CHAPTER 5

Intraventricular Conduction Abnormalities

NORMAL CONDUCTION

Many cardiac conditions cause electrical impulses to be conducted abnormally through the ventricular myocardium, producing changes in QRS complexes and T waves. Therefore, it is important to understand the conditions required to facilitate normal intraventricular impulse conduction. These are as follows:

- The left and right ventricles are not in an enlarged state that would prolong the time required for their activation and recovery (Chapter 4, "Chamber Enlargement").
- Myocardial ischemia or infarction is not present or is of insufficient magnitude to disrupt the spread of the activation and recovery waves (Chapter 7, "Myocardial Ischemia and Infarction").
- There is rapid impulse conduction through the right- and left-ventricular Purkinje networks so that the endocardial surfaces are activated almost simultaneously (as discussed later in this chapter).
- There are no accessory pathways for conduction from the atria to the ventricles (Chapter 6, "Ventricular Preexcitation").

BUNDLE-BRANCH AND FASCICULAR BLOCK

Since the activation of the ventricular Purkinje system is not represented on the surface electrocardiogram (ECG) (Fig. 1.9), abnormalities of its conduction must be detected indirectly by their effects on myocardial activation and recovery. The most specific changes indicative of such abnormalities occur within the QRS complex. A conduction disturbance within the right bundle branch (RBB), left bundle branch (LBB), left bundle fascicles, or between the Purkinje fibers and the adjacent myocardium may alter the QRS complex and T wave (Fig. 5.1). A conduction disturbance in the common bundle (Bundle of His) has similar effects on the entire distal Purkinje system, and therefore does not alter the appearance of the QRS complex or T wave.
Block of an entire bundle branch requires that its ventricle be activated by myocardial spread of electrical activity from the other ventricle, with prolongation of the overall QRS complex. Block of the entire RBB is termed complete right bundle-branch block (RBBB), while block of the entire LBB is termed complete left bundle-branch block (LBBB). In both of these conditions, the ventricles are activated successively instead of simultaneously. The other conditions in which the ventricles are activated successively occur when one ventricle is preexcited via an accessory atrioventricular (AV) pathway (Chapter 6, “Ventricular Preexcitation”) and when there are independent ventricular rhythms (Chapter 13 and Chapter 17). Under these conditions, there is a fundamental similarity in the distortions of the ECG waveforms: the duration of the QRS complex is prolonged and the ST segment slopes into the T wave in the direction away from the ventricle in which the abnormality is located (Fig. 5.2).
Figure 5.2. Comparison of patterns of QRS morphology in lead V1 when the two ventricles are activated successively rather than simultaneously: 


A ventricular conduction delay with only slight prolongation of the QRS complex could be termed incomplete RBBB or incomplete LBBB. However, it is important to remember from Chapter 4 ("Chamber Enlargement") that enlargement of the right ventricle may produce a distortion of the QRS complex that mimics incomplete RBBB (Fig. 4.9B), whereas enlargement of the left ventricle may produce a prolongation of the QRS complex that mimics incomplete LBBB (Fig. 4.8A). Since the LBB has multiple fascicles, another form of incomplete LBBB could be produced by a disturbance in one of its major fascicles.

The ventricular Purkinje system is considered trifascicular. It consists of the RBB and the anterior and posterior portions of the LBB. The proximal RBB is small and compact, and may therefore be considered either a bundle branch or a fascicle. The proximal LBB is also compact, but is too large to be considered a fascicle. It remains compact for 1 to 2 cm and then fans into its two fascicles. As Demoulin and Kulbertus have shown in humans, there are multiple anatomic variations in these fascicles among individuals. Based on their anatomic locations, the two fascicles are termed the left-anterior fascicle (LAF) and left-posterior fascicle (LPF), as seen in Figure 5.3. The LAF of the LBB courses toward the anterior-superior papillary muscle, and the LPF of the LBB courses toward the posterior-inferior papillary muscle. There are also Purkinje fibers that emerge from the very proximal LBB that proceed along the surface of the interventricular septum and initiate left-to-right spread of activation through the interventricular septum.
Rosenbaum and coworkers described the concept of blocks in the fascicles of the LBB, which they termed *left anterior and left posterior hemiblock.*3 However, these two kinds of block are more appropriately termed left anterior fascicular block (LAFB) and left posterior fascicular block (LPFB). Isolated LAFB, LPFB, or RBBB is considered *unifascicular* block. Complete LBBB or combinations of RBBB with LAFB or with LPFB are *bifascicular blocks*, and the combination of RBBB with both LAFB and LPFB is considered *trifascicular block.*

