The First Heart Sound in Left Bundle Branch Block: An Echophonocardiographic Study

G. W. Burggraf, M.D.

SUMMARY To examine the nature of the first heart sound in left bundle branch block (LBBB), 26 patients with LBBB were studied using echophonocardiography. Apexcardiograms and systolic time intervals were also obtained from these patients and the results were compared with 20 normal subjects. Mitral closure preceded tricuspid closure by 0.02–0.04 second in 19 of the 20 normal subjects. In contrast, in subjects with LBBB there was a variable delay in mitral closure so that in seven subjects the sequence of mitral and tricuspid closure was normal, in 11 it was reversed compared with the normal group, and in eight closure of both valves was simultaneous. The interval from the onset of ventricular depolarization to mitral closure was 0.074 ± 0.019 (mean ± sd) for the patients with LBBB and 0.057 ± 0.008 second for the normal group (p < 0.001). With LBBB the first sound tended to be of reduced intensity and separate mitral and tricuspid components could not be identified. Systolic time intervals and the apexcardiograms demonstrated a delayed onset of left ventricular contraction and an increased isometric contraction time in the LBBB group, both of which would contribute to a delayed mitral valve closure and decreased first heart sound intensity.

LEFT BUNDLE BRANCH BLOCK (LBBB) would logically be predicted to produce a delay in left-sided mechanical events and, thus, a reversal of the sequence of left- and right-sided contributions to the first heart sound (reversed splitting of the first heart sound). The nature of the first heart sound in LBBB has been studied using phonocardiography, apexcardiography, and measurement of intracardiac pressures. A delay in left-sided events has been inferred but not confirmed.

The contribution of valvular events to the first heart sound has been studied using echophonocardiography in normal subjects, in patients with right bundle branch block, complete atrioventricular block, atrial fibrillation, during right ventricular pacing and in various left- and right-sided cardiac abnormalities, but not in patients with LBBB. As the timing of both mitral and tricuspid closure (MVC and TVC) can be precisely detected with echocardiography, this technique would be particularly useful to determine whether the normal sequence of atrioventricular valve closure is reversed in LBBB. The present investigation is an attempt to elucidate further the nature of the first heart sound in LBBB by performing echophonocardiographic correlations in subjects with this conduction disturbance and comparing them with normal subjects.

Methods

The study population consisted of 26 patients with LBBB detected by surveying ECGs done on admission to hospital or requested from the outpatient department and 20 clinically normal subjects. Only subjects with adequate echograms of the mitral and tricuspid valves were included. Patients in the LBBB group were 44–81 years old (mean 62 years). Sixteen females and 10 males were in this group. The normal subjects were 15–79 years old (mean 29 years). Subjects with primary valvular heart disease were excluded. The causes of the LBBB were coronary atherosclerosis, hypertensive heart disease, cardiomyopathy and idiopathic degeneration of the conduction system in the remaining subjects. Because of the effect of PR interval prolongation on the intensity of the first heart sound, we excluded all subjects with a PR greater than 0.20 second. The PR interval in the patient group varied from 0.14–0.20 second (mean 0.16 second) and in the normal subjects from 0.12–0.20 second (mean 0.16 second). Three patients were in atrial fibrillation and the rest were in sinus rhythm.

Phonocardiograms were recorded using Cambridge microphones at the four standard valve areas and a Cambridge fiberoptic multichannel physiologic recorder. External carotid pulse recordings were obtained in 21 patients with apexcardiograms in nine patients using a hand-held rigid funnel connected with plastic tubing to a piezoelectric crystal transducer. The time constant for this pulse recording system was 1.6 seconds. Echocardiograms were recorded using a Smith Kline ultrasonoscope model 20A coupled with the Cambridge fiberoptic recorder to obtain strip-chart recordings. The mitral and tricuspid echograms were each recorded simultaneously with lead II of the ECG and a phonocardiogram recorded from the region of the cardiac apex at a paper speed of 100 mm/sec.

