Epicardial Mapping in the Wolff-Parkinson-White Syndrome

JOHN J. GALLAGHER, M.D., JACKIE KASELL, WILL C. SEALY, M.D., EDWARD L. C. PRITCHETT, M.D., AND ANDREW G. WALLACE, M.D.

The most elaborate studies have been obtained by in vitro methods using revived human hearts by Durrer et al. in adults\(^1\) and by Brusca and Rossetani in the fetus.\(^2\)

Presently accepted clinical applications have empirically resulted in almost exclusive use of isochrone maps to depict sequences of activation, but it should be emphasized that other potentially valuable information can also be recorded: potential distributions during depolarization and repolarization, waveform analysis etc.

In the investigation of the pre-excitation syndromes, most of the important practical observations can be derived from determination of local activation times. The following explanation of this determination has its foundation in the work of Wilson et al.\(^3\) and assumes uniform polarization of excitation boundaries. While this assumption is not yet established beyond question, it furnishes a conceptual framework on which to base further discussion. The electrical field produced by activation of a muscle layer was described by Wilson\(^4\) as a dipole area. The approach of the dipole gives rise to a positive deflection, and its passage results in a fast deflection in the opposite (negative) direction, with a final return to baseline. The rapid deflection was called the intrinsic deflection by Lewis.\(^5\) A unipolar recording from the epicardial surface reflects influences from the whole heart; the intrinsic deflection in the unipolar lead corresponds with activation of the subepicardial muscle immediately beneath the exploring electrode.\(^6\) An example is shown in figure 1. Two unipolar electrograms recorded from near contiguous points on the epicardium of a human heart are shown in the upper panel (unipolar #1 and #2). Note that in each, the electrograms record initial slow activity reflecting activation of more distant parts of the heart. A rapid component appears with activation immediately below the electrode and finally slow activity returns, reflecting again more distant activity. The configuration of two unipolar leads recorded in contiguous areas differ only in the detail of the electrogram recorded at the moment of local activation. If one unipolar lead is electrically inverted the two leads can be algebraically summed to form a bipolar lead as shown. The two electrograms will thus cancel each other except where differences occur resulting in a differential spike. The bipolar spike correlates with the rapid part of the intrinsic deflections; specifically, depending on polarity, the upstroke of the bipolar complex coincides with the intrinsic deflection of one unipolar complex, the downstroke with the

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From the Departments of Medicine and Surgery, Duke University Medical Center, Durham, North Carolina.

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Address for reprints: John J. Gallagher, M.D., P.O. Box 3816, Duke University Medical Center, Durham, North Carolina 27710.
Mapping Equipment

The lower panel of figure 1 shows how a hand-held mapping probe with two electrodes separated by 1 mm can be connected so as to simultaneously record both unipolar electrograms and their bipolar derivatives. Note that the unipolar recordings are made via a buffer amplifier between the left leg (distant ground) and the epicardial point, while the bipolar derivative is achieved by feeding both unipolar leads into a high gain differential amplifier which is itself grounded to the right leg. This latter feature has proved empirically useful in diminishing interference. Typical electrodes employed in surface mapping are shown in figure 2. The electrodes must be applied to the epicardium with a gentle touch and allowed to "ride" with the heart, thus avoiding the induction of ectopic beats by mechanical irritation. Excessive pressure on the recording site will slow local conduction, yielding spuriously delayed conduction times, and may be suspected when injury currents appear on the unipolar recording. Unipolar electrograms recorded by this technique (open chest, heart displaced) from normal ventricular muscle variable amplitudes ranging 20–60 mV on the ventricle and 5–15 mV on the atrium. A fast intrinsic deflection is one in which changes of 10 mV occur within 3 msec. The differential electrogram should ideally be less than 3 msec in duration.34, 35

The relation of the hand-held electrode to the rest of the system is shown in figure 3. The mapping and recording areas are linked by slaved monitoring oscilloscopes as well as by a two-way communication system.