**UNIFASCICULAR BLOCKS**
The term “unifascicular block” is used when there is ECG evidence of blockage of only the RBB, LAF, or LPF. Isolated RBBB or LAFB occur commonly, while isolated LPFB is rare. Rosenbaum and coworkers identified only 30 patients with LPFB, as compared with 900 patients with LAFB.3

**Right Bundle-Branch Block**
Since the right ventricle contributes minimally to the normal QRS complex, RBBB produces little distortion of the QRS complex during the time required for left-ventricular activation. Figure 5.4 illustrates the minimal distortion of the early portion and marked distortion of the late portion of the QRS complex that typically occurs with RBBB. The minimal contribution of the normal right-ventricular myocardium is completely subtracted from the early portion of the QRS complex and then added later, when the right ventricle is activated via the spread of impulses from the left ventricle.
This produces a late prominent positive wave in lead V1 termed R¢, because it follows
the earlier positive R wave produced by the normal left-to-right spread of activation
through the interventricular septum (Fig. 5.4 and Table 5.1).

**Figure 5.4.** The contributions from activation of the
interventricular septum and the right and left
ventricular free walls to the appearance of the QRS
complex in lead V1, with normal intraventricular
conduction (top) and with RBBB (bottom). The
numbers refer to the first, second, and third sequential
0.04-s periods of time. Only two 0.04-s periods are
required for normal conduction, but a third is required
when RBBB is present.

<table>
<thead>
<tr>
<th>Normal</th>
<th>Balanced septal + RV and LV</th>
<th>LV</th>
<th>—</th>
</tr>
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<tbody>
<tr>
<td>RBBB</td>
<td>L→R in septum + LV</td>
<td>LV</td>
<td>RV</td>
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**Table 5.1.** Criteria for Right Bundle Branch Block

<table>
<thead>
<tr>
<th>Lead V1</th>
<th>Late intrinsicoid, M-shaped QRS (RSR'); sometimes wide R or qR</th>
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<tr>
<td>Lead V6</td>
<td>Early intrinsicoid, wide S wave</td>
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<tr>
<td>Lead I</td>
<td>Wide S wave</td>
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RBBB has many variations in its ECG appearance, as illustrated by the examples in
Figure 5.5A, Figure 5.5B and Figure 5.5C. In Figure 5.5A, the RBBB is considered
“incomplete” because the duration of the QRS complex is only 0.10 s; but in Figure
5.5B and Figure 5.5C, the RBBB is considered “complete” because the duration of the
QRS complex is ≥ 0.12 s.
Figure 5.5. Twelve-lead ECGs from a 17-year-old girl with an ostium secundum atrial septal defect (A), an 81-year-old woman with fibrosis of the RBB (B), and an 82-year-old man with fibrosis of both the RBB and the anterior fascicle of the LBB (C). Arrows in A, B, and C indicate the prominent terminal Rs wave in V1, and asterisks in A and C indicate the rightward and leftward axis shifts, respectively.
**Left-Fascicular Blocks**

Normal activation of the left-ventricular free wall spreads simultaneously from two sites (near the insertions of the papillary muscles of the mitral valve). Wavefronts of activation spread from these endocardial sites to the overlying epicardium. Since the wavefronts travel in opposite directions, they neutralize each other's influence on the ECG in a phenomenon called *cancellation*. When block in either the LAF or LPF is present, activation of the free wall proceeds from one site instead of two. Since the cancellation is removed, the waveforms of the QRS complex change, as described below (Fig. 5.6 and Table 5.2 and Table 5.3).

