Epicardial Activation in Patients with Left Bundle Branch Block

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SUMMARY To elucidate the abnormalities in ventricular activation sequence in human left bundle branch block (LBBB), epicardial mapping was performed in five patients, ages 52–58 years, undergoing coronary bypass surgery, and the results were compared with similar data published from patients without conduction defect. Three patients had chronic and two patients intraoperative LBBB. ECGs during LBBB revealed a QRS duration of 130–160 msec and a mean QRS axis of −15° to +45°. Epicardial mapping revealed 1) anterior right ventricular (RV) epicardial breakthrough (5–26 msec after QRS onset), normal in site in all patients, but abnormally early in timing relative to QRS onset in three patients with chronic LBBB, and earlier compared with preoperative maps in two patients with intraoperative LBBB; 2) normal location of latest RV epicardial activation in four of five patients, but abnormally late occurrence of this event in 100, 108 and 110 msec after QRS onset in three of five patients; 3) absence of discrete left ventricular (LV) epicardial breakthroughs in all patients; 4) slow transseptal epicardial activation (crowded isochrones) from right to left, with anteroseptal crossing preceding inferoseptal crossing; 5) activation of the anterolateral left ventricle before the inferior LV epicardium; 6) more widely spaced isochrones, implying more rapid conduction, over the LV free wall epicardium; and 7) location and timing of latest LV epicardial activation in an abnormal site, and abnormally late relative to QRS onset (113–140 msec, mean 124 msec) in all patients. This event occurred a mean of 20 msec before the end of the QRS in the five patients.

In conclusion, with normal axis, human LBBB is associated with initiation of ventricular activation closer to anterior RV recording sites than is normal activation, slow leftward transseptal activation, a generally anteroinferior orientation of LV activation, and probable engagement of the distal LV Purkinje system during the latter part of the QRS.

THE CURRENT UNDERSTANDING of ventricular activation during left bundle branch block (LBBB) in humans is based on deductive electrocardiography, animal experimentation, and limited electrocardiographic-pathologic correlations in patients with LBBB.1–4 To our knowledge, only one example of directly recorded activation data in chronic human LBBB has been reported.9 In order to study systematically the mode of activation of the left ventricle during LBBB, five patients, three with chronic and two with acute LBBB, underwent epicardial mapping during open heart surgery.

Materials and Methods

Five patients undergoing coronary artery bypass surgery at the University of Illinois Hospital and the Westside Veterans Administration Hospital were approached preoperatively, and informed written consent was obtained for epicardial mapping under a protocol approved by the University of Illinois Human Investigation Committee. Patients 1, 2 and 3 had LBBB on preoperative ECGs. Patients 4 and 5 were studied on the basis of electrocardiographic left ventricular hypertrophy. These two patients developed transient LBBB intraoperatively, and postoperative mapping was also performed. All patients were assessed preoperatively by history, physical examination, ECG, chest x-ray, echocardiogram, treadmill stress testing and diagnostic cardiac catheterization with left ventriculography and selective coronary arteriography.

Epicardial Mapping

Epicardial mapping was performed by previously described techniques.10 In all patients, mapping preceded institution of cardiopulmonary bypass, and in patients 4 and 5, repeat mapping was performed after bypass was discontinued and hemostasis achieved. Simultaneous recordings of two or three of seven continuously available isolated monitored leads (I, II, III, aVR, aVL, aVF and V5), with an isolated bipolar right ventricular reference electrode and two or three atrial bipolar electrograms from a hand-held exploring probe (Elecath, Rahway, New Jersey) were made on a multichannel recorder (Electronics for Medicine VR-6 or VR-12, Minneapolis, Minnesota) at a paper speed of 100 mm/sec. Local ventricular activation times were measured in milliseconds from the onset of the QRS in surface or reference leads to the point at which the first high-frequency deflection crossed the baseline in the local electrogram, and were filtered between 40–500 Hz and 100–2500 Hz, as previously described10 (fig. 1).
Definitions

