An Electrophysiologic Approach to the Surgical Treatment of the Wolff-Parkinson-White Syndrome
Report of Two Cases Utilizing Catheter Recording and Epicardial Mapping Techniques

By ROBERT H. SVENSON, M.D., JOHN J. GALLAGHER, M.D.,
WILL C. SEALY, M.D., AND ANDREW G. WALLACE, M.D.

SUMMARY
The usefulness and limitations of electrode catheter and epicardial mapping techniques in the evaluation of the Wolff-Parkinson-White syndrome are described. In one case epicardial surface mapping was unsuccessful in localizing the accessory connection. However, a septal location was suggested by catheter recording techniques and confirmed by the point at which the surgical incision interrupted the accessory connection. In the second case catheter techniques could not confirm the role of the accessory connection in a re-entrant rhythm. Epicardial mapping of the atrium during PAT confirmed a return route to the atrium over the accessory connection, opposite the point of ventricular pre-excitation during sinus rhythm. In both cases, following surgery, epicardial maps, 12-lead ECGs and catheter recording techniques revealed no evidence of residual pre-excitation.

Additional Indexing Words:
His bundle electrocardiography Coronary sinus recordings
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TECHNIQUES which employ electrode catheters have been described for evaluating patients with the Wolff-Parkinson-White syndrome; and useful criteria have been established for demonstrating anomalous ventricular pre-excitation,1-9 mechanisms of tachycardia,6-9 the location of the accessory connection,4-7, 10 and the properties of anomalous atrioventricular conduction.2, 10 Epicardial mapping has been utilized to verify anomalous ventricular pre-excitation11-12 and to localize the accessory connection for surgical interruption.13-24

In several instances we have had the opportunity to combine electrophysiologic observations using catheter techniques and intraoperative mapping in the same patient. Two patients with the Wolff-Parkinson-White syndrome were of particular interest because these two approaches provided complementary data which were essential to their treatment. In the first patient an accessory connection within the septum, suggested by catheter data but not evident from epicardial mapping, was successfully divided at surgery. In a second patient, we were unable to delineate the site of the accessory pathway or to initiate tachycardia during the preoperative study. However, the location of the pathway between the left atrium and ventricle and its participation in the tachycardia were determined at surgery prior to its successful division.

Case 1
M.R. was a 47-year-old female with a history of tachycardia beginning at age 16. In 1969 episodes of tachycardia recurred more frequently and were accompanied by nausea, lightheadedness and tightness in the chest. During one such episode, supraventricular tachycardia was documented. Following conversion to sinus rhythm, the electrocardiogram revealed right bundle branch block. On alternate beats the PR interval shortened in association with delta waves compatible with ventricular pre-excitation. Treatment with digitalis and quinidine was instituted, but had to be discontinued because of intolerance. Over the succeeding three years, procaine amide in doses up to 2 g per day and/or propranolol up to 160 mg per day were unsuccessful in preventing supraventricular tachycardia. Procaine amide and propranolol were discontinued in December of 1972 because of severe sinus bradycardia and an episode of hypotension. The patient was admitted to Duke University Medical Center on July 15, 1973 at which time episodes of
tachycardia were occurring once a day despite treatment with quinidine polygalacturonate 275 mg t.i.d.

Physical examination was entirely within normal limits. Routine blood count, urinalysis, electrolytes, chest X-ray, and cardiac series and fluoroscopy were normal. The 12 lead electrocardiogram (fig. 1, upper panel) showed ventricular pre-excitation with a positive delta wave in leads I, II, III, aVF, and V; of interest was the small positive delta wave in lead V.

**Preoperative Electrophysiologic Study:** In the post-absorptive, unsedated state a quadripolar catheter was positioned in the coronary sinus. A second quadripolar catheter was positioned in either the right atrium (RA) or right ventricular apex (RV). A hexapolar catheter was positioned across the tricuspid valve to record from the bundle of His (HBE). No enhancement of pre-excitation was noted pacing the RA at cycle lengths (CL) from 800 to 450 msec. The His electrogram was obscured by early ventricular depolarization. At CLs shorter than 450 msec conduction occurred exclusively through the atrioventricular (A-V) node with a right bundle branch block pattern and an H-V of 45 msec. The effective refractory period of the anomalous pathway was determined with the extra stimulus method. During sinus rhythm (CL = 1,050 msec), the refractory period of the anomalous connection was 430 msec. During RA pacing at CLs of 800 and 700 msec, the refractory period shortened to 385 and 350 msec, respectively. Premature beats at coupling intervals less than the effective refractory period of the anomalous connection initiated PAT with antegrade conduction via the A-V node-His-Purkinje system and a return to the atrium over the accessory connection. During episodes of PAT, the range of CLs was 360-410 msec, notably shorter than the CL at which antegrade conduction blocked in the accessory connection during atrial pacing.

