Experimental Appraisal for Diagnosis of Right Bundle Branch Block Using the Body Surface Isopotential Maps

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This study was designed to detect reflection of epicardial breakthrough to body surface isopotential maps by recording epicardial and body surface maps simultaneously, and to estimate changes in epicardial breakthrough associated with complete and incomplete blocks induced by compression of the main stem of the right bundle branch or trans-sectioning of the lateral branches.

In the control, epicardial breakthrough appearing on the right ventricular surface was well detected on the body surface maps as a bend of isopotential lines localized at the mid sternum at 11.5 ± 1.6 msec (n = 5) after QRS initiation. At complete block immediately after compression of the main stem, the localized bend shifted inferiorly to the left at 17.8 ± 1.8 msec (n = 5) after QRS initiation, suggesting appearance of the left ventricular epicardial breakthrough. With progression of recovery from the compression, in addition to epicardial breakthrough on the left ventricle, the breakthrough on the right ventricle became detectable again with a delay of 5 msec, and then the former was faded away as time progressed.

After trans-sectioning of the lateral branches, sequential changes in the body surface maps were almost the same as in complete block of the main stem but they lapsed about 43 msec (n = 5) shorter in comparison with the complete block.

In conclusion, detection of the localized bend of the isopotential lines on the body surface can provide diagnosis of the site and degree of the right bundle branch block in detail.

Body surface isopotential maps\textsuperscript{1–3} which are constructed from electrocardiographic data through many lead points up to 400 over the chest surface, have proved successful in providing much topological information on the electrical phenomena of the heart. In particular, sequential analysis of the body surface map patterns can provide information about progression of the activation front over the epicardial surface of the ventricles\textsuperscript{4–6}

Recently, several papers\textsuperscript{7–9} reported the possibility of diagnosis of the block site of the conduction disturbances of the right bundle branch block, focusing on both chronological and topological changes in a localized depression described

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Key Words:
- Body surface map
- Epicardial breakthrough
- Main RBBB
- Lateral branch block

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as a second-minimum or niche.

However, there are few reports which directly correlate the epicardial activation sequences of potentials with the body surface maps.

Therefore, the purpose of this study is firstly to correlate the epicardial potentials directly with the body surface potentials, by analysing the simultaneously recorded epicardial and body surface isopotential maps of dogs, and secondly to determine whether the body surface maps can diagnose complete and incomplete right bundle branch blocks induced by graded injuries to the main stem of the right bundle or trans-sectioning of the lateral branches, i.e., the main branches extended directly from the main right bundle.

METHODS

1. Simultaneous recordings for the epicardial and body surface isopotential maps

Recordings of the epicardial isopotential maps

Five mongrel dogs, weighing 10 to 15 kg, were anesthetized with thiopental sodium 30 mg/kg. Under artificial ventilation, the heart was exposed through mid-sternal thoracotomy. The heart surface was completely covered with a female stocking net attached with insulated fine stainless steel wires. The bare distal end of each wire was used as an electrode for recording epicardial potentials. Thirty-two electrodes of this kind were equi-distantly placed mainly on the anterior surface of the heart, as shown in Figure 1A. The proximal end of each wire-electrode was connected to an input box assembled with 48 channels of a-c amplifiers.

After placement of the 32 electrodes on the epicardial surface of the ventricle, the chest was closed, and then aspirated to preserve the intrathoracic pressure between minus 5 to 10 cm H2O.

The epicardial potentials, which were taken as unipolar electrocardiograms with respect to the Wilsons' central terminal, were amplified 50 fold, and fed into a microcomputer processing system for the body surface map (HPM 5100, Chunichi Electric Corp.). This body surface mapping system can simultaneously acquire data of 96 channel electrocardiograms for 250 msec at a sampling interval of 1 msec and construct equipotential maps as well as scalar electrocardiograms of the standard leads and Frank lead system.

From the data processed by this system, epicardial potentials were displayed as equipotential maps on a cathode ray tube combined with a hard copy unit (Tektronic 4631).

