CHAPTER 6

Ventricular Preexcitation

HISTORICAL PERSPECTIVE

In the normal heart, there are no muscular connections between the atria and ventricles. In 1893, Kent described the rare occurrence of such connections, but wrongly assumed that they represented pathways of normal conduction.1 Mines suggested in 1914 that this accessory atrioventricular (AV) connection (Bundle of Kent) might cause tachyarrhythmias. In 1930, Wolff and White in Boston and Parkinson in London reported their combined series of 11 patients with bizarre ventricular complexes and short PR intervals.2 Then, in 1944, Segers introduced the triad of short PR interval, preexcitation of the ventricles characterized by a prolonged upstroke of the QRS complex (delta wave), and tachyarrhythmia that characterize the Wolff–Parkinson–White (WPW) syndrome.

CLINICAL PERSPECTIVE

Ventricular preexcitation refers to a congenital cardiac abnormality in which a part of the ventricular myocardium receives electrical activation from the atria before the arrival of an impulse via the normal AV conduction system (Fig. 6.1). AV myocardial bundles commonly exist during fetal life, but then disappear by the time of birth.3 When even a single myocardial connection persists, there is the potential for ventricular preexcitation. In some individuals, evidence of preexcitation may not appear until late in life, while in others with lifelong evidence of ventricular preexcitation on the electrocardiogram (ECG), the WPW syndrome may not occur until late in life. Conversely, infants with the WPW syndrome may outgrow any or all evidence of this abnormality within a few years.4
Figure 6.1. Schematic illustration of the anatomic relationship between the normal AV conduction system and the accessory AV conduction pathway provided by the Bundle of Kent. The solid bar represents the nonconducting structures (including the coronary arteries and veins, valves, and fibrous and fatty connective tissue) that prevent conduction of electrical impulses from the atrial myocardium to the ventricular myocardium. (AVN, AV node; HB, His bundle; RBB, right bundle branch; KB, Kent bundle; LBB, left bundle branch.)

Figure 6.2 illustrates the contrast between the alteration of the PR and QRS intervals that results from bundle-branch block (BBB) and from ventricular preexcitation. Right or left BBB (Fig. 6.2A) does not alter the PR interval, but prolongs the QRS complex by delaying activation of one of the ventricles. Ventricular preexcitation (Fig. 6.2B) shortens the PR interval and produces a “delta wave” in the initial part of the QRS complex. The total time from the beginning of the P wave to the end of the QRS complex remains the same as in the normal condition because conduction via the abnormal pathway does not interfere with conduction via the normal AV conduction system. Therefore, before the entire ventricular myocardium can be activated by progression of the preexcitation wave front, electrical impulses from the normal conducting system arrive to activate the remainder of the ventricular myocardium.

Figure 6.2. The two types of altered or “aberrant” conduction from the atria to the ventricles. The dashed line in A represents late activation of the ventricle served by the blocked bundle
branch, and the *dashed line* in B represents the early activation of the ventricle connected with the atria via an accessory muscle bundle.

Figure 6.3A illustrates the normal cardiac anatomy that permits AV conduction only via the AV node (the open channel at the crest of the interventricular septum). Thus, there is normally delay in the activation of the ventricular myocardium (PR segment), as noted in the ECG recording shown in the figure. When the congenital abnormality responsible for the WPW syndrome is present (Fig. 6.3B) the ventricular myocardium is activated from two sources: (a) via the preexcitation pathway (the open channel between the right atrium and right ventricle); and (b) via the normal AV conduction pathway. The resultant abnormal QRS complex (termed a *fusion beat*) is composed of the abnormal preexcitation wave and normal mid- and terminal QRS waveforms.

![Figure 6.3](image)

**Figure 6.3.** Relationship between an anatomic Bundle of Kent and physiologic preexcitation of the ventricular myocardium (*top*), and the typical ECG changes of ventricular preexcitation (*bottom*). Normal condition is presented (A) for contrast with the abnormal condition (B). (Modified from Wagner GS, Waugh RA, Ramo BW. Cardiac arrhythmias. New York: Churchill Livingstone, 1983:13.)