LBBB was defined by a QRS duration of greater than 0.12 second, rS or QS in V_{1} and slurred monophasic or notched R in V_{6}. The frontal plane QRS axis was computed as the mean axis for the total duration of the QRS. Epicardial breakthrough and latest epicardial activation were defined as previously described.¹⁰

Comparison with Normal Epicardial Activation

To delineate the abnormalities of the activation sequence in LBBB, we compared the findings of the present study with previously published data from 11 patients without conduction defect.¹⁰ In the patients without conduction defect, epicardial activation was characterized by 1) earliest epicardial breakthrough in the anterior paraseptal region of the right ventricle, 7-25 msec (mean 17 ± 7 msec) after the onset of the QRS complex; 2) two to four subsequent epicardial breakthrough events in all patients in the inferior right ventricle, inferior left ventricle or anterolateral left ventricle. Normally, one to three breakthroughs were invariably present in the left ventricular epicardium, inferiorly in seven of the patients and in the anterolateral wall in 10 of the 11 patients; 3) latest overall epicardial activation invariably at the base of the ventricles, at the right ventricular basal regions in nine patients and abutting the atroventricular groove in the inferior left ventricle in only two patients. In the left ventricle itself, the latest activation was always inferior in the 11 patients.

Statistical Methods

Comparative data are expressed as mean ± SEM. Means were compared by t test for unrelated means using pooled variance.

Results

Clinical and electrocardiographic data are summarized in Table 1. The five patients included four males and one female, ages 52-58 years (mean 56 years). All had coronary artery disease, and patients 1 and 5 also had aortic stenosis. Patient 2 had an inferior left ventricular aneurysm from previous myocardial infarction, and patients 2, 3 and 4 had a history of hypertension. All underwent two to four coronary artery bypass grafts. In addition, aortic valve replacement was performed in patients 1 and 5. Left ventricular hypertrophy was seen in all patients at operation.

ECGs corresponding to the epicardial mapping data to be presented (preoperative in patients 1, 2 and 3,

![Diagram](https://example.com/diagram.png)

**Figure 1.** Two mapping sites from patient 1, showing method of measurement of local epicardial activation times. A) Earliest breakthrough, 5 msec after onset of QRS in the paraseptal anterior right ventricle. B) Near-terminal activation, 110 msec after onset of QRS in the apical inferior left ventricle. $R - B_{L} = \text{local activation time in milliseconds}$. RV Ref = right ventricular reference electrogram. The exploring probe electrograms are shown as the three lowest channels.

**Table 1. Clinical and Epicardial Data**

<table>
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<tr>
<th>Pt</th>
<th>Age (years)</th>
<th>Sex</th>
<th>QRS duration (msec)</th>
<th>QRS axis</th>
<th>Epicardial breakthrough site and timing (msec)</th>
<th>Latest epicardial activation site and timing (msec)</th>
<th>Completion of septal crossing (msec)</th>
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Abbreviations: msec = milliseconds after onset of QRS; LEARV and LEALV = right and left ventricular sites of latest epicardial activation; ARV = anterior right ventricle; IRV = inferior right ventricle; RVOT = right ventricular outflow tract; ILV = inferior left ventricle; LVOM = left ventricular obtuse margin.
Typical Epicardial Maps

Figure 3 shows the epicardial map from patient 1, who had chronic LBBB. Anterior, left lateral and inferior views of the heart are shown. Only a single epicardial breakthrough in the anterior paraseptal right ventricle 5 msec after the onset of the QRS was present. The right ventricle was activated with normal sequence from this site. However, activation was markedly slowed, as judged by crowding of isochrones,11 as the right ventricular wave front approached the septal region of the inferior right ventricle, especially near the apex inferiorly, where a right-sided site was delayed to 110 msec after the QRS onset. There was no epicardial breakthrough on the left ventricle, which was activated transseptally from the right ventricle. Isochrones were crowded together, implying slowing of conduction11 over the septal region both anteriorly and inferiorly. Septal crossing by the leftward-moving wave front appeared to be mostly complete by about 50 msec anteriorly and by about 110 msec inferiorly. From these two moments, the anterior and inferior aspects of the left ventricle were activated by more rapidly moving fronts that spread laterally toward the obtuse margin of the anterolateral surface and moved in a basoapical direction on the inferior surface. The latest site of left ventricular activation was the apical region of the inferior surface, 120 msec after the QRS onset, which was 20 msec before the end of the QRS complex in this patient.