Recordings of body surface isopotential maps

Stainless steel needle electrodes for recording the precordial electrocardiograms were attached on 81 points equidistantly located over the anterior chest, illustrated as Figure 1B. The chest
Fig. 2. Compression of the main stem and trans-sectioning of the lateral branches of the right bundle branch.

Left panel: schematic illustration of the course of the bundle branches, right panel: photograph of the endocardial surface of the right ventricle. The main stem of the right bundle branch was compressed at the level just proximal to the base of the papillary muscle as indicated by the upper single arrow in the right panel. The lateral branches were trans-sectioned at the level indicated by the lower two arrows.

potentials, with Wilsons’ unipolar lead system, were obtained with two input boxes, each of which contains 48 channels of a-c amplifiers for electrocardiograms. The amplified electrocardiograms were processed in the same way as those from the epicardial surface by another mapping system of the same kind connected synchronously to the system for processing the epicardial potentials so as to record both epicardial and body surface potentials simultaneously and correlate both with each other.

2. Production of the right bundle branch block and construction of the body surface maps

Forty mongrel dogs were anesthetized and thoracotomized as described above. Twenty of these animals were used for production of the proximal right bundle branch block by compressing the main stem of the right bundle, and the other twenty animals for production of the distal block by sectioning the lateral branches, i.e., the distal branches divided from the main stem of the right bundle branch, as illustrated in Figure 2.

Transient block of the main stem of the right bundle branch

The main stem of the right bundle branch was compressed at the level just proximal to the base of the anterior papillary muscle indicated by an arrow in Figure 2 by use of a long curved cotton swab which was introduced into the right ventricular cavity via the right atrium. Completeness of conduction block was confirmed by electrocardiographic findings compatible with those obtained by the complete interruption of the main stem.

Trans-sectioning of the lateral branches

After sub-dividing from the main stem of the right bundle branch at the base of the anterior papillary muscle, the lateral branches traverse the right ventricular cavity, forming as false tendons, and attach to the endocardial surface of the free wall, as illustrated in Figure 2.

Trans-sectioning of the lateral branches was accomplished by cutting all the false tendons, using a narrow, flat bladed scalpel, sharpened at the tip only, and introduced trans-atrially into
Fig. 3. Anterior view of the electrode placement for recording the total thoracic potential distribution.

The anterior thorax was divided horizontally into 7 levels along the mid sternal line, and longitudinally into 7 lines. Solid circles denote the positions of the respective electrodes for recordings of electrocardiograms.

the right ventricular cavity. The completeness of the trans-sectioning was confirmed on the dissected heart after the experiment.

**Placement of the electrodes and construction of the body surface maps**

Placement of the electrodes for the body surface isopotential maps were different from that shown in Figure 1, but the same as previously reported. Figure 3 shows the anterior aspect of the electrode placement on the chest. Recordings of the body surface potentials and construction of the isopotential maps were performed as described above.

**RESULTS**

**Relationship between epicardial and body surface isopotential maps**

The effect of the operation of simple thoracotomy on the body surface potential distribution was examined first in the control experiments using 3 dogs.

The body surface isopotential maps recorded on the closed chest after simple thoracotomy were essentially similar to those before thoracotomy, even when the intra-thoracic pressure was maintained within the range stated before by aspiration.

Figure 4 shows a typical example of the simultaneous recordings of epicardial and body surface potentials, focusing on the phenomenon of the epicardial breakthrough of the ventricular activation front on the right ventricle. In this figure, the sequential changes in both isopotential maps on the anterior surface of the right ventricle and those on the corresponding precordium are correlatively illustrated at 12 to 17 msec after the initiation of the ventricular excitation determined from the left ventricular cavity potential. The top tracing of this figure shows the unipolar QRS complex on the lead point indicated by the solid circle on the schematic illustration of the heart. At 12 msec after initiation of the ventricular activation, the anterior surfaces of both right ventricle and chest were positively charged, suggesting reflection of progression of the activation front through the ventricular septum to the direction from the left to the right endocardial surface.