**Figure 5.6.** Schematic left ventricle viewed from its apex upward toward its base. The interventricular septum (*S*), left-ventricular free wall (*FW*), and anterior (*A*) and inferior (*I*) regions of the left ventricle are indicated. The typical appearances of the QRS complexes in leads *I* (**top**) and aVF (**bottom**) are presented for normal (**A**), LAFB (**B**), and LPFB left-ventricular activation (**C**). *Dashed lines* within the inner circles represent the fascicles; the two *wavy lines* crossing a fascicle indicate the sites of block. *Small crosshatched circles* represent the papillary muscles; *outer rings* represent the endocardial and epicardial surface of the left ventricular myocardium. *Arrows* within the outer rings indicate the directions of the wavefronts of activation as they spread from the unblocked fascicles through the myocardium.

| 1. Left axis deviation (usually ≥−60 degrees) |
| 2. Small Q in leads I and aVL, small R in II, III and aVF |
| 3. Usually normal QRS duration |
| 4. Late intrinsicoid deflection in aVL (>0.045 s) |
| 5. Increased QRS voltage in limb leads |

**Table 5.2.** Criteria for Left Anterior Fascicular Block
Left Anterior Fascicular Block.

If the LAF of the LBB is blocked (Fig. 5.6B), the initial activation of the left-ventricular free wall occurs via the LPF. Activation spreading from endocardium to the epicardium in this region is directed inferiorly and rightward. Since the block in the LAF has removed the initial superior and leftward activation, a Q wave appears in leads that have their positive electrodes in a superior/leftward position (i.e., lead I) and an R wave appears in leads that have their positive electrodes in an inferior/rightward position (i.e., lead aVF). Following this initial period, the activation wave spreads over the remainder of the left-ventricular free wall in a superior/leftward direction, producing a prominent R wave in lead I and a prominent S wave in lead aVF. This change in the left-ventricular activation sequence produces a leftward shift of the axis of the QRS complex to at least –45 degrees. The overall duration of the QRS complex may be normal (Fig. 5.7A) or prolonged by 0.01 to 0.04 s (Fig. 5.7B).4
**Left Posterior Fascicular Block.**

If the LPF of the LBB is blocked (Fig. 5.6C), the situation is reversed from that in LAF block, and the initial activation of the left-ventricular free wall occurs via the LAF. Activation spreading from the endocardium to the epicardium in this region is directed superiorly and leftward. Since the block in the LPF has removed the initial inferior and
rightward activation, a Q wave appears in leads with their positive electrodes in an inferior/rightward position (i.e., lead aVF) and an R wave appears in leads with their positive electrodes in a superior/leftward direction (i.e., lead I). Following this initial period, the activation spreads over the remainder of the left-ventricular free wall in an inferior/rightward direction, producing a prominent R wave in lead aVF and a prominent S wave in lead I. This change in the left-ventricular activation sequence produces a rightward shift of the axis of the QRS complex to at least +90 degrees.5 The duration of the QRS complex may be normal or slightly prolonged (Fig. 5.8).

**Figure 5.8.** Twelve-lead ECG from a healthy 77-year-old woman. Arrows indicate the deep S waves in leads I and aVL typical of both LPFB and RVH.

The consideration that LPFB may be present requires that there be no evidence of right-ventricular hypertrophy (RVH) from either the precordial leads (Fig. 5.8) or from other clinical data. However, even the absence of RVH does not allow diagnosis of LPFB, because RVH can produce the same pattern as LPFB in the limb leads, and RVH is much more common than is LPFB.

**BIFASCICULAR BLOCKS**

The term “bifascicular block” is used when there is ECG evidence of involvement of any two of the RBBB, LAF, or LPF. Such evidence may appear at different times or may coexist on the same ECG. Bifascicular block is sometimes applied to complete LBBB, and is commonly applied to the combination of RBBB with either LAFB or LPFB. The term “bilateral bundle-branch block” is also appropriate when RBBB and either
LAFB or LPFB are present. When there is bifascicular block, the duration of the QRS complex is prolonged to at least 0.12 s.

**Left Bundle-Branch Block**

Figure 5.9 illustrates the marked distortion of the entire QRS complex produced by LBBB. Complete LBBB may be caused by disease in either the main left bundle branch (LBB) (*predivisional*) or in both of its fascicles (*postdivisional*). When the impulse cannot progress along the LBB, electrical activation must first occur in the right ventricle and then travel through the interventricular septum to the left ventricle.

*Figure 5.9.* The format of Figure 5.4 is repeated to illustrate the contributions from activation of the various aspects of the ventricular myocardium to the appearances of the QRS complex in lead V1 with LBBB.