The ECG of an individual with ventricular preexcitation is abnormal in several ways:
1. In the presence of a normal sinus rhythm, the PR interval is abnormally short and the duration of the QRS complex is abnormally prolonged. Ventricular preexcitation...
produces a prolonged upstroke of the QRS complex, which has been termed a delta wave (Fig. 6.4).

2. In the presence of an atrial tachyarrhythmia, such as atrial flutter/fibrillation (Chapter 15, “Reentrant Atrial Tachycardias—The Atrial Flutter/Fibrillation Spectrum”), the ventricular rate also becomes rapid. The ventricles are no longer “protected” by the slowly conducting AV node (Fig. 6.5).
3. The abnormal AV muscular connection completes a circuit by providing a pathway for electrical reactivation of the atria from the ventricles. This circuit provides a continuous loop for the electrical activating current, which may result in a single premature beat or a prolonged, regular, rapid atrial and ventricular rate called a tachyarrhythmia (Fig. 6.6). In Figure 6.6B, an atrial premature beat has occurred which sends a wave of depolarization through the atria and toward the Bundle of Kent. Because this beat originated in such close proximity to the Bundle of Kent, the bundle has not had sufficient time to repolarize. As a result, the premature wave of depolarization cannot continue through this accessory AV conduction pathway to preexcite the ventricles. However, the premature wave is able to progress to the ventricles via the normal AV conduction pathway in the AV node and interventricular septum. This depolarization wave then travels through the ventricles, and since it does not collide with an opposing wave (as occurs with ventricular preexcitation in Figure 6.6A), it reenters the atrium through the Bundle of Kent, creating a retrograde atrial excitation (Fig. 6C).
The influence of ventricular preexcitation on the ventricular rate during atrial flutter/fibrillation and on tachyarrhythmias induced by an accessory pathway is discussed in Chapter 15 (“Reentrant Atrial Tachycardias—The Atrial Flutter/Fibrillation Spectrum”) and Chapter 16 (“Reentrant Junctional Tachyarrhythmias”), respectively. The combination of a PR interval of duration <0.12 s, a delta wave at the beginning of the QRS complex, and a rapid, regular tachyarrhythmia has been termed the Wolff–Parkinson–White (WPW) syndrome. The PR interval is short because the descending electrical impulse bypasses the normal AV-nodal conduction delay. The delta wave is produced by slow intramyocardial conduction that results when the descending impulse, instead of being delivered to the ventricular myocardium via the normal conduction system, is delivered directly into the ventricular myocardium via an abnormal or “anomalous” muscle bundle. The duration of the QRS complex is prolonged because it begins “too early,” in contrast to the situations presented in Chapter 4 (“Chamber Enlargement”) and Chapter 5 (“Intraventricular Conduction Abnormalities”), in which the duration of the QRS complex is prolonged because it ends too late. The ventricles are activated successively rather than simultaneously:
the preexcited ventricle is activated via the Bundle of Kent, and the other ventricle is then activated via the normal AV node and His-Purkinje system (Fig. 6.3). Various terms have been applied to the abnormal anatomic structure and resulting abnormal electrophysiologic function responsible for the WPW syndrome (Table 6.1).

<table>
<thead>
<tr>
<th>Anatomic Structure</th>
<th>Electrophysiologic Function</th>
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<tbody>
<tr>
<td>Myocardial bundle</td>
<td>Ventricular preexcitation</td>
</tr>
<tr>
<td>Bundle of Kent</td>
<td>Accessory AV conduction pathway</td>
</tr>
<tr>
<td>Bypass tract</td>
<td>AV nodal bypass pathway</td>
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</table>

