Interpretation of the Body Surface Isopotential Maps of Patients with Right Bundle Branch Block

Determination of the Region of the Delayed Activation within the Right Ventricle

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Summary

Body surface isopotential maps were produced by computer processing of the 85 electrocardiograms obtained from the entire thorax of 28 patients with complete or incomplete right bundle branch block (RBBB).

We divided the map patterns into the following 3 groups. Type I map pattern (10 cases): at the early stage of QRS, the maximum was located in the left chest. It shifted to the left from the normal position; at the instant of 44 msec, on the average, after the onset of QRS breakthrough minimum appeared over the left chest. Its appearance was delayed and its site shifted to the left as compared with the normal; at the late stage, the positive zone covered extensively the right chest and the right back; terminally, the maximum was positioned along the right parasternum. Type II map pattern (13 cases): at the early stage of QRS, the maximum was in the left chest as in Type I; breakthrough minimum appeared at 38 msec on the average, later than in the normal, but the site of breakthrough minimum varied from the left chest as in Type I to the midsternal region as in the normal; at the late stage, the positive zone covered the upper part of the right chest and the right back, less extensively than in Type I; the terminal maximum was in the upper sternal region. Type III map pattern (5 cases): the map pattern passed normally until the late stage, but thereafter a small positive zone survived over the upper sternal region.

In Type I the delayed activation was presumed to occur all over the right ventricle, in Type II mainly over the smaller area of the right anterior free wall, and in Type III over the localized area of the outflow tract.

Patients with complete RBBB showed Type I pattern. Patients with incomplete RBBB showed Type II or Type III pattern, although electrocardiograms failed to differentiate Type II patients from Type III patients. These findings suggest that the electrocardiographic pattern of incomplete RBBB probably arises from the various mechanisms.

Additional Indexing Words:
Activation sequence  Breakthrough  Breakthrough minimum
Latest activation  Terminal maximum  Main right bundle branch
THE diagnosis and the classification of right bundle branch block (RBBB) has been made by means of the electrocardiogram (ECG) or the vectorcardiogram. Nevertheless, these methods are, more or less, unsuitable for the detection of the electrical activity of the right ventricle (RV), because the recording points of these methods are fewer in the right side of the thorax than in the left.

In recent years the technique of body surface isopotential mapping has been developed. Body surface isopotential map (surface map) is constructed from a large amount of ECG data obtained from many recording points distributed over the entire thorax. It is therefore considered that surface maps may represent well the activity of individual parts of the heart, and hence will be more suitable for the detection of any change in the RV activity than any other conventional methods. In fact, the studies performed in animals and in human beings have demonstrated that the map pattern is related closely to the intraventricular activation sequence. Tacchetti et al. have suggested that the surface maps may enable the appreciation of the region or the extent of the delayed activation in RBBB hearts, and we have recently verified this experimentally.

We analyzed the map patterns of 28 patients with RBBB. The purpose of this paper is to determine through the analysis the region of the delayed activation in human RBBB and to examine the correlation between the map and ECG pattern.

**Materials and Methods**

Clinical materials (Table I):

This investigation was performed on 28 patients whose ECG indicated the RBBB pattern which satisfied the criteria described below and showed the absence of abnormal axis deviation (axis of less than -30°, more than +110°) and furthermore was not associated with any other electrocardiographic abnormalities. All patients were interpreted to be free from any overt cardiovascular disease by the routine examinations, i.e., physical examination, cardiac X-ray film, auscultation etc. Ages ranged from 20 to 62 years with an average of 42 years.

As a control group, 10 healthy adults with normal ECG (aged from 28 to 36 years, with an average of 32 years) were included.

ECG criteria:

RBBB was diagnosed on the basis of the presence either of, 1) a late R wave (R’ or r’), or 2) a notched S wave in the right precordial lead. This criteria is in accordance with the Sodi-Pallares. RBBB was also divided according to the conventional classification by QRS duration; complete RBBB (CRBBB) with that of 0.12 sec or more, and incomplete RBBB (IRBBB) with that less than 0.12 sec.

