Incomplete Left Bundle-Branch Block

The View from Transseptal Intraventricular Leads

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SUMMARY

The direction of the initial 20 to 30 msec forces of multiple unipolar and transseptal bipolar intraventricular electrocardiograms was examined in five patients with and five without incomplete left bundle-branch block (LBBB) and in one patient with complete LBBB. The data obtained were consistent with preservation of the normal left-to-right and apex-to-base direction of septal depolarization in patients with apparent, incomplete LBBB. Furthermore, no evidence of electromechanical delay in the left ventricle could be ascribed to this conduction defect. Until the minute details of normal and abnormal human septal excitation become known, it is suggested that the more descriptive and less mechanistic term “initial force abnormality” be substituted for “incomplete LBBB.”

Additional Indexing Words:
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Intraventricular conduction defect

INCOMPLETE left bundle-branch block (LBBB) continues to be a controversial electrocardiographic interpretation despite the accumulation of a considerable number of clinical and experimental observations concerning its existence.

The concept of incomplete block of a bundle branch originated nearly 50 years ago with the canine studies of Rothberger and Winterberg.1 They observed that delayed conduction through a bundle branch could give rise to QRS complexes transitional between complete bundle-branch block and normal conduction. Subsequently, Segers2 reported such complexes in the electrocardiogram of a patient with intermittent complete LBBB.

An experimental basis for the concept of incomplete LBBB was provided by the studies of Sodi-Pallares and associates3-7 beginning in the 1950’s. They obtained recordings from intramural and intracavitary electrodes in dogs with varying degrees of experimentally induced LBBB. They also made intracavitary recordings in humans with apparent incomplete LBBB. From these observations they concluded that there is a reversal in the direction of septal activation in incomplete LBBB similar to that found in complete LBBB, and they established criteria for diagnosis of the incomplete form from standard electrocardiograms.

Later Grant and Dodge8 challenged the existence of incomplete LBBB as a stable
Table 1
Clinical and Electrocardiographic Data, LBBB Patients

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yr), sex</th>
<th>Diagnosis</th>
<th>Standard leads</th>
<th>QRS duration (sec)</th>
<th>Extra leads</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 S.F.</td>
<td>22F</td>
<td>MS, mild</td>
<td>Notched P waves; no Q I, V5, V6; no R V1, V2</td>
<td>0.08</td>
<td>No Q V5, V6, V9; no Q V5-V9 up &amp; down 1 &amp; 2 incsp.; no R VR3, VR4; no R VR4-V5 up 1 &amp; 2 incsp.; no R VR4-V1 down 1 &amp; 2 incsp.;</td>
</tr>
<tr>
<td>2 B.B.</td>
<td>26F</td>
<td>MS, severe; AS, mild; pulmonary hypertension, moderate</td>
<td>LAE, RVH; no Q I, V9; qR VR3, VR4; Rs V1, V2</td>
<td>0.08</td>
<td>—</td>
</tr>
<tr>
<td>3 W.S.</td>
<td>39M</td>
<td>AS, severe; pulmonary hypertension, moderate</td>
<td>LVH; no Q I, V9, *6; QS V1 with initial notch</td>
<td>0.11</td>
<td>—</td>
</tr>
<tr>
<td>4 J.W.</td>
<td>54M</td>
<td>AI, severe; AS, mild</td>
<td>LAD; LVH; no Q I, V5, V6; no R V1, V2</td>
<td>0.09</td>
<td>—</td>
</tr>
<tr>
<td>5 E.L.</td>
<td>54M</td>
<td>AI, severe; MS, moderate; pulmonary hypertension, mild</td>
<td>Atrial fibrillation; LVH; no Q &amp; slurred R upstroke, I, V5, V6; QS with initial notch V1, V2</td>
<td>0.11</td>
<td>No Q &amp; slurred R upstroke; V5 up 1 &amp; down 1 &amp; 2 incsp., V6 up &amp; down 1 &amp; 2 incsp. QS with initial notch; V1 up &amp; down 1 &amp; 2 incsp., V2 up 1 &amp; 2 incsp., qrS, V5 down 1 incsp., rS, V2 down 2 incsp.</td>
</tr>
<tr>
<td>6 N.T.</td>
<td>37M</td>
<td>Congenital bicuspid aortic valve</td>
<td>Wandering pacemaker; complete LBBB; no Q &amp; slurred upstroke, I, V5, V6; rS in V1, V2</td>
<td>0.14</td>
<td>—</td>
</tr>
</tbody>
</table>

