV node, the accessory pathway, as well as portions of the His-Purkinje system and the atrial and ventricular myocardium. These long pathways provide the opportunity for pharmacologic intervention at multiple sites to terminate or prevent recriprocating tachycardia, which explains the multitude of drugs which have been shown to be useful in subsets of patients with the WPW syndrome. For patients capable of rapid antegrade conduction across an accessory pathway, the prevention of rapid atrial fibrillation is of paramount importance in the prevention of sudden death. Inappropriate therapeutic interventions can precipitate or worsen rapid atrial fibrillation in these patients, with potentially devastating consequences.

Quinidine, procainamide, and disopyramide are often drugs of first choice in the management of patients with the WPW syndrome (119-123). Their principal site of action is on the accessory pathway, where they prolong refractoriness and slow conduction. Disopyramide appears to be a more effective drug for patients with short effective refractory periods of the accessory pathway (123). Procainamide in its intravenous form is usually the drug of choice to acutely slow the ventricular response to atrial fibrillation. All of these agents are potentially useful in the prevention and termination of recriprocating tachycardias and atrial fibrillation in patients with accessory pathways. Intermittent therapy with procainamide may be useful in selected patients with occasional episodes of tachycardia (124).

Ajmaline is and experimental antiarrhythmic in this country with effects similar to procainamide (96). It also appears to be useful as an intravenous drug to slow conduction in accessory pathways (120,125,126).

Lidocaine has variable, but usually small effects on accessory pathway conduction (127-129). In patients with atrial fibrillation and a short antegrade refractory period of the accessory pathway, it can accelerate conduction with resultant hemodynamic deterioration (129). Lidocaine should therefore not be considered a drug of first choice in the management of arrhythmias incorporating anomalous AV pathways.

The class 1C antiarrhythmics flecainide and encainide appear to be very effective in the suppression of arrhythmias in many patients with the WPW syndrome (130-144). They have the effects of slowing conduction and increasing refractoriness in both the AV node and the accessory pathway. Recent concerns about the safety of these drugs in post-MI patients have prompted the FDA to restrict their use (145). Their long-term safety in the treatment of supraventricular tachycardias has yet to be adequately evaluated.

Amiodarone also has the effects of slowing conduction and increasing refractoriness in both the accessory pathway and the AV node. It is effective in preventing the recurrence of both atrial fibrillation and recriprocating tachycardias in the WPW syndrome (146-152). Because of its potentially severe side effects, its use should be reserved for patients who are refractory to other antiarrhythmics, and who are not good candidates for definitive surgical division or catheter ablation of the accessory pathway.

Beta adrenergic blockers are of limited utility in the management of WPW patients. They have little effect on the accessory pathway in most patients, and minimal to moderate effect on depressing AV nodal conduction (153-156). They occasionally can accelerate conduction across the accessory pathway during atrial fibrillation (156). They may be useful in selected patients in whom heightened adrenergic tone (as during exercise) is important in precipitating recriprocating tachycardia.

Digitalis has variable effects on the conduction properties of the accessory pathway, but can increase the ventricular response to atrial fibrillation in about one third of patients (157). It slows conduction and prolongs refractoriness in the AV node (158). Its use has been correlated with the development of ventricular fibrillation in patients with baseline short RR intervals during atrial fibrillation (157). The use of digitalis should therefore be avoided in patients with the WPW syndrome unless it has been demonstrated to safe in a given patient by electrophysiologic study.

Verapamil slows conduction and increases refractoriness in the AV node (159,160). Effects on conduction through the accessory pathway are variable, but acceleration of the ventricular response to atrial fibrillation with subsequent deterioration into ventricular fibrillation is well described (161-163). Although acute administration of verapamil may be useful in terminating episodes of orthodromic recriprocating tachycardia in certain patients (118), its use should be avoided in WPW patients with wide-complex tachycardias due to the propensity to accelerate antegrade conduction across the

accessory pathway. It should be borne in mind that, of all patients (not necessarily WPW patients) presenting with wide-complex tachycardias of uncertain mechanism, ventricular tachycardia is the most frequent cause (164,165). The administration of verapamil to such patients can be disasterous (165,166).