Normally, the interventricular septum is activated from left to right, producing an initial R wave in the right precordial leads and a Q wave in leads I, aVL, and the left
precordial leads. When complete LBBB is present, however, the septum is activated from right to left. This produces Q waves in the right precordial leads and eliminates the normal Q waves in the leftward-oriented leads. The activation of the left ventricle then proceeds sequentially from the interventricular septum, to the adjacent anterior and inferior walls, and then to the posterior-lateral free wall. This sequence of ventricular activation in complete LBBB tends to produce monophasic QRS complexes, with QS complexes in lead V1 and R waves in leads I, aVL, and V6 (Table 5.4).

<table>
<thead>
<tr>
<th>Lead</th>
<th>Criteria</th>
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<tbody>
<tr>
<td>V1</td>
<td>QS or rS</td>
</tr>
<tr>
<td>V6</td>
<td>Late intrinsicsoid, no Q waves, monophasic R</td>
</tr>
<tr>
<td>I</td>
<td>Monophasic R wave, no Q</td>
</tr>
</tbody>
</table>

Table 5.4. Criteria for Left Bundle Branch Block

LBBB has many variations in its ECG appearance, as illustrated by the examples in Figure 5.10A, Figure 5.10B and Figure 5.10C. Figure 5.10A shows the typical appearance of complete LBBB. In Figure 5.10B the extreme LAD indicates that conduction is even slower in the LAF than in the LPF, and only minimal R waves are seen in leads V1 through V4. In Figure 5.10C the aberration of a markedly prolonged QRS complex is present, suggesting the coexistence of left-ventricular hypertrophy (LVH).
Figure 5.10. Twelve-lead ECGs from an 82-year-old woman with no medical problems (A), a 71-year-old man with chronic heart failure (B), and a 74-year-old man with a long history of hypertension (C). Arrows in A and C indicate the typical characteristics of LBBB in leads I and V1, and arrows in B indicate the deep S waves in leads II, III, and aVF and decreased R waves in leads V2–V4.
Right Bundle-Branch Block with Left Anterior Fascicular Block

Just as LAFB appears as a unifascicular block much more commonly than does LPFB, it more commonly accompanies RBBB as a bifascicular block. The diagnosis of LAFB plus RBBB is made by observing the late prominent R or R¢ wave in precordial lead V1 of RBBB, and the initial R waves and prominent S waves in limb leads II, III, and aVF of LAFB. The duration of the QRS complex should be at least 0.12 s and the frontal-plane axis of the complex should be between –45 degrees and –120 degrees (Fig. 5.11). In Figure 5.11A only LAFB is present, while in Figure 5.11B the presence of RBBB indicates that a second fascicle has been blocked.

![ECG Diagram](image)

**Figure 5.11.** Twelve-lead ECGs from a 1-year previous (A) and a current (B) evaluation of a 73-year-old woman with no medical problems and no other evidence of heart disease. Arrows indicate the deep S waves in II, III, and aVF that are characteristic of LAFB in A, and a prominent R¢ wave characteristic of RBBB in V1 in B.

Right Bundle-Branch Block with Left Posterior Fascicular Block

The example of bifascicular block consisting of RBBB with LPFB rarely occurs. Even when changes in the ECG are entirely typical of this combination, the diagnosis should be considered only if there is no clinical evidence of RVH. The diagnosis of RBBB with LPFB should be considered when precordial lead V1 shows changes typical of RBBB and limb leads I and aVL show the initial R waves and prominent S waves typical of LPFB. The duration of the QRS complex should be at least 0.12 s and the frontal-plane axis of the complex should be at least +90 degrees (Fig. 5.12).
SYSTEMATIC APPROACH TO THE ANALYSIS OF BUNDLE-BRANCH AND FASCICULAR BLOCKS

The following steps should be taken in analyzing bundle-branch and fascicular blocks:

- Examine the contour of the QRS complex.

