Epicardial Mapping in a Variant of Type A Wolff-Parkinson-White Syndrome


SUMMARY
Electrocardiographic and epicardial mapping studies are reported in a child with an atrial septal defect and a variant of type A Wolff-Parkinson-White (WPW) syndrome. The vectorcardiogram suggested a posterior area of early activation and this was confirmed by epicardial mapping studies. Premature activation apparently occurred from a site near the endocardium of the basal region of the left ventricle and surgical interruption could not be planned on the basis of data provided by epicardial mapping.

The role of vectorcardiography in assessing patients with the WPW syndrome is discussed.

Additional Indexing Words:
Anomalous pathway Atrial septal defect Atrioventricular conduction Pre-excitation
Vectorcardiogram

With the demonstration that anomalous conduction and the susceptibility to supraventricular arrhythmia can be abolished surgically in some patients with the Wolff-Parkinson-White (WPW) syndrome investigation of conduction pathways has assumed a new significance. A number of studies have demonstrated early activation of the anterolateral surface of the right ventricle near the atrioventricular sulcus in type B WPW,1-4 but there is less information about the sequence of activation in type A and in intermediate types. We now report studies in a patient with a variant of type A WPW.

Case History

JB was referred to Green Lane Hospital at 13 months of age with a history of vomiting and failure to thrive. Height and weight were at the 3rd percentile. There was no frank heart failure but the right ventricle was overactive and there was a soft basal ejection murmur with narrow splitting of the 2nd sound and a short parasternal mid-diastolic murmur. The chest X-ray showed a cardiothoracic ratio of 0.67 with pulmonary plethora. Cardiac catheterization demonstrated the presence of an atrial septal defect with a pulmonary to systemic flow ratio of 1.7 and pulmonary arterial pressure of 20/10 mm Hg. Digitalis was commenced and the patient improved.

During the cardiac catheterization procedure there were five bouts of supraventricular tachycardia and from this time onwards bouts occurred at about four monthly intervals. They rarely lasted longer than one hour and required no treatment over the next nine years. At 10½ years of age the patient was admitted to the hospital during a bout of tachycardia which lasted 24 hr. At this time height and weight were between the 10th and 25th percentile, cardiac signs were typical of a secundum atrial septal defect and the cardiothoracic ratio was 0.62. Digoxin therapy, originally started at a dose of 0.015 mg/Kg body weight/day, had been allowed to run down to a dose of 0.005 mg/Kg/day (body weight 27.5 Kg) and this dose was now doubled. The bout of tachycardia resolved spontaneously but 12 further bouts occurred over the next two months. Digoxin was therefore stopped and no further bouts occurred during the next two weeks. Surgery was then undertaken with the patient on cardiopulmonary bypass. Access was by median sternotomy and following closure of the secundum atrial septal defect epicardial mapping was undertaken. During this procedure frequent bouts of supraventricular tachycardia occurred and were terminated by direct current shock applied across the atria. Twenty-four hours after the operation a single dose of 0.125 mg of digoxin was given, followed by a further bout of supraventricular tachycardia requiring direct current cardioversion. No further digoxin was given and no further arrhythmias occurred. Postoperative progress was complicated by fever, raised venous pressure and a pericardial rub, presumed due to a postpericardiotomy syndrome, which settled spontaneously. Physical signs indicated complete closure of the defect and subsequent progress was satisfactory.

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Epicardial Mapping

Unipolar epicardial tracings were made during sinus rhythm (with the delta wave present), and were recorded simultaneously with lead 2 of a Wilson central terminal. The exploring electrode was a steel ball 1.5 mm in diameter, connected to a flexible wire insulated with varnish. This permitted exploration of any region of the right and left ventricular surfaces without dislocation of the heart. For posterior sites the electrode was held against the epicardium with gentle pressure on the insulated wire by the surgeon's gloved finger. Once each electrode position was identified, the heart was allowed to rest in its normal position during recordings. Displacement of the heart tended to alter the QRS complex of the reference lead but this returned to normal when the heart was restored to its normal position. When a bout of tachycardia occurred, recordings were interrupted until the reference tracing returned to normal. Electrograms were recorded at 40 sites. Recordings from selected areas were made on three separate occasions, the pattern remaining quite constant on each occasion. Recordings were made with Electronics for Medicine amplifiers and an ultraviolet recorder with a paper speed of 500 mm/sec. The position of each electrogram was located on a diagram of the anterior and posterior ventricular surfaces. Tracings of each electrogram were made subsequently on the diagram (fig. 4). Timing of the intrinsicoid deflection at each area was made by comparison with the trough of the S wave of lead 2 which was recorded simultaneously. As the earliest recorded intrinsicoid deflection occurred 60 msec before the trough of the S wave.

