SPREAD OF THE EPICARDIAL EXCITATION IN RIGHT BUNDLE BRANCH BLOCK PATTERN

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Direct measurement of cardiac potentials provides various information concerning ventricular excitation which is not obtainable by such indirect methods as electrocardiography (ECG), vectorcardiography (VCG), or body surface mapping and useful for surgical treatment of arrhythmias. In 1930, Barker, MacLeod, and Alexander reported the first patient who underwent direct epicardial mapping during pericardiotomy for suppurative pericarditis.1 Rather recently several activation studies have been made on normal hearts2-5 and on hearts with Wolff-Parkinson-White syndrome6-9.

On the other hand, the electrocardiographic pattern of right bundle branch block (RBBB) is encountered in patients undergoing right ventriculotomy for correction of various cardiac lesions such as ventricular septal defect, tetralogy of Fallot, and pulmonary stenosis. The electrocardiographic pattern of RBBB is also observed commonly in patients with ostium secundum atrial septal defect (ASD) and ostium primum ASD. The electrocardiographic pattern of RBBB has been investigated by experimental RBBB10-14. However, comparative investigations surveying the epicardial activation of cardiac lesions exhibiting the RBBB pattern have been relatively few. The electrocardiographic RBBB pattern following surgical correction of ventricular septal defect has been explained in terms of delayed activation of the right ventricle resulting from either damage to the main bundle branch15 or interruption of peripheral right ventricular Purkinje system secondary to the ventriculotomy.15-17 The electrocardiographic pattern of RBBB in ostium secundum ASD was thought to be due to delayed activation of the right ventricle resulting from right ventricular hypertrophy.18-21 Whereas in ostium primum ASD, the ECG presents the RBBB pattern and left axis deviation. They were attributed to RBBB with left anterior hemiblock by Rosenbaum and associates22 while Durrer and associates reported early excitation in the posterobasal area of the left ventricular surface in ostium primum ASD23. Boineau and associates insisted that asymmetry of the conduction system was the cause of the characteristic pattern of the ECG24.

The present study was designed to investigate in detail the sequence of ventricular activation of normal hearts. This study was also designed to investigate the epicardial excitation sequence of various cardiac lesions exhibiting the electrocardiographic pattern of RBBB and to investigate the etiology of the RBBB pattern in the ECG.

MATERIALS AND METHODS

I. Experimental study

Twenty-nine healthy adult mongrel dogs ranging in weight from 8 to 27 Kg were examined for this study. Each dog was anesthetized with sodium pentobarbital (25 mg/Kg-intravenously). After intubation, a midsternal or left thoracotomy through the fourth intercostal space was performed and then the pericardium was opened. The heart was cradled in the pericardium to expose both ventricles. The standard ECG was monitored throughout the procedure. Warm iso-

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Key Words:
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Tonic saline solution was applied to the heart to prevent cooling and drying.

Epicardial exploration: Unipolar and bipolar epicardial electrograms were recorded from the ventricular surface by means of a hand-held electrode containing three silver terminals of 0.5 mm, mounted 1 mm apart each on a flat surface. This electrode was applied at numerous sites (100–150 sites) on the ventricular surface depending on the size of the heart. The epicardial sites explored were carefully noted on a map drawn during the experiment, on which anatomic landmarks, such as coronary blood vessels and the atrioventricular groove, were entered. This map was later compared at various angles with the photograph and the removed heart.

All epicardial electrograms were recorded together with the fixed reference, unipolar electrogram from a catheter electrode introduced into the left ventricular cavity through the apex. Bipolar electrograms were recorded from a electrode attached to the right atrium to assure normal atrio-ventricular conduction. The standard ECG lead (especially lead II) was also recorded as a time reference (Fig. 1).

Intramural exploration: Multiple-electrode needles, 1.0 mm in diameter, carrying 10 silver terminals of 0.1 mm diameter, each interterminal distance being 1 or 2 mm, were introduced into the wall of the ventricles and the interventricular septum through 100 sites of the ventricular surface. In total, 500 or so bipolar electrograms were recorded from these electrodes. After the experiment, the heart was removed and the location and the depth of the electrodes were carefully studied.

