SPECIAL ARTICLE

Wolff-Parkinson-White Syndrome
The Problem, Evaluation, and Surgical Correction

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The Wolff-Parkinson-White syndrome holds a fascination for those interested in cardiac electrophysiology because it seems to represent a naturally occurring event which, if adequately understood, would unmask answers to many fundamental questions concerning mechanisms and treatment of cardiac arrhythmias.¹ ³ The major purpose of this essay is to review the results and certain implications of an ongoing study of patients with the Wolff-Parkinson-White (WPW) syndrome at Duke University Medical Center. The focus of this study has been the development and evaluation of surgical techniques for the correction of the syndrome. A second and equally important objective has been to develop and evaluate new and improved techniques for assessing cardiac excitation in these patients and the potential benefit of either medical or surgical therapy.

We have chosen to present this experience as an essay, in part because it is a story which seems to unfold naturally, and in part because our approach to these patients is still evolving. The essence of our point of view is that the classical Wolff-Parkinson-White syndrome results from anomalous excitation over accessory connections between the atrium and ventricle, that these connections can be identified and localized by electrophysiological techniques, and that the anatomic fault and resulting arrhythmias can be corrected, when the symptoms warrant, by surgical intervention.

During the past five years we have had an opportunity to study 68 patients who had the Wolff-Parkinson-White syndrome and attacks of arrhythmias which were sufficiently frequent or of such severity as to be disabling or life endangering. This cluster of patients with arrhythmias which were difficult to manage has heightened our awareness of the serious consequences which occur in a small proportion of the population with WPW syndrome and has provided a rational stimulus for more intensive study of the problem and attempts to develop new and/or improved methods of treatment. This group was composed of 51 men and 17 women. The range of age within the group was from eight to 71 years. All patients were admitted to our Clinical Research Unit for examination. The nature and extent of studies performed have been modified and expanded slightly over the five-year period. These studies included a history, physical examination, chest X-ray and fluoroscopy, ECG, and VCG and more recently a phonocardiogram and echocardiogram. Fifty-eight of the 68 patients underwent detailed electrophysiological studies. Thirty patients underwent cardiac surgery at which time detailed epicardial mapping was conducted in an effort to locate the accessory muscle bridge between the atria and ventricles that would subsequently be divided. Follow-up data on all medical and surgical patients were acquired by re-examination at six month intervals at Duke University Medical Center.

It is not a major purpose of this paper to review in detail the clinical features of the WPW syndrome but rather to concentrate on those features of the syndrome which were manifest in our own group of patients and to highlight aspects which we believe are either new or deserve emphasis. Anomalous excitation of the ventricles resulting in the WPW electrocardiographic abnormality is of course often detected on a routine ECG in subjects with either no history of arrhythmias or with only infrequent and inconsequential episodes of tachycardia. For this reason, the syndrome was regarded for many years as benign and without effect on life expectancy. Over-all it can still be said that most persons with WPW syndrome follow...
a benign course. In recent years, however, it has become clear that some patients have frequent and prolonged episodes of tachycardia which may be totally disabling. In addition, syncope and sudden death have occurred in some patients.\(^4\)\(^5\) Within our group of patients are many who experienced a disabling course.

**Clinical Characteristics**

The median age of our patients was 37, and the average duration of symptoms indicating paroxysms of tachycardia was 20 years. Approximately 70% of the patients had had disabling attacks of arrhythmia prior to the age of 20. Sixty patients (88%) had documented attacks of regular paroxysmal supraventricular tachycardia with heart rates ranging from 120–230 beats/min. In 37 patients, the ventricular rate during the tachycardia exceeded 180 beats/min and in 23/37 the tachycardia was accompanied by chest pain, congestive failure, or syncope. Twenty-seven of our patients (39%) had documented atrial flutter-fibrillation with anomalous ventricular excitation on some or all beats. In these patients, the ventricular rate during atrial fibrillation ranged from 180–360 beats/min. Seven of our patients were documented to have had ventricular fibrillation. In eight additional patients, atrial flutter with 1:1 A-V conduction was documented.

It is also of interest that several of the patients referred to us because of catastrophic arrhythmias had never before experienced such an attack. In one such patient, documented ventricular fibrillation (VF) occurred in a hospital after two hours of recurrent syncope. Another patient had occasional attacks of palpitation over several years, but then developed syncope due to atrial fibrillation with a ventricular rate of 200 beats/min which progressed into VF spontaneously during monitoring. Two other patients had experienced frequent episodes of paroxysmal atrial tachycardia (PAT) which were interrupted by vagal maneuvers. Both developed atrial fibrillation attended by chest pain and syncope. Subsequently, while being treated with cardiac glycosides, they developed ventricular fibrillation. Thus, the transition from a benign to a life-threatening course can be abrupt. In our view, a "benign" history in a patient with electrocardiographic evidence of pre-excitation does not necessarily imply a "benign" future.

**Definition and Classification**

Durrer, Schuilenburg, and Wellens have defined "pre-excitation" as a condition in which all or some portion of the ventricular muscle is activated earlier, in relation to atrial events, than would be expected had the impulse reached the ventricle by way of the normal atrioventricular conduction system.\(^1\) This definition obviously includes the classical Wolff-Parkinson-White anomaly, and it also includes other electrocardiographic abnormalities which may or may not have the same anatomic basis. The variant forms of pre-excitation include instances of a short P-R interval, but a normal QRS duration (Lown-Ganong-Levine syndrome), and those with a prolonged QRS complex and delta wave, but a normal P-R interval. There are many reasons to consider these variants as possibly related to classical WPW. Breckenmacher et al. recently described abnormal fibers completely bypassing the A-V node in a patient with Lown-Ganong-Levine syndrome.\(^9\) The variant forms, however, will not be considered further in this essay.

Subsequent to the description of the syndrome by Wolff, Parkinson, and White,\(^8\) many electrocardiograms which met the criteria for that syndrome were published. It became apparent that the morphology of the QRS complex was quite variable in patients with the syndrome, and in 1945, Rosenbaum and associates suggested a classification of WPW records into type A and type B.\(^10\) In type A, the delta wave was upright in precordial leads V\(_1\) and V\(_2\), and the remaining QRS forces were also directed in an anterior direction. In type B, the delta wave was negative in V\(_1\) and V\(_2\), and the remaining QRS forces were also directed predominantly in a posterior direction. As a first approximation, the classification is useful, and tracings typical of type A and type B are presented in the upper portion of figure 1. The reason for subgrouping patients with WPW is based on the presumption that the spatial orientation of the initial vector (delta wave) will provide some insight regarding the site of ventricular pre-excitation.