Abbreviations: AI = aortic insufficiency; AS = aortic stenosis; MS = mitral stenosis; incsp. = intercostal space; LAE = left atrial enlargement; LAD = left axis deviation; RVH = right ventricular hypertrophy; LVH = left ventricular hypertrophy.

Conduction defect, mainly on the basis of the observed rarity of such an initial force abnormality in patients with transient complete LBBB. Others have objected to the inclusion of initial force abnormalities with normal QRS duration among the criteria for incomplete LBBB.9-11 Said and Bryant9 reported lack of Q waves or slurred R wave upstrokes or both in leads V5 and V6 in certain healthy young individuals and postulated normal variations in the order of ventricular activation as an explanation. Johnston and Willis10 ascribed the absence of Q waves in some instances to clockwise rotation of the heart. More recently, Burch and DePasquale11 have proposed electrical inertness of the septum due to fibrosis as a mechanism for similar initial force changes.

The purposes of the present study were to re-examine the evidence for reversal of septal activation by means of intracavitary electrocardiograms in patients with apparent incomplete LBBB, to extend these observations by recording multiple bipolar transseptal
### Table 2

**Clinical and Electrocardiographic Data, Control Patients**

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yr), sex</th>
<th>Diagnosis</th>
<th>Electrocardiogram</th>
<th>QRS duration (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>7 J.K.</td>
<td>29F</td>
<td>MS, mild</td>
<td>Normal</td>
<td>0.08</td>
</tr>
<tr>
<td>8 W.S.</td>
<td>20M</td>
<td>Innocent murmur</td>
<td>Suggests LVH; abnormal T waves</td>
<td>0.11</td>
</tr>
<tr>
<td>9 G.C.</td>
<td>48M</td>
<td>MS, severe; MI, mild; pulmonary hypertension, severe</td>
<td>Atrial fibrillation; incomplete RBBB; suggests RVH</td>
<td>0.07</td>
</tr>
<tr>
<td>10 N.M.</td>
<td>48M</td>
<td>AS, severe; AI, mild</td>
<td>LAD; complete RBBB</td>
<td>0.14</td>
</tr>
<tr>
<td>11 J.M.</td>
<td>57M</td>
<td>AS, moderate; P.O. ASHD</td>
<td>Old inferior infarct</td>
<td>0.10</td>
</tr>
</tbody>
</table>

Abbreviations: AI = aortic insufficiency; AS = aortic stenosis; P.O. = postoperative; LAD = left axis deviation; ASHD = arteriosclerotic heart disease; M.S. = mitral stenosis; M.I. = mitral insufficiency.