Recording: All electrical signals were subsequently amplified and printed in a 6 channel Siemens Elema Mingograf (type 82) at a paper speed of 100 or 250 mm/sec, and simultaneously recorded on a physiologic tape recorder (Teac R-351F). The frequency response of the system.
Fig. 2. Epicardial activation sequence in normal canine hearts. Either anterior or lateral and posterior views are shown. The zero time is the beginning of left ventricular cavity potential. Each zipitone pattern represents a 5-msec interval. The earliest epicardial breakthrough occurs in the anterior paraseptal area of the right ventricular surface.

was 5-1250 Hz. Minimum sampling rate which is sufficient to retain all of the information present in the original waveform from ventricular muscle is 1000 samples per second. Thus this system is considered suitable to study ventricular activation. Low-limit filters were used to increase base-line stability and to accentuate the electrical events of myocardial depolarization. In unipolar recording, the indifferent electrode was placed on the left leg.

Analysis: The main deflection in the bipolar electrograms was used as the moment of local activation at the recording site. The so-called "intrinsic deflection" in unipolar electrograms coincided with the main deflection of bipolar electrograms. However, unipolar electrograms

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Fig. 3. Intramural activation in a normal canine heart. A heart is cut in three planes parallel to the ventricular base (A, B, C). Much data which does not lie in the planes were taken to facilitate the drawing of the isochrones. Each zipitone pattern represents a 4-msec interval. Zero time is the beginning of left ventricular cavity potential.

were not used for measuring the time of local activation. All activation times were expressed in milliseconds following the time reference, i.e., the beginning of left ventricular cavity potential. For every site, at least 10 consecutive identical electrograms were recorded. Diagrams were constructed from the results obtained to show the sequence of ventricular excitation in 5 msec isochrones in epicardial exploration, and 4 msec isochrones in intramural exploration.

Production of RBBB: RBBB was produced in 10 canine hearts by cutting the main right bundle branch by means of a specially designed narrow scalpel, which was introduced into the right ventricular cavity through the free wall of the right ventricle. Bipolar electrograms from the right ventricular surface distal to the incision point were monitored to determined the influence of ventricular incision. However, their activation times were not changed by a small incision. RBBB was determined by elongation of QRS wave duration, development of a wide late R wave in lead aVR, and deep S waves in leads II, III, aVF by ECG monitoring throughout the procedure. After the study, the heart was removed and Lugol's iodine solution was applied to the endocardial surface, thereby staining the conduction system and conforming its exact relationship to the incision.

Ventricular pacing: Epicardial activation during ventricular pacing was investigated to compare with the epicardial activation pattern of

Epicardial Excitation of RBBB

RBBB and to determine the velocity of the muscle conduction. An intramural multi-electrode needle was introduced into the wall of the ventricle in 10 dogs. Unipolar and bipolar electrograms were taken to determine the proper depth of the electrode needle. The heart was paced by an external pacemaker at the endocardium and the epicardium at a slightly higher rate than that of the sinus rhythm (Fig. 5). Activation times were measured from the time of stimulation in pacing study.

II. Clinical study

Epicardial excitation of the ventricle was studied in 35 patients undergoing cardiac surgery in this series. These patients ranged in age from 1 to 59 years. Their diagnoses were ostium secundum ASD (10 cases), ostium primum ASD (3 cases), ventricular septal defect (10 cases), coronary heart disease (5 cases) and others (7 cases). A similar technique in the experimental study was used in the clinical study. Though the left ventricular cavity potential was used as the time reference in most of the cases, right ventricular cavity potential was used in some cases in which it was difficult to record the left ventricular cavity potential. The onset of QRS wave in lead II ECG was also used as a time reference in some cases including infants. Epicardial mapping was performed by means of the Elecath exploring electrode at as many sites as possible, from 42 to 70 sites of ventricular surface. The procedure required for 10–15 minutes and was carried out during the time of cannulation and after the intracardiac procedure. Intramural needle electrodes were not used in the clinical cases because of the risk of trauma to the myocardium and the conduction system. Surgical RBBB probably due to trauma to the main right bundle branch occurred in only one case out of 10 patients with ventricular septal defect in this series.

RESULTS

1. Epicardial excitation in the canine hearts

Some typical examples of isochronic representation of epicardial activation sequence are shown in Fig. 2. The zero time is the beginning of left ventricular cavity potential.