Figure 4 shows the epicardial maps obtained before and after surgery in patient 4. The map on the left (fig. 4A) departs from normal and shows changes possibly consistent with left ventricular hypertrophy. Normal right ventricular epicardial breakthroughs were pres-
Figure 3. Epicardial map from patient 1. Anterior, inferior and left lateral views are shown. A zone of overlap is present in anterior and left lateral views in the region of the left anterior descending artery. Isochrones are at 10-msec intervals.

Figure 4. Preoperative (A) and postoperative (B) epicardial maps from patient 4. Panels arranged as in figure 3. LVH = left ventricular hypertrophy; LBBB = left bundle branch block.
ent anteriorly and inferiorly, 26 msec after the onset of the QRS, and there were no normal left ventricular epicardial breakthrough events. The left ventricle was activated by anterior and inferior wave fronts proceeding from the right ventricle. Despite these abnormalities, the latest epicardial activation was not delayed, and occurred at a normal site in the inferobasal left ventricle 89 msec after the onset of the QRS. This event corresponded exactly to the end of the QRS on the surface leads that showed a QRS duration of 90 msec before cardiopulmonary bypass.

After technically successful triple aortocoronary bypass grafting to the left anterior descending, circumflex marginal and right coronary arteries, a LBBB pattern was noted. LBBB persisted for 2 days, when the ECG returned to the preoperative pattern without other evidence for perioperative myocardial necrosis.

The postoperative map (fig. 4B) showed slight changes in right ventricular and marked changes in left ventricular activation. The anterior right ventricular breakthrough was still present, but occurred 12 msec earlier than formerly, 14 msec after the QRS onset. No inferior right ventricular breakthrough was demonstrable postoperatively. Some delay in the activation of the inferior right ventricular wall was evidenced by the relatively later activation of the basal and septal regions inferiorly. The latest site of right ventricular epicardial activation was near the crux at 108 msec. Left ventricular epicardial activation was markedly delayed compared with normal and with the preoperative status. No left ventricular epicardial breakthroughs were identifiable. The left ventricular surface was activated by anterior and inferior fronts traversing the septal region slowly (crowded isochrones)\(^1\) from the right ventricle. Anteroseptal crossing appeared to be complete by about 40 msec, and inferiorly by about 110 msec after the onset of the QRS (table 1). Thus, the anterior left ventricular activation process preceded the inferior activation process, resulting in terminal left ventricular epicardial activation in the apical and septal region of the inferior left ventricle 124 msec after the onset of the QRS. This event occurred 6 msec before the end of the surface QRS.

Epicardial breakthroughs

Figure 5 shows the epicardial breakthrough data in the five patients. Only right ventricular breakthroughs were observed, initiated anteriorly in all five patients, 5–6 msec (mean 9 ± 4.9 msec) after the onset of the QRS. Patients 2 and 3 showed additional normal right ventricular breakthroughs in the inferior wall, near the acute margin at 17 msec in one patient, and near the apex in both patients, at 20 and 38 msec, respectively. The earliest right ventricular epicardial breakthrough was earlier than normal, 5 msec after QRS onset, in patients 1, 2, and 3, who had chronic LBBB. In patients 4 and 5, who had acute LBBB, the earliest right ventricular breakthrough anteriorly occurred 26 and 33 msec after the onset of QRS in the preoperative (electrocardiographic left ventricular hypertrophy) map, and 14 and 16 msec, respectively, in the postoperative (LBBB) map. Because in these two patients the earliest right ventricular epicardial breakthrough time was within the normal range, the group mean (9 ± 5 msec) for the five LBBB patients was not statistically shorter than the mean (17 ± 7 msec) for the 11 patients with normal QRS\(^10\) (p > 0.5).