Standard surface electrocardiographic leads are channelled, together with a reference epicardial lead and the data leads, by field effect amplifiers (input impedance 1014 ohms) to a set of high gain differential amplifiers. The overall frequency response of the system is 0.1–1200 Hz. A Hewlett-Packard 7 channel magnetic tape recorder records all data at 3¼–7½ inches/sec. The data are displayed on a slaved storage oscilloscope and can be also written out on a Honeywell 2106 ultraviolet oscillograph. The reference electrode has an array of five electrodes in a plaque which is

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**Figure 1.** Determination of local activation. The upper panel demonstrates a bipolar ventricular reference recorded together with the bipolar and unipolar electrograms from two closely spaced electrodes (on a data point). One of the unipolar electrograms, shown with a dotted outline, has been electrically inverted. The stippled areas between the two electrograms demonstrate the forces which cancel each other out. The bipolar spike corresponds to the rapid intrinsic deflection in the unipolar electrogram. The lower panel shows the circuit used to simultaneously record bipolar and unipolar data from two recording electrodes.
sutured to the atrium or ventricle. A junction box selects a pair from this plaque (or alternately an intramural needle) to serve as a reference and data electrodes can be used to trigger a special digital timer* which allows display of activation time on a beat-to-beat basis (relative to the reference). All data can be related to another parameter (onset of surface QRS, delta wave, cavity potential, P wave etc.) by relating the reference to this parameter and correcting the values accordingly. The constraints to be observed with this method of correction will be discussed later.

**Mapping in the Operating Room**

It is important to eliminate as many sources of 60 cycles/sec interference as possible during the map. This requires the use of shielded cables, adequate grounding of all equipment and usually temporary disconnection of the heating blanket, blood warmer and cautery equipment.

Induction of anesthesia can at times cause the disappearance or appearance of pre-excitation. We generally utilize a balanced anesthesia with barbiturate induction followed by morphine sulphate, nitrous oxide and oxygen, and a relaxant such as Pabylon. A supplemental halogenated anesthetic such as Ethrane is often used in addition.

**Method of Recording an Epicardial Map**

A variety of methods for relating recorded data to the epicardial surface are available. Polaroid photographs can be taken in multiple views (with a camera equipped with a sterile hood) and the data recorded directly on the landmarks. This has the disadvantage, however, that it is difficult to obtain complete views of the heart (especially of the diaphragmatic surface) under operating conditions. Sketches can also be made of the heart, employing conspicuous landmarks. The method we have found most useful is to use a grid composed of 53 points, such as shown in figure 4. This allows a preliminary scan of the heart to be

*Designed by Mr. Jackie Kasell
made with reasonable reproducibility. Once the area(s) of interest is identified, point-by-point continuous scanning of the area can be done, directly relating the data to surface landmarks.

The Normal Epicardial Map

Earliest epicardial breakthrough normally occurs over the area trabecularis on the anterior right ventricle 18–25 msec after the onset of the surface QRS (fig. 5). A second breakthrough often occurs nearly simultaneously at the apex because of the thinness of the ventricular wall in this area. The general sequence of activation following this is that of radial spread toward the apex and base with latest activation of the A-V groove and right ventricular outflow tract, the latter activation 70–80 msec after the onset of the QRS. The activation of the left ventricle is variable. Multiple areas of breakthrough are noted, usually over the middle portion of the left anterior and left posterior paraseptal regions, and occasionally, the left anterior septum near the base.

Mapping of Pre-excitation

The heart is generally exposed via median sternotomy. The method used to localize the accessory pathway intraoperatively will generally be dictated by the preoperative studies and the hemodynamic state of the patient.

We generally attempt to define both the atrial and ventricular insertions of the accessory pathway(s) by retrograde and antegrade mapping respectively. However, the presence of unidirectional block in the accessory pathway (antegrade or retrograde), recurrent reciprocating tachycardia, atrial fibrillation or hypotension during the mapping of an arrhythmia may require modification of the approach used.

Fixed electrodes are generally sutured to both the atrium and the ventricle for purposes of pacing and recording. Either electrode may serve a dual purpose in providing a reference to which the data are related. This also affords a convenient method for monitoring the atrioventricular conduction time, and permits prompt recognition of the development of a junctional rhythm or block in the accessory pathway due to inadvertent trauma during the mapping. The importance of the site of atrial pacing (when this is indicated) will be discussed later, but in general, the atrial appendages are useful sites for locating atrial electrodes. Electrodes are easily sutured to the appendages and in this position are not likely to interfere with any subsequent atriotomies. Moreover, hemostasis on their removal can be readily achieved.

For descriptive purposes, the most commonly encountered mapping situations will be characterized.