Right ventricle: The earliest epicardial breakthrough in the heart occurred in the anterior paraseptal area of the right ventricular surface at 8–10 msec. From this area, activation fronts spread in a more or less radial fashion on the ventricular surface. The excitation had two preferential directions; one toward the pulmonary conus, and the other toward the posterobasal area. Activation fronts formed a small dip near the atrioventricular groove. The pulmonary conus, the anterior high septal area and the posterobasal area were activated late at 40–60 msec depending on the size of the heart.

Left ventricle: Larger variations were visible in the left ventricular epicardial activation sequence. Early epicardial excitation was recorded at 15–25 msec in three areas; (1) an area near the posterior apex, (2) an anterior paraseptal area continuing the early activation area of the right ventricle, (3) a posterior paraseptal area. Area (1) and (2) were found in every canine
heart, while area (3) was not found in some hearts. Anyway, the epicardial breakthrough was always later in the left ventricle than in the right ventricle. The activation wave fronts then spread in a bizarre pattern toward the atrioventricular groove. The anterior lower paraseptal area of the left ventricle between two early activation areas was always activated a little later. The anterior and posterior septal areas near the atrioventricular groove were activated last (40–60 msec).

2. Intramural excitation in the canine heart.

Intramural exploration was carried out in 10 dogs. Isochronic representation of the intramural activation sequence in the canine heart is shown in Fig. 3. After intramural exploration, the heart was removed and cut by three planes parallel to the ventricular base (Fig. 3, A, B, C). Many data which does not lie in the planes were taken.

The earliest activation occurred coincidentally with the onset of left ventricular cavity potential in the endocardial region near the base of the anterior papillary muscle of the left ventricle. Within 8 msec, most part of the endocardial surface of the left ventricle near the apex and a small part of that of the right ventricle were activated. These areas to be activated during 0–8 msec were electrocardiographically silent as shown in lead II ECG in Fig. 3. In the left ventricle, the activation spread rapidly over the endocardial surface and also spread through the ventricular free wall toward the epicardium. Thus activation front made an irregularly truncated cone.

Activation of the right ventricle began at the endocardial region of the base of the anterior papillary muscle 4 msec later than that of the left ventricle. Activation then spread rapidly over the endocardial surface probably through subendocardial Purkinje networks and also spread toward the epicardium and appeared as a breakthrough in the anterior paraseptal area after 8–12 msec. The activation of the interventricular septum began in the endocardium of the left side, and then proceeded from both sides toward the center of the septum, and spread from the apex to the base. In the posterobasal septum, the activation spread from the left side to the right and behaved electrically as a part of the left ventricle. The last activation of the ventricle was recorded in the epicardial region of the anterosuperior and the posterobasal septal areas at 40–44 msec.

3. Epicardial excitation in canine RBBB

Isochronic representation of the epicardial activation sequence before and after the produc-

Fig. 6. Epicardial activation sequence of human hearts. Activation times were measured in msec after the beginning of left ventricular cavity potential in patients A, C, D, or after the onset of QRS wave in lead II ECG in patient B.

A: 53-year-old man with ischemic heart disease.
B: 4-year-old boy with ventricular septal defect with minimum ventricular hypertrophy.
C: 59-year-old woman with coronary artery fistel to the pulmonary artery.
D: 50-year-old man with mitral stenosis.

tion of RBBB is shown in Fig. 4. Activation times were measured from the beginning of the cavity potential of the left ventricle in which the left bundle branch was intact. The left figures show the control activation sequence before incision of the main right bundle branch and the right figures show the activation sequence after incision.

The activation sequence did not change by insertion of a narrow scalpel into the right ventricular free wall, however, it suddenly changed when the main right bundle branch was incised. The epicardial activation of the left ventricle showed the normal pattern ending in 40 msec, which did not change. In the right ventricle, the activation was markedly delayed and the right anterior paraseptal epicardial breakthrough was absent. The activation of the right ventricle began after completion of the left ventricular activation. Two activation fronts originating from the left ventricle invaded the right ventricular surface across the anterior and posterior septal areas, and then fomed a V-shaped pattern. Then the activation travelled toward the right side of the pulmonary conus, where the last activation was recorded after 90 msec. The total epicardial activation duration was slightly more then double the preoperative normal value. This was presented by marked elongation of QRS duration in the ECG. Slow conduction directed toward the pulmonary conus was represented by large, wide S waves in leads I, II, III, aVF and wide R wave in lead aVR. The isochrones were crowded in the anterior and posterior paraseptal areas and widely spaced in the right upper area of the right ventricle. These results suggested a participation of Purkinje system in the latter area of the right ventricle.