Ventriculo-atrial conduction was studied during RV pacing and premature stimulation. The V-A time remained constant (100 msec) at pacing CLs from 800 to 300 msec. During right ventricular pacing at a CL of 800 msec, V-A time during premature beats with coupling intervals of 750 to 305 msec remained constant at 100 msec. At coupling intervals less than 305 msec, premature beats failed to propagate retrograde.

The sequence of retrograde atrial activation during RV pacing in a subject without pre-excitation (fig. 2, upper panel) was compared with the observed sequence in this patient (fig. 2, lower panel). The upper panel in figure 2 shows the normal retrograde sequence of atrial activation with the atrial (septal) activity (RA) on the HBE preceding both low lateral right atrium (LLRA), and lateral left atrial activity (LA) recorded from the distal coronary sinus. This same sequence is noted with retrograde conduction over the accessory connection during RV pacing (fig. 2, lower panel) and during PAT (fig. 3). Of note is the shorter V-A time during RV pacing at comparable CLs in this patient with pre-excitation, 100 msec vs 230 msec as noted in figure 2.

In patients with proven lateral connections between left atrium and left ventricle activation of the lateral left atrium has preceded the atrial septum and lateral right atrium during PAT and RV pacing. With an accessory connection between the lateral RA and RV we have observed activation of the low lateral RA preceding atrial septum and lateral left atrium during PAT and RV pacing. In Case 1 the short V-A time (100 msec) during RV pacing and retrograde activation of the atrial septum followed by low lateral RA and lateral left atrium during RV pacing and PAT were most compatible with an accessory septal connection.

**Intra-operative Electrophysiologic Study:** On July 25, 1973 the patient was taken to the operating room. Mapping at the time of surgery revealed the earliest area of epicardial activation adjacent to the tricuspid annulus near the right ventricular outflow tract (fig. 4, upper panel). This early area occurred 10 msec after the onset of the delta wave. With the patient on cardiopulmonary bypass, the right atrium was opened. While in sinus rhythm, the atrial wall was divided along the tricuspid annulus beginning at the region indicated by the map. The delta wave did not disappear. The incision was then carried across the base of the aorta to within 3.5 mm of the His bundle at which point the delta wave was abolished. With the atrium closed and the patient off cardiopulmonary bypass, the epicardial map revealed RBBB and no evidence of pre-excitation (fig. 4, lower panel). Postoperatively, no arrhythmias occurred and the delta wave was abolished revealing sinus rhythm, a normal PR interval and right bundle branch block (fig. 1, lower panel).

**Figure 1**
Patient M.R., 12 lead ECG. Preoperative ECG (upper) shows a PR interval of .10 sec with large positive delta waves in I, II, III, aVF, and V; through V. Postoperative ECG (lower) shows a PR interval of .18 sec, QRS of .14 sec and right bundle branch block.

**Figure 2**
Retrograde atrial activation sequence during RV pacing at CL of 550 msec in a patient without pre-excitation (upper) compared with patient M.R. (lower). In each atrial septum (HBE) precedes low lateral RA (LLRA) and lateral LA recorded from distal coronary sinus (CS). However VA conduction time is 230 msec (upper) compared with 100 msec (lower) in patient M.R. with pre-excitation.
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Figure 3
Patient M.R. during an episode of PAT with right bundle branch block reveals the same atrial sequence as during right ventricular pacing (fig. 2).

Postoperative Electrophysiologic Study: On August 5, 1973 a repeat electrophysiologic study was performed. There was no evidence of preexcitation in sinus rhythm, during RA pacing, or with premature stimulation. The effective refractory period of the A-V node was 280 msec. In contrast to the preoperative study, complete ventriculo-atrial block was present during RV pacing. Atrial premature beats introduced during the entire diastolic interval did not initiate PAT.

Case 2

S.N. was a 30-year-old female with a history of occasional episodes of tachycardia which began at age 16. In 1968 the attacks became more frequent and prolonged, lasting 12-18 hr. Protracted episodes were associated with dyspnea and lightheadedness. Beginning in 1970, episodes of tachycardia were particularly frequent and associated on several occasions with syncope. A carotid sinus nerve stimulator, implanted elsewhere in 1971, was ineffective in terminating attacks. An attempt was also made to terminate episodes with a coronary sinus radio frequency pacemaker but the catheter could not be maintained in position. She was tried on digitalis, propranolol and quinidine sulfate, alone and in varying combinations, without sustained benefit. Prior to evaluation at Duke University Medical Center on August 5, 1973, she was taking procaine amide 3 gm daily, but was having 40-50 episodes of tachycardia per day despite this medication.

The physical examination, blood count, urinalysis, routine chemistries, chest X-ray and cardiac fluoroscopy were normal. The electrocardiogram (fig. 5, upper panel) was compatible with anomalous left ventricular pre-activation. A negative delta was present in leads I and aV1.

