Body Surface Map Patterns of Altered Depolarization and Repolarization in Right Bundle Branch Block

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SUMMARY Surface maps from 14 patients with right bundle branch block were analyzed throughout depolarization and repolarization. The abnormalities in depolarization found in all the subjects were 1) epicardial breakthrough that was delayed and shifted to the left, and 2) development of right upper anterior positivity during the midportion of depolarization. In eight patients, this positivity manifested as multiple peaks, suggesting a fragmented spread of depolarization. We believe these findings result not only from the delayed engagement of the right ventricle by the conduction process, but also from its nonuniform and dysynchronous spread.

The recovery phase displayed five abnormal patterns: 1) simultaneous negativity on the right and positivity on the left of the midline in six patients; 2) only negativity on the right of the midline in four; 3) only positive potentials in the left upper chest in two; 4) only negative potentials on the left side of the midline in one; and 5) negative potentials spread diffusely over the precordium in one. The different degrees of this altered repolarization, we believe, depend upon the degree of altered sequence of activation of the heart in addition to the changes produced by the underlying disease process.

BASIC SURFACE MAP patterns of altered depolarization in right bundle branch block (RBBB) in animals and humans have been described. The major cause of QRS prolongation in RBBB is thought to be the delay encountered by the activation front in crossing the interventricular septum from left to right. Some investigators have concluded that the Purkinje network of the right ventricle does not participate normally in the depolarization process in RBBB, whereas others that the depolarization process uses the conduction system after the activity reaches the right ventricle. It has also been reported that the spread of the activation front through the right ventricle may be nonuniform.

This report describes our experience in recording body surface maps in 14 subjects with RBBB, especially regarding the evidence that the spread of depolarization through the right ventricle is nonuniform and dysynchronous. Altered patterns of repolarization in RBBB, as they manifest on the body surface, are also described.

Methods

Total body surface maps were recorded in 14 patients in whom a diagnosis of RBBB was made from the total QRS duration of 0.12 second or more, terminal (0.04 second) forces directed to the right and anteriorly, indicated by a broad S wave in leads I, V₅, and V₆, terminal R wave in V₁ and V₆, and the frontal plane axis between +110° and −30°. These subjects were ages 17–83 years (mean 60 years). Clinical data from histories and physical examinations and posteroanterior and lateral chest x-rays were available. In addition, cardiac catheterization data were available in four subjects. Three subjects had clinical evidence of coronary artery disease and another three had angiographically proved hemodynamically significant coronary artery disease. One subject had mild aortic insufficiency clinically and one more was studied after repair of tetralogy of Fallot. The other six subjects had no evidence of overt heart disease. Patients with obstructive airway disease and mitral stenosis were excluded from the study.

In addition to the 12-lead ECGs and the vectorcardiograms, total body surface maps were constructed by recording potentials from 140 sites on the subject's torso, and from the head and the foot, using the methods described previously. The recording electrodes were arranged in seven rows and 20 columns, 18° apart. The maps obtained were unrolled from about the chest and tilted to produce a pseudo-three-dimensional isometric effect. Thus, the positive potentials appear as peaks and negative potentials as sinks or valleys. The upper boundary of the map is at the sternoclavicular junction and the lower boundary is just above the umbilicus.

The maps thus obtained were examined at 5-msec intervals throughout the depolarization sequence, and then at 20-msec intervals until the end of repolarization.

Departure Map Technique

The potentials from the subjects under study were compared with an expected range of normal of the data similarly obtained from a group of normal subjects by recording mean value ± 2 standard deviations at each recording site on the body and at each millisecond. Each subject's potentials from each recording site at each 5 msec during depolarization up to 90 msec, were time phased and subtracted from the expected range of normal. The remaining potentials...
were considered abnormal and such maps are designated as abnormal departure maps$^{14-16}$ (fig. 1).