4. Epicardial excitation in ventricular pacing

The velocity of ventricular muscle conduction determined by the rate of epicardial conduction during ventricular pacing was 36 cm/sec on the average.

Isochronic representation of the epicardial activation sequence during the ventricular pacing is shown in Fig. 5. Fig. 5A shows the normal epicardial activation before pacing. The canine heart was stimulated bipolarly at the epicardium (Fig. 5B) and the endocardium (Fig. 5C) with an intramural electrode that had been inserted near the apex. The activation spread in a radial manner from the stimulating site. The right ventricular activation was markedly delayed in comparison with the normal activation. The latest activation occurred at the pulmonary conus at 100–110 msec after the time of stimulation. The isochrones were crowded near the stimulation site and the septal area, whereas they became wide along the activation front moving toward the pulmonary conus. This rapid spreading of the activation was probably due to the involvement of the peripheral Purkinje system. The activation spread 10 msec faster during endocardial pacing than epicardial pacing.

5. Epicardial excitation in the human hearts

The hearts of patients with coronary heart disease without myocardial infarction and ventricular septal defect with minimum ventricular hypertrophy were examined as a control group. Fig. 6 shows the isochronic representation of the epicardial activation sequence in the human hearts. The activation sequence of the human hearts was similar to that of the canine hearts. However, the total activation time varied with the size of the hearts. In the right ventricle, the earliest epicardial breakthrough occurred at the right anterior paraseptal area 20–25 msec after the beginning of left ventricular cavity potential. From this area, the activation spread on the epicardial surface in a circular fashion. The pulmo-
Fig. 7. Epicardial activation sequence and ECGs of two patients with ventricular septal defect before and after operation. Activation times were measured in msec after the onset of QRS wave of lead II ECG in A, and after the beginning of left ventricular cavity potential in B. The left figures show preoperative activation sequence and ECGs and the right figures show postoperative ones.
nary conus was activated late after 50–60 msec. In the left ventricle, early activation was recorded at 30–40 msec in areas near the posterior apex, an anterior paraseptal area, and sometimes a posterior paraseptal area. The posterobasal area was activated last (70–80 msec). In comparison with the canine hearts, the anterior high septal area was not the latest area of the activation.

Despite atrial fibrillation in 2 patients including patient D of Fig. 6, epicardial mapping was thoroughly carried out. And his epicardial activation sequence presented an almost normal pattern. The beginning of left ventricular cavity potential coincided with the onset of ventricular depolarization and was not influenced by supraventricular arrhythmias including atrial fibrillation.

6. Epicardial excitation after right ventriculotomy

Epicardial activation pattern changed by repair of ventricular septal defect that required right ventriculotomy. Fig. 7 shows preoperative and postoperative epicardial activation sequence of 2 patients with ventricular septal defect. In patient A, the preoperative activation sequence showed a normal pattern, and then a vertical right ventriculotomy was performed in the dotted line. Following the ventriculotomy, the ECG showed elongation of QRS duration but no alteration of QRS axis. Postoperative epicardial activation showed marked delay at sites distal to the incision. However, the epicardial breakthrough was found in the anterior paraseptal area.
of the right ventricular surface with some delay by the influence of cardiopulmonary bypass, and no significant delay occurred proximal to the incision. From this area the activation spread slowly toward the atrioventricular groove. The isochrones were crowded around the area of ventriculotomy. These results suggested interruption of the peripheral Purkinje system in the right ventricular wall.

