Paradoxical Motion of Interventricular Septum in Left Bundle Branch Block

By Abdul S. Abbasi, M.B., M.R.C.P., Leslie M. Eber, M.D., Rex N. MacAlpin, M.D., and Albert A. Kattus, M.D.

SUMMARY
Abnormal interventricular septal motion, with pre-ejection posterior motion and anterior motion away from the posterior left ventricular wall during ejection, was demonstrated by echocardiography in 14 out of 17 cases with complete left bundle branch block (LBBB). Two of 14 cases had intermittent LBBB and showed abnormal septal motion only during LBBB. Of the control group of 49 patients without LBBB but with cardiac disorders similar to the cases with LBBB, only two showed abnormal septal motion. However, pre-ejection motion was not seen in these two cases. During right ventricular pacing abnormal septal motion was observed in three out of ten cases. It is suggested that conduction abnormalities are responsible for abnormal septal motion in LBBB; normal septal motion in most cases with right ventricular pacing may be due to different conduction pathways not affecting the septum.

Additional Indexing Words:
Noninvasive technique Cardiomyopathy Right ventricular pacing
Right ventricular volume overload Ultrasound

Left ventricular wall motion has been found to be normal in angiocardiographic studies of patients with complete left bundle branch block (LBBB) and no other cardiac abnormalities. However, motion of the interventricular septum in LBBB has not been investigated angiographically because of the difficulty in visualizing the septum without simultaneous opacification of both ventricular cavities.

Echocardiography is a convenient method for recording motion of the ventricular septum. This study was carried out to evaluate septal and posterior left ventricular wall motion using echocardiography in patients with LBBB.

Methods
Echocardiograms were analyzed in four groups of patients: group I consisted of 17 patients with LBBB; group II consisted of 49 patients without LBBB but with cardiac diseases similar to those present in group I; group III consisted of 30 healthy volunteers; group IV consisted of ten patients whose hearts were being paced electrically from the region of the apex of the right ventricle with an electrode catheter, and whose paced beats resembled LBBB.

Patients with suspected or proven coronary artery disease were excluded from groups I, II, and III. Diagnoses were confirmed by cardiac catheterization and coronary arteriography in all cases in group I, and all but eight cases of uncomplicated hypertension in group II.

The criteria for LBBB were those of the New York Heart Association: QRS \( \geq 0.12 \) sec; notching or slurring of the QRS which shows an initial R wave in I, aVL, and left precordial leads; in these cases the peak of the R wave or one of its prominent notches occurs relatively late in the QRS interval; displacement of the S-T segment and usually the T wave in a direction opposite that of the principal QRS deflection.

Echocardiograms were performed with commercially available equipment using a 2.25 MHz, 0.5 cm diameter transducer with a focus at 10 cm and a repetition rate of 1000 impulses per second. With the conventional technique described previously, the transducer was placed in the fourth or fifth intercostal space close to the left sternal border, and was directed antero-posteriorly and slightly medially to identify the mitral valve. The transducer was then pointed laterally and inferiorly, to obtain echoes simultaneously from the ventricular septum and posterior left ventricular wall just below the mitral valve. Care was taken to point the transducer inferiorly and not superiorly in order to avoid recording from the aortic root and membranous part of the interventricular septum. Records showing only a small part of the mitral valve were included. If the entire anterior mitral valve leaflet was seen in the record, the tracing was not included in the present.
study because this indicated that the transducer was pointed medially and the beam may have been close to the root of the aorta. In this position, abnormal septal motion has been seen by us and others even in normal individuals. Occasionally with a dilated left ventricle it was not possible to obtain an adequate record of the posterior wall without recording the mitral valve. However in such cases the transducer was directed enough laterally to avoid the aortic root. With adjustment of the gain controls it was possible to record endocardium of the posterior left ventricular wall in all but four cases. In these the epicardial-pericardial echoes were analyzed.

**Results**

**Patient Population**

The types of cases in groups I and II are shown in table 1. One patient in group I and two patients in group II with aortic stenosis had an aortic prosthetic valve, implanted at least one year prior to the study. Patients with hypertension had diastolic pressures of over 100 mm Hg. None of the patients with mitral insufficiency had associated tricuspid insufficiency. Congestive cardiomyopathy was diagnosed on the basis of cardiomegaly and episodes of clinical failure in the absence of valvular, atherosclerotic, or hypertensive heart disease. Echocardiograms were available with and without LBBB in one patient who had congestive cardiomyopathy and one normal person with intermittent LBBB. The ten patients in group IV were those requiring a temporary or permanent right ventricular pacemaker because of heart block or the sick sinus node syndrome. Four of these had clinical evidence of ischemic heart disease.