We have considered possible limitations of the departure map technique, especially in the presence of bundle branch block. Data from patients with myocardial infarction who had concomitant bundle branch block were excluded from our previous reports because of the expected influence of bundle branch block on the alteration produced by myocardial infarction.$^{14,15}$ However, we feel that in the presence of RBBB the departure map can still be relied upon to complement findings from directly recorded maps during the first 90 msec. An important consideration in using this technique, however, is the assurance of temporal fidelity. The reference point for digitizing, averaging and time alignment of data was obtained from a filtered, shaped, simultaneous control lead II. The earliest evidence of ventricular electrical activity was determined, by a rapid computer search of 142 surface leads, as the first of eight successive increases in the root mean square value of the total surface ECG. The level of the signal during the quiescent period immediately before the onset of the QRS complex was accepted as the zero or reference level for each recording site. Any temporal slippage, then, that may occur has not proved sufficient to cause false positivity in normal test subjects. These considerations help to assure us that the patterns of abnormality described are meaningful.$^{16-18}$

![Figure 1](image1.png)

**Figure 1.** On the left is a directly recorded map at 55 msec from a subject with right bundle branch block. The map has been unrolled from about the chest and tilted to produce a pseudo-three-dimensional effect. The positive potentials are displayed as peaks and negative potentials as valleys. The middle two maps are $+2$ SD (top) and $-2$ SD (bottom) from a normal population. The map on the right was obtained by subtracting the map on the left from the maps in the middle. The residual potentials are abnormal and this map has been designated as a departure map. Though the directly recorded map appears abnormal, the abnormalities become more evident in the departure map, especially the multiplicity of peaks of abnormal right upper anterior positive potentials. $VL =$ vertebral line; $R =$ right midaxillary line; $L =$ left midaxillary line; $S =$ midsternal line.

![Figure 2](image2.png)

**Figure 2.** An ECG showing a total QRS duration of 0.12 second, a mean frontal axis of +100°, delayed terminal forces directed anteriorly and to the right, indicated by terminal $R$ in lead $V_1$ and broad $S$ in lead I. These findings are consistent with the diagnosis of right bundle branch block.

**Results**

The ECG shown in figure 2 displays a total QRS duration of 0.12 second, a mean frontal plane axis of +100°, and terminal forces directed to the right and anteriorly, indicated by a broad $S$ wave in lead I and a broad terminal $R$ wave in $V_1$, and is, therefore, consistent with the diagnosis of RBBB. A vectorcardiogram from the same subject (fig. 3) shows normal initial

![Figure 3](image3.png)

**Figure 3.** A vectorcardiogram from the same subject whose ECG is shown in figure 1. Numbers in the transverse plane indicate the location of 40-, 60- and 90-msec vectors. The total duration of the vector loop is 125 msec and the initial forces are normally directed. The terminal forces show delay and rightward anterior direction.
forces directed anteriorly and to the right, a total duration of 125 msec, and a delayed terminal appendage directed anteriorly and to the right.19

Depolarization

Figure 4 shows mean maps derived from a group of normal subjects at 15, 25, 30 and 40 msec and directly recorded maps at the same instants from the patient whose ECG is shown in figure 2. The upper anterior chest manifested positive potentials early in the depolarization sequence in both the normal subjects and the patient with RBBB. These potentials became more pronounced, moved laterally and posteriorly and then diminished in intensity. These early forces appear normal in both the ECG and the vectorcardiogram of the RBBB subject under study (figs. 2 and 3).

The map at the right anterior chest became negative at 30 msec in normal subjects (right ventricular breakthrough)20 and this negativity increased in strength and area as the depolarization process progressed. No such pattern of relatively early negativity appeared in the RBBB maps, indicating the absence of normal right ventricular epicardial breakthrough in this condition.

Further progression of the depolarization sequence in the above subject is displayed in figure 5. Average normal maps at 45, 55, 60, 70, 80 and 90 msec are on the left and maps at the same instants from the patient with RBBB are on the right. By 45 msec the right anterior negativity in normal subjects has engulfed a large area of the map on the right of the midline. However, in the RBBB maps, such negativity never appeared on the right of the midline. On the contrary, negative potentials appeared in the 45-msec map on the left of the midline and then increased in strength and involved most of the precordium. Thus, the epicardial breakthrough was abnormally located and its appearance varied from 30–60 msec (average 45 msec) in all RBBB patients.

**Figure 4.** On the left are mean maps derived from a group of normal subjects and on the right are maps from a subject with right bundle branch block (RBBB) whose ECG is shown in figure 1 and whose vectorcardiogram is shown in figure 2. The upper anterior positive potentials are present both in normal subjects and in the patient with RBBB. The right anterior sink of negativity representing epicardial breakthrough (seen first in the 30-msec map of the group of normal subjects), however, is absent in the RBBB patient. VL = vertebral line; R = right midaxillary line; L = left mixaxillary line; S = midsternal line.