Stable Antegrade Pre-excitation During Sinus Rhythm

Assuming stable pre-excitation is present, the map may be performed in sinus rhythm, preferably using a ventricular reference. Atrial references are less reliable in this setting because a shift in the site of the spontaneous atrial pacemaker shifts the reference for all the data as well. An atrial reference is more useful if the atrium is being paced.
from a fixed site. The site for the ventricular reference should preferably be devoid of epicardial fat and blood vessels and yet be located as close as possible to the A-V ring in the area presumptively localized as the location of the accessory pathway by preoperative study. Conduction in the A-V node may vary considerably during mapping, and depending on the degree of fusion present, the activation time of a reference placed near the epicardial breakthrough of activation proceeding over the A-V node–His bundle could be quite variable. A useful safeguard during the antegrade pathway is to check periodically to see that the A-V interval and the interval between the onset of the delta wave and the ventricular reference remain reasonably constant. This indicates that a steady state is present and minimizes the likelihood of recording uninterpretable data.

If there is no clue to the location of the accessory pathway, a preliminary scan of the A-V ring by the epicardial probe can be undertaken to find a point somewhere early in the delta wave and the reference secured to an adjacent ventricular area.

The delta wave itself might appear to be the ideal reference but in practice its onset is difficult to discern on surface leads. In addition the delta wave, and indeed the entire QRS, may become difficult to interpret when the heart is displaced from the pericardial sac during mapping.

Another possible reference is a cavity potential recorded from a transmural electrode needle inserted in the epicardium. A cavity potential should theoretically provide the most accurate method of determining the onset of ventricular activation. However, the onset of cavity potential can vary depending on the position of the needle and isoelectric components may be present. In this respect, the cavity potential is subject to many of the same limitations as the delta wave recorded by surface leads.

In practice, we select a ventricular reference with a sharply defined local activation to trigger a digital timing device, and the data are subsequently corrected to relate to the onset of ventricular activation recorded by surface leads.

When the surface electrocardiographic evidence of pre-excitation (delta wave) is due to an accessory pathway of the atrioventricular type (so-called “Kent bundle”), an area of pre-excitation is almost invariably observed on the ventricular surface adjacent to the A-V groove, regardless of whether the pathway is septal or freewall in location. This might initially suggest that the most expeditious approach would be to confine epicardial mapping to sites adjacent to the A-V groove. This limited approach has several undesirable features: 1) At the base of the heart, the epicardial fat is often abundant, making it difficult in some cases to obtain optimal activation data at the site of pre-excitation. In such situations, it is usually quite apparent from the global activation sequence where pre-excitation is originating from. 2) Recognition of other types of pre-excitation (i.e. Mahaim fibers) may be obscured (see below).

If pre-excitation is not present at the beginning of the mapping procedures, it may be due to one of several possibilities: 1) Conduction over the A-V node is being favored by the site of origin of the spontaneous atrial rhythm; 2) interatrial conduction delay is retarding the arrival of atrial activation at the site of the accessory pathway; 3) conduction via the A-V node is being favored by a vagolytic state accompanying induction of anesthesia; 4) the accessory pathway has been inadvertently traumatized during preparation of the heart for mapping; 5) a rate-related or complete antegrade block is present in the accessory pathway. Most of these possibilities can be anticipated from the preoperative study. In some situations, it may be necessary to pace the atrium in the vicinity of the accessory pathway to elicit pre-excitation. The location of the pacing site can be established by pacing with a roving electrode until stable pre-excitation is obtained or by pacing at a more rapid rate, resulting in less contribution to ventricular activation by the His-Purkinje system. Pacing has certain limitations in that pre-excitation over one accessory pathway may obscure the possible detection of other accessory pathways.

An example of a map of antegrade pre-excitation recorded during right atrial pacing in a patient with a freewall endocardial accessory pathway between the lateral right atrium (RA) and right ventricle (RV) is shown in figure 6. Note that earliest ventricular activation is initiated at the base of the heart near the right lateral margin, and occurs 15

![Figure 6. Epicardial mapping of the ventricle in a patient with right lateral pre-excitation. The earliest area of pre-excitation occurs at the right lateral A-V groove 15 msec before the onset of the surface delta wave. The adjacent atrium activates 30 msec prior to appearance of ventricular pre-excitation. Activation spreads tangentially across the anterior and posterior right ventricle with the latest area of activation on the lateral base of the left ventricle.](image-url)
msec before the onset of the delta wave. The A-V interval in this region is 30 msec. Latest activation occurs over the anterolateral left ventricle.