Table 6.1. Structure and Function Terms

**ELECTROCARDIOGRAPHIC DIAGNOSIS OF VENTRICULAR PREEXCITATION**

Typically, with ventricular preexcitation, the PR interval is less than 0.12 s in duration and the QRS complex is greater than 0.10 s. However, the PR interval is not always abnormally short (Fig. 6.7A) and the QRS complex is not always abnormally prolonged (Fig. 6.7B). Conduction through the Bundle of Kent may be relatively slow, or the Bundle of Kent may directly enter the His bundle. Among almost 600 patients with documented ventricular preexcitation, 25% had PR intervals of 0.12 s or longer and 25% had a QRS-complex duration of 0.10 s or shorter.
Figure 6.7. Twelve-lead ECGs from a 57-year-old man without cardiac-related symptoms (A) and a 41-year-old woman with recurrent episodes of weakness and who sensed a rapid heart rate (B). Arrows in A indicate abnormally slow onset of the QRS complex following a normal PR interval (0.16 s) and arrows in B indicate an abnormally short PR interval preceding a QRS complex of normal duration (0.08 s).

When ventricular preexcitation is suspected in a patient with tachyarrhythmias but no ECG evidence preexcitation, the following diagnostic procedures may be helpful:

- Pace the atria electronically at increasingly rapid rates to induce conduction via any existing accessory pathway.
• Produce vagal nerve stimulation to impair normal conduction through the AV node so as to induce conduction via any existing accessory pathway.
• Infuse digoxin intravenously for the same purpose as in Procedure 2.

Ventricular preexcitation may mimic a number of other cardiac abnormalities. When there is a wide, positive QRS complex in leads V1 and V2, it may simulate right bundle-branch block (RBBB), right-ventricular hypertrophy (RVH), or a posterior myocardial infarction. When there is a wide, negative QRS complex in lead V1 or V2, preexcitation may be mistaken for left bundle-branch block (LBBB) (Fig. 6.8A) or left-ventricular hypertrophy (LVH). A negative delta wave, producing Q waves in the appropriate leads, may imitate anterior, lateral, or inferior infarction. As will be discussed in Chapter 10 (“Myocardial Infarction”), the prominent Q waves in leads aVF and V1 in Figure 6.8B could be mistaken for inferior or anterior infarction, respectively. Similarly, the deep, wide Q wave in lead aVF and broad initial R wave in lead V1 in Figure 6.8C could be mistaken for inferior or posterior infarction, respectively.

Figure 6.8. Twelve-lead ECGs from a 40-year-old woman admitted to a hospital emergency department for symptoms of dizziness (A), a 46-year-old woman admitted to a coronary care unit with chest pain but no clinical confirmation of a myocardial infarction (B), and a 31-year-old male medical resident without cardiac symptoms but an incorrect diagnosis of myocardial
infarction by computerized interpretation of a routine ECG (C). Arrows in A indicate delta waves producing QRS complexes mimicking LBBB, and arrows in B and C indicate delta waves producing QRS complexes mimicking myocardial infarction.

**ELECTROCARDIOGRAPHIC LOCALIZATION OF THE PATHWAY OF VENTRICULAR PREEXCITATION**

Many attempts have been made to determine the myocardial location of ventricular preexcitation according to the direction of the delta waves in the various ECG leads. Rosenbaum and colleagues\(^6\) divided patients into two groups (Group A and Group B) on the basis of the direction of the “main deflection of the QRS complex” in transverse-plane leads V1 and V2 (Table 6.2).

<table>
<thead>
<tr>
<th>ECG Appearance</th>
<th>Location of Abnormal Pathway</th>
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<tbody>
<tr>
<td>Group A: QRS mainly positive in leads V1 and V2</td>
<td>LA-LV</td>
</tr>
<tr>
<td>Group B: QRS mainly negative in leads V1 and V2</td>
<td>RA-RV</td>
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</table>