Figure 1

13 lead ECG recorded during sinus rhythm at 10% years. Heart rate was 85 beats/min. P-R interval 0.11 sec. QRS interval 0.11 sec. The delta wave and PRBB pattern are demonstrated.
wave in lead 2, all times were advanced 60 msec to simplify inspection.

Activation appeared first in the paraseptal region of the basal left ventricle 2-3 cm below the atrioventricular sulcus, spreading from there to the surrounding left ventricular surface. A second area of activation appeared some 20 msec after the first, in the anterior paraseptal region. This and most of the remaining areas of the ventricular surface were presumably activated by the normal conduction pathway.

Comment

From the anterior orientation of the delta wave and early QRS sE-loop it was anticipated that the anomalous pathway would occupy a posterior position; this was confirmed with epicardial mapping. QS potentials across the diaphragmatic surface of the left ventricle were unusual as qRs complexes are the common finding here.5 This unusual pattern presumably resulted from the combination of abnormal spread of activation from two foci, some delay in conduction through the right bundle and marked dilatation of the right ventricle secondary to the atrial septal defect.

With the onset of supraventricular tachycardia the duration of the P wave was shortened, and the P-R segment was lengthened by 20 msec so that the P-R interval was unchanged. During the tachycardia the delta wave was lost and the QRS pattern was “normal” for the clinical situation. In the light of studies of arrhythmia in the WPW syndrome6 it is likely that a circus rhythm was present, with forward conduction through the normal pathway and retrograde conduction through the anomalous pathway.

The modest shortening of the P-R interval with pre-excitation and the site of early activation on the left ventricular surface are compatible with anomalous conduction through Mahaim fibres arising from the distal atrioventricular node or the main bundle, and passing to the postero-basal region of the interventricular septum. Such an abnormal pathway was considered responsible for the delta wave in the case described by Lev et al.7 in which a second anomalous bundle bypassing the atrioventricular node produced a short P-R interval. However a circus rhythm involving Mahaim fibres has not been described and it is perhaps more likely that the route of premature conduction is through an anomalous bundle crossing the atrioventricular

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Figure 2

13 lead ECG recorded during supraventricular tachycardia 24 hr after the tracing shown in figure I. Heart rate 215 beats/min. P-R interval 0.11 sec. QRS interval 0.09 sec. Q-P interval 0.16 sec. The delta wave is not present but PRBB persists.
Figure 3
Vectorcardiogram recorded during sinus rhythm with premature conduction. (Top) Complete complexes. (Bottom) QRS complexes only. Broad arrows denote beginning of delta wave. Curved arrows indicate direction of inscription of the QRS sE-loop. Dots are 4 msec apart. H = horizontal, F = frontal, S = left sagittal.

Figure 4
Diagrams of the anterior and posterior surfaces of the ventricles to show complexes recorded during epicardial mapping. Figures show the timing of the intrinsicoid deflection for each area. Time 0 is the earliest intrinsicoid deflection (postero-basal LV), occurring 60 msec before the trough of the S wave of lead 2, which was recorded simultaneously with each tracing. The intrinsicoid deflection of the anterior paraseptal region occurred some 20 msec after that of basal LV. When a QS complex was recorded, no figure is given. The timing of the onset of epicardial activation in each area showed a similar distribution. Relative to time 0 in the diagram, activation began at about −10 msec in the basal LV, +10 msec in the surrounding LV, +10 msec in the anterior paraseptal region and +25-30 msec in the surrounding RV. LV = left ventricle; RV = right ventricle.
sulcus and initiating ventricular excitation near the endocardium in the basal region of the left ventricle. The precise route of the anomalous pathway could not be determined by epicardial studies alone, but it is noteworthy that the earliest area activated was 2-3 cm below the atrioventricular sulcus. The early positive deflection in this region shows that excitation began deep in the muscle and spread in an endocardial-epicardial direction. This would suggest that, whatever the route followed by the anomalous pathway, it is situated deep in the muscle. Even if its location were known precisely, surgical interruption would be particularly difficult (perhaps impossible) and certainly surgical approach could not be planned on the basis of epicardial mapping data alone.

Many cases of the WPW syndrome can be classified as type A or type B as defined by Rosenbaum et al., but Boineau and Moore found it necessary to extend the classification to include cases where the initial and terminal segments of the QRS sE-loop were divergent. Difficulties which may occur in predicting the site of anomalous conduction from the electrocardiogram are illustrated by a recent report in which the electrocardiographic pattern was interpreted as showing type B WPW syndrome and yet the area of early activation was close to that shown in the present case. Information about early activation can be expected only from the initial part of the QRS sE-loop and in this connection it might be anticipated that carefully recorded vectorcardiograms would provide better information than scalar tracings. An assessment of the accuracy of vectorcardiography in predicting the site of premature activation, however, awaits correlation in a series of patients between vector tracings, detailed electrophysiological studies, epicardial mapping studies and, in some cases, surgical interruption of anomalous pathways.

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References
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