RBBB which was thought to be due to trauma to the main right bundle branch occurred in one patient out of 10 ventricular septal defect closure. Characteristic alteration of epicardial activation sequence is shown in Fig. 7B. The sequence of epicardial activation before operation showed an almost normal pattern, but postoperatively it changed with development of RBBB pattern in the ECG, which revealed elongation of QRS duration, wide and slurred S waves in leads II, III, and aVF, and rsR' complex in lead V1. In postoperative RBBB, the right anterior paraseptal epicardial breakthrough was absent. Epicardial activation in the left ventricle was similar to the excitation before operation. The activation wave fronts spread from the left ventricle to the right ventricle both anteriorly and posteriorly and formed a V-shaped pattern. Then the activation spread toward the right side of the pulmonary conus, where the latest activation occurred at 95 msec. The isochrones were crowded at the ventriculotomy area as well as the anterior and posterior paraseptal areas. They were widely spaced in the right upper region of the right ventricle as in the case of canine RBBB. Epicardial activation pattern was similar to that of the canine heart with experimental RBBB. In this case, damage to the main right bundle branch would pay a major role in the electrocardiographic pattern of RBBB.

7. Epicardial excitation in ostium secundum

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TABLE I  HEMODYNAMIC DATA AND TOTAL ACTIVATION TIME IN OSTIUM SECUNDUM ASD

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Sex</th>
<th>RV pressure (mmHg)</th>
<th>L-R shunt ratio (%)</th>
<th>Total activation time (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>K.Y.</td>
<td>23</td>
<td>male</td>
<td>36/6</td>
<td>82</td>
<td>100</td>
</tr>
<tr>
<td>S.H.</td>
<td>56</td>
<td>male</td>
<td>35/2</td>
<td>68</td>
<td>80</td>
</tr>
<tr>
<td>I.A.</td>
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<td>male</td>
<td>40/5</td>
<td>80</td>
<td>95</td>
</tr>
<tr>
<td>H.Y.</td>
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<td>male</td>
<td>42/0</td>
<td>67</td>
<td>100</td>
</tr>
<tr>
<td>S.N.</td>
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<td>60/8</td>
<td>78</td>
<td>110</td>
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<tr>
<td>E.D.</td>
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<td>male</td>
<td>35/1</td>
<td>40</td>
<td>130</td>
</tr>
<tr>
<td>M.U.</td>
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<td>48/1</td>
<td>72</td>
<td>80</td>
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<td>M.C.</td>
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<td>30/6</td>
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<td>90</td>
</tr>
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<td>S.N.</td>
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<td>80/10</td>
<td>67</td>
<td>120</td>
</tr>
<tr>
<td>M.K.</td>
<td>55</td>
<td>female</td>
<td>45/5</td>
<td>44</td>
<td>105</td>
</tr>
</tbody>
</table>

ASD

In patients with ostium secundum ASD, the total epicardial activation times were 95–130 msec, as compared to the normal value of 60–80 msec. Fig. 8 shows some examples of the epicardial activation sequence of the hearts with ostium secundum ASD. Each patient showed right ventricular hypertrophy in chest x-ray and during operation. In patients A and B, the earliest epicardial activation was recorded 10 msec later than the control group in the anterior paraseptal area of the right ventricle. The activation spread in the right ventricle in a normal fashion, but it was markedly prolonged (95–100 msec). Only half of the right ventricle was activated within 60–70 msec in these patients, whereas the activation was almost completed in the right ventricle of the control group. In patient C, the earliest area of epicardial activation was not located in the right ventricle, but in the left anterior paraseptal area. From the left ventricle the activation front invaded the right ventricular surface both anteriorly and posteriorly, but its pattern was different from a V-shaped pattern of RBBB and the activation speed was not so slow as in muscle conduction which was seen in cases of conduction disturbance. The last epicardial activation was recorded at the inferior area near the atrioventricular groove (110 msec). In ostium secundum ASD, activation delay of the right ventricle was not due to conduction disturbance of the main right bundle branch.

Fig. 9. shows the epicardial activation sequence of the heart with ostium secundum ASD providing left axis deviation on the standard ECG (−10°). The intraoperative diagnosis was ostium secundum ASD with prolapse of the posterior leaflet of the mitral valve, which did not cause mitral regurgitation. The VCG findings were superior orientation of the predominant QRS forces and counterclockwise rotation in the frontal VCG loop, which are the usual diagnostic findings in cases of ostium primum ASD. The earliest epicardial breakthrough occurred at the right and left anterior paraseptal areas and the activation fronts were directed toward the atrioventricular groove. The activation speed was as slow as in muscle conduction and then it increased in the right half of the right ventricle. The total activation time of the right ventricle was markedly prolonged (130 msec). In the left ventricle, the initial epicardial activation was recorded at the anterior paraseptal area and the posterior apex and not at the posterobasal area. Then the activation spread toward the atrioventricular groove in the same sequence as a normal left ventricle. The epicardial activation sequence of this patient suggested conduction disturbance of Purkinje system in right ventricle.