RBBB and LBBB have opposite effects on the contour of the QRS complex. RBBB adds a new waveform directed toward the right ventricle following the completion of slightly altered waveforms directed toward the left ventricle (Fig. 5.4). Therefore, the QRS complex in RBBB tends to have a triphasic appearance. In lead V1, which is optimal for visualizing right-versus left-sided conduction delay, the QRS in RBBB has the appearance of “rabbit ears” (Fig. 5.5). Typically, the “first ear” (R wave) is shorter than the “second ear” (R¢ wave). (Although the term “rabbit ears” in this context refers to a triphasic QRS, it can also refer to two peaks found in monophasic QRS complexes.) When RBBB is accompanied by block in one of the LBB fascicles, the positive deflection in lead V1 is often monophasic, as in Figure 5.12.

In LBBB, a sequential spread of activation through the interventricular septum and left-ventricular free wall replaces the normal, competing and simultaneous
spread of activation through these areas. As a result, the QRS complex tends to have a monophasic appearance that is notched rather than smooth.

Although LBBB and LVH have many ECG similarities, they also show marked differences. Whereas the normal Q waves over the left ventricle may be present or even exaggerated in LVH, they are absent in LBBB. When the LBB is completely blocked, the septum is entirely activated from its right side. Figure 5.13 illustrates the appearance of incomplete (Fig. 5.13B) and complete (Fig. 5.13C) LBBB in a patient with LVH (Fig. 5.13A).
Figure 5.13. The five representative ECG leads illustrate the evolving ECG changes in a patient with severe hypertension as the LVH is complicated by LBBB. A. Age 60 years. B. Age 63 years. C. Age 67 years.

- Measure the Duration of the QRS Complex.

Complete RBBB increases the duration of the QRS complex by 0.03 to 0.04 s, and complete LBBB increases the duration of the complex by 0.04 to 0.05 s. Block within the LAF or LPF of the LBB usually prolongs the duration of the QRS complex by only 0.01 to 0.02 s (Fig. 5.7B and Fig. 5.8).4
• Measure the Maximal Amplitude of the QRS Complex.

Bundle-branch block (BBB) produces QRS waveforms with lower voltage and more definite notching than those that occur with ventricular hypertrophy. However, the amplitude of the QRS complex does increase in LBBB because of the relatively unopposed spread of activation over the left ventricle.

One general rule for differentiating between LBBB and LVH is that the greater the amplitude of the QRS complex, the more likely is LVH to be the cause of this. Similarly, the more prolonged is the duration of the QRS complex, the more likely is LBBB to be the cause of this effect. Klein and colleagues8 have suggested that in the presence of LBBB, either of the following criteria are associated with LVH:

- S wave in V2 + R wave in V6 > 45 mm.
- Evidence of left-atrial enlargement with a QRS-complex duration > 0.16 s.

• Estimate the Direction of the QRS Complex in the Two Planes of the ECG.

Since complete RBBB and complete LBBB alter conduction to entire ventricles, they might not be expected to produce much net alteration of the frontal-plane QRS axis. However, Rosenbaum studied patients with intermittent LBBB in which blocked and unblocked complexes could be examined side by side.4 LBBB was often observed to produce a significant left-axis shift and sometimes even a right axis shift. The axis was unchanged in only a minority of patients. However, block in either the LAF or LPF of the LBB alone produces marked axis deviation. The initial 0.20 s of the QRS complex is directed away from the blocked fascicles, and the middle and late portions are directed toward the blocked fascicles, causing the overall direction of the QRS complex to be shifted toward the site of the block (Fig. 5.7 and Fig. 5.8).5 When block in either of these LBB fascicles is accompanied by RBBB, an even later waveform is added to the QRS complex, thereby further prolonging its duration. The direction of this final waveform in the frontal plane is in the vicinity of 180 degrees, as a result of the RBBB (Fig. 5.5C).5