However, within the next one millisecond, a localized area surrounding the solid circles on the schema suddenly reduced its positive potential, and then was replaced by a negative potential (see the map at 14 msec), indicating occurrence of breakthrough of the activation front on the epicardial surface of the right ventricle.

The negative area associated with the epicardial breakthrough increased in size and amplitude, resulting in development of a new minimum at the point corresponding to electrode D7 on the body surface map at 17 msec after initiation of the ventricular activation. However, if the distributed potentials around electrode D7 are retrospectively examined, a precursory sign of the new minimum is observable as a localized bend of the equipotential lines around the electrode as early as 14 msec, i.e., the time of the epicardial break-through, indicated by arrows in the respective maps of Figure 4. Thus, it was proved in this experiment that the time of the epicardial breakthrough could be recognized indirectly on the body surface maps by detecting the beginning of the bend of isopotential lines which developed finally into a new minimum.

*Japanese Circulation Journal Vol. 45, April 1981*
Fig. 4. Simultaneous recordings of distribution of epicardial and body surface potentials. Sequential changes in the epicardial potential distribution on the anterior surface of the right ventricle are depicted on schematic illustrations of the heart (middle panels). Each equipotential line is depicted at an interval of 1 mV and development of the negative area, i.e., breakthrough of the activation front to the epicardial surface of the ventricle is illustrated as a shaded area.

Potential distribution on the anterior chest (bottom panels) is represented by isopotential lines drawn at an interval of 0.1 mV. The vertical line crossing point D correspond to the mid sternal line. Top tracing illustrates QRS complex recorded from the lead point of the ventricular surface marked by a solid circle in the schematic diagram. In accordance with the intrinsic deflection of the QRS complex, the breakthrough develops on the right ventricle, and correspondingly a localized bend of isopotential lines appeared as indicated by arrows.

**Transient block of the main stem**

In all 20 animals used for this experiment, we succeeded in producing the complete block of the main stem of the right bundle branch. However, in 15 of them the block was either permanent during experiment or completely recovered before completion of recordings of the body surface potentials. Therefore, analysis of the remaining 5 animals was undertaken (Table 1). Figure 5 shows a typical example comparing differences in the sequential changes of the localized bend of the isopotential lines (the reflection of the epicardial breakthrough to the chest surface) on body surface maps taken before and immediately after compression of the main stem and during recovery from the compression.

In the control, as is shown by arrows in the top row in Figure 5, the localized bend of the isopotential lines, which reflects the breakthrough of the activation front on the epicardial surface of the right ventricle, began to appear around electrode E4 at 12 msec after initiation of the ventricular activation, and developed into a new minimum within a few milliseconds.

Immediately after compression of the main stem of the right bundle branch with a cotton swab, the QRS of lead V1 changed from an RS configuration in the control to a wide notched R with a small initial component followed by a tall late component, which is compatible with the QRS pattern of the complete right bundle branch block obtained after trans-sectioning of the main stem.5,13,14

During complete block of the main stem, the body surface isopotential maps changed significantly from those of the control. As is shown in the second row of Figure 5, no potential changes reflected from the epicardial breakthrough of the right ventricle were discernible but, in place of them, a localized bend of isopotential lines began to appear around electrode H2 at 17 msec after initiation of the ventricular excitation and developed into a new minimum at 20 msec, which corresponds to the localized potential depres-
**TABLE I DETECTION OF EPICARDIAL BREAKTHROUGH ON BODY SURFACE MAPS**

<table>
<thead>
<tr>
<th>Case</th>
<th>Appearance Time of Breakthrough</th>
<th>QRS Prolongation (% increase)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RV (msec)</td>
<td>LV (msec)</td>
</tr>
<tr>
<td>Control (n = 5)</td>
<td>11.8 ± 1.6</td>
<td>—</td>
</tr>
<tr>
<td>CRBBB (n = 5)</td>
<td>—</td>
<td>17.8 ± 1.3</td>
</tr>
<tr>
<td><strong>IRBBB, advanced</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. 1</td>
<td>—</td>
<td>21</td>
</tr>
<tr>
<td>4</td>
<td>—</td>
<td>19</td>
</tr>
<tr>
<td>4</td>
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<td>17</td>
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<tr>
<td>5</td>
<td>—</td>
<td>20</td>
</tr>
<tr>
<td><strong>IRBBB, mild</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. 3</td>
<td>16</td>
<td>17</td>
</tr>
<tr>
<td>3</td>
<td>15</td>
<td>18</td>
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<td>2</td>
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<td>5</td>
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</table>