Epicardial activation sequence of other 6 patients resembled to that of A, B or C. The hemodynamic data was shown in Table I. Summarizing the patients with ostium secundum ASD, the total epicardial activation time provided a reasonable correlation with the systolic pressure of the right ventricle, except in the case of left axis deviation.

8. Epicardial excitation of ostium primum ASD

The sequence of epicardial activation of a patient with ostium primum ASD is shown in Fig. 10. The ECG and VCG findings provided left axis deviation (−15°), counterclockwise rota-
Fig. 10. Epicardial activation sequence of ostium primum ASD. Anterior and posterior views are shown. Activation times were measured in msec after the beginning of left ventricular cavity potential. The earliest epicardial breakthrough was recorded in the area indicated by the stippled mark. The lower left figure shows the VCG (top; frontal loop, middle; sagittal loop, bottom; horizontal loop). The lower right figure shows the ECG.

...tion and superior orientation of the QRS forces in the frontal plane. The chest x-ray showed right ventricular hypertrophy which was observed during operation. In epicardial mapping the earliest epicardial breakthrough occurred in the left posterior paraseptal area at 20 msec after the beginning of left ventricular cavity potential. The activation time of this area was 20 msec faster than that of a control group (40 msec). The activation front spread toward the apex and the atrioventricular groove as well as toward the right ventricle. Epicardial breakthrough in the right ventricular surface was recorded in the anterior paraseptal area 10 msec later than that of the left ventricle, after which the activation spread in a circular fashion. Two activation fronts originating from epicardial breakthroughs of both ventricles fused on the posterior area of the right ventricle. The pulmonary conus was activated late at 70 msec, and the last epicardial activation occurred in the posterobasal area of the right ventricle (80 msec). The epicardial activation sequence of ostium primum ASD suggested that activation delay in the right ventricle was not due to conduction disturbance of the right bundle branch. It was considered due to asymmetry of the conduction system characterized by early activation of left ventricular posterior paraseptal area and maybe due to right ventricular hypertrophy.

DISCUSSION

This study in canine hearts showed the activation occurred in both ventricles with initial activation in the endocardium of the terminals of Purkinje system, which was also observed in previous publications. The earliest ventricular activation occurred at the base of the anterior papillary muscle of the left ventricle and slightly later at that of the right ventricle. Early activation of these regions is good for proper ventricular function associated with early papillary muscle contraction to close the mitral and tricuspid valves.
The spread of excitation from the endocardium to the epicardium varied because of the difference of wall thickness and distribution of the Purkinje system. The earliest epicardial breakthrough occurred in the mid-anterior paraseptal area of the right ventricle and the activation spread on the right ventricular surface in a circular fashion. Early epicardial activation of the left ventricle occurred at three different areas, that is, the area near the posterior apex, the anterior paraseptal area continuing the early area of the right ventricle, and the posterior paraseptal area. The activation fronts from these areas fused and formed an irregular pattern and then spread toward the atrioventricular groove.

The activation of the interventricular septum proceeded first from the left side and then from both sides and the final activation of the septum occurred in the center. In the posterobasal region of the septum, the activation proceeded only in a left to right direction. This area behaved electrically as a part of the left ventricle and was probably excited by Purkinje system of the left ventricle.

Generally the sequence of the epicardial activation reflected well the corresponding intramural activation sequence except for that of the interventricular septum.

In the clinical study intraoperative epicardial mapping was hampered by lack of time. The human ventricular epicardial surface was explored at 42–70 sites, which was considered adequate to obtain the sequence of epicardial activation. However, it was not so complete as the canine study, and intramural exploration was not recommended in the clinical study because of the risk of trauma to the myocardium and the conduction system. Epicardial activation sequence of the control group was in agreement with the results of Durrier and associates and it was similar to the canine hearts. There were some differences, however, between ventricular activation in canine and human hearts. The total activation time was longer in human hearts than canine hearts because of the difference of the size of the heart. In dogs, the Purkinje system is more extensive and penetrates more deeply into the ventricular wall than in human hearts, so the activation spreads more rapidly through the Purkinje system. The activation of the interventricular septum occurs predominantly in a left to right direction in human hearts while it occurs from both sides in canine hearts.