Figure 1 illustrates vectors which depict the direction of the first 0.03 seconds of the delta wave in 34 of our patients. The purpose of the illustration is to demonstrate that the spatial direction of these initial forces is quite variable. While patients can be classified into type A and type B using Rosenbaum's criteria, there are in fact many intermediate types and the picture is one of a continuum. This continuum is certainly consistent with the highly variable location of the accessory tracts responsible for anomalous excitation as will be discussed subsequently. Despite such variability, the anatomic studies which have been reported and the surgical results which will be discussed in this essay support the view that the direction of the delta forces provide an important and usually reliable index of the site of anomalous pre-excitation.

**Associated Congenital Disease**

Many reports of Wolff-Parkinson-White syndrome from clinics which see patients referred because of suspected heart disease note a high association
between WPW and several forms of congenital heart disease. When WPW and other forms of congenital heart disease exist in the same patient, the type of WPW may have some diagnostic value in predicting the nature of the associated abnormality.

Although two of the original 11 patients reported by Wolff, Parkinson, and White had cardiomegaly (one of these probably had hypertensive heart disease), none was thought to have congenital heart disease. In more recent articles it has been emphasized that 5–10% of patients with Ebstein’s anomaly have the WPW syndrome.11 Scheibler et al.12 found that 24 of 83 patients with congenital heart disease and WPW syndrome had Ebstein’s anomaly, and in a report by Świderski et al., four of 20 patients with heart disease and WPW had Ebstein’s anomaly.13 In addition, sporadic reports of the association between WPW and other forms of congenital heart disease have appeared including tricuspid atresia, corrected transposition of the great vessels, ventricular septal defect, atrial septal defect, and familial myocardial disease.14 In a recent report, Giardina et al. described 62 infants and children with the WPW electrocardiogram of whom 20 had associated congenital heart disease.15

Our group of patients is of considerable interest from the point of view of associated anomalies. Six of our patients had Ebstein’s anomaly of the tricuspid valve, and four of these six had an atrial septal defect of the secundum type documented by cardiac catheterization. Another patient had a secundum ASD without any abnormality of the tricuspid valve. Four patients in our group had cardiomyopathy of the non-obstructive type and one patient eight years of age had an ejection-type murmur at the left sternal border and an echocardiogram which met criteria for asymmetric septal hypertrophy.16 In addition, seven of our patients had a midystolic click with or without an apical murmur, and ballooning of the posterior mitral leaflet was documented by echocardiogram, by left ventriculogram, or both. All of our patients with Ebstein’s anomaly had a type B electrocardiogram while all with the balloon mitral valve had type A electrocardiograms. Other coincidental anomalies included: RBBB (2); sick sinus syndrome (3); coronary artery disease (4); mitral valve disease due to rheumatic fever (2).
James has argued\textsuperscript{17} that the association of Ebstein's anomaly with the WPW syndrome does not favor lateral connections between the right atrium and right ventricle as a basis for pre-excitation in such patients. However, an association of WPW with Ebstein's anomaly is apparent and points to the possibility that both conditions may result from some structural fault that occurred during the development of the tricuspid ring. The finding that 90% of the reported cases of this association have had a type B electrocardiogram points to something more than a mere chance relationship. Moreover, the electrocardiogram in most cases not only has been classified as type B, but has demonstrated large negative delta forces in leads II, III and aVF with either horizontal or left axis deviation.\textsuperscript{18} These findings suggest that the area of pre-excitation is along the inferior margin of the right ventricle. An association of the WPW syndrome with ballooning of the posterior mitral leaflet has not to our knowledge been reported previously. Seven of our patients had this association, and as noted above, all had a type A electrocardiogram.

\textbf{Anatomic Findings}

In 1932, Holzmann and Scherf\textsuperscript{19} first postulated that one or more muscular bridges between the atrium and ventricle, similar to those described by Paladino\textsuperscript{20} in 1876 and by Kent in 1893,\textsuperscript{21} might account for the WPW syndrome. The first direct evidence for such an accessory bridge in a patient with WPW syndrome was provided in 1943 by Wood, Wolferth, and Geckeler.\textsuperscript{22} These authors identified three muscular bridges laterally, connecting the right atrium and ventricle in a 16-year-old boy who had WPW with attacks of paroxysmal tachycardia and an ECG which showed a delta vector directed to the left and posterior, compatible with type B. In 1944, Öhnell described a muscular bridge between the left atrium and left ventricle.\textsuperscript{23} In 1971, Rosenberg and his colleagues presented two cases of WPW syndrome and co-existing congenital heart disease.\textsuperscript{24} In addition to their two cases, they reviewed the literature and summarized the anatomic findings in 11 other cases of WPW syndrome. Of the total of 13 cases, eight had accessory connections from the right atrium to right ventricle, one was from left atrium to left ventricle, two had bilateral connections and two had septal connections. In 1972, Lunel reported two additional cases of the WPW syndrome and gave detailed postmortem studies.\textsuperscript{25} One patient who had demonstrated both type B and type A complexes in life had numerous muscular connections between the right atrium and right ventricle, as well as between the interatrial and interventricular septum. The second patient, with type B WPW on the basis of a single ECG, had parietal connections on both sides of the heart as well as in the septum. In 1973, Mann et al. reported a 15-year-old boy with recurrent episodes of tachycardia and type A WPW who died suddenly.\textsuperscript{26} At autopsy two separate muscle bundles were found connecting the left atrium and left ventricle approximately 2 cm lateral to the interventricular groove. In 1974, Brechenmacher et al.\textsuperscript{8} reported a patient with type A in whom three abnormal bundles were found linking the left atrium and left ventricle posteriorly. Finally, James has recently described a patient with WPW type A with an anomalous A-V pathway located just beneath the left atrial appendage in the left ventricular free wall.\textsuperscript{27} The available anatomic studies appear to lend credence to the hypothesis that embryological faults in partitioning of the atrium from the ventricle, with persisting muscular bridges passing through the faulty ring, underlie many if not most cases of WPW syndrome.