**Lead V1**

Fig.5. Body surface isopotential maps reflecting the epicardial breakthrough in complete and incomplete block due to compression. From top to bottom, QRS complex and development of localized bend of isopotential lines reflecting the epicardial breakthrough in the control, complete and incomplete block of the main stem. As indicated by arrows, in the control, the bend of isopotential lines, reflecting the right ventricular epicardial breakthrough, appeared around electrode E4 at 12 msec after QRS initiation, in incomplete block it shifts downward to the left (electrode H2) with a delay of 5 msec, indicating that disappearance of the right ventricular epicardial breakthrough unmasks the breakthrough of the activation front to the left ventricular epicardial surface, and in incomplete block, the maps suggest co-existence of both right and left ventricular epicardial breakthrough.

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**Japanese Circulation Journal Vol. 45, April 1981**
as is shown in the bottom row of Figure 5. In addition to the distorted isopotential lines around electrode H2, a potential depression localized around electrode E4 reappeared at 19 msec after initiation of the ventricular activation, indicating that the right ventricular epicardial breakthrough occurred with a delay of 7 msec from the control. This delay might be attributed to incomplete recovery of the impulse conduction along the compressed region of the main stem.

Table I summarizes the appearance time of the epicardial breakthrough determined by the localized characteristic bend of the equipotential lines on the body surface maps.

In the early phase of recovery of the impulse conduction along the main stem after the compression, the epicardial breakthrough was not detectable on the right ventricle but that on the left ventricle was observable in the same way as in complete block of the main stem, and QRS duration increased by 25 to 50 percent of the control.

However, with progression of recovery of the impulse conduction along the main stem from the compression, the right ventricular epicardial breakthrough became detectable with a delay of several milliseconds in addition to the left ventricular epicardial breakthrough, which finally faded away from the body surface maps when the QRS prolongation was restored to 15 percent or less of the control.

Figure 6 illustrates the potential distributions on the anterior chest in the mid and late phases of the ventricular excitation of the same case as shown in Figure 5, respectively. In the control, in both mid and late phases the negative potential covered the left anterior chest and the positive potential the left lateral chest. During complete block of the main stem, the left lower part of the chest showed negative potential and the right upper part positive potential, both of which were increasing in amplitude with time, suggesting that the activation front progressed through the right ventricular free wall late in the ventricular activation.

On the body surface isopotential maps during recovery of the main stem from the compression, their potential distribution in the mid phase showed an intermediate pattern between those during the control and the complete block but
that in the late phase was similar to the control pattern.

Sectioning of the lateral branches
The lateral branches traverse the right ventricular cavity, forming free-running false tendons, and attach to the endocardial surface of the right ventricular free wall.

Figure 7 illustrates a typical example comparing changes in the QRS complex of lead V1 and the body surface maps before and after complete trans-sectioning of the false tendons. The right ventricular epicardial breakthrough, which were observed around electrode E4 at 13 msec after initiation of the ventricular activation, disappeared after trans-sectioning of the lateral branches, and, in place of it, a new localized bend of the isopotential lines consistent with the left ventricular epicardial breakthrough became discernible around electrode F3 at 17 msec after initiation of the ventricular activation. In the late phase of the ventricular activation the potential distribu-
tion manifested a pattern similar to that observed in complete block of the main stem of the right bundle branch as well as in the early to mid phase of the ventricular activation. However, the potential patterns compatible with those in complete block of the main stem were completed within 44 msec after initiation of the ventricular excitation, as revealed by the duration of the QRS complex depicted in Figure 7.

Table II summarizes results of the successfully transsected 5 cases. In all cases the epicardial breakthrough shifted to the left ventricle as in the complete main stem block but percent prolongation of QRS duration was only increased by an average of 36 percent.