A number of other experimental antiarrhythmic drugs have been studied and shown to be of potential use in patients with the WPW syndrome. These include lorcainide (167,168), pimrenol (169), propafenone (170-172), and sotalol (173,174).

B. Surgical therapy. Although most patients with arrhythmias related to the WPW syndrome can be managed medically, the surgical interruption of accessory AV connections offers the possibility of cure of the condition to patients who have medically refractory arrhythmias or are in a high risk group for sudden cardiac death. Surgery also represents a desirable alternative to possible life-long medical therapy for many patients. Both endocardial and epicardial approaches have been successfully utilized to surgically divide accessory pathways, with several recent modifications.

The usual surgical approach is via a median sternotomy. Following exposure of the heart, intraoperative electrophysiologic mapping is performed. This can be performed using a single electrode serially positioned at multiple different epicardial sites, using multielectrode bands positioned along the AV groove, or may involve the production of detailed ventricular activation sequence maps using multielectrode arrays (175,176). The site of earliest ventricular preexcitation is determined during atrial pacing. The site of earliest retrograde atrial activation is then determined during orthodromic recriprocating tachycardia or ventricular pacing. Endocardial mapping is also sometimes performed following bypass when using a right atriotomy to approach right sided or septal pathways.

The endocardial approach was pioneered at the Duke University Medical Center and is the technique most widely utilized (7,9,177-184). This technique requires the use of cardiopulmonary bypass and cardioplegia. Left free wall pathways are approached via a left atriotomy. Right free wall and septal pathways are approached through a right atriotomy. For free wall accessory pathways, the technique involves making an endocardial incision slightly to the atrial side of the mitral or tricuspid valve at the site of the accessory pathway predicted by intraoperative electrophysiologic mapping and extending the incision considerably on each side of the predicted location. A plane of dissection is then carried all the way to the epicardium, being careful to avoid damage to the coronary arteries and coronary sinus running in the AV groove. The techniques for septal pathways are similar in principle, although anatomically more complex. The results of surgery for septal pathways have improved dramatically as experience has grown, facilitated by the use of continuous monitoring of the His bundle during the initial incision to avoid damage to this structure (9,178).

ENDOCARDIAL SURGICAL APPROACH

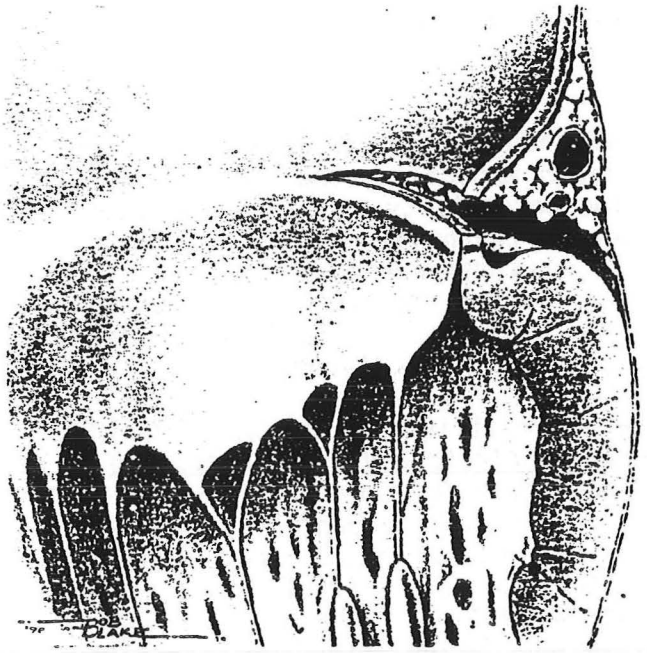


Fig. 6 Diagram of the plane of dissection used during the endocardial approach to accessory pathway division (177).

