Comparison of Total Body Surface Map Depolarization Patterns of Left Bundle Branch Block and Normal Axis with Left Bundle Branch Block and Left-axis Deviation

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SUMMARY  Total body surface maps from 15 subjects with left bundle branch block and normal axis (LBBB-NA) and 10 subjects with left bundle branch block and left axis (LBBB-LA) were analyzed and compared with maps from normal subjects. In 19 of the 25 subjects with LBBB, the timing of early upper sternal positivity was similar to that of normal subjects, indicative of timely but oppositely directed septal activation.

The right ventricular breakthrough was normally located in all, but was earlier after the onset of QRS than expected in some. The initial portion of the positivity produced by left ventricular activation was located in the upper anterior chest in both LBBB-NA and LBBB-LA, but its onset was generally delayed compared with that in normal subjects, presumably because of the time taken by the right-to-left septal activation. Also, the total duration of this positivity was longer than in normal subjects and extended considerably beyond 90 msec, indicating prolonged activation of the anterior free wall of the left ventricle. In LBBB-NA, this upper anterior positivity remained anterior throughout depolarization, but in LBBB-LA it moved toward the left shoulder and the left upper back, presumably due to the posterior orientation of the terminal portion of depolarization. This terminal orientation in patients with LBBB-LA was thought to be due to the additional delay in the activation of the anteriorobasal portion of the left ventricle caused by selective involvement of the left anterior fascicle.

THE ALTERATIONS in the ventricular depolarization produced by left bundle branch block (LBBB) have been basically understood for a long time. Recently, additional knowledge has been gained from studies based on recordings of the epicardial and endocardial spread of excitation. In this report, we describe the depolarization process in LBBB as deduced from total body surface mapping, as well as the distinguishing features of LBBB with normal axis (LBBB-NA) and with left-axis deviation (LBBB-LA).

Methods

Total body surface maps were recorded in 25 subjects in whom an electrocardiographic diagnosis of left bundle branch block was made from the presence of a total QRS duration of 0.12 second or more, absence of a Q or an S in leads I, aVL, V5, and V6, and the presence of an RR' pattern in V5 and V6. Fifteen subjects had a normal frontal QRS axis of +90° to −29° (LBBB-NA) and 10 a left axis of −30° or less (LBBB-LA). The LBBB-NA subjects were 45–95 years old (mean 67 years) and 11 were males. All the subjects in this group had clinical evidence of left ventricular dysfunction and five had cardiac catheterization evidence of severe three-vessel coronary artery disease. The subjects in LBBB-LA group were 33–81 years old (mean 62 years); five were males and five females. Similarly, all the subjects in this group had clinical evidence of left ventricular dysfunction, and one also had hypertrophic obstructive cardiomyopathy.

In addition to the clinical data, chest x-rays, cardiac catheterization data in six, ECGs and vectorcardiograms in most, total body surface maps from 140 sites were recorded. No intracardiac electrophysiologic studies were performed.
To obtain a body surface map, potentials are recorded from 140 sites on the thorax, a point on the head and another point on the left lower extremity. The thorax is divided into 20 columns 18° apart and seven rows 5 cm apart. The topmost column starts at the cranial extreme of the manubrium sternii and the last column is located at the unibicus. The data acquired from a given point on the torso consist of an average of 50 cardiac cycles. The recordings are made sequentially, one column at a time. These data are recorded on a magnetic tape at frequencies of 0.01–3 kHz and stored at sampling rates of 1000 samples/sec after analog-to-digital conversion. To determine the fiducial mark for time alignment, simultaneously recorded lead II is used after it is filtered to eliminate extraneous noise (e.g., respiratory movements, muscle tremor and alternating current). The level of signal in the quiescent period immediately preceding the QRS complex is taken as the zero level and forms the baseline for that QRS. A total surface ECG is obtained by time aligning and averaging all the 142 leads. The first of the programmatically determined eight successively increasing root mean square (RMS)* values of this total ECG is taken as the onset of the ventricular activity.

Final processing in the form of isometric projection maps at each 5 msec was done by computer (the PDP-9 computer used originally has been replaced by a PDP-11 computer). The map is unrolled from the chest and slightly tilted, which produces a pseudo three-dimensional effect. The peaks indicate positive potentials and the negative potentials are manifested by dips or valleys. We display data in the isometric form because of the ease of visual interpretation by readers used to looking at the ECG. Others display map data in isopotential form where contour lines are produced by joining points having the same potentials of the same polarity.

**Results**

Figure 1 is an ECG from a subject with LBBB-NA. The total QRS duration is 0.18 second, there is no Q or S in leads I, aV\textsubscript{1}, V\textsubscript{3}, and V\textsubscript{6}, and an RR' pattern is present in lead V\textsubscript{5}, which indicates LBBB. The mean frontal QRS axis is +50°. The ECG in figure 2 is from a subject with LBBB-LA. The total QRS duration is 0.12 second, there is no Q or an S in lead I or aV\textsubscript{1}, and the axis is −45°. This patient was in atrial fibrillation with a controlled ventricular response when the data were recorded.