Although precise quantitative phonocardiography was not performed, the loudness of the first heart sound was roughly estimated from the phonocardiographic tracings as low, normal or increased in intensity. We also ascertained whether the first heart sound was single or had two or more distinct bursts of vibrations, and the onset of these components was timed relative to the onset of the QRS. Using the simultaneous external carotid pulse and phonocardiographic recordings, systolic time intervals were obtained as de-
scribed by Weissler and Garrand. The isometric contraction time (ICT) was determined by the difference between the prejection period (PEP) derived from the systolic time intervals and the interval from the beginning of the QRS of lead II of the ECG to the onset of left ventricular contraction determined from the apexcardiogram. Closure of the mitral and tricuspid valves was defined as the point of apposition of both leaflets, or if only the anterior leaflet was visualized, as the abrupt termination of the posteriorly directed closing motion after the onset of ventricular systole. The time from the onset of the QRS to the closure points of the two atrioventricular valves was averaged over five cardiac cycles.

The time intervals used in the results and discussion are:

- \( QS_1 \) = interval from onset of the QRS complex of lead II to the onset of the first high-frequency vibrations of the first heart sound.
- QMVC = time from onset of QRS of lead II of the ECG to MVC.
- QTVC = time from onset of QRS of lead II of the ECG to TVC.

QC = interval from onset of QRS of lead II of the ECG to onset of contraction of the left ventricle as determined from the apexcardiogram.

Statistical comparisons were performed between the LBBB and normal groups using the two-tailed \( t \) test.

## Results

### Characteristics of First Heart Sound

The first heart sound characteristics in both groups are summarized in table 1.

For the 20 normal subjects, \( QS_1 \) was 0.057 ± 0.007 second (mean ± sd). The first sound was single in seven subjects and split in 13. When a second component of the first heart sound was identifiable, it followed the first component by 0.02–0.04 second (mean 0.024 second). The intensity of the first sound was reduced in three and normal in the remaining subjects.

In the patients with LBBB, the first heart sound was decreased in intensity in 14 subjects, normal in nine

### Table 1. First Heart Sound Characteristics

<table>
<thead>
<tr>
<th>Group</th>
<th>( QS_1 ) (seconds)</th>
<th>Intensity</th>
<th>Splitting</th>
<th>Intervals (seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ( \pm ) sd</td>
<td>Range</td>
<td>Normal</td>
<td>Soft</td>
</tr>
<tr>
<td>Normal</td>
<td>0.057 ± 0.007</td>
<td>0.05-0.07</td>
<td>17</td>
<td>3</td>
</tr>
<tr>
<td>(n = 20)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LBBB</td>
<td>0.075 ± 0.014∗</td>
<td>0.05-0.11</td>
<td>9</td>
<td>14</td>
</tr>
<tr>
<td>(n = 26)</td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>

∗Significantly different (\( p < 0.001 \)) from normal group.
Abbreviations: LBBB = left bundle branch block; \( QS_1 \) is defined in Methods.

### Table 2. Atrioventricular Valve Closure Times in Normal Subjects and Patients with Left Bundle Branch Block

<table>
<thead>
<tr>
<th>Group</th>
<th>QRS Duration (sec)</th>
<th>Frontal axis (degrees)</th>
<th>QMVC (sec)</th>
<th>QTVC (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Range</td>
<td>Mean</td>
<td>Range</td>
</tr>
<tr>
<td>Normal (n = 20)</td>
<td>0.08</td>
<td>0.08-0.11</td>
<td>-51</td>
<td>-30 to +120</td>
</tr>
<tr>
<td>All LBBB (n = 26)</td>
<td>0.14</td>
<td>0.12-0.20</td>
<td>+3</td>
<td>-60 to +75</td>
</tr>
<tr>
<td>LBBB MVC before TVC (n = 7)</td>
<td>0.13</td>
<td>0.12-0.16</td>
<td>0</td>
<td>-60 to +45</td>
</tr>
<tr>
<td>LBBB MVC and TVC simultaneous (n = 8)</td>
<td>0.13</td>
<td>0.12-0.15</td>
<td>+17</td>
<td>-30 to +75</td>
</tr>
<tr>
<td>LBBB MVC after TVC (n = 11)</td>
<td>0.15*</td>
<td>0.12-0.20</td>
<td>-7</td>
<td>-45 to +60</td>
</tr>
</tbody>
</table>

∗NS compared with LBBB MVC before TVC.
†NS compared with normal group.
‡\( p < 0.001 \) compared with normal group.
Abbreviations: LBBB = left bundle branch block; TVC = tricuspid valve closure; MVC = mitral valve closure. Intervals are defined in Methods.
and loud in three. Two distinct components were recorded in only four subjects, with the second component 0.11–0.15 second after the QRS, and these were shown to be ejection clicks rather than tricuspid closure sounds. QS1 for the LBBB group was 0.075 ± 0.014 second, significantly later than that in the normal subjects (p < 0.001).

