Incomplete Left Bundle-Branch Block

A Definite Electrocardiographic Entity

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SUMMARY

Rate-dependent intermittent incomplete and complete left bundle-branch block (LBBB) is described in a patient without other evidence of heart disease. Continuous electrocardiographic tracings revealed QRS complexes intermediate in configuration between normal conduction and complete LBBB during the transitional phase from normal to abnormal conduction. This provided an opportunity to study the various grades of incomplete LBBB in man. During the incomplete LBBB phase, the morphology of the QRS complexes bore a close resemblance to the QRS form in the Wolff-Parkinson-White syndrome (type B). The differences between these two entities are emphasized.

Additional Indexing Words:
Critical rate phenomenon Wolff-Parkinson-White syndrome Delta wave
Intrinsicoid deflection Septal activation

INCOMPLETE left bundle-branch block (LBBB) has been produced and studied in experimental animals, but its occurrence in man has been conclusively established only recently. Prior to 1964, when Schamroth and Bradlow described a patient with intermittent incomplete LBBB of gradual onset and disappearance, this abnormality received little documentation. Schamroth and Bradlow's report provided for the first time convincing evidence of the existence of incomplete LBBB. The purpose of the present communication is to report the same phenomenon in an additional patient who exhibited various grades of incomplete LBBB, thus presenting further evidence of the existence of such an entity in man.

Report of Case

A 41-year-old woman was referred to Mount Sinai Hospital for investigation of short-lived episodes of paroxysmal tachycardia in the last 2 years. She had no other symptoms referable to the cardiovascular system. On physical examination she was normotensive, and there was a very soft ejection systolic murmur (grade I/IV in intensity) along the left sternal border. The rest of the examination was unremarkable. Chest x-rays were normal. The electrocardiograms, which disclosed intermittent incomplete and complete LBBB, are illustrated in figures 1 to 4. The conduction disturbance was clearly rate-dependent, appearing with an increase in heart rate and disappearing with a decrease in heart rate (figs. 1 and 2). The full progression from normal conduction to complete LBBB became evident when sinus tachycardia was induced by exercise, excitement, and the inhalation of amyl nitrite. The configuration of the ventricular complexes in leads I and V1 to V4, during normal intraventricular conduction, and during intermittent and complete LBBB, is displayed in figure 3. Figure 4 demonstrates the various patterns observed during the progression from normal conduction to complete LBBB. During normal conduction there was T-wave inversion from V4 to V6 (fig. 3). The vectorcardiograms recorded by the Frank system are illustrated and described in figures 5 and 6. Right and retrograde left heart catheterization, together with
left ventricular angiography and coronary arteriography were carried out. There was no demonstrable abnormality, and the cardiac index was within normal limits (2.8 L/min/m²). Five months later, the conduction abnormality was still present.

**Discussion**

Rodriguez and Sodi-Pallares and associates, on the basis of extensive experimental work, proposed a physiological classification of the conduction disorders of the left bundle branch according to the degree of functional impairment. Their experimental data indicated that minor degrees of LBBB do not alter the direction of normal septal activation from left to right. However, as the degree of block increases, reversal of the direction of septal activation occurs. Their data also indicated that a change in the direction of the initial vector is responsible for the loss of the initial q wave and for the appearance of slurring of the initial part of the R wave in leads I, aVL, V₅, and V₆. Further delay in the transmission of activation along the left bundle presumably allows a

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**Figure 1**

Continuous recording of V₆ showing rate-dependent incomplete and complete LBBB. The cycle lengths which are measured from the beginning of the QRS to the beginning of the next QRS, are indicated in hundredths of a second. When intraventricular conduction is normal (last two complexes in strip 1), the P-R interval is 0.16 sec. During incomplete LBBB the P-R interval measures 0.18 to 0.19 sec (strip 3) and lengthens slightly to 0.20 sec during complete LBBB (strip 4). For full explanation see text.
The greater part of the left septal region to be activated by the excitation travelling from the right septal surface. Electrocardiographically, this delay is manifested by a greater amplitude and longer duration of the initial slurring. When the block becomes complete, activation of the left ventricle, including the left septal surface is entirely dependent upon right bundle-branch conduction. In this situation the leads facing the left ventricle exhibit a widened QRS complex with an R wave, followed by a plateau which is notched. Sodi-Pallares and co-workers concluded that the morphology, rather than the duration of the QRS complex, provides the most valuable way of assessing the functional capacity of the left bundle branch. They considered that the duration and amplitude of the initial slurring of the ascending limb of the R wave are the most useful diagnostic features of incomplete LBBB. Sodi-Pallares and co-workers also felt that although the intrinsicoid deflection is generally prolonged in incomplete LBBB, it is of little significance in making the diagnosis; the duration of the QRS complex was considered to be of lesser importance than its morphology, as complete LBBB may exist with a QRS of only 0.11 sec, and incomplete LBBB may prolong the QRS to as much as 0.15 sec.8