Body surface isopotential mapping:

Details of the method utilized were described previously. In summary,
ECGs were recorded from the 85 recording points arranged over the entire thorax; the highest row of recording points passed the level of the suprasternal notch, and the bottom row passed approximately the level of the navel. All data were recorded on a magnetic tape and processed by a mini-computer. The surface maps were produced for every 1.5 (or 3) msec during QRS phase. The distribution of positive

Table I. ECG Findings in Patients with RBBB

<table>
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<tr>
<th>Pt</th>
<th>Age</th>
<th>Sex</th>
<th>QRS duration (sec)</th>
<th>Shape of $V_1$</th>
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<th>Wide S and deep S* (wide and deep S**)</th>
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Type II Patients

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Type III Patients

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<td>25</td>
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<td>rS (notched S)</td>
<td>aVR, V2</td>
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<td>26</td>
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<td>Mean</td>
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and negative zone, the locations of maximum and minimum, and the time course of change of these features were analyzed.

**RESULTS**

The map patterns of 28 patients could be classified into 3 groups (Type I, Type II, and Type III).

**Surface maps of Type I** (Fig. 1 A, B, C, D, E, and F)

All of 10 cases with CRBBB displayed this pattern. Fig. 1 shows a representative time course of Type I map pattern. Surface maps obtained at the instant of 10, 25, 40, 55, 100, and 120 msec after the onset of QRS are presented. The left half of the map represents the anterior chest surface

![Fig. 1](image)

Fig. 1. The typical time course of Type I map pattern. A to F show the surface map obtained at the instant of 10, 25, 40, 55, 100, and 120 msec after the onset of QRS, respectively. In the right upper inset is shown the QRS complex of lead $V_1$, expressing the instants at which the surface maps were obtained. In the right lower inset are the conventional ECGs of the same patient. *See text.*
and the right half the back; the interrupted line illustrates the zero line (the potential of Wilson’s central terminal) and the solid lines denote isopotential lines for each 0.4 mV.

At the stage of 10 msec after the onset of QRS (A), the positive potential zone, which is illustrated by a shaded area, covered the entire anterior chest surface and negative potential zone, which is illustrated by a white area, covered the back. The maximum potential (marked by a plus sign) was located over the left chest; it shifted to the left as compared with the normal. The minimum potential (a minus sign) was seen in the right back.

At the stage of 25 msec (B), negative potential invaded also the right chest and the positive potential remained mainly on the left chest. A maximum was located in the left chest and a minimum was in the right. This minimum had its origin in the back minimum seen in the previous stage (10 msec).

At 40 msec (C), another minimum newly appeared over the left chest (see arrow); 2 minima existed at this stage. In association with the appearance of this new minimum, an area of relatively higher potentials developed between the 2 minima, and then the zero line projected into the left lower chest and encompassed the minimum. These phenomena have been described “saddle” and “niche”, respectively. It has been demonstrated that such a minimum is related closely to the intracardiac event of epicardial breakthrough, which occurs when the ventricular excitation has reached initially the epicardial surface. Therefore, we will call this mini-

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Fig. 2. The sites (A) and the times (B) of the occurrence of breakthrough minimum. Normal cases (○), Type I patients (●), Type II patients (▲), and Type III patients (●). LAL = Left mid-axillary line, RAL = right mid-axillary line. * p<0.001, ** N.S.
maximum as a breakthrough minimum hereafter. In the normal subjects, breakthrough minimum appeared near the midsternal region at the instant of 26 msec on the average (SD=4.0, range of 18 to 30 msec) after the onset of QRS, as indicated by open circles in Fig. 2. Meanwhile, in 8 cases of Type I it appeared over the left chest at 44 msec on the average (SD=6.8, range of 36 to 57 msec), as indicated by black squares in the same figure; its appearance was delayed significantly (p<0.001) and its site was to the left.