Echocardiograms of Atrioventricular Valves

The closure times for the atrioventricular valves as determined by echocardiography and QRS characteristics are summarized in table 2.

In the normal subjects, MVC preceded TVC in all cases except one, in whom these two events were simultaneous. The values for QMVC and QTVC were 0.057 ± 0.008 second and 0.080 ± 0.011 second, respectively. The onset of the first high-frequency component of the first heart sound was coincident with echocardiographic MVC, and when a second component was visible it similarly timed with TVC.

In contrast, the subjects with LBBB had equal mean QMVC and QTVC values of 0.074 second and the order of atrioventricular valve closure was variable. MVC was significantly later in the LBBB group (p < 0.001), while there was no significant difference in TVC time between the LBBB and normal groups. The sequence of atrioventricular valve closure was mitral before tricuspid in seven subjects, simultaneous in eight subjects, and in the remaining 11 there was reversal of the normal order. The values for closure time in these three groups and the mean QRS duration for each subgroup are listed in table 2. Subjects with the greatest delay in MVC tended to have the longest intraventricular conduction, although this difference did not reach statistical significance. The onset of the first heart sound timed with either MVC or TVC on the echocardiogram, but in no case with LBBB was a second component of the first heart sound timed with an atrioventricular valve closure. In the four patients with an apparent second component of the first heart sound, closure of both atrioventricular valves preceded this sound by 0.03–0.06 second, confirming that these components were in fact ejection sounds.

In figures 1–3, the variable relationship between MVC and TVC in subjects with LBBB is illustrated. Figure 1 is an echophonocardiogram from a patient who had LBBB and shows reversal of the normal sequence of atrioventricular valve closure, with a QMVC of 0.08 and a QTVC of 0.06 second. In figure 2, a patient with LBBB shows a delay in both atrioventricular valve closures, which occur almost simultaneously. In figure 3, mitral and tricuspid valve echograms are shown from a patient with intermittent LBBB. During normal conduction, QMVC was 0.04 second and QTVC was 0.06 second, which is the normal sequence of valve closure (fig. 3A). When this patient has LBBB, these intervals do not change. Thus, no delay occurred in MVC in this patient despite the development of LBBB.

Systolic Time Intervals and Apexcardiograms

Systolic time intervals obtained from the normal subjects and those with LBBB are summarized in table 3. PEP was prolonged, at 140 ± 20 msec, in the group with LBBB, compared with 92 ± 12 msec in the normal subjects (p < 0.001). The PEP index (PEP + 0.4 X heart rate) was also significantly longer in the LBBB group (p < 0.001). Mean PEP was similar in the three subgroups with LBBB (range 138–144 msec) (table 2). Satisfactory apexcardiograms were obtained in nine patients and 16 normal subjects. QC was 58 ± 15 msec and ICT was 86 ± 17 msec in the LBBB patients and 37 ± 8 and 54 ± 15 msec, respectively, in the normal subjects. These differences were significant (p < 0.001). Thus, PEP was prolonged in LBBB due to an increase in both QC and ICT.

Figure 4 is a comparison of the apexcardiograms in a subject with LBBB and intermittent pacing via a right ventricular endocardial electrode. With LBBB, QC was 40 msec, which is similar to QC in subjects

Figure 1. Mitral and tricuspid echophonocardiograms in a patient with left bundle branch block. Tricuspid closure (right) occurs earlier than mitral closure (left). Vertical lines drawn at onset of QRS and time of atrioventricular valve closure. QMVC = interval from onset of QRS to mitral closure (seconds); QTVC = interval from onset of QRS to tricuspid closure (seconds).
with normal conduction. During artificial pacing, QC prolongs to 100 msec, which suggests that in this subject the LBBB was incomplete. The degree of completeness of LBBB must vary considerably in different patients, which would explain the variable relationship between MVC and TVC.