\textbf{Associated Coronary Disease}

Well over 80% of the reported cases of the WPW syndrome have been described in patients with either no associated heart disease or as an incidental finding in patients with coronary or hypertensive heart disease. The occurrence of WPW in patients with coronary disease is worthy of mention. First, the delta wave abnormality may simulate myocardial infarction by producing deep broad Q waves and it may produce a broad R in V\textsubscript{1} and V\textsubscript{2}. Subtle degrees of pre-excitation may also produce secondary ST-T wave abnormalities which mimic ischemic changes. On several occasions we have found it useful to suppress pre-excitation with atropine in order to obtain a "normal" electrocardiogram for diagnostic purposes or preliminary to exercise stress testing in patients with chest pain. In addition, we have also seen patients with WPW and no prior history of arrhythmia develop myocardial infarction. In that setting, ventricular ectopic beats, presumably secondary to the infarction, initiated frequent and sometimes catastrophic bouts of supraventricular tachycardia. Thus, the WPW syndrome may mask or mimic the diagnostic criteria for ischemic heart disease, and when the two exist in the same patient, the WPW syndrome may render the patient vulnerable to arrhythmias which would either not occur, or occur less frequently in the absence of WPW.

\textbf{Electrophysiological Evaluation}

Over the past five years we have developed a protocol for the study of patients with the Wolff-Parkinson-White syndrome. Although not all patients were subject to this protocol, particularly prior to 1972, it seems appropriate to present the results of our
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studies in accord with the questions which the current protocol is designed to answer. Hopefully, this approach will be useful to others interested in the study of these patients.

a) Is pre-excitation present and is ventricular activation anomalous, i.e., does it occur over some pathway other than the His bundle?

To answer this question in the affirmative, we have sought to demonstrate that the onset of the delta wave on the surface electrocardiogram either occurs, or can be made to occur, before the rapid deflection which represents local activity recorded from the bundle of His. This observation, in conjunction with pacing the bundle of His, also excludes the possibility that appearance of the delta wave actually represents depolarization of Mahaim fibers arising from the His bundle. In all of our patients with the WPW syndrome who underwent His bundle studies we succeeded in demonstrating this phenomenon. In several patients it was difficult to record a discrete His bundle electrogram when the degree of anomalous excitation was marked. We found the use of electrode catheters with contacts separated by 3–5 mm rather than the usual 1 cm was helpful in these cases.

b) Is the ventricular complex observed in WPW during periods of sinoatrial rhythm the result of fusion, with the initial phase of ventricular activation representing excitation via the accessory pathway and later forces produced by excitation of residual portions of the ventricles via the His-Purkinje system?

To answer this question in the affirmative we have sought to demonstrate that the degree of pre-excitation could be increased either by prolonging conduction time through the normal A-V node by pacing at progressively faster rates or by pacing from a site which is closer to the presumed accessory pathway than to the sinoatrial (SA) node (fig. 2). In the majority of our patients we have been able to increase the degree of anomalous excitation by one or both of the techniques noted above. In each case, an increase in the degree of anomalous excitation at faster heart rates was accompanied by a prolongation of the interval between the stimulus and the His bundle electrogram (presumably due to A-V nodal delay) without a concomitant change in the interval between the stimulus and the onset of the delta wave. In the seven patients who failed to demonstrate this phenomenon, one of the following explanations seemed to fit: in three patients, the interval between the stimulus and the His complex failed to lengthen with an increase in heart rate. This phenomenon has been reported in some patients with Lown-Ganong-Levine syndrome, and presumably represents a functional difference within the A-V node or an anatomic bypass of the portion of the A-V node that would normally be delayed.

In all three of these patients, the A-H interval during sinus rhythm was short (i.e., less than 60 msec). In the remaining four patients, anomalous excitation of the ventricles disappeared with increasing heart rate due to block in the accessory pathway. In each of these four patients, the effective refractory period of the accessory pathway was subsequently shown to be long (i.e., greater than 500 msec). Another potential reason for failure to increase pre-excitation with pacing at higher heart rates would be coexistent A-V nodal conduction delay. Such patients would already be fully pre-excited, even at basal heart rates.

We have increased the degree of pre-excitation in several patients with type B tracings by pacing the low lateral right atrium, though this finding was not looked for until recently. We have compared to a much more systematic manner the effects of right and left atrial pacing. As observed by others, the degree of anomalous excitation in patients with type B WPW was usually greater with right atrial pacing than during left atrial pacing. In none was the degree of anomalous excitation greater during left atrial pacing. These observations were in marked contrast to those in patients with a type A electrocardiogram. In

![Figure 2](http://circ.ahajournals.org/)

**Figure 2**

Effect of pacing site on the degree of pre-excitation. Pacing of the right atrium results in greater pre-excitation than pacing of the left atrium (at the same cycle length) in the presence of RV pre-excitation, while the opposite is true of LV pre-excitation.
each patient with type A WPW, it was possible to enhance the degree of anomalous excitation by left atrial pacing. In each case, this was accompanied by a shorter interval between the stimulus and the onset of the delta wave (when left atrial pacing was compared to right atrial pacing), with little or no change in the interval from the stimulus to the His complex. We feel that this is presumptive evidence for left ventricular pre-excitation, as suggested by others. In cases of septal accessory pathways, changing the site of atrial stimulation did not alter significantly the degree of pre-excitation. In general, we have interpreted the observations noted above as consistent with the view that the morphology of the QRS complex in most patients with WPW is a consequence of fusion.

c) **What is the effective refractory period (ERP) of the accessory atrioventricular pathway and how does it contribute to the genesis of arrhythmias?**

In 1967, Durrer and his colleagues published a report of four patients in whom atrial and ventricular premature beats were induced at progressively earlier times during the diastolic interval to measure the refractoriness of the accessory bundle. During antegrade conduction, the refractory periods ranged from 270–365 msec. Recently, Wellens and Durrer reported more extensive experience and compared the refractory periods of the accessory pathway during antegrade and retrograde conduction in the same patients. A wide range of antegrade refractory periods was noted by Wellens, as well as significant differences between antegrade and retrograde measurements. A histogram of the antegrade refractory periods obtained in 44 of our patients during either sinus rhythm or atrial pacing at rates just in excess of the sinus rate is presented in figure 3. The wide range of values in these 44 patients is noteworthy, as is the exceedingly short refractory period observed in some patients.