### Table 3

**Intraventricular QRS Characteristics**

<table>
<thead>
<tr>
<th>Case</th>
<th>Group</th>
<th>RV mid</th>
<th>RVOT</th>
<th>RVIT</th>
<th>RV apex</th>
<th>LV</th>
<th>Bipolar transseptal, LV to</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>RV mid</td>
</tr>
<tr>
<td>1</td>
<td>ILBBB</td>
<td>QS*</td>
<td>rsR'S' to qRs</td>
<td>—</td>
<td>—</td>
<td>QS</td>
<td>rSr'</td>
</tr>
<tr>
<td>2</td>
<td>ILBBB</td>
<td>qrS</td>
<td>—</td>
<td>QS</td>
<td>—</td>
<td>QS</td>
<td>Rs</td>
</tr>
<tr>
<td>3</td>
<td>ILBBB</td>
<td>rS</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>QS*</td>
<td>rSr'</td>
</tr>
<tr>
<td>4</td>
<td>ILBBB</td>
<td>QS</td>
<td>rS</td>
<td>—</td>
<td>—</td>
<td>qRs</td>
<td>Qr</td>
</tr>
<tr>
<td>5</td>
<td>ILBBB</td>
<td>QS</td>
<td>QS to qrs*</td>
<td>—</td>
<td>—</td>
<td>QS</td>
<td>Qr</td>
</tr>
<tr>
<td>6</td>
<td>CLBBB</td>
<td>QS</td>
<td>QS</td>
<td>—</td>
<td>—</td>
<td>RS</td>
<td>Qr</td>
</tr>
<tr>
<td>7</td>
<td>Control</td>
<td>rS</td>
<td>rSr's' to QS</td>
<td>—</td>
<td>—</td>
<td>QS</td>
<td>R</td>
</tr>
<tr>
<td>8</td>
<td>Control</td>
<td>rS</td>
<td>rS</td>
<td>—</td>
<td>—</td>
<td>rS</td>
<td>Qr</td>
</tr>
<tr>
<td>9</td>
<td>Control</td>
<td>qrS</td>
<td>rsR'S'</td>
<td>—</td>
<td>qRs</td>
<td>QS</td>
<td>Rs</td>
</tr>
<tr>
<td>10</td>
<td>Control</td>
<td>rsR'S'†</td>
<td>rsR'S'‡</td>
<td>RS‡</td>
<td>—</td>
<td>QS</td>
<td>Rs‡</td>
</tr>
<tr>
<td>11</td>
<td>Control</td>
<td>rS</td>
<td>rsR'S'</td>
<td>—</td>
<td>—</td>
<td>QS</td>
<td>rSr'</td>
</tr>
</tbody>
</table>

*With initial notch
†With terminal slur
‡Initial and terminal slur

Abbreviations: RVOT = right ventricular outflow tract; RVIT = right ventricular inflow tract; — = lead not recorded.

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The usual positions of the electrodes during intracavitary recordings are illustrated. Three are in the PA projection while the bottom right film is a left anterior oblique view. In each instance the tip of the transseptal catheter is in the left ventricle, having crossed the mitral valve. The tip of the right ventricular electrode catheter is in the mid-ventricle in the top left panel, in the outflow tract in the top right panel, and in the apex in the bottom panels.

leads, and to look for evidence of electro-mechanical delay in the onset of left ventricular contraction.

Methods

The 11 patients included in this study were selected from a larger group undergoing diagnostic
Case 3. The external electrocardiogram is recorded at 25 mm/sec. $V_6$, $V_4$, and $V_5$ are at half normal standardization. The tracing is consistent with left ventricular hypertrophy with a QRS duration of 0.11 sec. The initial force abnormality of incomplete LBBB is present. The intracardiac complexes are recorded simultaneously with limb lead I at 100 mm/sec. Both unipolar and bipolar intraventricular tracings indicate the initial forces are directed left-to-right, although the downstroke of the Q wave is slurred in the LV. The sensitivity for each intracardiac lead is $I_{mv} = 8 mm$.

Cardiac catheterization for evaluation of valvular heart disease. Pertinent clinical details are found in tables 1 and 2.

Five patients had initial force abnormalities consistent with incomplete LBBB, one had complete LBBB, and five others who served as controls had either normal conduction or electrocardiographic abnormalities other than LBBB (table 2).

Body surface electrocardiograms were taken with an Elema-Schonander Mingograf recorder. The frequency response of this machine is flat to 400 cps. The electrocardiographic criteria used to characterize the incomplete LBBB group of patients were generally those of Sodi-Pallares and associates.\textsuperscript{3, 6} They include absence of Q waves (with or without slurring of the upstroke of the R wave) in leads "facing" the left ventricle ($I$, a$V_L$, $V_6$, and $V_6$), absence of the R wave in $V_1$, and a QRS duration of 0.08 to 0.11 sec. The electrocardiographic findings for each patient appear in tables 1 and 2.