**Figure 5.** More maps from the depolarization sequence begun in figure 4. The right anterior negativity progresses further in normal subjects but is absent in right bundle branch block (RBBB). However, negative potentials appear on the left of midline and the appearance is delayed. Another finding seen in RBBB is the right anterior upper positivity appearing in the 60-msec map and continuing until the end. VL = vertebral line; R = right midaxillary line; L = left midaxillary line; S = midsternal line.
The second finding of interest is the appearance of positive potentials at the right anterior chest illustrated in the 60-msec map of the RBBB subject. This positivity moves superiorly and rightward as the depolarization sequence progresses. This abnormal positivity was seen in all the subjects and its onset varied from 45–70 msec (average 60 msec) and lasted until the end of depolarization. The vectorcardiogram at this time shows delay and rightward shift of the terminal forces.

These anterosuperior abnormal positive potentials manifested as multiple peaks in eight of 14 patients (two peaks in five patients and three peaks in three). Representative directly recorded maps from three subjects with this finding are displayed in figure 6. The abnormal departure maps at 70 and 90 msec from the patient with RBBB, whose directly recorded maps are shown in figures 4 and 5, are displayed in figure 7. In the beginning, the abnormal positive potentials appeared as a single peak at the right anterosuperior part of the chest (not shown). As the depolarization progressed, more peaks appeared (70-msec map). The abnormal location of the delayed epicardial breakthrough on the left of the midline can also be seen (90-msec map). In all patients with multiple peaks, this phenomenon was evident in the directly recorded maps (60- and 70-msec maps in figure 5 and maps in figure 6), but in some instances, the multiplicity of peaks could be better appreciated in the departure maps, as in the example shown above by the 70-msec maps in figures 4, 5 and 7. In the other six subjects, only a single hump of positive potentials was seen.

Repolarization

Figure 8 displays a sequence of repolarization in a normal subject at 428, 468, 488, 508, 528 and 548 msec. The surface manifestation of repolarization in this subject is a smooth, slowly developing and slowly regressing uniform expression of positive potentials. In RBBB, five different patterns of abnormal repolarization were seen. The most common pattern is shown in figure 9 in maps at 125, 165, 205, 245, 285 and 325 msec from a patient with RBBB. The onset of the repolarization process manifested as a negativity that was anterior and to the right of the midline. As the process progressed, a second front appeared anteriorly in the form of a positivity in the left chest. Both of these manifestations coexisted until the end of repolarization. The pattern of simultaneous presence of positive and negative potentials in the precordium

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**Figure 6.** Representative directly recorded maps, at the instants shown, from three subjects with right bundle branch block (RBBB). Multiplicity of peaks (marked by arrows) of right anterior upper positive potentials can be seen.

**Figure 7.** Abnormal departure maps from the subject whose directly recorded maps are shown in figure 4 and 5. Multiplicity of the right upper positive potentials becomes more evident. The epicardial breakthrough is located abnormally on the left of the midline. VL = vertebral line; R = right midaxillary line; L = left midaxillary line; S = midsternal line; RBBB = right bundle branch block.

**Figure 8.** Sequence of repolarization from a normal subject is manifest as a smoothly progressing front of positive potentials in the left anterior chest. VL = vertebral line; R = right midaxillary line; L = left midaxillary line; S = midsternal line.
was seen in six subjects. The second most common pattern, seen in four subjects, consisted simply of a negative front on the right of midline, without the associated positive potentials described above. The process manifested as positive potentials only in the anterior left upper chest in two. In yet another, the surface manifestation of abnormal repolarization was located in the precordium, left of the midline, but was in the form of negative potentials.

Finally, a subject who had undergone repair of tetralogy of Fallot showed the onset of the repolarization process on the right, which later moved to the left of the midline (fig. 10). Multiple valleys are visible as well as multiple peaks in the right upper positivity. Positive and negative potentials simultaneously present in the 105-msec map indicate that the repolarization process commenced while the depolarization process in the form of positive potentials was still going on. This coexistence of both processes was also seen in another subject.

Discussion

Studies of the activation of the animal interventricular septum showed that the depolarization begins in the area of the junction of middle and lower thirds on the left, and at the base of the anterior papillary muscle on the right. From then on the major portion of the interventricular septum is depolarized from the left side. In humans, activation begins simultaneously at two points on the left side of septum, one anteriorly in the middle third and another posteriorly near the posterior wall in the lower third. The contribution from the right side comes from the activity that started in the middle third anteriorly.