The mean age of groups I and II was comparable (49 years, with range of 28–72 for group I, and 45 years, with range of 25–68 for group II). The mean age of the healthy volunteers was younger (30 years, with a range of 22 to 45). The group IV patients were slightly older (mean age of 52 years, with a range of 40–80).

**Table 1**

<table>
<thead>
<tr>
<th>Types of Patients</th>
<th>Group I (with LBBB)</th>
<th>Group II (without LBBB)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic stenosis</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>Hypertension</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>Mitral insufficiency</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td>5</td>
<td>16</td>
</tr>
<tr>
<td>Normal</td>
<td>2</td>
<td>—</td>
</tr>
<tr>
<td>Total</td>
<td>17</td>
<td>49</td>
</tr>
</tbody>
</table>

**Electrocardiograms**

The duration of QRS in group I was between 0.12 and 0.14 in seven cases, between 0.15 and 0.17 in eight cases, and 0.18 in two cases. The mean frontal plane QRS axis was normal in seven cases and leftward (superior to −30°) in ten cases.

**Echocardiograms**

Representative echocardiograms from the various groups are presented in figures 1 to 4 and show left ventricular wall and ventricular septal motion.

The letters used to represent different intervals in this study were those introduced by Edler and Gustafson to describe the timing of mitral valve motion and by Kraunz and Kennedy for left ventricular wall motion.

Normally, following the P wave of the electrocardiogram, the posterior left ventricular wall and ventricular septum move away from each other as

![Figure 1](http://circ.ahajournals.org/)

**Figure 1**

Diagrammatic echocardiographic patterns of interventricular septum (IV septum) and posterior left ventricular wall (Post. LV) in normal and in LBBB. The echocardiograms were recorded at different scales; thus the amplitudes of wall motion are not directly comparable. The time markings in each record are 1.0 sec apart.

![Figure 2](http://circ.ahajournals.org/)

**Figure 2**

Normal echocardiograms showing interventricular septal and posterior left ventricular wall motion. ECG = Electrocardiogram; Phono = Phonocardiogram; Chordae = Chordae tendineae.
At the beginning of ventricular diastole a notch in the septal motion is commonly seen. This general pattern of the posterior left ventricular wall and the septal motion was seen in all the normal subjects in group III, and all but two cardiac cases of group II. These two cases had congestive cardiomyopathy without LBBB, and had abnormal septal motion similar to that seen in group I except for one important difference described below.

The patients with LBBB (group I) showed three patterns of ventricular septal motion: 1) normal motion in three cases; 2) paradoxical septal motion in ten cases; 3) intermediate septal motion in four cases. There was no relationship between the type of cardiac disease and the type of septal motion (table 2).

The analysis of paradoxical septal motion in cases with LBBB (figs. 1 and 3) showed an anterior displacement of the septum following the P wave, as was seen in normal cases. However, during pre-ejection phase there was a rapid posterior septal motion, often quite prominent. Following this, there was a major anterior (paradoxical) septal motion away from the posterior left ventricular wall during ejection, instead of a normal posterior septal motion; the posterior left ventricular wall moved normally, that is, anteriorly at this time. At the beginning of ventricular diastole, following the T wave of the electrocardiogram, both the septum and the posterior wall moved posteriorly, as compared to the normal anterior septal motion during ventricular diastole. One healthy adult and one patient with cardiomyopathy, both with intermittent LBBB showed abnormal septal motion as described above only during a LBBB pattern. During normal conduction the septal motion was normal.

The intermediate type of septal motion was more complex (figs. 1 and 4). It had a similar anterior motion following the P wave, as seen in normal cases or in cases with LBBB. Following the QRS there was a prominent pre-ejection posterior septal motion as seen in paradoxical septal motion.

### Table 2

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Interventricular septal motion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>1</td>
</tr>
<tr>
<td>Hypertension</td>
<td>0</td>
</tr>
<tr>
<td>Mitral insufficiency</td>
<td>1</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td>1</td>
</tr>
<tr>
<td>Normal</td>
<td>0</td>
</tr>
</tbody>
</table>
described above. However, the subsequent anterior (paradoxical) septal motion was much reduced in amplitude and was often interrupted by one or two mid- to late systolic posterior motions, toward the posterior left ventricular wall.

Although the initial rapid pre-ejection motion was seen in both the types of abnormal septal motion associated with LBBB, it was absent in two cases of cardiomyopathy that showed abnormal septal motion without LBBB.

A careful analysis of the echocardiogram in three patients with apparently normal septal motion showed a delayed posterior left ventricular wall motion in one. No significant difference in QRS duration or axis deviation was noted in these three patients as compared to those with LBBB and paradoxical septal motion.

In patients in group IV, only three out of ten showed paradoxical septal motion.