Stable Antegrade Pre-excitation Not Obtainable — Retrograde Conduction Over the Accessory Pathway Intact

If no stable antegrade pre-excitation is present, the accessory pathway can be localized by retrograde mapping during reciprocating tachycardia (RT) or ventricular pacing.

An example of a map performed during RT (same patient as in figure 6) is shown in figure 7. Incomplete right bundle branch block (IRBBB) is present. In the presence of a normal QRS in RT, mapping can reasonably be confined to the atrium. Note that earliest epicardial ventricular activity now occurs near the apex at the area trabecularis, 20 msec after the onset of the QRS (normal). Some delay along the right ventricular base is present because of IRBBB aberration. The retrograde atrial activation times are indicated by the numerals on the atrial side of A-V ring. Retrograde activation is initiated in the lateral RA opposite the previously demonstrated (fig. 6) site of ventricular pre-excitation. The local V-A interval is 30 msec.

Some patients will not have stable RT intraoperatively, or may not tolerate the hemodynamic consequence of this arrhythmia without partial cardiopulmonary bypass. In this situation, a retrograde map recorded during ventricular pacing from a site near the accessory pathway can be used to localize the accessory tract. This method has the same advantages and limitations as atrial pacing in the demonstration of antegrade pre-excitation. Namely, the pacing site may favor conduction over one accessory pathway while obscuring the presence of other pathways; in addition, retrograde conduction over the A-V node—His bundle may result in atrial fusion. The latter may be minimized by more rapid ventricular pacing, but the rate of pacing is often limited by hypotension or induction of RT.

An example of a retrograde atrial map (same patient as figures 6, 7) recorded during right ventricular pacing at a cycle length of 500 msec is shown in figure 8. Prior to division of the accessory pathway, earliest retrograde atrial activation occurred in the lateral RA, where the interval from the
ventricular stimulus on the RV to the atrial site was 105 msec. (The local V-A interval in the region of the A-V ring was again 30 msec.) Following division of the accessory pathway, retrograde activation was initiated in the septal region at a long V-A interval, followed by symmetrical lateral spread. Incremental pacing now caused a retrograde Wenckebach phenomenon at a cycle length of 450 msec and no arrhythmia could be induced.

**Presence of Intractable Arrhythmias**

If attempts at antegrade mapping are repeatedly punctuated by episodes of (orthodromic) RT, mapping may have

![Diagram](https://example.com/diagram.png)

**Figure 8.** Demonstration of the site of pre-excitation by ventricular pacing with atrial mapping. This panel is a schematic representation of the atrial activation times recorded at the level of the A-V ring during right ventricular pacing at a cycle length of 500 msec. Note that the earliest area of retrograde activation occurs in the lateral right atrium at a V-A interval of 105 msec with spread to the septum and lateral left atrial regions. Following incision of the accessory pathway, all ventriculo-atrial activation times at the same cycle length of pacing are longer and spread now occurs symmetrically from the region of the atrial septum compatible with retrograde conduction to the A-V node.

to be restricted to atrial mapping in RT. In the unusual situation of antidromic RT (antegrade conduction over the accessory pathway with retrograde conduction occurring via the A-V node or a second accessory pathway), mapping of both ventricle and atrium is indicated. An example of this (same patient as figures 6–8) is shown in figure 9. The patient had demonstrated this arrhythmia preoperatively, and electrophysiological study at that time confirmed the presence of a retrograde His bundle deflection in the ventricular electrogram during tachycardia with pre-excited complexes, followed at a long H-A interval by symmetrical activation of both atria. The arrhythmia was elicited in the operating room and demonstrated a pattern of ventricular pre-excitation similar to that obtained with atrial pacing (fig. 6), but with more marked pre-excitation because of the absence of His-Purkinje participation in antegrade activation. The retrograde atrial sequence demonstrates that atrial activation is occurring at a long V-A interval. The complete sequence demonstrated origin of activation in the atrial septum and was identical to that recorded during retrograde mapping following division of the accessory pathway (fig. 8), suggesting retrograde conduction over the A-V node-His bundle.