Figure 1 shows the critical rate phenomenon (phasic aberrant conduction),9-12 above

Figure 2

Continuous tracing of V6 taken 35 sec after the recording of figure 1. Normal intraventricular conduction resumes with slowing of the heart rate (see text).
which left bundle-branch conduction becomes impaired, and below which normal conduction occurs. This phenomenon, caused by an anatomic or functional lesion of the left bundle which prolongs its refractory period, reflects functional fatigue of the bundle when a critical rate of conduction is reached. The progressive impairment of left bundle-branch conduction with increasingly faster rates, permits analysis of the various grades of incomplete LBBB as the transition from normal conduction to complete LBBB is gradual. Correspondingly, when the rate slows, the gradual return to normal conduction is associated with diminishing degrees of LBBB. Figures 1 and 2 demonstrate that when intraventricular conduction is normal, the ventricular complexes (0.07 sec) in $V_6$ are preceded by a small q wave and have an intrinsicoid deflection of 0.04 sec (fig. 1, last two complexes in strip I). The slightest recognizable degree of incomplete LBBB is characterized by the disappearance of the small initial q wave and a small increase in the voltage of the R wave. When the degree of incomplete LBBB increases, slurring of the initial portion of the R wave appears and the intrinsicoid deflection becomes prolonged. In the fourth strip of figure 1, the first and second complexes, which measure 0.10 sec, exhibit no initial q wave, slurring of the ascending limb of the R wave, 3.5 mm in height, and have an intrinsicoid deflection of 0.06 sec. In advanced incomplete LBBB, the duration and amplitude of the initial slurring is increased (fig. 1, fourth beat in strip 4). When the pattern of complete LBBB supervenes, a notched plateau after the upstroke of the R wave becomes evident (fig. 1, strip 5: QRS duration, 0.13 sec). The gradual development of incomplete LBBB is illustrated in figure 4, which consists of various QRS complexes selected from a long recording of $V_6$. The complexes are arranged to demonstrate the morphology of the QRS complex during the evolution of incomplete to complete LBBB.

The case reported herein is similar to the one reported by Schamroth and Bradlow and fulfills their criteria that to establish convincing clinical evidence of incomplete LBBB, it is necessary to demonstrate in the same patient: (a) tracings with normal ventricular conduction, (b) subsequent tracings that show various degrees of incomplete LBBB, (c) tracings that eventually show complete LBBB, and (d) transitions occurring during a short-term interval. Some of the tracings published to illustrate intermittent complete LBBB exhibit, in the brief transitional phase from normal conduction to complete LBBB, only a few ventricular complexes with slurring of the initial upstroke.

Figure 3

Leads I, $V_1$ to $V_4$ during normal conduction, incomplete and complete LBBB.
of the R wave, suggestive of incomplete LBBB. In contrast to the published instances of incomplete LBBB,2,4,11,12 our patient at rest exhibited long periods of incomplete LBBB. The incomplete LBBB progressed to complete LBBB only when sinus tachycardia was induced by exercise, excitement, and the inhalation of amyl nitrite.

Sanchez and associates14 described conduction delay in the initial deflection of the vectorcardiographic loop in some patients with hypertensive or coronary artery disease who exhibited a stable form of incomplete LBBB diagnosed according to the criteria of Sodi-Pallares.15 The vectorcardiograms of our patient during the incomplete LBBB phase are similar to the ones published by Gardberg and Rosen,4 who analyzed the vectorcardiograms of the various grades of incomplete LBBB in a patient displaying an intermittent form of this abnormality. Conduction delay was absent in Garberg and Rosen’s case and our case during incomplete LBBB.

The occasional occurrence of a normal P-R interval in the Wolff-Parkinson-White (WPW) syndrome16,17 and the similarity of the slurring of the initial upstroke of the R wave of incomplete LBBB to the delta wave of the WPW syndrome may cause difficulty in distinguishing the two conditions if the period of incomplete LBBB is sustained. The differences between incomplete LBBB and the WPW syndrome (type B)18,19 are outlined in table 1. Sodi-Pallares and associates6 warned that many cases interpreted as WPW syndrome may actually be examples of incomplete LBBB. Indeed, in our case, the clinical history of paroxysmal tachycardia and the presence of normal intraventricular conduction, alternating with incomplete LBBB only in the initial electrocardiogram

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**Figure 4**

QRS complexes selected from V₆ demonstrating various grades of incomplete LBBB observed during the gradual transition from normal conduction to complete LBBB.
Figure 5

Vectorcardiograms in the horizontal plane: (A) Normal vectorcardiogram. (B₁) Magnified initial and late components of the loop when a minor degree of incomplete LBBB is present. (B₂) Greater magnification of B₁ showing the direction of the initial forces. The P wave has been electronically excluded. (C) Incomplete LBBB with a clockwise loop. (D₁ and D₂) Virtually identical loops. D₁ shows clearly the efferent limb and D₂ the afferent limb of the loop with an advanced degree of incomplete LBBB. (E) Complete LBBB.