The clinical significance of a short antegrade ERP of the accessory pathway is that it may constitute a basis for a very rapid ventricular response should either atrial flutter or fibrillation develop. In this regard, it is noteworthy that 20 of the 68 patients in this series had documented atrial fibrillation at least one time in their clinical course. The antegrade ERP should determine the maximum rate at which the accessory pathway will conduct impulses to the ventricle during atrial fibrillation. This relationship was confirmed recently by Castellanos et al. A potential limitation to the validity of findings based on this technique is the dependence of the effective refractory period on cycle length. Shortening of the ERP might occur with the decrease in cycle length which accompanies atrial fibrillation and thus allow a more rapid ventricular response than would have been anticipated based on measurements at a slower heart rate. For this reason, we determine the ERP of the accessory pathway at several cycle lengths, including a cycle length comparable to one recorded during any documented PAT. In 14 of our patients in whom detailed measurement of the ERP was made at multiple cycle lengths, the ERP decreased in nine (maximum 45 msec), was unchanged in three, and increased in two (maximum 35 msec). It should also be noted that the determination of the ERP of an accessory pathway should be made with right atrial pacing in patients with a right-sided connection and with left atrial pacing in cases of left-sided pre-excitation to exclude the possibility of interatrial delay as a source of measurement error.

In our studies, the correlation of the shortest antegrade ERP of the accessory pathway and the cycle length of the ventricular response during atrial fibrillation (r = 0.74) has not been as strong as that reported by others. Long ERPs have been associated with moderate rates of ventricular response, while short ERPs have been associated with rapid rates. However, some patients with short ERPs developed only moderately fast ventricular rates during atrial fibrillation with most beats conducting normally. The explanation for this may lie in the phenomenon of concealment in the accessory pathway. Because of this, all patients with a short ERP of the accessory pathway are subjected to atrial fibrillation induced by rapid atrial stimulation. To date it has been necessary to perform a DC cardioversion following induction of atrial fibrillation in only three instances.

We have only recently begun to compare the refractory period of the accessory pathway during both
antegrade and retrograde conduction and have confirmed that the antegrade ERP of the accessory pathway is usually (nine of 12 patients) longer than the retrograde ERP at comparable cycle lengths. When the retrograde ERP of the accessory pathway (determined at a cycle length comparable to PAT) is shorter than the cycle length of PAT, the accessory pathway can be implicated in the development and maintenance of circus movement tachycardia. This relationship was confirmed in all patients in whom these parameters were studied.

In agreement with the work of others, we have consistently found that episodes of supraventricular tachycardia utilizing the bypass tract can be induced when the antegrade refractory period of the accessory pathway is longer than that of the A-V node, and when premature beats either of atrial or ventricular origin are initiated during the interval or “window” separating these refractory periods. Such appropriately timed premature beats dissociate the two pathways and evoke a re-entrant rhythm. To demonstrate the potential for such dissociation, it is sometimes necessary to introduce premature beats from several different sites (RA, LA, RV, LV) at several different basic driving cycle lengths.38

d) Where is the accessory pathway located?

Published anatomic studies have demonstrated accessory muscular bridges between the right atrium and ventricle or between the atrial and ventricular septae in the hearts of patients who had a type B electrocardiogram and have found left-sided connections when the patients had type A electrocardiograms. In our operated series of 30 patients, the electrocardiogram was useful as a first approximation of the site of pre-excitation but did not allow discrimination of free wall accessory pathways (18 patients) from septal accessory pathways (11 patients). Additional studies are thus required to predict the site of the accessory pathway prior to surgical intervention.

As noted above, a comparison of the degree of anomalous excitation during right and left atrial pacing can be helpful. Left and right ventricular pre-excitation can be further distinguished by recording activity from the base of the left ventricle during anomalous excitation via a catheter electrode in the distal coronary sinus.39 Such records have consistently revealed left ventricular activity closely coinciding with the onset of the delta wave in patients with type A WPW and left ventricular pre-excitation confirmed at surgery. In patients with type B WPW, activity from the base of the left ventricle was recorded well after initiation of the delta wave and usually during the terminal phases of the QRS complex. Distinguishing left from right ventricular pre-excitation on this basis is reliable only during maximal pre-excitation.

The site of pre-excitation is also indicated by the pattern of retrograde atrial activation during supraventricular tachycardia.40–42 Since atrial activation in this situation proceeds from the point of insertion of the accessory pathway into the atrium, identification of the earliest point of retrograde atrial activation furnishes a presumptive location of the pathway. Routinely, simultaneous electrograms from the low lateral right atrium (RA), the low septal RA (via the His bundle catheter), and the lateral left atrium (LA) (via a coronary sinus catheter) are recorded during PAT (fig. 4) as well as during determination of ventricular refractory periods. More detailed localization is obtained by mapping of atrial activation at the level of the tricuspid valve by a special preformed electrode catheter. Similar recordings are obtained from most of the circumference of the mitral valve by continuous recording during withdrawal of the coronary sinus catheter from a distal position back to the orifice. It has been our experience in normal subjects, as well as those with paroxysmal supraventricular tachycardia due to re-entry in the A-V node, that earliest retrograde atrial activation occurs on the low septal RA simultaneous with or followed closely by activation of the os of the coronary sinus. In patients with anomalous A-V connections between the atria and ventricles, retrograde excitation of the atria over the accessory pathway results in eccentric atrial depolarization during PAT and results in a fusion of atrial depolarization, resulting from normal and AP input, during ventricular pacing.

Septal accessory pathways constitute a special problem, since in this situation, the pathway may be close to the normal conduction system. We have found that simultaneous recording of electrograms

Figure 4
Retrograde atrial activation during paroxysmal atrial tachycardia (PAT) in patients with RV, septal, and LV pre-excitation. Recordings from above down in each panel are ECG lead V1, with bipolar electrograms recorded from the low lateral right atrium (LRA), the His bundle electrogram (HBE), and lateral left atrium (LA). In RV pre-excitation, retrograde atrial activation is earliest in the LRA. In septal pre-excitation, the medial RA as recorded on the HBE is earliest. In LV pre-excitation, the LA activity is earliest.
from the medial right atrium and the orifice of the coronary sinus is useful. In patients with documented septal connections anterior to the His bundle, activation from the medial RA precedes that from the os of the CS, while in septal connections posterior to the His bundle, activation of the os of the CS is simultaneous or slightly earlier than that from the medial RA. Special efforts must be taken in the latter situation to exclude the possibility that conventional A-V nodal re-entry is present. Finally, this method has allowed us to identify an accessory pathway in two cases with PAT which had no evidence of antegrade pre-excitation during sinus or paced rhythms.

Thus the standard electrocardiogram, when combined with electrogrograms during atrial pacing, recording of electrograms from the coronary sinus during anomalous excitation, and determination of the site of initial atrial retrograde excitation during supraventricular tachycardia, can be used to determine the site of anomalous excitation in patients with the WPW syndrome.

e) Is the accessory pathway used during episodes of tachycardia?