Earlier experimental studies in animals by Wilson and associates and others provided some clarification of the patterns of electrocardiographic changes in RBBB. Later investigators addressed themselves to the question of normal patterns of activation of the right ventricle and the relative role of the interventricular septum and the right ventricular free wall in the genesis of changes produced in RBBB. Such studies indicate that in experimental and human RBBB, the early portion (40 msec) of the QRS complex generally remains unaltered, probably because the block in RBBB occurs beyond the supply to the septum from the right bundle. In clinical situations where the early portion of the QRS complex was found to have been altered, the block was thought to be proximal. These studies, therefore, show that depolarization of the right septum does contribute to the earliest portion of the normal QRS complex. In this study, the early anterior positivity was indistinguishable from normal in all the subjects studied, indicating that the septal depolarization both from the left and the right remained normal in this group of patients. This suggests that the block might have occurred beyond the point of supply of the right side of septum from the right bundle. However, this cannot be stated with certainty from the findings on the surface map.

Pathologic studies performed in acquired RBBB in the presence of clinical evidence of coronary artery disease have shown extensive disease involving the right bundle and its branches.
The reason for the delay involving the middle and late parts of the QRS complex in RBBB, shifting the terminal forces anteriorly and to the right, is thought to be the time taken by the electrical activity to traverse the septum. The role of the Purkinje system once the depolarization process reaches the right ventricle is controversial. Some investigators feel that the propagation of depolarization is via the right ventricular myocardium, and that Purkinje network does not participate in the usual manner, if at all, thus contributing to the prolongation of the QRS complex in RBBB. We believe that the right ventricular Purkinje network is used, wherever encountered, as the advancing wave front proceeds from the left branch system to invade the anterior and inferior attachments of the right ventricular free wall to septum. Otherwise, QRS durations in RBBB would be much longer. In our computer models of the activation process, ventricular activation via muscle alone takes longer, sometimes approaching 280 msec, if the faster Purkinje transmission system is not used.

An exact translation of the electrical potentials on the surface of the heart to the ones recorded on the surface of the body has been questioned, because alteration of the electrical signals generated on the heart does occur in the process of their transfer to the surface of the body. However, good correlation has been found between these two locations. 29-42

After producing RBBB in dogs, Sugenoya et al. 1 obtained surface maps that showed the epicardial breakthrough to be delayed and shifted to the left of midline. They also found abnormal positive potentials in the superior and right regions of the chest anteriorly in the middle and late parts of depolarization. Similar surface map findings have been reported in human RBBB. 2, 8 Our data corroborate these two findings; we found delay and lateral shift of the epicardial breakthrough and terminal rightward, anterior and superior forces in all 14 patients.

Some experimental studies indicate that depolarization may spread nonuniformly through the right ventricle in RBBB. 7 In eight of our 14 subjects, the right anterior terminal forces manifested as multiple simultaneous peaks that proved to be abnormal. The possible explanations are that the activation wave travels its abnormal route at different speeds, alternately encountering specialized, fast conduction fibers with velocities up to 5 m/sec, then slower, non-specialized fibers with velocities down to 0.5 m/sec. This nonuniformity of spread may be further emphasized as a result of involvement of the conduction pathways to different degrees, thus increasing the fragmentation of spread. In one subject who has tetralogy of Fallot, the right ventricular hypertrophy probably contributed to the nonuniformity of right ventricular activation. 43

The recovery phase displayed multiple patterns, the most common being simultaneous negativity on the right of the midline and positivity in the precordium (six of 14 patients). This pattern occurred primarily as a result of the altered recovery sequence of the right ventricle secondary to its abnormal sequence of activation, with the normal recovery pattern of the left ventricle maintained. In four patients where probably most or all the myocardium recovered abnormally, the process manifested as only single receding negativity to the right of the sternum. In two subjects, recovery occurred in a more expected manner. However, from this study we could not completely distinguish primary changes produced in repolarization by the underlying disease process from the secondary changes produced by the altered activation sequence of RBBB. Two negative sinks in the maps of the patient with tetralogy of Fallot again indicate abnormal direction of repolarization, but the process goes on at multiple sites simultaneously with different degrees of intensity.

This study, therefore, confirms the reported surface map findings produced by the altered activation sequence of the heart due to the delayed engagement of the right ventricle in RBBB. In addition, we believe, in most cases of RBBB, the spread of the electrical front is nonuniform in speed and abnormal in direction after it engages the right ventricle, as indicated by the multiple maxima on the front of the chest superiorly that occur outside the expected range.

Surface map patterns of repolarization vary. We believe that the degree of alteration in repolarization depends upon the degree of alteration in the activation sequence produced by RBBB in addition to the changes produced by the underlying disease process.

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