An epicardial approach for division of accessory pathways has recently been developed that utilizes a combination of surgical dissection and cryoablation (185-187). This technique has the advantage that it can be performed on the beating heart without the necessity of cardiopulmonary bypass and cardioplegia. The technique is not suitable for patients with anterior septal pathways, or occasional patients with "atypical" posterior septal locations, but is highly successful for other pathway locations (188,189).

Other recent innovations in surgical therapy include cryoablation from an endocardial location or via the coronary sinus (left sided pathways) with or without the use of cardiopulmonary bypass (190-192), and the use of an argon laser from an endocardial approach (193).

With the considerable surgical experience which has been accumulated, and with concomitant improvement in techniques, surgical division of an accessory pathway can now be accomplished with a mortality rate of less than 1 to 2% in patients without other structural heart disease, a success rate of 90% or more, and an incidence of complete heart block during operations for posterior septal pathways of less than 5% (194). Patients who undergo successful surgery often experience signifigant improvements in the quality of life (184).

C. Catheter ablation of accessory AV connections is a fairly recent development which is currently being intensively explored. It is attractive in that it offers an non-surgical alternative with the potential for definitive cure. Interest in the use of this technique for accessory pathway ablation has been encouraged by the success of catheter ablation or catheter modification of the AV junction, which has largely replaced surgical AV junction ablation (195-200).

Initial trials of this technique have utilized standard recording and pacing electrodes and standard defibrillators to deliver an electrical shock through the electrode tip at the site of accessory pathway location predicted by electrophysiology study. The result is what has been termed a small explosion at the electrode tip which traumatizes the adjacent myocardium by as yet poorly understood combinations of electrical and thermal injury and barotrauma (201,202), leading to the eventual fibrosis in the area of the lesion (203,204).

The feasibility of ablating left-sided and posterior septal pathways via a catheter placed within the coronary sinus was demonstrated in dogs (204-206). An initial series of 8 patients with left-sided pathways treated with this approach was disappointing, with accessory pathway conduction returning in all patients after the procedure (though modified in some), and one episode of cardiac tamponade requiring surgical drainage (207). Higher rates of success have been reported using the coronary sinus approach for posterior septal pathways (208-211), however, in the largest of these series, 3 of 19 patients required emergent surgery for cardiac tamponade, and one suffered a posterior myocardial infarction possibly related to spasm in the right coronary artery near the ablation site (211). The results of the Percutaneous Cardiac Mapping and Ablation Registry were recently reported (212). Of 26 patients reported to this registry (predominantly with posterior septal and left-sided pathways), successful ablation of the accessory pathway was reported in 58%, and there were 4 cases of cardiac tamponade, one fatal. Little detail of the techniques or complications were reported.

Very promising results demonstrating the successful ablation of accessory AV connections in any location have recently been reported (213-215). Warin, et al (214,215), achieved a success rate of 90% with relatively few long-term complications (4 cases of ventricular fibrillation-successfully defibrillated, 3 cases of pericarditis without effusion, 2 cases of persistent complete AV block) in their accumulated series of 70 patients. They avoided the use of ablation catheters within the coronary sinus or near its os, and instead approached left-sided pathways through a patent foramen ovale or with trans-septal catheterization.

A variety of new techniques are being developed which may further improve the results of catheter ablation. One technique which shows considerable promise uses radiofrequency current to selectively induce thermal injury (216-224).

Another approach is to design catheters whose electrodes have a larger surface area and more uniform electric fields, and as a result have a lower tendency for arcing (fulguration) to occur, permitting the delivery of more voltage to the tissue and less barotrauma (225,226).

Laser techniques utilizing "hot tip" thermal catheters and photoablation are also being investigated (227-231).