When incomplete LBBB supervenes, the initial forces change direction and are written anteriorly and slightly to the left, while the major part of the QRS loop is inscribed posteriorly. Slight degrees of incomplete LBBB exhibit counterclockwise rotation of the loop (B). Greater degrees of incomplete LBBB and complete LBBB cause clockwise rotation of the loop (C, D, and E). Conduction delay is only evident in complete LBBB, which displays conspicuous slowing of the middle and terminal portions of the loop (E).
Vectorcardiograms in the frontal (left) and right sagittal (right) planes during normal intraventricular conduction and incomplete LBBB. The initial forces change direction from right to left, and most of the loop is inscribed posteriorly when incomplete LBBB is present. The middle vectorcardiograms were recorded at half sensitivity.

Figure 6
### Table 1

Distinguishing Features Between Incomplete LBBB and Wolff-Parkinson-White Syndrome (Type B)

<table>
<thead>
<tr>
<th>Pathogenesis</th>
<th>Incomplete LBBB</th>
<th>WPW (Type B)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physiological effect</td>
<td>Delayed transmission of activation in the left bundle branch</td>
<td>Pre-excitation of the right ventricle</td>
</tr>
<tr>
<td>Right ventricular</td>
<td>At the normal time</td>
<td>Early</td>
</tr>
<tr>
<td>activation</td>
<td>Delayed¹</td>
<td>At the normal time²</td>
</tr>
<tr>
<td>Left ventricular</td>
<td>Normal</td>
<td>Short; occasionally normal¹</td>
</tr>
<tr>
<td>activation</td>
<td>May become longer during abnormal conduction²,¹²</td>
<td>Becomes longer during normal conduction¹</td>
</tr>
<tr>
<td>Effect of P-R interval</td>
<td>Increase</td>
<td>May restore normal atrioventricular conduction²³⁻²⁵</td>
</tr>
<tr>
<td>Effect of heart rate</td>
<td>Increase</td>
<td>Usually constant</td>
</tr>
<tr>
<td>Slurring of initial</td>
<td>Varies in amplitude and duration according to the degree of incomplete LBBB¹,²</td>
<td>Present²⁶</td>
</tr>
<tr>
<td>portion of R wave</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vectorcardiogram</td>
<td>Absent in <em>intermittent</em> incomplete LBBB¹; absent in <em>intermittent</em> incomplete</td>
<td></td>
</tr>
<tr>
<td>Initial slowing</td>
<td>LBBB¹; has been described in some cases of the <em>stable</em> forms of incomplete</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LBBB¹; diagnosed according to the criteria of Sodi-Pallares¹⁵</td>
<td></td>
</tr>
</tbody>
</table>

(unfortunately this tracing is not available), led to consideration of the possible diagnosis of a variant of the WPW syndrome (type B) with a normal P-R interval. The correct diagnosis became evident when another electrocardiogram (figs. 1 and 2) revealed the entire progression to LBBB.

The slight prolongation of the P-R interval during the incomplete and complete LBBB phases (figs. 1 and 2) is explicable on the basis of abnormal initial ventricular activation. The P-R interval reflects the time needed for the cardiac impulse to travel from the sino-atrial node to that part of the septum which is activated first. Normally, the left septal surface is activated first, 0.01 to 0.015 sec before the right septal surface. When the left bundle is cut in dogs, there is some delay with a correspondingly slight prolongation of the P-R interval in the propagation of the impulse from the sino-atrial node to the right septal surface, which becomes activated before the left.²¹,²²

The increased QRS voltage, with the onset of incomplete LBBB, has been noted by other investigators.¹⁴ Sanchez and associates¹⁴ suggested that the increased QRS voltage may reflect unopposed activation of some portions of the left ventricular free wall or may be the consequence of somewhat aberrant activation of the left ventricular free wall.

This study indicates that the configuration of the QRS complexes of the various grades of incomplete LBBB corresponds very closely to the pattern described in experimentally produced lesions, thus providing further clinical evidence of the existence of this entity.

### References

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