The most common arrhythmia in patients with the WPW syndrome is paroxysmal supraventricular tachycardia. A logical basis for therapy, whether pharmacological or surgical, rests upon a clear demonstration that the accessory pathway which is responsible for anomalous excitation during sinus rhythm also participates in the genesis of the tachycardia. There is no a priori reason why patients who have anomalous excitation should be any less susceptible to arrhythmias of traditional causes than the population at large. In the laboratory, paroxysmal supraventricular tachycardia can be induced in patients with the WPW syndrome by appropriately timed premature atrial or ventricular beats. The subsequent rhythm is called ‘‘PAT’’ when the morphology of the QRS is normal for that patient (i.e., no delta wave), each QRS complex is preceded by a His bundle deflection at an interval of at least 30 m sec, and each QRS complex is associated with a single atrial depolarization. The chief problem is to distinguish this rhythm from a reciprocating mechanism within the A-V node.

In most patients with WPW, the initiation of tachycardia is uniquely related to block of a premature beat in the accessory pathway (AP). When the ERP of the AP is long relative to the ERP of the A-V node, these initiating beats may propagate through the A-V node with little or no delay. This is in contrast to the situation of re-entry within the A-V node where conduction delay is a requisite condition for re-entry. Thus, the finding that PAT can be induced by a premature beat which conducts with no antegrade conduction delay suggests that a bypass tract may be involved in the re-entry circuit. On the other hand, induction of PAT at critical A-H intervals, or achieved by pacing-induced A-V nodal Wenckebach suggests A-V nodal re-entry as the mechanism.43

Important differential observations can be made during ventricular pacing and premature ventricular stimulation. The V-A conduction time during ventricular pacing (at a cycle length comparable to that of observed tachycardia) should be comparable to the V-A interval during spontaneous tachycardia. Likewise, ventricular premature depolarizations (VPDs) which induce tachycardia should: a) be associated with V-A intervals comparable to those of control beats and b) have the same sequence of retrograde atrial activation as observed during tachycardia. Both of these observations require that measurement of V-A intervals be made using recordings at an atrial site close to the bypass. The latter can be obtained by the multiple recording technique alluded to above. Where local electrograms from locations near the bypass are not available, measurement of the V-A intervals is less meaningful.

Another observation which favors participation of the AP is the demonstration of eccentric retrograde atrial activation during tachycardia. When retrograde atrial activation is near the atrial septum, it is difficult to distinguish retrograde AP conduction from retrograde conduction over the His-A-V node system. In the latter situation, two further observations favoring use of an AP are pivotal: a) the demonstration that early VPDs can conduct retrogradely to the atria with a ‘‘septal’’ activation sequence without intervening conduction over the His-A-V node (i.e., retrograde His appears after atrial depolarization), and b) demonstration that VPDs can be induced during PAT which retrogradely pre-excite the atria without disturbing the preceding His deflection.

Finally, slowing of PAT associated with the appearance of a bundle branch block suggests that a bypass tract located in the ventricle showing evidence of block is participating in the PAT.44 This slowing of the cycle length of tachycardia must be due to prolongation of the V-A interval to be significant. Failure of bundle branch block to lengthen the cycle length suggests the existence of a septal pathway or A-V nodal re-entry.

The other important and not uncommon dysrhythmia encountered in the WPW syndrome is atrial flutter-fibrillation with a rapid ventricular response. Here the AP is more readily incriminated, since the rapidly conducted beats manifest pre-excitation if conducted over the AP. Of note is the finding that of the 15 patients with either ventricular fibrillation or atrial flutter with 1:1 conduction, 14 were found to have an AP in the left ventricle or in-
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terventricular septum. Of these 15 patients, six would have been categorized electrocardiographically as type B, in contrast to a previous report suggesting that atrial flutter-fibrillation was unusual in patients with type B WPW.45

Whenever episodes of supraventricular tachycardia (SVT) are initiated by beats which are followed by "echo" responses compatible with A-V nodal re-entry or when one cannot establish V-A conduction with appropriate V-A intervals at cycle lengths comparable to those found during tachycardia, the participation of the accessory pathway should be seriously questioned and alternative explanations sought. In our 68 patients, only one failed to meet these criteria and appeared to have re-entry within the A-V node.

Therapy

Drug Therapy

Paroxysmal tachycardia in the WPW syndrome occurs when at least three conditions are satisfied: a) the episodes of tachycardia are initiated by a premature beat which may be atrial, junctional, or ventricular in origin, b) the premature beat must occur at a time when conduction over the A-V node and bypass can be dissociated, and c) after the premature beat is conducted over one pathway from its chamber of origin, the other pathway must be excitable to permit initiation of circus movement. In the classic situation a premature atrial beat blocks antegrade in the Kent bundle and is conducted to the ventricle across the A-V node. On reaching the ventricle it then returns retrograde over the Kent bundle to the atrium initiating circus movement. Pharmacological therapy is aimed at modifying one or more of these links in the circuit: a) by reducing the number of premature beats, b) by narrowing the "window" or interval in which premature beats can dissociate the two pathways and c) by prolonging refractoriness so that the returning impulse blocks either in the accessory pathway or the A-V node. To our knowledge, there are no data which demonstrate the effect of pharmacological agents on the frequency of spontaneous premature beats in patients with WPW. It is clear from our studies and those of others46,47 that digitalis and propranolol either singly or in combination will prolong refractoriness of the A-V node and either reduce or abolish the window created by discrepant refractory periods in the two pathways. We have demonstrated this action in several patients and have utilized this approach in the prophylactic treatment of paroxysmal tachycardia with considerable success.

In other patients with the Wolff-Parkinson-White syndrome, the refractory period of the accessory bundle is short and presumably shorter than that of the A-V node. In such patients, single APDs do not initiate episodes of supraventricular tachycardia because, even though the two pathways may be dissociable, concealed conduction of the APD in the A-V node appears to block the return path to the atrium after anomalous excitation of the ventricle over the Kent bundle. On the other hand, in patients with a very short refractory period of the accessory bundle, it is frequently possible to initiate episodes of supraventricular tachycardia by appropriately timed ventricular premature beats. In such patients, the premature ventricular beat propagates to the atrium over the accessory pathway and returns to the ventricle over the normal A-V node and His-Purkinje system, thus initiating an episode of supraventricular tachycardia. We treat such patients with propranolol and procainamide/quinidine. Procainamide was selected because we have found, in agreement with a recent report by Wellens,48 that it usually prolongs antegrade refractoriness in the accessory pathway. Propranolol has been used because of its demonstrated ability to prolong refractoriness in the A-V node and thus reduce the likelihood that an early PVD which propagates to the atrium via the accessory bundle will be able to return to the ventricle via the A-V node. In an occasional patient, a paradoxical increase in frequency of PAT had been noted following institution of therapy with procainamide, presumably due to prolongation of the antegrade refractory period of the accessory pathway with resultant widening of the "window."