Figure 3 shows maps at 15- and 30-msec instants from a normal subject, a subject with LBBB-NA and a subject with LBBB-LA. Further progression of depolarization in the same subjects is shown in 50- and 70-msec maps in figure 4. The ECGs of the latter two subjects are shown in figures 1 and 2. As in normal subjects, early upper sternal positivity appeared in 12 of 15 patients with LBBB-NA and seven of 10 with LBBB-LA. Its onset, however, was earlier than normal (at 5 msec) in 14 of 25 subjects.

Normally located negativity caused by right ventricular breakthrough was seen in both LBBB-NA and LBBB-LA. It had appeared by 20 msec on the average, which was earlier than expected in some. The right ventricular breakthrough was associated with upper chest positivity anteriorly, the onset of which was occasionally simultaneous with the epicardial breakthrough, but generally followed it by 15 msec on the average. This positivity lasted well beyond 90 msec. It persisted anteriorly in LBBB-NA throughout depolarization, but moved toward the left shoulder and back in LBBB-LA as the depolarization sequence progressed. The progression in location and intensity of this positivity had reached maximum by the 70-msec instant in both subjects. Thereafter, it slowly decreased in voltage without change in location until the end of depolarization.

**Discussion**

The role of each bundle branch in activating portions of the septum and the ventricular myocardium and the surface electrocardiographic morphology produced by bundle branch block have been described.\textsuperscript{1-4, 13-15} However, controversy has existed for almost as long regarding the mechanism and the location of delay of the electrical forces in bundle branch block and whether the Purkinje system of the blocked side participates in the depolarization process once the activity reaches that ventricle. This delay has been thought to be due to the slow passage of the electrical front across a synaptic barrier at the junction of the right septal and left septal masses;\textsuperscript{16} part of the septal delay may be caused by engagement of the Purkinje system at multiple sites on the left of the septum.\textsuperscript{17, 18} Others have concluded that the mechanism of delay is the slow but uniform conduction through the septum (conduction through the myocardium being slower than through the Purkinje cells), rather than delay at a particular point in the septum.\textsuperscript{19, 20} Recent canine experiments in chronic LBBB by van Dam\textsuperscript{5} and by others suggest that latter is the case.

According to van Dam,\textsuperscript{5} activation of the septum and the left ventricle in LBBB can be divided into three phases: phase I (0–45 msec) encompasses the activation of the right side of the septum, which is earlier than normal; phase II (45–70 msec) encompasses the activation of the left septum and anterior and posterior insertions of the left ventricular wall; and phase III (beyond 70 msec) encompasses the meeting of the anterior and posterior activation fronts, which depolarize the remaining left ventricle (the lateral basal area is depolarized last). Most investigators agree that the Purkinje system participates in depolarizing the left ventricle. According to van Dam, this occurs in the third phase.

Recently, epicardial mapping has been performed in human subjects with LBBB: in one subject studied by van Dam,\textsuperscript{5} in five subjects studied by Wyndham et al.,\textsuperscript{6} and five by Horowitz et al.\textsuperscript{7} Horowitz et al. also per-

\[\text{RMS} = \sqrt[2]{\frac{\sum X_i^2}{n}} \]

where \(i\) = each electrode number (1–142), \(X\) = the respective potential value and \(n\) = the total, 142.
formed endocardial mapping in 15 subjects with LBBB. The right ventricular epicardial breakthrough in LBBB was normal in location but was earlier in timing than in normal hearts. The left ventricular septal endocardial activity usually started in the middle portions of the septum, but in some subjects it occurred anteriorly or posteriorly. The septal crossing of the electrical activity was slow but uniform. The earliest left ventricular epicardial activity generally occurred anteriorly. The latest portion of the left ventricle to show epicardial activity was the lateral base in the subject in van Dam's experiment; the latest epicardial activity was inferior in four patients and lateral near the atrioventricular groove in the fifth in the studies of Wyndham et al., and was described by Horowitz et al. as posterior or lateral. These studies suggest that transfer of electrical activity across the septum occurs at a uniform speed, in contrast to the conclusion drawn by Sodi-Pallares et al. Involvement of the Purkinje system in depolarization of at least part of the left ventricle is again suggested.

The subjects in the above reports had either coronary artery disease, often with myocardial involvement, or left ventricular hypertrophy. Myocardial disease may have modified the electrical spread through the left ventricle. The majority of patients with LBBB have pathologically evident severe coronary artery disease or clinically overt heart disease.

Experience from our laboratory and others indicates that significantly more detail is available from the surface map than from the ECG or the vectorcardiogram. Although the epicardial events cannot be directly translated to the electrical expression of those events recorded from the surface of the body, a definite, predictable relationship between electrical activity recorded from these two sites does exist.