At 55 msec (D), the positive zone occupied abnormally the right chest and the right lower back. A maximum was located in the right parasternal region, whereas a minimum was observed on the left chest. Thereafter until the end of QRS (139 msec) through 100 msec (E) and 120 msec (F), the extent of positive potential remained virtually unchanged. The positivity over the right chest, however, became largest at the stage of 100 msec (E). Also, at the terminal stage of QRS, the maximum remained in the right parasternal region. In all patients with Type I the terminal maximum was consistently noted in the right chest, as shown by black squares in Fig. 3.

Surface maps of Type II (Fig. 4 A, B, C, D, E, and F)

Fourteen out of 19 IRBBB patients showed this pattern. At 10 msec (A), the positive zone covered the left upper chest and the left upper back. This distribution seems to be somewhat different from that of Type I. However, the maximum was situated in the same location as in Type I. At 25 msec (B), the maximum had moved slightly to the left. The minimum was positioned in the right upper chest. This minimum hereafter disappeared.

Instead of this minimum, breakthrough minimum appeared at 42 msec (see arrow in C). In 9 cases breakthrough minimum could be observed
Fig. 4. The typical time course of Type II map pattern.

(black triangles in Fig. 2). The locations where this minimum appeared varied widely, from the left chest as seen in Type I to the midsternal region as seen in the normal. The time when the minimum appeared averaged 38 msec after the onset of QRS (SD=3.8, range of 34 to 42 msec), and was significantly later (p<0.001) than in the normal, but significantly earlier (p<0.001) than in Type I.

At 55 msec (D), the positive zone occupied the right chest and the right back. A maximum was present over the right chest; a minimum was over the left. This pattern had much similarity to that of Type I. Subsequently, the maximum, unlike Type I, moved upward and slightly to the left, until it reached the upper part of the sternum (70 msec, E). (In some cases, this maximum maintained its upper sternal position even after the stage of 55 msec, and its subsequent movement was not observed.) At the same time, the positive zone diminished in extent and remained only over the upper chest; the zero line tended to travel transversely. A similar pattern persisted until the end of QRS (84 msec, F).
In this group, the terminal maximum was located in the upper sternal region (black triangles in Fig. 3), higher and shifted to the left as compared with the Type I cases, but similar or somewhat lower as compared with the normal.

**Surface maps of Type III (Fig. 5 A, B, C, D, and E)**

Five out of 19 cases with IRBBB exhibited this pattern. At 10 msec (A), positive potentials were evident mainly over the left chest. A maximum was normally located in the left chest; a minimum was in the upper back. At 25 msec (B), 2 minima were apparent; the lower one (see arrow) was breakthrough minimum. In Type III, this minimum appeared near the midsternal region at 24 msec in average (SD=3.0, range of 21 to 27 msec) as shown by closed circles in Fig. 2. No significant delay nor shift could be observed as compared with the normal.

At 40 msec (C), the negative zone expanded to cover almost entirely the anterior chest, whereas the positive zone extended over the back. A maximum was now located in the left subaxillary region. Subsequently it moved to the back (55 msec, D). Another maximum, at this time, occurred in the

![Fig. 5. The typical time course of Type III map pattern.](image-url)
upper sternal region; positive potentials appeared over the upper chest as well as the back. Thus the map pattern of Type III passed through quite normally, from the onset of QRS until the stage of 55 msec. Subsequently, in the normal, both the positive and the negative potential became smaller and approached zero, while in Type III, the positive potential became larger only over the upper chest with the survival of a small positive zone, as seen in 70 msec (E).

In this type, terminal maximum was located in the upper sternal region (closed circles in Fig. 3), almost the same region as seen in Type II.