The presence of atrial fibrillation constitutes a special problem since retrograde mapping is not possible and varying degrees of normal and accessory pathway conduction are
manifest in ventricular responses. If a ventricular reference has been situated near the presumed site of the accessory pathway, variable activation times can be recorded at any given ventricular site, depending on the degree of pre-excitation. However, if attention is directed to beats having early activation times (and manifest pre-excitation), a map can still be constructed. A second reference electrode can be simultaneously used to monitor the area trabecularis of the right ventricle in order to identify the degree of fusion present on any recorded complex.

The unipolar morphology of the ventricular data in the region of the accessory pathway may be of further help in delineating the site of the accessory pathway, as will be discussed subsequently.

**Special Problems Encountered**

**Septal Accessory Pathways**

A septal accessory pathway should be suspected whenever the earliest area of ventricular activation overlies the anterior or posterior septum. Recognition of the possible septal location of the pathway is important since local dissection of the anulus adjacent to the earliest epicardial breakthrough will not necessarily modify the pre-excitation. Indeed, the activation may have coursed a considerable distance before reaching the epicardium. Certain features of the epicardial map may suggest a septal accessory pathway. An example of a map recorded in such a case is shown in figure 10. Ventricular pre-excitation is initiated on the left

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**Figure 10.** Epicardial mapping of a septal accessory pathway. Earliest ventricular activation occurs in the left crux of the heart with the onset of activation 24 msec after the appearance of the surface delta wave, characteristic of a septal accessory pathway. In the lower panel, following division of the accessory pathway, this early area is now activated late and epicardial breakthrough first occurs over the lower anterior right ventricle.
side of the crux of the heart, overlying the posterior inter-
ventricular septum. The earliest recorded activity on the
epicardial surface occurs + 24 msec after the onset of the
delta wave. Following division of the pathway, normal ac-
tivation is present.

We have found the timing and morphology of the unipolar
epicardial data recorded at the earliest site of pre-excitation
useful in localizing the accessory pathway.

In patients with freewall A-V connections, the earliest
epicardial activation has been noted to occur before or
simultaneous with the onset of the delta wave, while in
patients with septal connections, the area of earliest epicar-
dial activation occurs after the onset of the surface delta
wave. Figure 11 shows some representative examples. The
unipolar morphology is of further significance. The panel on
the left was recorded from a patient who had anterior right
ventricular pre-excitation. Extensive endocardial incision at
the atrial margin of the tricuspid anulus through to the
epicardial fat surrounding the right coronary failed to
abolish pre-excitation or RT. When epicardial dissection of
the fat overlaying the site of pre-excitation was performed, all
pre-excitation disappeared. The unipolar morphology of the
earliest recorded data had a QS morphology suggesting
epicardial to endocardial activation. This is in contrast to
the more usual finding of an Rs complex, shown in the center
panel recorded from a patient with a right sided endocardial
accessory pathway in the freewall (same patient as figures
6–9). The panel to the right was recorded from a patient
(same in in figure 10), with pre-excitation of the left crux due
to a septal accessory pathway. Note that the earliest record-
able data occur after the onset of the delta wave, and have
an Rs morphology.

Another advantage of examining the unipolar data is that
premature ventricular beats are more readily recognized.

Figure 12 shows an epicardial recording made in an area of
ventricular pre-excitation. Note that during paced beats, an
RS configuration is recorded near the onset of the delta
wave. The third complex is a premature ventricular beat
resulting from mechanical stimulation of the probe and
resembles the paced beats because it arises in the vicinity of
the accessory pathway. However, it has a QS morphology
and the intrinsic deflection occurs at the exact onset of ven-
tricular depolarization.

A serious deficiency of using the timing of the early area
as an indication of the presumed site of the accessory
pathway is the difficulty mentioned earlier in defining the
onset of the delta wave. When the delta wave is clearly
defined, it should be possible to record activity at or before
its onset on the epicardial surface and failure to achieve this
should suggest the possibility of septal pathway.

Examination of the unipolar morphology can be of addi-
tional value in the study of atrial fibrillation. Mapping can
be performed looking for areas demonstrating rS or QS
morphology. An attempt should be made to find an area
demonstrating complexes in which the intrinsic deflection
occurs early in the total unipolar complex. Particular care
must be exercised to avoid induction of premature ventric-
ular beats by mechanical stimulation of the probe, a situa-
tion difficult to distinguish from pre-excitation in the setting
of atrial fibrillation.