**Discussion**

The results of this study have demonstrated a variable relationship between MVC and TVC in LBBB. Only seven of the 26 patients with LBBB had a normal mitral and tricuspid closure sequence; in the rest it was simultaneous or reversed. The usual interval between mitral and tricuspid first heart sound components using both echocardiography and phonocardiography, determined in other reports and in our normal subjects, is 0.02–0.04 second.6, 9, 12 Thus, the simultaneous MVC and TVC in eight of our patients with LBBB indicates a delay in MVC, as does the reversed split in 11 others.

Baragan and associates studied the first heart sound with phonocardiography and apexcardiography in LBBB and concluded that mitral valve closure was delayed.1 Using intracardiac micromanometer techniques, O'Toole and co-workers also demonstrated a reversed sequence of atrioventricular valve closure in LBBB.3 In contrast, Haber and Leatham reported that most subjects with LBBB did not have a prolonged QC and first heart sound splitting was not reversed.6 They suggested that, most patients with LBBB had an arborization block rather than a complete interruption of the main left bundle.

Several hemodynamic studies have been performed to determine whether there is a delay in onset of left ventricular contraction in LBBB. In a single case report of the hemodynamics in intermittent LBBB, Bourassa and associates could not demonstrate a

<table>
<thead>
<tr>
<th>Group</th>
<th>Heart rate (beats/min)</th>
<th>PEP (msec)</th>
<th>PEPI (msec)</th>
<th>QC (msec)</th>
<th>ICT (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n = 20)</td>
<td>69 ± 11</td>
<td>92 ± 13</td>
<td>120 ± 13</td>
<td>37 ± 8</td>
<td>54 ± 15</td>
</tr>
<tr>
<td></td>
<td>(20)</td>
<td>(19)</td>
<td>(19)</td>
<td>(16)</td>
<td>(16)</td>
</tr>
<tr>
<td>LBBB</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n = 26)</td>
<td>75 ± 15</td>
<td>140 ± 20</td>
<td>170 ± 23</td>
<td>58 ± 15</td>
<td>86 ± 17</td>
</tr>
<tr>
<td></td>
<td>(26)</td>
<td>(21)</td>
<td>(21)</td>
<td>(9)</td>
<td>(9)</td>
</tr>
</tbody>
</table>

Values are mean ± sd. The number of subjects having each measurement is given in brackets. Except for heart rate all measurements are significantly different (p < 0.001) in the two groups.

Abbreviations: LBBB = left bundle branch block; PEP = pre-ejection period; PEPI = PEP index; ICT = left ventricular isometric contraction time. Intervals are defined in Methods.
change in QC with onset of the conduction abnormality. Adolph et al. studied 25 patients with LBBB hemodynamically and found no delay in onset of left ventricular contraction. Braunwald and Morrow did not find a delay in onset of left ventricular contraction using hemodynamic studies in five patients with LBBB. Thus, the hemodynamic evidence indicates no delay in onset of left ventricular contraction in LBBB, and on this basis, paradoxical first heart sound splitting would not be expected to occur in this conduction abnormality. The apparent conflict between previous studies and the demonstration of a variable delay in
mitral closure in this investigation may be explained by varying degrees of completeness of the LBBB in different subjects.

The delay in MVC in the majority of the patients with LBBB was not clinically apparent; reversed splitting was not seen on the phonocardiograms. In most of our subjects with LBBB, the first heart sound was single, of reduced intensity and low frequency. Subjects with reversed atrioventricular valve closure had a longer mean QRS (0.15 second) compared with that of normal of simultaneous closure (0.13 second). This suggests that subjects with a reversed split had a more complete degree of LBBB. We do not have hemodynamic studies in our subjects, but we do have indirect evidence from the apexcardiograms suggesting a delay in the onset of contraction in some subjects with LBBB. It would thus appear that MVC is often delayed and decreased in intensity because of a delay in onset of left ventricular contraction, which allows for a greater atrial contribution to valve closure. Closure of an atrioventricular valve by atrial systole is usually silent.7