In BBB, the T wave is usually directed opposite to the later portion of the QRS complex (e.g., in Figure 5.14A, the T wave in lead I is inverted and the later part of the QRS complex is upright; in Figure 5.14B the T wave is upright and the later part of the QRS complex is negative). This opposite polarity is the natural result of the depolarization–repolarization disturbance produced by the BBB, and is therefore termed secondary. Indeed, if the direction of the T wave is similar to that of the terminal part of the QRS complex (Fig. 5.14C), it should be considered abnormal. Such T-wave changes are primary and imply myocardial disease. The diagnosis of
myocardial infarction in the presence of BBB is considered in Chapter 10 ("Myocardial Infarction").
Figure 5.14. Twelve-lead ECGs from an 89-year-old woman during a routine health evaluation (A), a 45-year-old pilot during an annual health evaluation (B), and a 64-year-old woman on the first day after coronary bypass surgery (C). Arrows indicate the concordant directions of the terminal QRS complex and of the T wave in leads V2–V4 in C.
One method of determining the clinical significance of T-wave changes in BBB is to measure the angle between the axis of the T wave and that of the terminal part of the QRS complex. Obviously, if the two are oppositely directed (as they are with secondary T-wave changes), the angle between them will be wide and may approach 180 degrees. It has been proposed that if this angle is less than 110 degrees, myocardial disease is present. In Figure 5.14B, the angle is about 150 degrees, whereas in Figure 5.14C it is only a few degrees.

**CLINICAL PERSPECTIVE ON INTRAVENTRICULAR CONDUCTION DISTURBANCES**

Both RBBB and LBBB are often seen in apparently normal individuals. The cause of this is fibrosis of the Purkinje fibers, which has been described as Lenegre's disease or Lev's disease. The process of Purkinje fibrosis progresses slowly: a 10-year follow-up study of healthy aviators with BBB revealed no incidence of complete AV block, syncope, or sudden death. The pathologic process may be accelerated by systemic hypertension: it preceded the appearance of BBB in 60% of the individuals in the Framingham study. The mean age of onset of the BBB was 61 years.

Insight into the long-term prognosis for individuals with chronic BBB but no other evidence of cardiac disease comes from studies of the ECG changes preceding the development of transient or permanent complete AV block. Friedberg and associates have documented the common presence of some combination of bundle-branch or fascicular block immediately before onset of the AV block. The most common combination was RBBB with LAFB.

The combined results of these studies suggest that Lenegre's or Lev's disease is a slowly developing process of fibrosis of the Purkinje fibers that has the ultimate potential of causing complete AV block because of bilateral bundle-branch involvement. Since the Purkinje cells lack the physiologic capacity of the AV-nodal cells to conduct at varying speeds, a sudden progression from no AV block to complete AV block may occur. When this does occur, ventricular activation can result only from impulse formation within a Purkinje cell beyond the site of the block. Several clinical conditions may result, including syncope and sudden death.

Bundle-branch or fascicular block may also be the result of other serious cardiac diseases. In Central and South America, Chagas disease, produced by infection with *Trypanosoma cruzi*, is almost endemic and is a common cause of RBBB with LAFB. As indicated in Chapter 4, RBBB is commonly produced by the distention of the right ventricle that occurs with volume overloading. Transient RBBB may be produced during right-heart catheterization, as illustrated in Figure 5.15.
Any combination of the bundle branches or proximal fascicles may be blocked during an episode of myocardial cell death in a patient with coronary atherosclerosis. These structures receive their blood supply via the proximal septal perforating branch of the left anterior descending coronary artery (Fig. 5.16). Therefore, the bundle branches and their proximal fascicles become involved when there is an occlusion in either the left main coronary artery or the origin of its anterior descending branch. Individuals who survive to reach the hospital after occlusion of such a major coronary artery may have any combination of bundle-branch or fascicular blocks complicating extensive myocardial infarction. Since the acute and long-term mortality rates in these patients are very high, they do not represent a significant portion of the overall population of individuals with chronic bundle-branch and fascicular block.17
Figure 5.16. The proximal portion of specialized conduction system is shown in relation to its blood supply from a right anterior oblique view: A, AV node; B, Common bundle; C, LPF; D, LAF; E; RBB. Note the lengths of the septal perforating branches of the left anterior descending (LAD) coronary artery in contrast to those of the posterior descending artery (PDA). (From Rotman M, Wagner GS, Wallace AG. Bradyarrhythmias in acute myocardial infarction. Circulation 1972;45:703–722, with permission. Copyright 1972 American Heart Association.)

Intermittent BBB (prolonged QRS complexes present at some times but not at others) usually represents a transition stage before permanent block is established. Figure 5.17A and Figure 5.17B show examples of the sudden onsets of LBBB and RBBB, respectively.
Figure 5.17. Precordial leads V1 and V5 are shown from a 62-year-old woman during routine ECG monitoring after uncomplicated abdominal surgery (A) and a 54-year-old man during 24-hour ECG monitoring for a complaint of dizziness (B). Arrows indicate the onsets in the V1 leads of typically appearing LBBB in A and RBBB in B.