An important feature of all the patients we have studied with atrial fibrillation and a rapid ventricular rate due to conduction over the accessory bundle has been a short antegrade refractory period of the accessory bundle. Most of these patients have undergone surgery. In several, however, we evaluated the effects of procainamide on refractoriness in the accessory pathway and on the ventricular rate during induced episodes of atrial fibrillation. In some, but not all of these patients, procainamide prolonged the antegrade refractoriness of the accessory pathway when administered in 50 mg boluses every two minutes to a total of 10 mg/kg. Other investigators have reported that propranolol has little or no effect on refractoriness on the accessory pathway,46 and that digitalis glycosides may actually shorten it.47 The best current interpretation of these observations is that agents such as procainamide or quinidine should be used in patients with the WPW syndrome and atrial fibrillation and that digitalis may be contraindicated. Further studies are needed, however, before the actions of pharmacological agents on the accessory pathway are fully clarified.

Implantation of Pacemaker

The circus movement model of supraventricular tachycardia in patients with the WPW syndrome
suggests that it should be possible to terminate episodes of tachycardia with an appropriately timed premature atrial or ventricular beat. An APD should interrupt the tachycardia if it renders the atrium refractory to the impulse returning from the ventricle via the accessory bundle, yet is itself sufficiently premature to block in the A-V node. Similarly, a ventricular premature depolarization should interrupt the tachycardia if it renders the ventricle refractory to the next impulse arriving via the His-Purkinje system, yet itself is sufficiently premature to block in the accessory bundle. In 1967, Durrer et al.26 reported that in patients with type B WPW tachycardias could be easily terminated by single electrically induced right atrial or right ventricular premature depolarizations. In 1971, Wellens et al.27 reported that in patients with type A WPW, episodes of tachycardia could be easily terminated by delivering single appropriately timed premature stimuli to either the left atrium or left ventricle.

The observations noted above suggested to us that an implantable pacemaker could be used to terminate episodes of tachycardia. Five of our patients gave a history of recurrent episodes of PAT which had not been satisfactorily controlled with medication. Although these attacks were of only moderate frequency, each of the patients had had extreme difficulty in terminating the attacks and in fact had undergone several cardioversions after medical measures failed to interrupt the tachycardia. All five patients had type A WPW and were seen before we were reasonably confident that accessory bundles on the left side of the heart could be localized and surgically divided. For these reasons we decided to insert a pacemaker. In each of the patients it was determined at the time of the preoperative electrophysiological study that a single appropriately timed premature depolarization would consistently terminate episodes of tachycardia. Pacing was equally effective from the right atrium, right ventricle, or left atrium in one patient. In the remaining four, episodes of tachycardia could be easily interrupted with left-sided pacing, but not from the right side. Accordingly, one patient had a transvenous right ventricular pacemaker, three had transvenous coronary sinus pacemakers and one had a transthoracic left ventricular pacemaker. In each case the rationale was to provide the patient with a unit which could be turned on in a fixed rate mode when episodes of tachycardia occurred, and that when an appropriately timed capture beat occurred, the tachycardia would terminate. This therapeutic goal was achieved in all five patients.

Surgical Treatment

Despite scattered reports of successful and unsuccessful attempts to surgically divide an accessory A-V bridge in patients with WPW,48-56 the role of this procedure and its potential long-term benefit remains to be clarified. Our experience with the surgical approach is substantially greater than any other which has been reported, but it is still small and in the formative stage. That experience is sufficient, however, to indicate that surgical intervention clearly offers the potential for correction of WPW syndrome and it is also sufficient to form the basis for useful lessons worth sharing.

In 1967, stimulated by the reports of Durrer26 and Burchell49 which demonstrated anomalous excitation of the right ventricle in type B WPW, we performed epicardial mapping in a patient with type B WPW and successfully divided a right lateral bundle of Kent.50 The operation was successful and abolished both the delta wave and episodes of tachycardia. That patient has continued to do well and has had no further episodes of tachycardia in six years of follow-up.

In the original description, epicardial mapping was performed to define the area of earliest epicardial activation. The right coronary was then dissected free in the region of the presumed tract, and a transmural incision was made through the A-V ring, while the patient was on cardiopulmonary bypass. This procedure proved less than optimal for left-sided connections, however, where problems with hemostasis were encountered due to the many tributaries to the coronary sinus and the difficulty of dissecting the distal circumflex coronary. A new technique67 which utilizes an intra-atrial incision has been developed and applied to the last 21 consecutively operated patients. This approach circumvents many of the above technical problems and allows a more complete division of the A-V ring in areas not accessible from the external approach previously used.

After cannulation for cardiopulmonary bypass, epicardial mapping is performed.40,41,49,50,54-68 To achieve maximal pre-excitation, the atrium is paced at a site close to the bypass tract. The placement of this pacing electrode is determined on the basis of preoperative studies and by slowly passing a handheld stimulating probe around the A-V rings until maximum pre-excitation is noted. A plaque electrode is also sutured to the ventricle as close as possible to the presumed site of pre-excitation. This latter electrode is used as a reference for timing and for ventricular pacing. We routinely display oscilloscopically and record on tape a surface ECG wave demonstrating a pronounced delta wave, the bipolar reference, and the unipolar and bipolar recordings from a handheld bipolar electrode with terminals 1 mm apart (fig. 5).

An analog computing device continually displays in digital form the interval (in msec) between the reference and bipolar recordings from the probe and
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EARLY AREA

**Figure 5**

Area of earliest epicardial activation data in a case of a free wall accessory pathway. Tracings from above down are ECG lead II, a bipolar ventricular reference, and bipolar and unipolar data from the point of earliest epicardial activation. Note that during pre-excitation (before incision), the earliest activation occurs 15 msec before the onset of the surface delta wave. After incision of the accessory pathway, this point now occurs late in the QRS.

**Figure 6**

Earliest epicardial activation data in a case of septal pre-excitation. The earliest activation recorded from the epicardial surface occurs 10 msec after the onset of the surface delta wave.

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greatly facilitates on-line data acquisition. The delta-reference interval is carefully measured, so that data can be referred on-line to the delta wave. Periodically, the delta-reference interval is checked to ensure that it is stable and not influenced by possible fusion. Activation is then explored by measurement of approximately 55 epicardial sites, and the data plotted on drawings on the heart. In addition, a continuous sweep is made of the entire circumference of the A-V ring with beat-by-beat measurement of the activation times.