Though activation velocity decreased by a cardiomulmonary bypass, the pattern of epicardial activation did not change. The beginning of left ventricular cavity potential coincided with the onset of ventricular depolarization in a such condition, so it was reliable as a time reference in order to compare with the preoperative and postoperative epicardial activation.

The electrocardiographic pattern of RBBB occurs commonly in patients who underwent repair of ventricular septal defect through ventriculotomy, but opinions differ regarding the cause of RBBB. RBBB was due to either trauma (suture placement, hemorrhage, or infarction) to the main right bundle branch or interruption of distal right ventricular Purkinje fibers. Epicardial activation pattern changed following operative correction in these patients. From the results of experimental RBBB in canine hearts, the characteristic pattern of epicardial activation in RBBB due to the damage to the main right bundle branch is summarized as below: (1) The activation of the left ventricle was normal. (2) The epicardial breakthrough at the right anterior paraseptal area was absent. (3) Total activation time was prolonged resulting from the marked delay in the right ventricular activation. (4) The right ventricular epicardial activation showed a V-shaped pattern. (5) The last activation occurred in the pulmonary conus. (6) The activation proceeded slowly in the anterior and posterior paraseptal areas and then proceeded much faster in the right upper area and the pulmonary conus of the right ventricle. Epicardial activation in the human heart with RBBB was similar to that of the canine heart. The right ventricular activation was markedly delayed due to the proximal conduction disturbance and the right ventricle was excited by the impulse of myocardial slow conduction from the left ventricle. These results of RBBB are in basic agreement with the result of van Dam and associates.

Erickson and associates reported that the impulse reached the right ventricle only by slow conduction across the septum. The slow conduction in both anterior and posterior septal areas found in this study assists the muscle conduction across the septum. On the other hand, faster conduction in the right upper area and the pulmonary conus suggests the involvement of a local Purkinje system. A comparison of epicardial activation sequence with the ECG finding suggests that accentuation of the terminal QRS forces in a superior direction which was represented by deep S waves in leads II, III, aVF,
results from the latest activation in the pulmonary conus.\textsuperscript{12}

Right ventriculotomy caused interruption of the peripheral Purkinje system in the right ventricular free wall. Following vertical right ventriculotomy, right ventricular activation showed marked delay at sites distal to the incision. However, they showed no significant delay at sites proximal to it\textsuperscript{17} and epicardial breakthrough was found in the right anterior paraseptal area. Epicardial activation pattern was much different from that of RBBB due to proximal disruption of the right bundle branch. From a comparison of two types of epicardial activation sequence, it was suggested that central right bundle branch injury was able to be differentiated from distal Purkinje system injury. Central injury affected both initial and terminal activation of the right ventricle, whereas peripheral Purkinje injury affected only the terminal activation. Okoroma and associates suggested that these two types of RBBB could be differentiated by changes in the initial vectorcardiographic electromotive forces.\textsuperscript{15}

In the canine study of left ventricular pacing the right ventricular activation was markedly delayed. The epicardial activation sequence showed a radial pattern around the stimulating site, and was different from the pattern of RBBB. The activation spread at a slow speed near the stimulating point and moved rapidly away from it. On the basis of 30 to 50 cm/sec, accepted as the rate for muscle conduction,\textsuperscript{3} secondary involvement of the right ventricular Purkinje system probably played a role in the area of fast conduction.\textsuperscript{28} The activation spread at a faster speed during the endocardial pacing than the epicardial one. These findings also suggest participation of the conduction system.