A prolonged ICT was demonstrated in our LBBB patients and has also been reported in hemodynamic studies.13,14 The prolonged ICT, which indicates a decreased rate of rise of left ventricular pressure, may be the result of left ventricular disease or the abnormal sequence of depolarization. The reduced rate of rise of left ventricular pressure would be expected to produce both a decrease in first heart sound intensity and a small delay in the onset of MVC. The variable relationship between mitral and tricuspid closure in our subjects might then be explained by depressed left ventricular function, causing delay of MVC in some and not in others. With this possibility in mind, we compared the left ventricular dimensions as measured by echocardiography in the group with a normal sequence and in those with a reversed atrioventricular valve closure. There was no significant difference between these groups. In addition, the distribution of subjects who were asymptomatic and those with compensated heart failure was similar in both groups. Thus, the variable relationship in atrioventricular valve closure could not be explained by differences in ventricular function.

This study has shown a variable relationship between MVC and TVC in LBBB. There was a tendency for a delayed and softer MVC in LBBB compared with normal subjects and this seemed to relate to the degree of completeness of LBBB. Reduced left ventricular contractility may also contribute to the reduced intensity of the first heart sound in LBBB, but is probably not as important a factor in the delay in MVC.

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Effect of Lido
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Via the Accessory Pathway in Patients
with Wolff-Parkinson-White Syndrome

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SUMMARY Electrophysiologic effects of i.v. lidocaine were evaluated in 10 patients with Wolff-Parkinson-White (WPW) syndrome during atrial fibrillation (AF) (eight of 10) or programmed atrial stimulation (nine of 10). The shortest RR intervals during AF were 190–415 msec (mean 217.8 ± 64.5 msec) before lidocaine and decreased to 250.0 ± 85.4 msec (range 180–435 msec, p = NS) after the drug. In six of eight patients, the shortest RR interval decreased and in the remaining two patients it increased by 20 msec after lidocaine. After lidocaine, the average RR intervals during AF for all eight patients decreased from 351.1 ± 45.9 msec to 335.6 ± 68.0 msec (p = NS). After lidocaine, the RR interval shortened in five of eight patients, lengthened in two and did not change in one. In two of eight patients, acceleration of ventricular rate after lidocaine was accompanied by hemodynamic deterioration, necessitating DC cardioversion in one. The control effective refractory period of the accessory pathway (ERP-AP) was 300 msec or less in all patients, and lidocaine prolonged this variable in only one case. In the remaining patients, after lidocaine the ERP-AP either shortened (two of nine), did not change (two of nine) or atrial refractoriness precluded its determination. Similarly, during incremental pacing, the atrial cycle length that produced block in the AP shortened in five patients, lengthened in one and did not change in the others.

In patients with WPW syndrome and relatively short ERP-AP (i.e., ≤ 300 msec), lidocaine generally has no significant effect or produces acceleration of ventricular response during AF. In patients with AF and a rapid ventricular rate due to antegrade conduction over the AP, lidocaine is unlikely to have beneficial effects and may be deleterious.

ATRIAL FIBRILLATION (AF) can be a life-threatening arrhythmia in patients with the Wolff-Parkinson-White syndrome.1–4 The rate of preexcited ventricular responses during AF in these patients is primarily determined by the electrophysiologic properties of the AP in the antegrade direction.3,4 Several investigators evaluated effects of various pharmacologic agents upon the functional properties of the AP.5–17 Most studies so far have dealt with the responses of AP during programmed cardiac stimulation. Rarely have the efficacy and possible deleterious effects of a given drug been assessed during actual AF in patients with ventricular preexcitation. Surprisingly, rather limited data are available dealing with the response of AP to the commonly used antiarrhythmic agents during AF.14,15 With the exception of a single case report, even the effects of lidocaine have not been tested17 in this setting.

The present study was undertaken to systematically evaluate the effect of lidocaine on the electrophysiologic properties of the AP, with particular emphasis on ventricular response during AF in 10 patients with WPW syndrome.

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