Septal Depolarization

Table 1 shows the timing of completion of anterior and inferior septal crossing by the leftward-moving epicardial wave fronts initiated in the right ventricle. The anteroseptal region was activated completely by approximately 40–80 msec (mean 56 ± 14 msec) in the five patients. The inferoseptal region was crossed later in all patients, and was completed by approximately 70–120 msec (mean 102 ± 17 msec) (p < 0.01). The left inferior wall was activated later than the left anterior wall in all patients, as shown by the following analysis. We compared the timing of activation of all 96 sites in the anterolateral left ventricular wall with that of all 59 sites in the inferior left ventricular wall in the five patients, and found that anterolateral sites were activated 64 ± 2 msec after the onset of QRS, compared with inferior wall activation, which occurred 106 ± 2 msec after the onset of QRS (p < 0.0001). In all patients, after emergence of the septal epicardial wave fronts onto the epicardial surface of the left ventricular free wall, a marked widening of isochrones occurred, in contrast to the crowding of isochrones over the septal regions.\(^11\) The widely spaced isochrones persisted until the moment of latest recordable epicardial activity.

Latest Epicardial Activation

Figure 6 summarizes the sites of latest epicardial activation in the five patients. Both right and left ven-
tricular latest activation sites are indicated. The right ventricular terminal epicardial activity was recorded from the paraseptal inferior wall in four patients, three near the crux and one near the apex, and from the right ventricular outflow tract in one patient. The timing of the latest right ventricular activation ranged from 50–110 msec (mean 87 ± 24 msec) after the onset of QRS, and occurred 22–110 msec (mean 57 ± 18 msec) before the end of the QRS. Thus, the latest right ventricular epicardial activation did not correspond to the latest QRS forces, but was delayed compared with normal (range 60–96 msec) in patients 1, 4 and 5, in whom the latest right ventricular activation occurred at 110, 108 and 100 msec, respectively.

The latest left ventricular activation occurred at sites removed from the base of the heart in the inferior wall in four patients; the sites were near the apex in three patients and in the midparaseptal region in two patients. One of the latter two patients (patient 4, fig. 4) had simultaneous latest activity in both inferior left ventricular sites. Patient 3 had latest left ventricular activation at the obtuse margin, adjacent to the atrioventricular groove. The timing of the latest left ventricular epicardial activation ranged from 113–140 msec (mean 124 ± 9 msec) after the onset of QRS, which was later than normal (range 60–87 msec) in all patients and occurred within 6–47 msec (mean 20 ± 7 msec) of the end of the QRS complex. Thus, the latest left ventricular epicardial activity accompanied the terminal forces of the QRS.

A comparison of the mean latest right and left ventricular activity between our 11 patients with normal QRS and the five patients with LBBB shows that LBBB was associated with a slightly later occurrence of terminal right ventricular activation (75 ± 10 msec vs 87 ± 24 msec; p < 0.1), and also with markedly later left ventricular terminal activation (70 ± 9 msec vs 124 ± 9 msec; p < 0.0001).

Discussion

Mapping before and after production of experimental bundle branch block in canines and primates has been used to elucidate the patterns of normal ventricular activation, as well as for the study of activation derangements due to bundle branch block. Lewis believed that ventricular activation in right bundle branch block resulted from a wave front moving uniformly through the myocardium from left to right. Rodriguez and Sodi-Pallares and Sodi-Pallares et al. found evidence that the ventricular septum was activated almost entirely from the left bundle branch, except for a thin layer on the right septal surface, and that in many canine hearts there was a region of the apical right septal surface that was actually depolarized exclusively from the left. They concluded that the prolongation of the QRS in LBBB was due not to diffuse slowing of the impulse crossing from the right to left through the septal mass, but rather to a septal “barrier” that was approximately 1–2 mm thick and within the septum close to the right side. They also concluded that the remainder of septal activation occurred at normal velocity, and that activity within the left ventricular free wall occurred with normal sequence and normal endo-epicardial and apical-basal direction. Erickson et al. subsequently used an increased number of recording sites with more advanced recording technology to show that in acute experimental canine bundle branch block, the excitation wave front spread uniformly from the contralateral ventricle across the septum, with slower-than-normal septal velocity, to the blocked ventricle, and that changes occurred in the site and extent of early endocardial activation in the blocked ventricle. Similar findings were reported by van Dam in chronic canine LBBB. Pruitt et al. suggested that the mechanism of slow transseptal conduction was due to the perpendicular direction of movement of the septal front relative to the orientation of the myocardial fibers in the long axis of the septum, a view with which Scher did not concur. Activation during the latter portion of the QRS after segmental left bundle branch lesions was shown by Gelband et al. to be associated with late activation of the left bundle branch peripheral network beyond the lesions. This resulted in rapid spread of the impulse through the subendocardial layers, albeit with an abnormal activation sequence, resulting in endo-epicardial activation of at least part of the left ventricular free wall.