The epicardial activation sequence of ostium secundum ASD was characterized by the prolonged right ventricular activation.\textsuperscript{18–20} In a previous study of the ventricular excitation of dogs in which atrial septal defects were produced surgically, Boineau and associates reported that hypertrophy of the right ventricular free wall accounted for the prolonged activation time and that there was no disturbance of conduction in the Purkinje system.\textsuperscript{18} In this study epicardial activation of the right ventricle exhibited various patterns. The earliest epicardial breakthrough was found at the right anterior paraseptal area in some hearts but not in others. A and B of Fig. 8 showed normal activation sequence with delay over the entire right ventricular free wall. The epicardial activation sequence of the patient C were different from those of A and B. But also in C, delay of epicardial activation was found over the entire free wall of the right ventricle, not in a part of it. Its activation pattern did not exhibit a V-shaped pattern of RBBB with the last activation in the pulmonary conus. The activation speed was not so slow as in conduction disturbance. Thus the activation delay of the right ventricle in this case was thought a consequence of ventricular hypertrophy.

Generally in ostium secundum ASD activation delay of the right ventricle can be accounted for by ventricular hypertrophy, not by conduction disturbance. It is not certain whether there was a consequence of the stretch of the peripheral Purkinje fibers or not.

The data obtained in this study of ostium primum ASD were characteristic as Durrer and associates have reported.\textsuperscript{23} From the electrocardiographic changes induced in tetralogy of Fallot, Rosenbaum and associates assumed that RBBB with left anterior hemiblock was the cause of the electrocardiographic changes spontaneously occurring in ostium primum defects.\textsuperscript{22} However, the epicardial activation sequence of the right ventricle of ostium primum defect did not exhibit a RBBB pattern, but rather a normal activation pattern with some delay over the entire wall of the right ventricle. In addition, Boineau reported that a dog with ostium primum ASD presented asymmetry of the conduction system. They described that shortening of a posterior division of left bundle and elongation of a left anterior division of left bundle and right bundle branch were due to the posterior-inferior displacement of the conduction system.\textsuperscript{24} The data in this study strongly suggest that asymmetry of the conduction system is the cause of the abnormal epicardial activation pattern in ostium primum defects.

The ECG pattern of superior orientation of the predominant QRS forces with counterclockwise frontal VCG loop has been a diagnostic hallmark of ostium primum defect.\textsuperscript{29} In this study the patient with ostium secundum ASD associated with mitral valve prolapse showed ECG and VCG findings compatible with an ostium primum defect. However, the earliest epicardial breakthrough was absent in the posterobasal area of the left ventricle, and left ventricular activation showed a normal pattern similar to that of the ostium secundum defects. On the other hand, the right ventricular activation provided a pattern

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which was considered the consequence of the conduction disturbance of the Purkinje system. Therefore, the earliest epicardial breakthrough in the posterior surface of the left ventricle was characteristic of ostium primum ASD. No relationship was found between left axis deviation and prolapse of the mitral valve.

SUMMARY

Electrophysiologic mapping was performed on 29 dogs and 35 patients to investigate the ventricular excitation sequence of normal hearts and various cardiac lesions exhibiting the electrocardiographic pattern of right bundle branch block (RBBB). The activation times of epicardial surface were referenced to the onset of left ventricular cavity potential or QRS wave of lead II ECG. Epicardial activation sequence was represented by isochrones.

1. In normal hearts, the earliest epicardial breakthrough occurred at the mid-anterior paraseptal area in the right ventricle and the activation spread in a circular fashion. In the left ventricle, the epicardial activation occurred at three areas and then spread to the posterobasal area. The epicardial activation sequence was a good representation of the ventricular excitation.

2. In RBBB due to trauma to the main right bundle branch, the right ventricular activation showed marked delay and the characteristic V-shaped pattern.

3. Following vertical ventriculotomy, the right ventricular epicardial activation showed marked delay at sites distal to the incision but no significant delay proximal to it. Regarding postoperative RBBB, central right bundle branch injury was able to be differentiated from distal Purkinje injury due to right ventriculotomy by means of epicardial mapping.

4. In left ventricular pacing, the activation spread in a circular fashion with the prolonged right ventricular activation.

5. In ostium secundum defect, the right ventricular epicardial activation sequence showed various patterns of activation delay resulting from right ventricular hypertrophy. In ostium primum defect, the earliest epicardial activation was found in the left posterior paraseptal area, and the right ventricular activation showed a normal pattern with some delay.

Epicardial mapping has been the precise representation of ventricular excitation by direct measurement of cardiac potentials. Cardiac lesions exhibiting the electrocardiographic RBBB pattern provided various patterns of the right ventricular activation delay according to the genuses of RBBB.

REFERENCES

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