Retrograde atrial mapping in RT or during ventricular
pacing of the patient with a septal pathway usually
demonstrates initiation of activity over the anterior and
posterior atrial septum, not unlike the situation encountered
during normal retrograde conduction or during re-entry in
the A-V node. The magnitude of the V-A interval has not
allowed us to distinguish between a posterior septal
accessory pathway and a right paraseptal freewall accessory

![Figure 11](http://circ.ahajournals.org/content/57/5/862/F11.large.jpg)

**Figure 11.** Epicardial data at the earliest site of pre-excitation. In each panel are shown recordings of surface electro-
cardiographic leads together with a bipolar reference electrogram in addition to a unipolar and bipolar electrogram
recorded from the earliest site of pre-excitation. The left and center panels were recorded from patients with freewall
accessory pathways. Note that the earliest data recorded occur well before the surface delta wave while in the panel to the
right, recorded in a patient with a septal accessory pathway, the earliest point occurs after the onset of the surface delta
wave. In the case of an epicardial freewall accessory pathway (left panel), a QS morphology is recorded on the unipolar
channel. In the case of freewall endocardial accessory pathways and septal accessory pathways (center and right panels),
an initial positive deflection is generally observed on the unipolar electrogram as indicated by the open arrows.
pathway. A significantly longer V-A interval during ventricular pacing as compared to that recorded in RT should of course suggest a re-entrant mechanism in the A-V node. Figure 13 demonstrates the retrograde atrial activation sequence of the septal pathway shown in figure 10 recorded during right ventricular pacing at a cycle length of 500 msec. Earliest retrograde activation occurs just to the left of the crux at a V-A interval of 109 msec. In contrast to freewall accessory pathways, however, it can be seen that retrograde activation cannot have been initiated by the adjacent ventricle since the latter activates later than the atrium. These findings are representative of what we have found with septal accessory pathways and point out the limitations of mapping septal pathways from outside the heart.

An area of the heart deserving of special emphasis is the anterior RV epicardial surface. The crista supraventricularis can be pre-excited by anterior septal accessory pathways. The resultant zone of pre-excitation is invariably located on the anterior RV wall, near the pulmonary outflow tract and can mimic findings of a right ventricular freewall pathway.

All of the remarks considered above pertain to mapping performed on the epicardial surface. If more detailed localization is necessary, as in the case of septal pathways, intracardiac mapping (antegrade or retrograde) may be performed during cardiopulmonary bypass. The same equipment can be used to localize the location of the His bundle in the setting of a septal accessory pathway.

Multiple Accessory Pathways

The possibility of multiple accessory pathways underling the pre-excitation syndrome must constantly be looked for and anticipated. As previously mentioned, pacing of the atrium or ventricle preparatory to mapping can obscure the presence of multiple pathways. An example is shown in figure 14 recorded in a patient with a left lateral accessory pathway during pre-excitation induced by left atrial pacing. Because of interatrial conduction delay, the anterior right atrium is not activated until a considerable amount of ventricular activation has occurred. If an additional accessory pathway was present on the right side, it would not have been evident.

Other Technical Problems

A variety of other problems may confront the mapper during the procedure. The surface lead configurations change markedly when the heart is lifted up and the accompanying hypotension not infrequently increases the degree of fusion present. This can be minimized by pacing the atrium.
and mapping with a ring electrode on the hand, localizing the landmarks by palpation without physically displacing the heart. Some patients demonstrate a tendency to develop atrial fibrillation repeatedly with a rapid ventricular response. Cautiously administered intravenous doses of procainamide will often stabilize the rhythm without abolishing pre-excitation. There is often a temptation to speculate on the location of the accessory pathway based on the ability of digital pressure on a portion of the A-V ring to terminate re-entry. There are serious contraindicates to be made on such observations. Mechanical distortion of the ring may cause block in the A-V node, and premature atrial or premature ventricular beats are often evoked by attempts to terminate re-entry by digital pressure, giving a false impression that pressure on the accessory pathway site was responsible. We have terminated re-entry on several occasions by pressing on the accessory pathway site, but this is not a reliable finding. It is essential that the accessory pathway not be traumatized by retractors or blunt dissection. Trauma of this kind can inactivate the pathway resulting in normal ventricular activation temporarily.