Very little information about the ventricular activation sequence in human LBBB is available. No systematic epicardial mapping studies of the left ventricle have been reported, to our knowledge. A single case report of epicardial and plunge electrode data in a patient with LBBB and aortic stenosis is included in van Dam's review of activation sequence in bundle branch block. In his patient, right ventricular activation was normal. Septal activation proceeded slowly from right to left beginning in the apical region. Left ventricular free wall activation was also oriented from apex to base, with isochrones proceeding at more or less equally spaced intervals. The latest area to be ac-
tivated was in the basal anterolateral left ventricle, 110 msec after the onset of the QRS, which measured 120 msec in duration in his patient.

Our previous studies in patients without intraventricular conduction defects have shown the following salient features of the epicardial activation sequence of the normal QRS: 1) three to five epicardial breakthroughs in all patients; 2) initial epicardial breakthrough in the anterior right ventricle adjacent to the septum 7–25 msec after the onset of the QRS; 3) two to four subsequent breakthroughs in all patients in the inferior right ventricle and in the inferior and anterolateral left ventricle 18–48 msec after the onset of QRS; 4) a tendency for the anterior septal region to be activated in a right-to-left direction, and for the posterior septal region to be activated in a left-to-right direction; 5) latest right ventricular epicardial activation localized to the outflow tract, anterobasal region or the posterobasal region; 6) latest left ventricular epicardial activation invariably in the posterobasal region; and 7) latest epicardial activation (63–96 msec after the onset of the QRS) that measured 75–95 msec.

Supporting the validity of conclusions based on the epicardial activation sequence in patients with intraventricular conduction defects are our reported observations in patients with electrocardiographic left anterior fascicular block, who had 1) no normal basal anterolateral left ventricular epicardial breakthrough and 2) abnormal delay in activation to the same region, with latest left ventricular activation in the basal anterolateral region, which was never the site of latest activity in the patients with normal QRS. We concluded that the epicardial sequence in patients with marked left-axis deviation was consistent in the presence of block or delay in the left anterior border fibers or fascicle of the left bundle branch.

In the present study, we examined epicardial activation sequence in five patients with LBBB and normal axis who underwent surgery for coronary artery disease. In three of the patients, LBBB was chronic and in two it occurred intraoperatively and persisted transiently after operation. The epicardial activation sequence was abnormal and similar in all, with the following major features: 1) right ventricular initial breakthrough anteriorly was normal in location in all patients, but earlier than normal in the three patients with chronic LBBB, and earlier than the corresponding site of initial breakthrough in the “control” preoperative map in the two patients with intraoperative LBBB; 2) location of latest right ventricular activation in a normal site in four of the five patients, but delay of activation of this site to beyond expected normal limits in three of the patients, including one with an abnormal location of latest right ventricular activity inferiorly at the apex; 3) absence of any discrete left ventricular epicardial breakthroughs in all patients; 4) activation of the epicardium overlying the septal regions by slowly moving wave fronts (crowded isochrones) activating the anterior septal region ahead of the posterior septal region by a mean of 46 msec; 5) activation of the anterolateral wall ahead of the inferior wall by a mean of 42 msec; 6) widely spaced isochrones over the free wall of the left ventricle; and 7) location of latest left ventricular activation 113–140 msec (mean 124 msec) after the onset of QRS, or 6–47 msec (mean 20 msec) before the end of the QRS, which measured 130–160 msec (mean 144 msec) in these patients with LBBB.