To examine more closely the initial force abnormality in two patients (cases 1 and 5) in the incomplete LBBB group, external electrocardiograms were taken at a paper speed of 50 mm/sec, and extra precordial leads were obtained ($V_7$, $V_8$, $V_9$, and $V_1$ to $V_6$ one and two intercostal

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Case 1. The standard electrocardiogram is recorded at a paper speed of 50 mm/sec and each lead is taken at the same sensitivity. Its complexes have been retouched for clarity. Although the QRS duration is normal, the direction of initial forces suggests incomplete LBBB. The intracavitary leads are recorded simultaneously with limb lead II, at a paper speed of 75 mm/sec. The unipolar recording from the right ventricle has no initial R wave, but the tracing from the left ventricle is normal. The bipolar lead confirms normally directed left-to-right initial forces.

Intracavitary electrocardiograms from the right side of the heart were obtained using a 7-F bipolar electrode catheter with the recording nickel-silver electrode at the distal tip. Intracavitary pressures were monitored through the central lumen and were used along with fluoroscopy to confirm the catheter tip location prior to each recording.

Unipolar recordings in the right ventricle were usually made from at least two sites. In four patients, these were obtained from three sites: the first at a point midway in the right ventricle (at a level opposite the tip of the left ventricular lead), the second from the outflow tract, and the third from either the apex or inflow tract (fig. 1; table 3).

Intracavitary tracings from the left ventricle were obtained by passing a 2-F platinum electrode probe through an 8-F Ross Teflon transseptal catheter. The electrode tip was advanced 1 or 2 cm beyond the catheter (fig. 1). Pressure could be monitored through the fluid-filled transseptal catheter. No attempt was made to record from more than one site in the left ventricle.

In sequence, unipolar recordings were first made from the left ventricle and from one or more right ventricular sites. Bipolar transseptal tracings...
were then obtained by connecting the two intracavitary electrodes so that the one at each of the right ventricular sites was positive while the other electrode in the left ventricle was negative. Intracavitary recordings were made at times when the electrode tips were not impinging on endocardial surfaces, as evidenced by lack of premature beats or S-T segment elevations. The paper speed was either 75 or 100 mm/sec with some additional recordings made at 200 mm/sec. An Electronics for Medicine DR-8 recorder (frequency response flat to 200 cps) was used for all but one study. A Sanborn 560 recorder with 350-2700 preamplifiers (frequency response flat to 400 cps) was used once.

The high speed left ventricular pressure tracings of three patients with incomplete LBBB, the one patient with complete LBBB, and four controls were examined for electromechanical delay. The time from QRS onset (from external limb lead 1 or II) to the onset of left ventricular pressure rise was measured to the nearest 5 msec (table 4).

Results

Data on the form and duration of the QRS complexes as noted in the records made with intracardiac leads are summarized in table 3. Only the initial 20 to 30 msec forces were examined and compared.
Case 5. The paper speed for the standard electrocardiogram is 50 mm/sec. V₂ is at half normal standardization. Its complexes have been retouched for clarity. The tracing is consistent with left ventricular hypertrophy. The QRS duration is 0.11 sec. The initial force direction and slurred R wave upstroke in V₅ and V₆ suggest incomplete LBBB. The intracavitary leads were recorded at 75 mm/sec. Standardizations are as follows: 1 mv = 3 mm for the first and third panels, 14 mm for the second panel and 6 mm for the fourth panel. While initial positivity could not be found in the RV or on the bipolar lead connected to the mid-RV, the LV complex is a normal QS and initial positivity is found on the bipolar lead to the RV outflow tract.

Unipolar Left Ventricular Tracings
The recordings from the left ventricle all showed the initial negativity anticipated in normals, except in the one patient with complete LBBB (case 6). There was no difference in the direction of the initial forces recorded in the incomplete LBBB and control patients with one exception. In case 3 there was an initial notch on the descending limb of the QS complex (fig. 2). In another patient (case 4) initial negativity, though present, was of small magnitude and was followed by a tall positive deflection.

Unipolar Right Ventricular Tracings
Among the patients with incomplete LBBB the direction of initial forces was variable. In two patients (cases 1 and 4) initial negativity, present in the mid-right ventricle, was replaced by initial positivity in the outflow tract. As the catheter tip moved with cardiac pulsations, the small initial R wave
was intermittently present on continuous recordings.