Relationship between the surface map and ECG (Table I)

QRS duration: In cases showing Type I pattern, QRS duration ranged from 0.12 to 0.16 sec (mean±SD=0.14±0.014), and all of these belonged to CRBBB according to the ordinary ECG classification. In Type II cases it ranged from 0.08 to 0.11 sec (0.09±0.011) and these belonged to IRBBB. And, in Type III it ranged from 0.09 to 0.10 (0.09±0.004) and these fell within IRBBB. A significant difference was noted between the groups of Type I and Type II or III (p<0.001), but not between the groups of Type II and Type III.

Shape of QRS complex of V1: An rsR', rSR', and rR' pattern were noted in Type I cases; an rsr', rSr', Rsr', and rS with a notching in S wave in Type II; an rsr' and rS with a notching in S wave in Type III. An R' wave was present characteristically in Type I, and an r' wave or notching of S wave was evident both in Types II and III.

Late R wave: The delayed activation of RV due to RBBB would produce a late R wave (R' or r') in leads facing the RV, and a wide or deep S wave in leads facing the left ventricle. Herein, we analyzed the development of late R wave. In Type I group, a late R wave was present in leads aVR and V1 in all cases, and also in lead III or V2 in many cases. Whereas in Type II group, it was noted in lead V1 as well as aVR in most cases, and also in lead V3 and V5 in several cases, and further in lead III in a few cases. And, in Type III group it existed in some leads of V1, V3, and aVR. Thus no appreciable correlation was observed between the appearance of late R wave and the type of map pattern, especially Types II and III.

Wide or deep S wave: The presence of wide and deep S waves was analyzed; in the present study the former abnormality was defined when the duration of the S wave is more than 0.04 sec14 and the latter was when the amplitude of the S wave is greater than the upper limits of the normal value15 which was determined from the percentile distribution (97.5 percentile).

In almost all patients with Type I, a wide S wave developed with a notching in leads I, aVL, V5, and V6. In 5 out of 13 patients with Type
II, a deep S developed in some of leads I, II, V5, and V6; in 2 out of these 5 the S wave was also widened abnormally. In 3 out of 5 cases of Type III, a deep and wide S developed in leads II, aVF, V5, and V6. Thus, the appearance of abnormal S waves was also independent of the type of map pattern, especially Types II and III.

**DISCUSSION**

1. The region of the delayed activation and the site of the conduction block in RBBB

The surface potentials are influenced by many factors; the generator activity, the shape of the torso, the position of the heart, the conductivity of the medium within the torso and so on. As for our materials, the influences from the latter 3 factors could be negligible. Therefore it can be accepted that the varied pattern of surface map observed in our RBBB patients represents the varied type of the intracardiac activity. It is of importance that the surface map may represent mainly the myocardial activity, but not directly the activity of the conduction system.

In previous work, we produced RBBB experimentally by cutting the main right bundle branch (RBB) of the dog, and the surface maps and the epicardial activation sequence before and after RBBB were obtained. Between the epicardial and the surface events, the following relationships were observed. 1) When the RBB was cut, breakthrough shifted from the anterior epicardium of the RV free wall to the anterior epicardium of the left free wall, and its occurrence delayed. This change of breakthrough resulted in a shift of breakthrough minimum from the midsternal region to the left chest, and in a long delay of the occurrence. 2) When the RBBB was cut, the RV excitation was delayed markedly. As an effect of the delay, a positive zone developed over the right chest during the late stage of QRS. 3) The localization of the latest activation was represented on the location of the terminal maximum. These relationships were extrapolated to the results of the human mapping.

Type I: First, breakthrough minimum appeared in the left chest. It shifted to the left as compared with that in the normal, and delayed in appearance (Fig. 2). This resembles the results in the RBBB dog, suggesting that in this type breakthrough occurs over the left anterior wall, in other words, there is an activation delay in the right anterior free wall, where breakthrough normally occurs.