**Discussion**

Our study showed abnormal septal motion in 14 out of 17 cases with LBBB. No abnormality was found in posterior left ventricular wall motion. In two of the cases showing abnormal septal motion, no further abnormality was detected on physical examination or at cardiac catheterization. That the abnormal septal motion was secondary to abnormal conduction was further demonstrated by two cases with intermittent LBBB. In both, abnormal septal motion occurred only during LBBB. During normal conduction, the ventricular septal motion was normal. The relationship of abnormal septal motion to LBBB was further supported by findings in our control group II without LBBB, all but two of which showed normal septal motion, even though cardiac disorders similar to those in group I were present. In these two cases abnormal septal motion may have been related to the intrinsic cardiac disease, which was cardiomyopathy in both. Motion abnormalities of left ventricular wall have been found secondary to cardiomyopathy or ischemic heart disease. Abnormal septal motion has also been reported in ischemic heart disease with or without LBBB. It is possible that abnormal septal motion may also occur in cardiomyopathy unassociated with LBBB. Our study did not include cases with coronary artery disease. The incidence of paradoxical septal motion without LBBB in our series of cardiomyopathy was low (12.5%); while with LBBB, a significant number of patients with cardiomyopathy (four out of five) had abnormal septal motion. This suggests that paradoxical motion in cardiomyopathy is more often related to LBBB. Moreover, an important difference between the abnormal septal motion associated with LBBB and that occurring with cardiomyopathy, but without LBBB, was noted; this was the presence of pre-ejection posterior septal motion with LBBB and its absence without LBBB. Thus it appears that the pre-ejection motion of the septum may be the more specific abnormality associated with LBBB.

An abnormal septal motion has been found in patients with right ventricular volume overload which was not present in any of our cases.

The mechanism of paradoxical septal motion in LBBB is uncertain. It may be that LBBB results in early pre-ejection posterior motion of the septum due to displacement from rising right ventricular isovolumic pressure that exceeds the pressure developed by the delayed left ventricular contraction. However, normal septal motion during apical right ventricular pacing does not favor this hypothesis. A better explanation may be an initial septal activation on the right side of the septum as occurs in LBBB causing septal contraction first when the rest of the left ventricle is quiescent. When the rest of the left ventricle contracts, the septum is relaxed and is pushed anteriorly away from the posterior left ventricular wall because of rising left ventricular pressure.

The three patients with LBBB who had apparently normal septal motion could have a more peripheral block, as has been suggested to occur in some cases with LBBB. Thus the septal activation may occur normally, and septal motion may remain normal. A delayed posterior left ventricular wall motion may be expected in these cases; however, it was only seen in one of our three cases.

It is not clear why only three of ten patients who had right ventricular pacing and a "LBBB pattern" on the electrocardiogram during pacing showed abnormal septal motion. However, all these patients had pacing performed from the apex of the right ventricle. It is possible that several conduction pathways exist in different areas of the right ventricle, and paradoxical septal motion may be seen only when an appropriate region of the right ventricle is stimulated.

The question may be raised whether LBBB with abnormal motion of the interventricular septum has any hemodynamic consequences. A reduction of cardiac output has been reported during LBBB in patients with intermittent LBBB. However, we did not find any hemodynamic abnormality in two of our cases with permanent LBBB who had no
associated cardiac disease. It may be that during acute LBBB, as occurs in patients with intermittent LBBB, stroke volume may drop, and in more chronic cases of established LBBB, compensatory mechanisms may normalize the hemodynamics.

Present evidence in the literature suggests that LBBB which is unassociated with cardiac disease may be benign.17,18 This may be so in otherwise normal patients, even though the interventricular septum may be asynergic. Whether or not patients with LBBB, occurring concurrently with other cardiac pathology, e.g., aortic stenosis or cardiomyopathy, are at risk because abnormal septal motion has not yet been determined.

Since completion of this study, a paper by MacDonald, “Echocardiographic demonstration of abnormal septal motion in left bundle branch block” has appeared in print.19 Our findings are similar to his. After submission of this manuscript for publication, Dillon and his associates reported studies that showed that the abnormal septal motion in LBBB can be distinguished from that seen in right ventricular overload.20

Acknowledgment

We wish to acknowledge the technical assistance of Mrs. Nancy Ellis.

References

Paradoxical Motion of Interventricular Septum in Left Bundle Branch Block
ABDUL S. ABBASI, LESLIE M. EBER, REX N. MACALPIN and ALBERT A. KATTUS

Circulation. 1974;49:423-427
doi: 10.1161/01.CIR.49.3.423

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1974 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on
the World Wide Web at:
http://circ.ahajournals.org/content/49/3/423

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally
published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not
the Editorial Office. Once the online version of the published article for which permission is being
requested is located, click Request Permissions in the middle column of the Web page under Services.
Further information about this process is available in the Permissions and Rights Question and Answer
document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/