We therefore attempted to analyze the electrical activity on the surface of the body in LBBB. Preda et al. studied 12 patients with LBBB. Eleven subjects with LBBB and normal axis were thought to show three different types of right ventricular activation. In type I (five subjects) the right ventricular breakthrough was normal and in type II (four subjects) the epicardial breakthrough was lower, at the apex of the right ventricle; however, in type III (two subjects), the initial portion of right ventricular activity pointed posteriorly. Types II and III were thought to indicate some degree of right bundle branch block in addition to LBBB. The activation of the left ventricle was similar in all the three types: traveling through the apex it involved the lateral region, posterobasal region and, finally, the anterobasal region.

The left-axis deviation in left anterior fascicular block is thought to be an electrical effect of the interruption of the anterior fascicle or many of its distal arborizations. Similarly, the coexistence of left-
axis deviation in LBBB may indicate selective slowing of conduction because of a structural or functional block in the left anterior fascicle, in addition to the proximal left bundle branch block. This sequence could also be due to earlier engagement of the left posterior fascicle than of the left anterior fascicle.

None of the human subjects reported in the above studies had left-axis deviation. Patients with LBBB-LA have a greater incidence of myocardial impairment and prolongation of AH and HV intervals. To our knowledge, no data are available on the endocardial, epicardial, or surface map recordings in human LBBB-LA.

The very early high sternal positivity reported here is thought to represent septal activation via the right bundle branch in the absence of normal septal activation via the left bundle branch. The timing of this positivity was similar to that in normal subjects, but it was generally diffuse and was located somewhat more to the left, probably because of the oppositely directed septal depolarization compared with normals and because the septum is engaged at different sites in different subjects. Some right bundle branch disease, the degree of which is unknown because of the lack of electrophysiologic studies, may have played a part.

As expected, the right ventricular epicardial breakthrough was normally located. However, in a few subjects it was earlier than normal. This observation is in accordance with the findings of Wyndham et al. and with the expectation that the earliest site of ventricular depolarization is on the right side of the septum in LBBB patients, as opposed to the left side in normal subjects. Thus, the earliest site of ventricular depolarization is closer to the site of right ventricular epicardial breakthrough, which leads to its earlier-than-normal appearance.

Body surface maps of normal subjects show positive potentials at about 20 msec in the upper sternal area, indicating the onset of activation of the left ventricle; right ventricular breakthrough occurs about 10 msec later. In LBBB, the left upper sternal positivity occurred simultaneously with the right ventricular breakthrough in five subjects and followed it by an average of 16 msec in the remaining 20 subjects, reflecting the time required for right-to-left septal activation and thus the delay in left ventricular activation. Diffuse disease of the conduction system and unknown degree of disease in the right bundle branch block may have played a role in producing variable time relationship between right ventricular breakthrough and onset of left ventricular activation. Anterior positivity persisted until 90 msec or longer and its total duration, average 55 msec, was considerably longer than normal. Thus, not only is the onset of left ventricular activation delayed, but its total duration is prolonged, which indicates slower-than-normal depolarization of the anterior left ventricle in LBBB. Slowed depolarization is probably due to the involvement of the distal arborizations to a varying degree and to the involvement of the left bundle and its main branches by the disease. In subjects with LBBB-LA, the positivity persisted mostly anteriorly throughout depolarization, and moved toward the left shoulder in some. In subjects with LBBB-LA, the positivity was also located at the left shoulder and back, which indicates further selective slowing of depolarization.

Figure 3. Body surface maps at 15-msec and 30-msec instants from a normal subject, from a patient with left bundle branch block and a normal axis (LBBB-NA) whose ECG is shown in figure 1, and from a subject with left bundle branch block and left-axis deviation (LBBB-LA) whose ECG is shown in figure 2. Early upper anterior positivity indicating septal activation is present in the 15-msec maps in all the three subjects. By the 30-msec instant, the right ventricular breakthrough in LBBB subjects is in an advanced stage, ahead of the normal subject, but its location is normal. VL = vertebral line; R = right midaxillary line; L = left midaxillary line; S = midsternal line.

Figure 4. Further progression of depolarization in the subjects depicted in figure 3. In the normal subject, the left ventricular activation indicated by left upper positivity is complete by the 70-msec instant. In the patients with left bundle branch block (LBBB), ventricular activation still persists. The left upper positivity in LBBB patients with a normal axis (LBBB-NA) stays in the left anterior upper chest, whereas in those with left-axis deviation (LBBB-LA), it moves toward the left shoulder and left upper back.
anterobasally and direction of the terminal positive potentials posterosuperiorly. This mechanism is speculative and remains to be proved by direct epicardial and surface recordings in the same subjects. In conclusion, the present study indicates that in LBBB, the septal activation occurs on time but is oppositely directed; the right ventricular breakthrough occurs in the normal location, but is earlier in timing; the delay occurs both at the septum and across the left ventricular myocardium; the anterior and anterobasal regions of the left ventricle are the last to be depolarized; and in LBBB-LA further selective involvement of the terminal arborizations of the left anterior fascicle leads to further delay in the activation of the anterobasal segments of the left ventricle.

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Comparison of total body surface map depolarization patterns of left bundle branch block and normal axis with left bundle branch block and left-axis deviation.
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Circulation. 1983;67:660-664
doi: 10.1161/01.CIR.67.3.660

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