Preoperative Electrophysiologic Study: Catheter positioning was similar to patient M.R. except that a coronary sinus catheter could not be maintained in position. Atroventricular conduction was assessed with RA pacing and premature stimulation. At pacing CLs of 700 to 500 msec, 1:1 propagation of impulses occurred over the accessory connection. At shorter CLs conduction occurred exclusively through the A-V node. Using the extra stimulus method (pacing CL 700 msec), the effective refractory periods of the accessory connection, A-V node and atrium were 390, 360, and 240 msec, respectively. The length of the study prohibited evaluation of the refractory period of the anomalous pathway at different cycle lengths. Single atrial premature beats at coupling intervals less than the effective refractory period of the accessory connection captured the atrium but failed to initiate PAT. Stable catheter position in the coronary sinus could not be maintained and thus attempts to initiate PAT by premature stimulation of the left atrium were unsuccessful.

Ventriculo-atrial conduction was assessed during RV pacing and premature stimulation. At RV pacing CLs of 700 to 350 msec, the stimulus to RA interval remained constant at 110 to 130 msec. The sequence of right and left atrial events during RV pacing could not be evaluated and we were uncertain whether retrograde conduction was occurring exclusively via the A-V node or via an anomalous pathway to the left atrium with subsequent RA depolarization. However, during RV pacing (CL 700 msec), premature beats at coupling intervals from 600 to 250 msec propagated to the right atrium with conduction delay (115-280 msec) which was related to the degree of prematurity. The longer V-A conduction with shorter coupling intervals seemed to be most consistent with retrograde conduction through the A-V node.

In this patient anomalous ventricular excitation had been established by demonstrating ventricular depolarization prior to His bundle depolarization. However, single RA or

Figure 4
Epicardial map in patient M.R. Prior to incision (upper), the earliest epicardial activation occurring 10 msec after onset of delta was recorded along the A-V ring near the pulmonary outflow tract. Following incision (lower), epicardial activation sequence is compatible with RBBB.

Figure 5
Patient S.N., 12 lead ECG. Preoperative tracing (upper) shows PR interval of .08 sec with positive delta in V1 and negative delta aV1. Postoperative tracing (lower) shows a normal PR interval (.12 sec) and absent delta wave.
RV premature beats never initiated PAT: Stimulation and recording of left atrial activity was not possible and the ventriculo-atrial events during right ventricular pacing did not clearly implicate retrograde conduction over an accessory pathway. However, the disabling nature of the patient's arrhythmia seemed to justify an attempt to localize the anomalous connection and to establish with epicardial mapping the role of the anomalous connection in the reentrant rhythm pathway during PAT.

Intra-operative Electrophysiologic Study: On August 14, 1973 intra-operative epicardial mapping was performed through a left thoracotomy. Pre-excitation was not present at the time of initial mapping and it could not be evoked by left atrial pacing. In contrast to right ventricular premature stimulation in the preoperative study, left ventricular premature stimulation induced PAT. A map of retrograde conduction to the atrium was therefore done during the arrhythmia. During PAT the earliest area of retrograde activation of the left atrium was at the acute margin (fig. 6). The conduction time from left ventricle to left atrium at this site was 30 msec. The tachycardia was then converted to sinus rhythm and after conversion pre-excitation was present. An epicardial map during anomalous excitation demonstrated that the earliest area of ventricular excitation was adjacent to the area of earliest atrial activation during PAT (fig. 7, upper panel).

The patient was placed on cardiopulmonary bypass. Through a left atriotomy, the left atrial wall was disconnected from the annulus of the mitral valve from the posterior trigone laterally over a distance of 2 cm. With the atriotomy closed and the patient off cardiopulmonary bypass, epicardial mapping showed a normal ventricular activation pattern (fig. 7, lower panel). Following surgery, the delta wave disappeared (fig. 5, lower panel) and PAT did not recur.

Postoperative Electrophysiologic Study: On August 22, 1973 the patient was restudied following the preoperative protocol. There was no evidence of pre-excitation. During sinus rhythm, RA pacing and atrial premature stimulation, the H-V remained constant at 45 msec. Ventriculo-atrial conduction times during right ventricular pacing were identical to those observed preoperatively. Multiple catheter induced premature beats did not initiate PAT. The patient has been free from symptoms for 3 months and the ECG remains normal.

Discussion

Accessory connections between the atrial and ventricular septae have been proposed as an explanation for some cases of Type B WPW, and such connections have been demonstrated histologically in postmortem examinations of hearts. In case 1, the atrial activation sequence during retrograde conduction was consistent with a septal location, showing atrial activation on the HBE which preceded low lateral RA and lateral LA activity recorded from the coronary sinus. This same sequence was observed during PAT. Constant and short ventriculo-atrial conduction times with increasing ventricular rates and premature stimulation suggested that atrial activation resulted from retrograde conduction over an accessory connection. Complete ventriculo-atrial block following successful surgical correction, without any significant change in the antegrade effective refractory period of the A-V node, confirms this interpretation.