While initial force direction also varied in the control patients, four of the five showed initial positivity from more than one site in the right ventricle. In one instance (case 7) initial positivity alternated with negativity in the mid-right ventricle. Another control patient (case 9) had initial negativity at two sites and initial positivity only in the outflow tract. Thus two of the five control records did not consistently show the anticipated initial R wave in the mid-right ventricle.

**Bipolar Transseptal Tracings**

Three patients from the incomplete LBBB group had initial positivity, and two had initial negativity on transseptal left ventricle to mid-right ventricle recordings. In the latter two (cases 4 and 5), when the right ventricular outflow tract site was substituted for the mid-right ventricle in the bipolar lead, initial positivity was noted in each (fig. 5). The control patients invariably showed initial positivity on transseptal recordings while the patient with complete LBBB consistently had initial negativity. This was true even though multiple right ventricular sites were connected to the positive terminal. With two exceptions both the magnitude and the duration of the initial R wave was greater in the control group (average R wave duration: 40 msec) than in the group with incomplete LBBB (table 3).

**Pre-contraction Times**

The data on left ventricular pre-contraction times are summarized in table 4. These measurements were not corrected for the delay in pressure transmission through the catheter system and are therefore presented as comparative rather than absolute values. While the values for the three incomplete LBBB patients (all of whom had left ventricular hypertrophy) were longer than for the two controls with apparently normal left ventricles (cases 8 and 9), they were in the same range as those of the two controls (cases 10 and 11) who had left ventricular hypertrophy. All were considerably shorter than those of the patient with complete LBBB.

**Autopsy Data**

Two patients (cases 3 and 4) from the incomplete LBBB group subsequently expired and autopsy was performed. Both had left ventricular hypertrophy and patchy fibrosis throughout the myocardium of the left ventricle. The fibrosis was most marked in the posterior wall close to the apex in case 3 and in the interventricular septum in case 4. Since detailed studies of the conduction system were not done and because the myocardial fibrosis involved large areas of the left

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*Table 4*

**Left Ventricular Pre-contraction Period**

<table>
<thead>
<tr>
<th>Case</th>
<th>Group</th>
<th>Diagnosis</th>
<th>Q to LVp (msec)</th>
<th>Heart rate</th>
<th>Rhythm</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>ILBBB</td>
<td>AS</td>
<td>50</td>
<td>74</td>
<td>Sinus</td>
</tr>
<tr>
<td>4</td>
<td>ILBBB</td>
<td>AI, AS</td>
<td>40</td>
<td>91</td>
<td>PAT, 2:1 A-V block</td>
</tr>
<tr>
<td>5</td>
<td>ILBBB</td>
<td>AI, MS</td>
<td>50</td>
<td>78</td>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td>6</td>
<td>CLBBB</td>
<td>AS (mild)</td>
<td>95</td>
<td>56</td>
<td>Sinus to nodal</td>
</tr>
<tr>
<td>8</td>
<td>Control</td>
<td>Innocent murmur</td>
<td>35</td>
<td>76</td>
<td>Sinus</td>
</tr>
<tr>
<td>9</td>
<td>Control</td>
<td>MS, MI</td>
<td>30</td>
<td>80</td>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td>10</td>
<td>Control</td>
<td>AS, AI</td>
<td>60</td>
<td>65</td>
<td>Sinus</td>
</tr>
<tr>
<td>11</td>
<td>Control</td>
<td>AS, ASHD</td>
<td>45</td>
<td>79</td>
<td>Sinus</td>
</tr>
</tbody>
</table>

Abbreviations: LVp = onset of the rise of left ventricular pressure; PAT = paroxysmal atrial tachycardia. Other abbreviations as in tables 1 and 2.
ventricle in each case, the postmortem findings were not helpful in defining the conduction defects.