By the process just described, a point of earliest epicardial activation is identified. Correlation between the timing of this event and that of the delta wave in the conventional electrocardiogram has proved valuable in localizing the accessory pathway. In patients with free wall A-V connections, the earliest epicardial activation occurred before the onset of the delta wave (fig. 5), while in patients with septal connections, the area of earliest epicardial activation occurred 5–15 msec after the onset of the delta wave (fig. 6). In patients with A-V connections in the free wall, the area of earliest ventricular epicardial excitation identifies the site at which the surgeon should carry out the dissection of the A-V ring.

After completion of the antegrade map, the ventricle is then paced through the ventricular electrode and adjacent ventricular and atrial points around the A-V ring are mapped to determine the earliest point of retrograde atrial excitation. In every case of a free wall connection the site of earliest retrograde atrial excitation during ventricular pacing or PAT has corresponded closely to the site of earliest ventricular activation during atrial pacing when maximal pre-excitation was present. If further localization of the site of the accessory pathway is required, intra-atrial activation (during ventricular pacing or PAT), or intraventricular activation (during sinus rhythm or atrial pacing) can be performed after the patient is placed on cardiopulmonary bypass.

A median sternotomy is used to obtain exposure in
cases where the anomalous bundle is thought to lie on the right side or in the septal region, based on preoperative recordings. Approach for suspected left lateral connections is via a left lateral thoracotomy. Cardiopulmonary bypass is then instituted, an atriotomy performed, and the point on the annulus fibrosus bracketed by the earliest ventricular and atrial points identified during antegrade and retrograde mapping respectively is identified. The heart is fibrillated, and an incision made through the atrial wall just at its cephalad attachment to the annulus. By taking advantage of a large and consistently present fat pad beneath the coronary vessels, the surgeon can define a safe plane of dissection which allows complete division of atrium from ventricle on either the tricuspid ring, from the right trigone clockwise to the coronary sinus, or on the mitral ring, over the entire circumference of the valve save the small portion between the right and left trigones (corresponding to the attachment of the aortic ring). As soon as the plane of dissection is developed, the heart can be vented and defibrillated, and the electrocardiogram monitored as the incision is lengthened. The incision has usually been at least 2 cm in length. When the electrocardiogram normalizes, the area in which early activation had previously been present is reexamined. If that area is found to be late, incisions in the annulus and atrial wall are closed, and cardiopulmonary bypass terminated. The entire sequence of antegrade and retrograde mapping is then repeated before the cannulas are removed to insure that pre-excitation has been abolished and that no pathways remain.

To date, 30 patients have undergone epicardial mapping and attempts at surgical resection. The illustrative examples that follow have been chosen to demonstrate the spectrum of sites of pre-excitation and their electrocardiographic manifestations. Several general comments are in order regarding this relationship:

1) Relationship of ECG to the area of epicardial pre-excitation.

Patients whose ECGs present predominantly anteriorly directed forces in V1 during maximal pre-excitation were found to have an area of epicardial pre-excitation on the left ventricular free wall or immediately to the left side of the crux, while those patients whose ECGs showed predominantly negative complexes in V1 were found to have an area of epicardial pre-excitation on the right ventricle overlying the anterior interventricular septum, the free wall, or just to the right of the crux posteriorly. It should be noted that the term "crux" in this context is defined as the posterior border of the interventricular septum as determined by palpation, and subsequently by direct visualization. It is important to note that the posterior descending coronary was not a reliable landmark for locating the septum itself (This may explain the unusual finding in the case reported by Lister et al.89 Mapping a limited number of points along the posterior sulcus, they found early activation to the left of the posterior descending in a patient with type B WPW.). Patients exhibiting similar electrocardiograms were found to have similar areas of epicardial pre-excitation.

2) Relationship of area of epicardial pre-excitation to location of the accessory pathway.

When the area of epicardial pre-excitation was localized to the free wall of either ventricle, pre-excitation was generally abolished by incising the atrial muscle at its attachment to the annulus fibrosus underlying this point. However, pre-excitation overlying the anterior or posterior interventricular septum could result from an accessory pathway located within the adjacent free wall or the septum itself. When the earliest epicardial data recorded occurred after the onset of the delta, a septal location was strongly suspected.

3) Localization of the anatomic site of the accessory pathway from the electrocardiogram.

Because of the observations noted above, it was not unexpected that difficulty would be encountered in any attempt to directly relate the ECG to a unique anatomic site. Thus, of five patients whose ECGs showed completely negative forces in V1 (type B), two were found to have an AP in the septum (s), one in the posterior RV free wall and two in the lateral RV free wall. Among the 16 patients predominantly positive in V1 (type A), however, 13 were found to have a distinctive pattern associated with a left lateral free wall connection. During maximal pre-excitation, all had a negative delta wave present in leads I and aVL. Septal pre-excitation in 11 patients was associated with forces ranging from negative to isoelectric to completely upright in V1. A very good prediction of the presumptive site of the accessory pathway was obtained when the ECG was interpreted in conjunction with the preoperative electrophysiological studies previously described.

Because of the limitations of the ECG alone as a basis for locating the AP, we will refer to the localization of the pathways for the examples to follow as RV (free wall), septal, and LV (free wall).

Illustrative Surgical Cases

Figure 7 shows a 12 lead ECG from a 17-year-old male with refractory PAT. Note the positive delta in leads I, II and rS morphology in V1. His vectorcardiogram demonstrated slow initial forces directed leftward and anteriorly. The preoperative electrophysiologic study demonstrated the following atrial sequence during PAT: low anterior RA → low lateral RA-medial (septal) RA → medial LA → lateral LA.
Antegrade mapping at surgery demonstrated epicardial breakthrough on the anterior RV (fig. 8). Recording of this earliest point demonstrated recordable ven-

Figure 7

Standard 12 lead ECG obtained during high right atrial pacing preoperatively, demonstrating pre-excitation.

tricular activity 15 msec before the onset of the delta wave (fig. 5). During RV pacing, earliest retrograde activity appeared at a contiguous atrial site (fig. 9). With the heart beating in sinus rhythm, on bypass, the annulus was dissected adjacent to the earliest point of epicardial activation resulting in abolition of the delta wave.