An example of block induced in an accessory pathway by local pressure on the A-V ring is shown in figure 15. Left ventricular pacing is being performed in a patient with a left lateral accessory pathway. The hand-held probe is recording the left atrium near the accessory pathway, and a relatively short ventriculo-atrial conduction time of 110 msec is present. With local pressure on the A-V ring near the pathway, the V-A time prolongs to 240 msec. Further study suggested that retrograde conduction to the atrium now occurred over the A-V node.

Trauma to accessory pathways by local pressure generally resolves within minutes but block can last up to several hours. In cases where the accessory pathway has been traumatized during unsuccessful operative attempts to divide it, it has been noted that block can persist for hours up to several months. Such patients can demonstrate normal antegrade and retrograde conduction intraoperatively and postoperatively and remain free of arrhythmias until the accessory pathway recovers function. This calls attention to the need for precise localization of the accessory pathway so that a very deliberate dissection can be carried out in a reasonably circumscribed area.

**Surgical Implication of the Mapping Procedure**

The rationale for precise localization of the accessory pathway intraoperatively is based on the experience that conduction in the accessory pathway can be abolished for prolonged periods of time by local trauma. A very careful and vigorous dissection in a localized area appears preferable to a more superficial attempt carried out randomly over a wider circumference.

An attempt is generally made to keep the outer epicardium intact during endocardial dissection in order to secure hemostasis and facilitate closure. However, we have more recently encountered three patients with right freewall accessory pathways in whom extensive endocardial dissection above the tricuspid valve annulus initially abolished pre-excitation, which then returned. This was finally abolished by a discrete epicardial incision. This finding suggests that accessory pathways may in some instances be quite superficial. In subsequent work, we have actually been able to record an electrogram (presumably arising in the accessory pathway) in the A-V groove in four instances. Ex-ternal cryosurgical ablation was attempted in three of these and resulted in permanent ablation of pre-excitation.\(^{41}\) If there is no large coronary vessel present at the site of ventricular pre-excitation, local cooling is used to determine if the pathway is indeed superficial. If evidence of pre-excitation disappears at 0°C and reappears on rewarming, then the area is ablated by cooling to -60°C. If cooling at 0°C fails to modify pre-excitation we assume that the pathway is not superficial and proceed with a conventional dissection.

In a few cases, there has been discordance between the location of the atrial and ventricular ends of the accessory
pathway, suggesting either a circuitous route or multiple pathways. For this reason, we prefer to localize both ends of the accessory pathway(s) and dissect the A-V groove between these two sites.

When an appropriately situated incision fails to modify pre-excitation the map should be repeated at representative sites to assure that persistence of pre-excitation is not due to a second accessory pathway. In four cases to date, we have documented the presence of a Mahaim fiber as the cause of persistent pre-excitation. In two of these, the additional presence of enhanced conduction in the A-V node resulted in both a short P-R interval and a delta wave. Ventricular mapping in each case demonstrated earliest breakthrough near the apex and later activation of all areas adjacent to the A-V ring. Prompt recognition of this situation eliminated further unnecessary surgery.

When accessory pathways are situated near the His bundle, very precise localization by intracardiac atrial mapping during RT with simultaneous mapping of the His bundle can minimize the extent of the lesion required to ablate pre-excitation and thus ensure the integrity of normal conduction.\textsuperscript{41}

### Studies Performed after Incision of the Accessory Pathway

A few representative measurements are performed following completion of the dissection before the patient is rewarmed and the intracardiac incision is closed, to determine if pre-excitation is still present. Before withdrawal of bypass cannulae however, it is advisable to perform an antegrade and retrograde map during pacing of the atrium and ventricle respectively to exclude the presence of additional pathways in addition to documenting block in the pathway under study. It appears advisable to pace the atrium and the ventricle to determine at what cycle length the Wenckebach phenomenon occurs, and as a final measure, to electively induce atrial fibrillation in order to anticipate the ventricular response which might be observed if this arrhythmia appears postoperatively.

### Summary

Epicardial mapping provides a method for defining antegrade and retrograde sites of pre-excitation. It is best undertaken only after a careful, detailed preoperative electrophysiological study has been performed. The potential pitfalls of the technique are many and technical expertise must be constantly available to maintain a functioning system. For these reasons, it is not likely to lend itself to widespread application. The same techniques can be applied to localization of the site of origin of atrial or ventricular dysrhythmias, localization of myocardial ischemia and infarction, as well as to differentiate between epicardial delays due to conduction delay and those caused by intramural myocardial delay.\textsuperscript{39, 42}

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