D. Antitachycardia pacing is a consideration in a small subset of WPW patients who do not respond well to medical therapy and who do not desire surgery. Since the premature atrial stimuli or atrial burst pacing used by most of these devices to terminate tachycardias can trigger atrial fibrillation, antitachycardia pacing should not be considered for patients capable of rapid antegrade conduction over the accessory pathway, and should only be considered for patients with poor antegrade accessory pathway conduction or concealed accessory pathways (232-238). The long term results with antitachycardia pacing are somewhat disappointing, with moderate to poor long-term success rates, and a high percentage of patients continuing to require medical therapy (232-234).

VI. Summary of Recommendations for Evaluation and Therapy.

The management of patients with preexcitation syndromes is currently an area of intensive investigation. Guidelines for the management of patients with the WPW syndrome have recently been proposed, although there continue to be many areas of debate (239,240).

A. Indications for electrophysiologic testing in the WPW syndrome:

1. Presentation with life threatening arrhythmia (e.g rapid preexcited atrial fibrillation) or sudden death

2. Medically refractory arrhythmias in a patient being considered for surgery, catheter ablation, or antitachycardia pacing

3. Patients with accessory AV connections who are to undergo cardiac surgery for other reasons (e.g. congenital heart disease)

4. To assess the response to pharmacologic therapy or other interventions

5. Assessment of patients with wide complex tachycardias of unknown type or associated with an irregular rhythm.

6. Assessment of tachycardia mechanism and appropriate therapy of patients with narrow complex tachycardia refractory to empiric therapy

7. Asymptomatic patients with a family history of sudden death

8. Assessment of risk in patients who participate in vigorous sporting activities, in airplane pilots, and in patients who operate mass transportation vehicles

Areas which are as yet ill-defined include the question of how vigorously to pursue risk stratification of WPW patients who are asymptomatic or who are symptomatic but easily controllable with medication. It would be inappropriate to tell these patients that they have a minor cardiac defect of no consequence,

due to the small but documented chance of sudden death in such patients. Non-invasive risk stratification can further help to define the patient as being in a high-risk or low-risk group but may miss a signifigant percentage of patients defined to be at increased risk by electrophysiologic testing. The induction of atrial fibrillation by intracardiac or esophageal pacing remains the most reliable means of predicting which patients are at risk for sudden death, although the predictive value of a short accessory pathway refractory period or a short RR interval during atrial fibrillation is fairly low. The decision as to whether or not to pursue invasive testing in this group must be made on an individual basis until these issues can be further clarified.

B. Indications for surgical therapy. Although medical therapy is the mode of treatment for most patients with the WPW syndrome, surgery offers the potential for life-long cure of the condition. The morbidity and mortality of surgery have greatly decreased as experience has grown. Surgery is the treatment of choice for patients who present with sudden death or rapid atrial fibrillation (shortest preexcited RR interval $\leq$250 msec). Surgery also represents an attractive option for many patients with symptomatic recriprocating tachycardias who are refractory to medical therapy. It may be considered as an alternative to possible life-long medical therapy by others, especially if electrophysiologic testing discloses an accessory pathway capable of rapid antegrade conduction. Asymptomatic individuals with WPW may be considered for surgery under special circumstances. These include work occupations which may not allow the presence of preexcitation on the ECG, and patients with a family history of sudden death (32), or in patients participating in vigorous athletics who have rapid antegrade accessory pathway conduction.

C. Indications for catheter ablation. It is too early to recommend catheter ablation as a routine alternative to surgical therapy, although the field is rapidly changing. At present, catheter ablation of posterior septal pathways is a reasonable alternative for patients who are surgical candidates but do not desire surgery, but carries an appreciable morbidity and only a moderate success rate. If results as favorable as those recently reported (213-215) can be duplicated, and with the development of new catheter techniques, catheter ablation will undoubtedly assume a larger role in the management of the WPW syndrome.

VII. Summary

The intensive investigative attention which has been recently been given to the WPW syndrome has resulted in dramatic improvements in our understanding of the tachycardia mechanisms and proper management of patients with this condition. A number of important issues remain regarding the identification and treatment of patients at risk for sudden death. We should be able to look forward to continued rapid improvement in the pharmacologic and non-pharmacologic treatment options for patients with this condition.

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