Table 6.2. Relationship Between Pathway Location and ECG Changes

Other classification systems consider the direction only of the abnormal delta wave in attempting to better localize the pathway of ventricular preexcitation. Since curative surgical and catheter ablation techniques for eliminating it have become available, more precise localization of the accessory pathway is clinically important,\(^7\) and many additional ECG criteria have therefore been proposed for achieving this. However, precise localization of an accessory AV pathway is made difficult by several factors, including minor degrees of preexcitation, the presence of more than one accessory pathway, distortions of the QRS complex caused by superimposed myocardial infarction, or ventricular hypertrophy. Nevertheless, Milstein and his associates\(^8\) devised the algorithm presented in Figure 6.9 that enabled them to correctly identify the location of 90% of more than 140 accessory pathways.
Although accessory pathways may be found anywhere in the connective tissue between the atria and ventricles, nearly all are found in three general locations (Fig. 6.10), as follows.
between the atria and the ventricles. The ventricular outflow aortic and pulmonary valves are located anteriorly, and the ventricular inflow mitral (bicuspid) and tricuspid valves are located posteriorly. The three general locations of Bundles of Kent are: 1, LA-LV free wall; 2, posterior septal; and 3, the right anteroseptal and right lateral locations of Milstein and colleagues combined as RA-RV free wall. (Modified from Tonkin AM, Wagner GS, Gallagher JJ, et al. Initial forces of ventricular depolarization in the Wolff-Parkinson-White syndrome. Analysis based upon localization of the accessory pathway by epicardial mapping. Circulation 1975;52:1031.)

- Left laterally, between the left-atrial and left-ventricular free walls (50%).
- Posteriorly, between the atrial and ventricular septa (30%).
- Right laterally or anteriorly, between the right atrial and right ventricular free walls (20%).

Tonkin and associates presented a simple method for localizing accessory pathways to one of the foregoing areas on the basis of the direction of the delta wave (Table 6.3).

<table>
<thead>
<tr>
<th>Direction of Preexcitation</th>
<th>Location of Pathway</th>
<th>Incidence Correct</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rightward</td>
<td>LA-LV free wall</td>
<td>10 of 10</td>
</tr>
<tr>
<td>Leftward and superior</td>
<td>Posterior septal</td>
<td>9 of 10</td>
</tr>
<tr>
<td>Leftward and inferior</td>
<td>RA-RV free wall</td>
<td>6 of 7</td>
</tr>
</tbody>
</table>

Table 6.3. Consideration of Delta Wave at QRS Onset + 0.02 s

ABLATION OF ACCESSORY PATHWAYS

Figure 6.11A and Figure 6.12A illustrate the typical ECG appearances of preexcitation of the right ventricular free wall and the interventricular septum, respectively. Successful ablation of the accessory pathways (Fig. 6.11B and Fig. 6.12B) revealed the underlying presence of normal QRS complexes.
Figure 6.11. Serial 12-lead ECGs from a 44-year-old woman with a history of recurrent symptoms of dizziness and shortness of breath just before (A) and 1 week after (B) catheter-induced radio-frequency ablation of her Bundle of Kent. Arrows indicate delta waves in A and a normal appearance of the QRS complex in B.

Figure 6.12. Serial 12-lead ECGs from a 28-year-old woman with recurrent episodes of rapid heart beat 1 day before (A) and 1 day after (B) catheter-induced radio-frequency ablation of her Bundle of Kent. Arrows indicate delta waves in A and a normal appearance of the QRS complex in B.

GLOSSARY

Bundle of Kent:
a congenital abnormality in which a bundle of myocardial fibers connects the atria and the ventricles.

Delta wave:
a slowing of the initial aspect of the QRS complex caused by premature excitation (preexcitation) of the ventricles via a Bundle of Kent.

Fusion beat:
activation of the ventricles by two different wave fronts, resulting in an abnormal appearance of the QRS complexes on the ECG.

**Preexcitation:**
premature activation of the ventricular myocardium via an abnormal AV pathway called a Bundle of Kent.

**Tachyarrhythmia:**
an abnormal cardiac rhythm with a ventricular rate ≥100 beats/min.

**Woff–Parkinson–White syndrome:**
the clinical combination of a short PR interval, an increased duration of the QRS complex caused by an initial slow deflection (delta wave), and supraventricular tachyarrhythmias.

**REFERENCES**


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Version: rel9.3.0, SourceID 1.10284.1.251