Second, at the late stage of QRS, the positive zone extended over the right chest and the right back (Fig. 1, D to F). Furthermore, this zone
remained there in Type I for about 60% of the total QRS duration. These findings resemble those in the RBBB dog.6)

However, a minor difference can be recognized as to the position of the terminal maximum. In Type I, it was located in the right chest at the level of the 4th or 5th intercostal space (Fig. 3), while in the RBBB dog it was located at the 1st or 2nd intercostal space.6) This discrepancy, however, can be readily explained on the basis of the structural or the positional difference between the canine and the human heart. In the RBBB dog, the latest activation occurred in the anterolateral base of RV.6) Since the heart is positioned much more horizontally in man than in the dog, the latest activation of that part, if present in man, is expected to be represented in much lower portion of the map than in the dog. This predicted portion is consistent with the observed location in Type I. Thus, we concluded that the serial maps of the Type I case correspond to those of the RBBB dog. This conclusion implies a similarity between the intraventricular activation sequence of the Type I case and that of the RBBB dog. In our RBBB dog,6) the right free wall excitation initiated at the apical region (mostly in its posterior wall). Subsequently, the wave front parallel to the anterior interventricular groove progressed upward and to the right over the right anterior free wall, and finally reached the anterolateral base,16) which is most distant from that groove; a remarkable delay of activation was noted over the entire RV.

Most recently, van Dam et al reported the epicardial map obtained from a patient with CRBBB.17) Interestingly their data agree with the activation sequence of Type I which we described above. Herein, we believe that such an extensive delay may arise either from the lesion localized in the main RBB such as we produced in the dog, or from the extensive lesion of the right Purkinje network.18) However, from the surface data, no more information than this could be provided concerning the cause or the mechanism of this type.

Type II: In this type, breakthrough minimum appeared later than in the normal, but earlier than in the Type I group. The location at which the minimum appeared varied from the left chest to the sternal region (Fig. 2). This indicates that breakthrough occurs on the left anterior wall in some cases, and on the right wall in the others. With the latter cases, it is considered that the degree of delay is relatively less so that the earliest activation of the right epicardial surface occurs prior to that of the left. It may be concluded that in Type II an activation delay, although its degree varies, exist in the anterior region of the right free wall.

The other characteristic of Type II is that the terminal maxima were present over the upper sternal region, and were located in the normal position.
Fig. 6. The area of delay. The area in which the difference between the voltage of a normal map and that of the RBBB map is significant. The left, the middle and the right columns indicate those from the typical case of Type I, Type II, and Type III, respectively, maps of which are presented in Fig. 1, Fig. 4, and Fig. 5, respectively.

or in a little lower position (Fig. 3). Normally, this upper sternal maximum is due to the latest activation of the pulmonary conus. \(^{31,19}\) Judging from this, the latest activation in this type probably occurs in the pulmonary conus region as in the normal, or in a little lower (i.e., more lateral) part of it. However, as indicated by the fact that there is no appreciable prolongation of QRS, the delay of that part appears to be small.

During the late stage of QRS, the positive zone covered mainly the upper part of the right chest and the right back (Fig. 4, D to F). This zone is, of course, due to the delayed RV activation. In order to determine the exact extent of the delayed activation, a mathematical technique was employed; the mean voltage and the standard deviation (SD) of every 85 recording points for each 1.5 msec after the onset of QRS were calculated in 10 healthy adults. Then the mean voltage was subtracted from the individual patient's voltage, and the difference obtained was furthermore divided by the SD. And, the area in which the value finally obtained is more than \(+2.0\) (or less than \(-2.0\)) was determined and illustrated (the darker zone in Fig. 6). This area (the area of delay) is considered to be an effect of the delayed activation.