DISCUSSION

Detection of the epicardial breakthrough on the body surface isopotential map

The breakthrough of the ventricular activation front on the epicardial surface of the ventricle results in a sudden inversion of the polarity of the epicardial potential distributed over the breakthrough area\(^4\,5\,11\,15\). These sudden changes in the epicardial potential distribution is transmitted to the body surface through the volume conductor of the complex of intervening tissues including the lung, muscle and subcutaneous adipose tissue, and necessarily affects the distribution of the body surface potential.

Taccardi\(^4\,16\) who was interested in the reflection of the breakthrough to the body surface potential maps, first observed that in accordance with the breakthrough a localized depression of the body surface potential described as a second-minimum, saddle or niche, appeared on the limited area of the chest wall which directly covers the epicardial breakthrough area on the right ventricle.

These observations have been replicated by several investigators\(^5\,6\,8\,11\) but it has remained undetermined as to what extent the pattern of the localized potential depression might change in association with progression of the epicardial breakthrough.

Therefore, in this experiment the isopotential maps were simultaneously recorded from both epicardial and body surfaces in order to determine precisely the relationship between expansion of the breakthrough and resultant potential changes in the body surface maps. As shown in Figure 4, the breakthrough of the ventricular activation front on the antero-inferior part of the right ventricle was detected on the body surface as a localized depression on the isopotential map, patterns of which changed from niche, a simple bend of the equipotential lines, to development of a new minimum as the niche, a simple bend of the equipotential lines, to development of a new minimum as the ventricular activation progressed. Therefore, if inspection is performed on the body surface maps in retrospective sequence, focussing on the area of the maps where a new minimum develops in later stages, the examiner can determine initiation of the epicardial breakthrough by detecting a bend in isopotential lines around the focussing point.

Boineau et al\(^5\) reported that the area of the breakthrough at least 2 cm\(^2\) in size was needed for production of any discernible potential depression on the body surface. In all cases in this experiment, the breakthrough extended over 2 to 3 cm\(^2\) on the epicardial surface within one millisecond after occurrence. The reason why the epicardial breakthrough develops so rapidly may be explained from results of the experiment in which the activation sequence on the right ventricular endocardium was measured in detail on the superfused preparation dissected from the isolated canine heart\(^17\). The ventricular activation was initiated on the endocardial surface of the inferior part of the paraseptal region of the right ventricular free wall by the impulse conducted through the lateral branches, i.e., the direct extension of the main bundle branch. The ventricular activation extended over the endocardial surface about 2 cm\(^2\) within one millisecond. Therefore, it is concluded that such a sudden widespread breakthrough as is shown in Figure 4 may develop through the direct progression of the activation front elicited by the impulse through the lateral branches.

Diagnosis of conduction block of the main stem

Right bundle branch block is usually diagnosed\(^18\) by the morphology of the QRS complex on the right precordial unipolar electrocardiograms, but neither standard 12 leads nor vector cardiographic leads can provide any information about the site of block, only the former providing conventional criteria to classify the degree of the block into “complete” and “incomplete”, and the latter proving presence of a delayed, slow progression of the terminal ventricular activation, which is suggested by the terminal bunchings.

The body surface maps, as described previously, can directly reflect the epicardial potential distributions, in particular those associated with the right ventricular epicardial breakthrough, which is elicited through direct progression of
the impulse through the lateral branches, i.e., the direct extension of the main right bundle branch.

Sugenoya et al. proved experimentally that after trans-sectioning of the main stem of the canine right bundle branch, the localized potential depression, i.e., the reflection of the epicardial breakthrough to the body surface changed its location from the mid sternal line to the left anterior chest surface, resulting from shift of the epicardial breakthrough from the right ventricular surface in the control to the left ventricular surface after the trans-sectioning. Clinically, Taccardi et al. and Sugenoya attempted to classify the human right bundle branch block by the characteristics of the body surface maps, after confirming that all cases manifesting the QRS complex wider than 0.12 sec were diagnosed as main stem block of the right bundle branch as a result of demonstration the leftward shift of the breakthrough.