At times, intermittent BBB is determined by the heart rate. As the rate accelerates, the RR interval shortens and the descending impulse finds one of the bundle branches still in its refractory period (Fig. 5.18). With this tachycardia-dependent BBB, slowing of the heart rate allows descending impulses to arrive after the refractory period of the entire conduction system, and normal conduction is resumed.

Figure 5.18. Precordial leads V1 and V5 during routine ECG monitoring of a 47-year-old woman after breast cancer surgery. Arrows indicate the appearance of incomplete RBBB following the shorter cycle intervals.
A rarer form of intermittent BBB, which develops only when the cardiac cycle lengthens rather than shortens (Fig. 5.19), is termed bradycardia-dependent BBB. Intermittent BBB is a form of intermittent aberrant conduction of electrical impulses through the ventricular myocardium.

Figure 5.19. All beats are conducted sinus beats grouped in pairs. Those ending the shorter cycles are conducted normally, while those ending the longer cycles are conducted with LBBB.

GLOSSARY

Atherosclerosis:
a thickening of the inner arterial wall caused by the deposition of fatty substances.

AV block:
a block in the cardiac conduction system that causes a disruption of atrial-to-ventricular electrical conduction.

Bifascicular block:
an intraventricular conduction abnormality involving any two of: the RBB, the anterior division of the LBB, and the posterior division of the LBB.

Bilateral bundle-branch block:
an intraventricular conduction abnormality involving both the right and left bundle branches, as indicated either by the presence of some conducted beats with RBBB and others with LBBB, or by AV block located distal to the common bundle.

Bradycardia-dependent BBB:
RBBB or LBBB that is intermittent, appearing only with a slowing of the atrial rate.

Cancellation:
Elimination of an abnormality produced by a particular cardiac problem by a similar abnormality in another part of the heart or by a different abnormality in the same part of the heart, since the ECG waveforms represent the summation of the wavefronts of activation and recovery within the heart.

Chagas disease:
a tropical disease caused by the flagellate organism *Trypanosoma cruzi*, which is marked by prolonged high fever, edema, and enlargement of the spleen, liver, and lymph nodes, and is complicated by cardiac involvement.

**Fascicle:**
a group of Purkinje fibers too small to be called a “branch.”

**Fibrosis:**
a condition in which Purkinje fibers are transformed into nonconducting interstitial fibrous tissue.

**Left anterior fascicular block:**
a conduction abnormality in the anterior fascicle of the LBB.

**Left posterior fascicular block:**
to a conduction abnormality in the posterior fascicle of the LBB.

**Lenegre’s (Lev’s) disease:**
both Lenegre and Lev described variations of fibrosis of the intraventricular Purkinje fibers in the absence of other significant cardiac disease.

**Predivisional and postdivisional:**
terms referring to block within the LBB either “pre-“ or proximal to its division into fascicles, or “post-“ and involving both the anterior and posterior fascicles.

**Primary and secondary T-wave changes:**
in the presence of RBBB or LBBB, the term “primary T-wave changes” refers to abnormal T waves that are directed similarly to the latter portion of the QRS complex, and “secondary T-wave changes” refers to normal T waves that are directed opposite to the latter portion of the QRS complex.

**Refractory period:**
the period following electrical activation during which a cardiac cell cannot be reactivated.

**RR interval:**
the period between successive QRS complexes.

**Septal Q wave:**
a normal, initially negative QRS waveform that appears in leftward-oriented ECG leads because of earliest activation of the interventricular septum via the septal fascicles of the LBB.

**Syncope:**
a brief loss of consciousness associated with transient lack of cerebral blood flow.

**Tachycardia-dependent BBB:**
RBBB or LBBB that is intermittent, appearing only with an acceleration of the atrial rate.

**Trifascicular block:**
an intraventricular conduction abnormality involving the RBB and both the anterior and posterior fascicles of the LBB.

**Unifascicular block:**
an intraventricular conduction abnormality involving only one of the three principal fascicles of the intraventricular Purkinje system.

REFERENCES


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