Discussion

Intracavitary electrocardiograms were recorded from the right ventricle in humans soon after the introduction of cardiac catheterization as a useful procedure in cardiovascular studies. The first reports of such data by Lenègre and Maurice in 1945 were soon followed by additional observations by Hecht, Battro and Bidoggia, and Sodi-Pallares and Kossman and their associates. Although a QS complex was found in the right ventricle of patients with complete LBBB in contrast to the normal rS form, certain exceptions were noted. Instances in normal patients of change from rS to an initial Q wave were found to be associated with changes in the position of the electrode within the right ventricle, with continuous recording while the electrode was moved from outflow tract to inflow tract, and with phases of respiration.

In the early 1950’s unipolar tracings from the left ventricle in man were first reported by Sodi-Pallares and Gilbert-Queralto and their associates, soon followed by the studies of Zimmerman and Hecht, and Coelho, Steinberg, and Mas and their co-workers. While the typical normal QRS complex had a QS form, isolated instances of initial positivity in the left ventricular cavity in individuals without evidence of LBBB have been reported by several observers.

A bipolar lead between left and right ventricular terminals has been used almost exclusively by Sodi-Pallares and associates for the purpose of determining the direction of transeptal forces in cases of suspected incomplete LBBB. They have reported a few cases of initial right-to-left forces on such a lead when QS complexes were present on the unipolar recordings from within each ventricle and the external electrocardiograms suggested incomplete LBBB. The only other intracavitary electrocardiographic studies of incomplete LBBB have been unipolar right ventricular tracings reported by Latour and Puech. They found normal initial positivity in the right ventricle in some cases in which incomplete LBBB was present on the external electrocardiograms.

Numerous objections have been raised to the use of intracardiac leads for detailed investigation of ventricular excitation. (1) The exact location of the exploring electrode, its proximity to the endocardium, papillary muscles, and interventricular septum is not known. (2) The electrode’s position is not stable, but changes continuously within the beating heart under the phasic influence of respiration. (3) Such an electrode merely reflects the sum total of multi-directional electrical forces; it thus includes many cancellation effects and cannot indicate actual depolarization pathways. (4) A considerable degree of normal variation may occur in ventricular excitation. (5) In patients with conduction defects, control observations have not been made in the absence of defective conduction.

Granted that these objections all have some validity, nevertheless, multiple trans-septal leads should be expected to indicate the direction of early septal forces better than body surface leads for several reasons: (1) because of proximity to the septum; (2) because left and right-sided body leads cannot accurately depict the direction of forces across a septum which separates ventricles lying both anteriorly and posteriorly as well as leftward and rightward; and (3) because certain early free wall excitation forces may be separated from septal forces.

An example of this last reason is provided by the tracings of case 6, the man with complete LBBB (fig. 6). While initial positivity persists in leads V₁ and V₂, initial negativity is present at multiple sites in the right ventricle both on unipolar and bipolar leads. This phenomenon has been noted previously and the right precordial R wave has been attributed to right ventricular free wall excitation.
Case 6. Patient had complete LBBB. The external electrocardiogram is recorded at 25 mm/sec. V₂ is taken at half normal standardization. The intracardiac tracings are recorded at 100 mm/sec. Unipolar tracings from other sites in the right ventricle were identical in configuration with the RV mid. A bipolar lead connecting electrodes in the LV and the RV inflow tract was identical to the two bipolar leads shown. All intracardiac leads were consistent with initial right-to-left forces.

The methods of recording intracardiac electrocardiograms in the present study differ from those of previous investigations both in the transseptal approach to the left ventricle and in the use of more than one bipolar lead. Consequently recordings were made in patients suspected of having incomplete LBBB and, for comparison, in patients with normally directed initial forces.

None of the five patients with indications of incomplete LBBB on body surface electrocardiograms appeared to have consistent reversal of septal activation on the intracardiac recordings. The significance of the lesser magnitude of initial positivity on the bipolar recordings of the incomplete LBBB patients, compared to the controls, is uncertain. The longer duration of the initial R wave in the control patients (who did not have normal hearts) suggests that excitation of the ventricular free wall may contribute more to this deflection than to the smaller R wave of the patients with incomplete LBBB. The possibility must also be considered that septal activation really is abnormal in the incomplete LBBB group.
Among the lead systems used in this study, the unipolar left ventricular leads were the most consistent in indicating normally directed initial forces in both the incomplete LBBB and control groups. On the bipolar and unipolar right ventricular tracings of both groups, initial positivity was more consistent in the outflow tract than lower in the ventricle.