However, what pattern of the body surface maps might develop in the incomplete block of the main stem has not been reported. The iso-potential maps taken during recovery from the complete block of the main stem of the right bundle branch were classified into two groups by the difference in appearance of the potential depression reflected from the epicardial breakthrough as shown in Table I. In the first group, the advanced incomplete block of the main stems, the epicardial breakthrough of the right ventricle is not yet discernible and that of the left ventricle is still detected on the body surface maps namely as in the complete block, suggesting that the body surface maps in the early to middle stage of the ventricular activation might manifest patterns similar to those during the complete block. However, the maps in the late stage may manifest a variety of patterns in the range between those from the control and the complete block, depending on QRS prolongation, a parameter estimating the degree of the conduction delay in the right ventricle. In the second group, the mild incomplete block of the main stem, the right ventricular epicardial breakthrough becomes discernible again, showing a delay of 3 to 7 msec. In three cases having around 20 percent increase in the QRS duration, both right and left ventricular epicardial breakthroughs are discernible as is shown at the bottom of Figure 5, suggesting that the body surface maps manifest a mixture of the control and complete block patterns in the early to mid stage of the ventricular activation and the control pattern in the late stage.

Therefore, it is concluded that analysis of the body surface maps, focussing on the epicardial breakthrough in the early stage of the ventricular activation and the distribution of the positive and negative potentials, makes it possible to diagnose not only the complete but incomplete block of the main stem of the right bundle branch.

**Trans-sectioning of the lateral branches**

Uhley et al. after confirming that the main right bundle of the dog spreads into three peripheral branches, consisting of the anterior primary branches, the lateral branches and posterior primary branches, examined changes in the configuration of the QRS complex induced by incision of the lateral branches. The incision resulted in an increase in the amplitude of the superiorly orientated terminal forces by 30 percent without any significant increase in the QRS duration. The effects of the dissection of the lateral branches on the vector cardiograms were also investigated by Moore et al. who observed that the vectors at 8 to 16 msec after initiation of QRS loop were shifted superiorly to the left with an increase in the QRS duration of less than 10 msec.

In this experiment, electrocardiographic changes involving an increase in amplitude of the S wave and QRS prolongation of 10 to 15 msec are compatible with those reported previously. The body surface maps after incision of the lateral branches are comparable to those of the complete block of the main stem, but they develop so rapidly that they terminate about 43 msec shorter in comparison with the complete block.

**Clinical implication**

Among a variety of conduction disturbances which may complicate following repair of ventricular septal defects and tetralogy of Fallot, the right bundle branch block pattern develops most frequently but its clinical importance has not been determined yet. Some papers insist that the surgical right bundle branch block is one of important warning signs against sudden death or development to complete A-V block, in particular if it complicates the left anterior hemi-block, and others deny its prognostic importance.

The reason why the argument has not been settled yet may be ascribed to the difficulty in investigating the causes of the surgical blocks. The surgical right bundle branch block may result from 1) trauma to the main stem of the

right bundle branch associated with closure of the ventricular septal defect, 2) disruption of the lateral branches and the terminal Purkinje network due to the right ventriculotomy and 3) damage to peripheral Purkinje network following infundibular resection. 

Therefore, the prognostic value of the surgical right bundle branch block may be clarified if the patients are followed up after the site of conduction block is differentiated. At present, construction of the epicardial activation sequence of the right ventricle during operation or recordings of the right ventricular apical electrogram by use of catheter electrodes have proved successful in classifying the surgical right bundle branch blocks into the proximal block due to damage to the main stem of the right bundle branch or the distal block owing to disruption of the peripheral Purkinje network. Hattori et al. recently correlated the body surface maps with the epicardial activation maps in patients with surgical right bundle branch block, and suggested that the body surface maps could make differential diagnosis between the proximal and distal blocks of the right bundle branch as well as the epicardial activation maps.

Therefore, it is concluded that the body surface maps can diagnose the block site of right bundle branch block in detail.

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