In the representative patient of Type II (Fig. 6, middle), the area of delay is present at 55 msec over the right chest with a relatively similar extent to Type I (Fig. 6, left), suggesting that the activation delay of the right anterior wall is as extensive as in Type I. At 70 msec the area of delay, unlike Type I, rapidly decreases in extent, also suggesting that the right anterior activation, once occurred, progresses rapidly.
Moore et al\textsuperscript{20} have demonstrated in the dog that the incision of the false tendon (lateral branch of the RBB) may produce an activation delay of the area confined within the anterior and lateral part of the right free wall with no appreciable prolongation of QRS. Furthermore, they have proposed as a mechanism of IRBBB the functional disorder of the moderator band (corresponding to the false tendon in the dog) resulted from its distension due to the RV dilatation. Our RBBB patients have no clinical sign of the RV dilatation. A few investigators\textsuperscript{8,21} have reported the surface maps of patients with ostium secundum type of atrial septal defect. Interestingly, the maps which Blumenschein et al\textsuperscript{8} depicted are very similar to our Type II maps. This fact gives a suggestion that our Type II might be caused by the lesion located in the lateral branch of RBBB.

On the other hand, there is other evidence suggesting the lesion in the main RBB. Using a simulation method,\textsuperscript{22} surface maps were reconstructed on the assumption that the lesion is in the main RBB, and the impulse is transmitted from the left side of the heart to the right Purkinje network, through which the right free wall is activated. The simulated maps were very similar to the Type II pattern.

In conclusion, we believed that the cause of Type II RBBB may be multiple, rather than single.

Type III: In this type, the map pattern passed quite normally up to the terminal stage of QRS (70 msec in the representative case). Thereafter, however, a small positive zone remained in the upper sternal area for a relatively long time. The maps of the area of delay (Fig. 6, right) indicates that the significant delay is apparent in the smallest area of the upper sternum and the left clavicle, and, in addition, its extent become largest at the stage of 70 msec, later than in Type II. These findings suggest the presence of activation delay localized in the outflow region of RV. That is, Type III would be identical to the "focal block"\textsuperscript{11,14,23,24} which has been referred electrocardiographically. We consider that this type of RBBB arises from the lesion localized more distally: the block in the Purkinje fiber supplying the outflow tract; or the conduction disturbance due to the focal hypertrophy developed in that region, as has been found in the canine hearts by Moore et al.\textsuperscript{25} As to the mechanism arising this type, the surface data are far from decisive.

We have found out the fact that the Type III pattern can be derived also from a healthy adult with normal QRS. This points out a possibility that Type III is a normal variant. In this way, it is still questioned whether this type is truly due to the conduction "block" within the right bundle branch.
2. Electrocardiographic manifestation of the delayed activation of RV
RBBB is usually diagnosed by the presence of R’ wave in QRS complex
of lead V₁, and furthermore divided into 2 categories, complete and in-
complete RBBB, on the basis of the degree of QRS prolongation. The cor-
relative analysis between the type of map pattern and the QRS duration
revealed that CRBBB patients belonged to Type I, whereas IRBBB fell under
Type II or Type III; but no significant difference could be noted between
Types II and III. Further correlations were examined between the type of
map pattern and the other electrocardiographic findings characteristic of
RBBB, namely, the appearance of R’ (r’) wave and the wide or deep S wave,
or the shape of QRS complex of V₁. However, the result revealed that there
was no appreciable relationship, especially in Types II and III. Here we
arrive at a conclusion that IRBBB pattern may arise from the different types
of conduction defect, which can be hardly differentiated from the conven-
tional ECGs.

In conventional ECGs, the tracing from the right side of the thorax is
usually lead V₁ alone. Therefore, it can readily be understandable that the
information obtained concerning the RV activity is very limited. Thus, the
surface mapping can provide the useful information which is not obtained
from the ECG.

ECG, however, can show a constant relationship with the type of map
pattern, when some additional leads are applied. For example, the potentials
of the right subclavicular region and the right hypochondrial part during
the late stage of QRS differ characteristically among the types of RBBB.
Therefore, it is recommended that additional lead points are positioned in
these regions. These additional leads may probably contribute to the establish-
ment of a diagnosis or a classification of RBBB based on the activation se-
quence of RV, which has never been established by the conventional 12
leads system.

APPENDIX

Type I, Type II, and Type III are designated in our preliminary report as Type Ia, Type Ib, and Type II, respectively.

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