The tracing in figure 10 was recorded from a 47-year-old lady with similar electrocardiographic features, with a positive delta in leads I, II, and an Rs morphology in V1. While this ECG initially suggested a right lateral site for pre-excitation, a sequence of retrograde atrial activation was noted during FAT on the preoperative study which suggested a septal bypass. Epicardial mapping (fig. 11) demonstrated a site of pre-excitation comparable to that of the patient, described above. Breakthrough occurred on the anterior RV, adjacent to the pulmonary artery. In contrast to the first patient, however, the earliest recordable activity on the epicardial surface was 10 msec after the onset of the delta wave (fig. 6). This suggested that contiguous bipolar electrodes placed on the epicardium of the anterior RV free wall failed to record the earliest excitation potentials. With the heart beating in sinus rhythm, dissection adjacent to

Figure 8

Epicardial mapping performed before (above) and after (below) division of an accessory pathway in the anterior RV free wall. (Same patient as figure 7). All values are corrected to the onset of the delta wave and Q wave during anomalous and normal conduction respectively. During pre-excitation, epicardial breakthrough occurred at the base of the RV 15 msec before the onset of the delta wave. Following division of the accessory pathway, breakthrough occurred 30 msec after the onset of the QRS, at three separate epicardial sites.

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the area of earliest epicardial excitation did not modify the delta wave. The incision was then extended along that portion of the tricuspid annulus which is attached to the septal leaflet. When the incision reached a point which was at one and the same time a few millimeters anterior to both the right trigone and the site from which the His deflection was recorded, pre-excitation disappeared. The differentiation of free wall and septal accessory pathways in the region of the crux is illustrated by the patients shown in figures 12–15. Figure 12 demonstrates negative delta waves in leads II, III and aVF, with an RS complex in V1, recorded in a 20-year-old man with recurrent PAT. Preoperative studies demonstrated the earliest retrograde atrial activation during PAT in the posterolateral RA, with spread later to the septal RA, and the lateral IA. Epicardial mapping (fig. 13) demonstrated an area of epicardial pre-excitation to the right of the crux, with activation occurring well before the onset of the delta wave. Following incision of the free wall annulus in this area, pre-excitation was abolished.

The electrocardiogram shown in figure 14 is remarkably similar to the above patient’s and was recorded in a 43-year-old man presenting with atrial flutter associated with 1:1 A-V conduction down the bypass tract. Preoperative study, however, demonstrated early and near-simultaneous activation of the septal RA and the medial LA during both ventricular pacing and during closely coupled premature ventricular beats. Epicardial mapping (fig. 15) revealed that the earliest recordable ventricular activity occurred 5 msec after the onset of the delta and RV cavity potential. Incision of the annulus underlying the early epicardial point failed to normalize the ECG. With the heart beating in sinus rhythm, the incision was extended beneath the coronary sinus. Pre-excitation disappeared when the annulus was incised just posterior to the membranous septum and the recorded His potential.

Figure 16 demonstrates the tracings obtained during sinus rhythm and left atrial pacing from a 54-year-old male.

Figure 10

12 lead ECG recorded in sinus rhythm preoperatively.

Figure 11

Epicardial mapping performed before and after division of an accessory pathway in the anterior septum. (Same patient as fig. 10). During pre-excitation, epicardial breakthrough occurred 10 msec after the onset of the delta wave. Following division of an accessory pathway in the anterior septum, breakthrough occurred 25 msec after the onset of the QRS, over the posterior left ventricle. Delay in activation over the RV is compatible with right bundle branch block, which had been noted during intermittent normalized conduction prior to surgery.

Figure 12

Preoperative 12 lead ECG of a patient who presented with recurrent PAT.
old man who characteristically had minimal pre-excitation in sinus rhythm. During LA pacing, negative delta waves developed in I and aVL, associated with tall R waves in V1-V6. During PAT, earliest atrial activity was recorded from the lateral coronary sinus. Mapping during sinus rhythm (fig. 17) at surgery demonstrated an area of pre-excitation on the left lateral free wall, occurring before the onset of the delta. In addition, a second later breakthrough occurred over the area trabecularis of the right ventricle due to activation proceeding over the normal conduction system. During an intra-operative episode of PAT, atrial activation was noted to proceed from a point on the lateral LA corresponding to the previous demonstrated site of antegrade pre-excitation. Incision of the free wall annulus between these two points normalized the activation and eliminated further episodes of PAT.

Figure 13
Epicardial mapping before and after division of a posterior RV free wall accessory pathway. (Same patient as in fig. 12). The map was obtained during pacing of the right atrial appendage. Pre-excitation initially occurred at the posterior base of the RV, 22 msec before the onset of the delta wave. Following surgical interruption of the accessory pathway, normal breakthrough was noted on the anterior RV. $\Phi$ = site of pacing electrode.

Figure 14
Preoperative 12 lead ECG of a man who presented with atrial flutter and 1:1 A-V conduction down the bypass tract.

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Figure 16

Preoperative 12 lead ECG during sinus rhythm (NSR) and left atrial pacing in a patient with minimal pre-excitation in sinus rhythm.

Figure 17

Epicardial map prior to division of a lateral LV free wall accessory pathway. Pre-excitation was present during sinus rhythm. (Same patient as in fig. 16). Earliest activation occurred 13 msec before the onset of the delta wave.

Characteristics of the accessory pathway. There have been no arrhythmias noted in a follow-up of 4–48 months.

In three patients, the delta wave was unchanged, and arrhythmias persisted. His bundle division was undertaken in one of these, abolishing PAT.

One additional death occurred postoperatively in a patient operated on early in the series as an emergency. She presented with intractable PAT, and because of the urgent nature of her arrhythmia did not undergo electrophysiological study. She never demonstrated a typical delta wave. Epicardial mapping at the time of surgery failed to provide definitive evidence of pre-excitation. Division of the A-V ring failed to abolish the arrhythmia, and she died on the second postoperative day of hypoxia and digitalis intoxication. An autopsy was performed. Detailed review of the histologic studies of the A-V ring failed to demonstrate an accessory pathway. She was included in this series because she was thought preoperatively to possess the WPW syndrome, although it is uncertain, based on the operative and postmortem findings, that she did in fact have WPW.

Summary

Physiological studies of the type we have described, when performed in patients with the WPW syndrome, can yield diagnostic information regarding the mechanism of arrhythmia, demonstrate functional properties of therapeutic import, facilitate therapeutic
decision-making about drug regimens and presumptively localize the site of pre-excitation as a basis for possible surgical intervention. Based on our experience, we feel that in selected patients, surgical correction of the WPW syndrome is entirely feasible, and can be accomplished in the majority of patients in whom free wall A-V connections are present. The continuing challenge of identification and correction of septal accessory pathways directs our present work with the WPW syndrome.

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