If data on ventricular activation from canine studies are applied to the normal excitation process of the human interventricular septum, there is some experimental evidence to account for the finding of initial positivity in the right ventricular outflow tract. Such studies, both in intact hearts and in the isolated perfused septum, have shown that the septum is depolarized mainly in an apex-to-base direction as well as a left-to-right direction. Both Scher and Amer and their associates, using multiple septal surface electrodes in the dog heart, found that the earliest electrical activity on both surfaces is nearly simultaneous, the right side following the left by 1 to 2 (at the most 5) msec. Septal excitation then proceeds by double envelopment and is completed in 25 msec, at the time when the base of the right septal surface is activated. The faster excitation of the left septal surface probably accounts for the overall left-to-right direction of normal initial transseptal forces. Scher and Young felt that depolarization values for humans should be about 2.5 times those of the dog.

If this depolarization sequence is true for humans without LBBB, the apex-to-base direction of the initial 20 to 30 msec of septal forces might be more consistent than the left-to-right direction. The latter would depend not only on the arrival time of the excitation process on each septal surface but also on the subsequent velocity of spread on each surface. Either a slight acceleration of events on the right septal surface or a slight retardation of events on the left might be sufficient to diminish the total left-to-right sense of septal depolarization. An electrode in the right ventricle might not be perpendicular to the vector representing early septal forces but might actually be parallel to such a vector. One might, therefore, normally find considerable variation in the direction of the initial forces recorded by a unipolar lead from such an electrode, with more likelihood of positivity in the outflow tract. While this seems to be true for our patients, the experiences of others have been variable. By taking the electrical difference between two close transseptal terminals, a bipolar lead tends to amplify the initial forces and should show their directions more consistently than do unipolar leads.

The observation from intracavitary leads of normally directed initial forces (though small in magnitude) in these patients with “incomplete LBBB” suggests at least two possible mechanisms. Either the conduction disturbance is not within the main left bundle or these patients may have a partial form of LBBB which is characterized by diminution in magnitude of the early septal forces rather than by actual reversal of their direction.

Transient incomplete LBBB has been observed in dogs during recovery from experimental LBBB. In such circumstances degrees of right-to-left direction less than that seen in complete LBBB have been documented for septal forces on intramural, septal surface, and intracavitary leads. It does not necessarily follow, however, that a stable initial force abnormality of similar configuration in humans is due to incomplete block of the left bundle-branch system. Indeed, the common association of this initial force abnormality with left ventricular hypertrophy has suggested to other investigators the possibility of a segmental anomaly of activation within the left ventricle. Until more evidence is accumulated to incriminate the left bundle branch as the site of defective conduction, “initial force abnormality” seems a more appropriate term.

Adding further uncertainty to the concept of incomplete LBBB is the lack of electromechanical delay in the left ventricle of patients with this conduction defect beyond that seen in control patients with left ventricular hypertrophy (table 3). Although measuring
the period from the Q wave to the onset of left ventricular pressure rise clearly distinguished the patient with complete LBBB from the other patients in this series, others have not had the same experience. The values for this period given by Braunwald and associates are only slightly longer for patients with complete LBBB than for those with normal conduction, and there is considerable overlap between the two groups. Similar variations have been found in the pre-contraction phase of canine left ventricles following the experimental production of LBBB.

The reasons for these discrepancies remain unclear. One possibility suggested by Braun-Menendez and Solarí is that the duration of the pre-contraction phase provides a better index of the completeness of LBBB than does the QRS configuration; this implies that variations in this phase signify varying degrees of LBBB. Although the present study provides no information about this matter, further catheterization studies of patients with LBBB may yield such information and should lead to a better definition of the entire spectrum of left ventricular conduction defects.

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