The epicardial map in case 1 is also worthy of special note. In our experience with 11 other patients who had lateral left or right accessory connections, proven at surgery, the earliest epicardial activation recorded from the ventricle adjacent to the accessory bridge either coincided with the onset of the delta wave or preceded it by 10-15 msec. Detailed epicardial mapping in the region of the A-V ring in case 1 failed to reveal ventricular activity earlier than 10 msec after the onset of the delta wave on any surface ECG lead. Thus, ventricular activation began

Figure 6

Epicardial map in patient S.N. during PAT seen in left lateral (left) and posterior views (right). Earliest activation occurs in LA 86 msec following onset of QRS. The time between ventricular and atrial activation at this point is short (30 msec) compared to other points along A-V ring.

Figure 7

Epicardial map in patient S.N. Prior to incision (upper) in sinus rhythm early epicardial activation is recorded along the left lateral A-V ring 5 msec prior to onset of delta wave on the surface ECG. This point lies adjacent to earliest atrial point during PAT (fig. 6). Post-incision map (lower) shows normal epicardial activation sequence.
somewhere, at least 10 msec and perhaps 20-25 msec, earlier than the earliest site of epicardial breakthrough. This raised two possibilities at the time of surgery: either there was initial endocardial excitation of the right ventricle with an unusually long delay to the overlying epicardium, or the bypass was located within the septum. The latter was strongly suggested by the preoperative catheter data and was confirmed by the site (3-5 mm anterior to the His bundle) at which division of the A-V ring abolished the delta wave.

The electrophysiologic data in case 1 are of interest from another point of view. The influence of cycle length on the refractory period of myocardium and of the specialized ventricular conduction system is well established. To our knowledge this is the first report of the influence of cycle length on the refractory period of an accessory A-V connection. In case 1 and in other patients with the WPW syndrome we have shown that the antegrade refractory period of the accessory pathway may be reduced by an increase of heart rate. We have also observed that at any given heart rate the refractory periods for antegrade and retrograde conduction over the accessory pathway may differ by as much as 100 msec. It has been suggested \(^26,27\) that the accessory pathway may not participate in episodes of PAT if the bypass does not support conduction from atrium to ventricle during pacing and/or premature coupling intervals comparable to the cycle length during PAT. Our observations would suggest that it is retrograde conduction which is critical to the question of participation of the bypass in re-entrant tachycardia and that the appropriate cycle length at which to determine retrograde refractoriness of the bypass is one which approximates the cycle length during spontaneous episodes of tachycardia.

Case 2 demonstrated several potential limitations of catheter techniques in the evaluation of patients with the WPW syndrome. First, premature stimulation of the right atrium or of the right ventricle failed to induce episodes of PAT. This phenomenon has been noted before in patients with Type A WPW and a presumed bypass track on the left side of the heart.\(^4,8\) In addition, the atrial responses to right ventricular pacing and to premature stimulation of the right ventricle did not provide clear cut evidence for retrograde conduction over an accessory connection. Finally, despite long efforts a catheter could not be maintained in the coronary sinus to record left atrial and ventricular activity and to evaluate the responses to left atrial pacing. At surgery, however, it was clear that retrograde conduction to the left atrium over the accessory pathway could be demonstrated during episodes of PAT and, furthermore, premature stimulation of the left ventricle consistently induced episodes of PAT. These observations support the view that the site of stimulation and the site of origin of premature beats are critically important in evaluation of the role of accessory connections in re-entrant arrhythmias. Case 2 and several others in our recent experience suggest that when surgical intervention is considered in patients with the WPW syndrome, one must be prepared not only to map the sequence of ventricular activation during sinus rhythm, but also to pace from various sites and to map retrograde atrial activation either during PAT or during ventricular pacing. In case 2, atrial mapping during PAT was essential to localize the accessory connection prior to surgical division.

In summary, the electrophysiologic evaluation of two patients with anomalous ventricular pre-excitation has been presented. The first case represents the first surgical correction of anomalous pre-excitation which resulted from a septal connection, and that location was suggested principally by catheter techniques preoperatively. In the second case surgical correction of anomalous left ventricular pre-excitation was undertaken only after epicardial mapping identified the site of the accessory connection and its role in the re-entrant tachycardia. This report illustrates the importance of studies of retrograde conduction over the bypass and highlights the complementary information which can be obtained by preoperative and intra-operative studies.

Postoperative epicardial maps, electrocardiograms, electrophysiologic studies, and the absence of arrhythmias during the follow-up period indicate that both patients have undergone successful surgical treatment.

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