The observation of earlier-than-normal right ventricular epicardial breakthrough is consistent with the initiation of ventricular depolarization on the left septal surface in patients with normal conduction, and on the right ventricular endocardial surface in patients with LBBB. In LBBB, the epicardial site recording right ventricular breakthrough is anatomically closer to the site of early endocardial right ventricular depolarization than to the left septal surface. Hence, that recording site would be expected to record activity earlier than normal during the QRS in LBBB. Durrer et al. demonstrated that in the normal isolated human heart, left septal activation precedes the onset of right ventricular activity by 5–10 msec, which is consistent with most but not all canine data. If conduction time in the right bundle branch were longer than that in the left bundle branch, as implied by these observations, patients with intermittent LBBB should have a longer HV interval during LBBB than during normal conduction. In two small series, Berkowitz et al. and Narula showed that this could occur. However, subsequent studies by Schuilenburg et al., Rosen et al. and Denes et al. have shown no consistent significant change in HV intervals between normal conduction and intermittent LBBB. One possible explanation for this inconsistency is that the left bundle branch in patients with intermittent LBBB has a slightly prolonged conduction time during normal conduction, sufficient to equal that in the right bundle branch, so that no further lengthening of HV occurs with LBBB.

Delay in the occurrence of the latest right ventricular epicardial activation during LBBB compared with normal is consistent with data from studies in dogs suggesting that part of the right septal surface is normally dependent upon the left bundle branch for depolarization, as originally postulated by Sodi-Pallares et al. Scher et al. showed that this tendency was particularly marked in the posterior one-eighth of the septum and that, in an occasional animal, the creation of LBBB delayed activation of some portion of the posterior right septal surface by 3–7 msec. If, during LBBB, this area of the septum in humans were not dependent upon the right bundle branch for its activation, relatively late occurrence of paraséptal epicardial activation would be expected, as we have found. In the absence of direct recordings from the septum, we cannot shed any light on the controversial question of the septal “barrier” of Sodi-Pallares. Though our data show smooth activation of the septal region of the epicardium, this does not necessarily reflect passage of a smooth wave front through the septum itself. However, data of others in the dog and in one instance of human LBBB suggest that there is no discrete narrow zone where septal delay takes place.
The epicardial activation of the anteroseptal region and of the anterolateral left ventricular wall before that of the posteroseptal region and inferior left ventricular wall in our patients may relate to their normal frontal QRS axis. Assuming that the time course of epicardial potential distribution during the QRS approximates that of the isochrones during LBBB, a normal mean QRS vector would be seen. It could be speculated that the presence of left-axis deviation in patients with LBBB might reflect relatively later anterior left ventricular activation compared with inferior activation. LBBB with left-axis deviation has been associated with more extensive myocardial disease and with a longer HV interval (implying bilateral bundle branch disease) than in patients with LBBB and normal axis. In these patients, delay in the anterior region of the left ventricle might reflect delay in activation of the anterior relative to the inferior left ventricular wall, due to asymmetric pathologic processes in the anterior vs inferior left bundle branch fibers, or to intrinsic myocardial delays secondary to fibrosis or infarction, resulting in disordered septal crossing. This hypothesis should be testable using epicardial mapping techniques.

Based on the more widely spaced isochrones over the free wall of the left ventricle after completion of septal crossing, it seems probable that upon reaching the left ventricular Purkinje tissue, the impulse accelerates endocardial activation of at least part of the left ventricle, late in the QRS in LBBB. This is consistent with the data of Gelband et al. showing circuitous return to specialized tissues distal to sites of experimental block in the left bundle branch system in neonatal puppies. This phenomenon probably accounts for the fact that in human LBBB, in which pathologic changes are found predominantly in the proximal portion of the bundle, the QRS duration seldom exceeds 0.18 second. Considering the dimensions of normal human hearts, the increased dimensions of most hearts with LBBB and the known velocity of conduction through healthy myocardium, it is probable that if left ventricular activation in LBBB were to depend entirely on intramyocardial conduction, it would be delayed to a much greater extent than is usually seen in LBBB.

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