Preexcitation
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Key Points

- Wolff, Parkinson, and White described patients with electrocardiograms (ECGs) showing a short PR interval, a delta wave, and a wide QRS complex. Wolferth and Wood postulated the presence of an accessory AV pathway to explain the unusual ECG and the frequently occurring tachycardias in these patients.

- Ventricular preexcitation should be understood as activation of the ventricles occurring over two atrioventricular (AV) pathways, resulting in a fusion QRS complex whose configuration depends on the contribution of each of the two activation fronts.

- Orthodromic circus movement tachycardia (CMT) has AV conduction over the AV node and ventricular-atrial conduction over the accessory AV pathway. A tachycardia moving in the reverse direction, that is, AV conduction over the accessory pathway and ventricular-atrial conduction over the His-AV node, is called an antidromic one.

- In patients having a rapidly conducting accessory pathway, atrial fibrillation can be extremely dangerous.

- An accessory AV pathway with a short anterograde refractory period may lead to life-threatening high ventricular rates if atrial fibrillation supervenes.

- The patient with preexcitation and arrhythmias can usually have these arrhythmias cured by catheter ablation of the accessory pathway.

- There is controversy about how to manage the asymptomatic patient.

In 1930, Wolff, Parkinson, and White described patients with electrocardiograms (ECGs) showing a short PR interval, a delta wave, and a wide QRS complex. In 1933, Wolferth and Wood postulated the presence of an accessory atrioventricular (AV) pathway to explain the peculiar ECG and the frequently occurring tachycardias in these patients.

Subsequently, epicardial mapping, electrophysiologic investigations, surgical findings, anatomic studies, and outcomes of catheter ablation have shown that there are several pathways by which a part of or the whole ventricle can be activated earlier than expected. The old and new nomenclature is given in Table 93.1. By far the most common type of extra connection leading to ventricular preexcitation is a rapidly conducting accessory AV pathway, formerly called a Kent bundle. The other connections are rare and require sophisticated intracardiac stimulation techniques and intracardiac recordings to be identified. Emphasis in this chapter, therefore, is on the recognition, consequences, and treatment of patients who have a rapidly conducting accessory AV pathway.

Understanding the Electrocardiogram

Essential in our understanding of the ECG findings in ventricular preexcitation is the awareness that when activation of the ventricles occurs over two AV pathways, a fusion QRS complex results whose configuration depends on the contribution of each of the two activation fronts. As shown in Figures 93.2 and 93.3, a left-sided accessory AV pathway is present. In Figures 93.2A and 93.3A, because of the time required to conduct the impulse from the sinus node to the ventricles over the two AV pathways, an important part of the ventricle is preexcited, leading to a short PR interval, a delta wave (representing activation of the left ventricular free wall), and a widened QRS complex. In Figures 93.2B and 93.3B, contribution to ventricular activation over the accessory AV pathway is minor, and therefore, the PR interval is longer, the delta wave small, and the QRS complex relatively narrow.

The conclusion is that during sinus rhythm, an ECG showing a classic Wolff-Parkinson-White (WPW) syndrome
FIGURE 93.1. Schematic of the atrioventricular (AV) conduction system and the possible accessory connections partially or totally bypassing that system. AV, atrioventricular; LBB, left bundle branch; RBB, right bundle branch.

TABLE 93.1. Nomenclature of accessory connections

<table>
<thead>
<tr>
<th>Old Connection</th>
<th>New Connection</th>
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<tbody>
<tr>
<td>Kent bundle</td>
<td>Accessory AV pathway</td>
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<tr>
<td>Mahaim fiber</td>
<td>Atriofascicular pathway</td>
</tr>
<tr>
<td>Mahaim fiber</td>
<td>Nodoventricular pathway</td>
</tr>
<tr>
<td>Mahaim fiber</td>
<td>Fasciculoventricular pathway</td>
</tr>
<tr>
<td>Atrio-His fiber</td>
<td>Atrio-Hisian bypass</td>
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BB, bundle branch, R, right.

FIGURE 93.2. Factors determining the degree of preexcitation in a left-sided accessory atrioventricular (AV) pathway during sinus rhythm. Left: Because of a shorter conduction time from the sinus node to the ventricle over the accessory pathway \(65 + 30 = 95\) ms versus \(35 + 80 + 45 = 160\) ms over the normal AV conduction system, an important part of the ventricle is preexcited, resulting in an electrocardiogram (ECG) with a short PR interval, a clear delta wave, and a widened QRS complex (top left). Right: In contrast, activation of the ventricle starts simultaneously over the normal AV conduction system \(30 + 60 + 35\) and the accessory pathway \(90 + 35 = 125\) ms. This leads to an ECG with a longer PR interval, hardly any delta wave, and a more narrow QRS complex (top right). CS, coronary sinus, HRA, high right atrium.

FIGURE 93.3. Electrocardiographic examples of the two schematic drawings shown in Figure 93.2. A and B correspond to the left and right sides of Figure 93.2. (A) ECG shows a prominent delta wave. (B) ECG shows a small delta wave. In A, a much larger area of ventricle is preexcited than in B.

[P-delta interval less than 0.12 sec] is not always present. The form of the ECG depends on the amount of ventricular muscle activated over the accessory AV pathway. Several factors may play a role here, such as location of the accessory pathway, the site of atrial impulse formation in relation to the location of the accessory pathway, the size of the atria, and the transmission characteristics of the AV node and of the accessory AV pathway. These considerations should be kept in mind, when one tries to locate the accessory pathway on the 12-lead ECG. Only when an important amount of ventricular preexcitation (a clear delta wave) is present can dependable predictions be made.
Incidence of Preexcitation

The true incidence of preexcitation is unknown; the reported figures vary from 0.1 to 0.3/1000 ECGs. In one study, the incidence of new cases of WPW syndrome was 4.4/1000/year, and was much higher in males (6.8/1000/year) than in females (2.2/1000/year). There is a fourfold increase in family members of WPW patients. The WPW syndrome is undoubtedly underdiagnosed because the contribution to ventricular activation over the accessory pathway may vary.

Arrhythmias

Pathways leading to ventricular preexcitation can be classified as shown in Figure 93.1. These pathways may be incorporated into several different types of reentry circuits, but from a practical point of view, a direct connection between the atrium and the ventricle, a true accessory AV pathway, is most commonly involved in the tachycardia mechanism. The incidence of tachyarhythmias in the WPW syndrome is unknown. The reported figures range from 12% to 80%.

Although any type of arrhythmia, such as atrial, AV nodal, or ventricular tachycardia, may occur in the presence of accessory connection, the clinically most common types of arrhythmia in patients with preexcitation are circus movement tachycardia and atrial fibrillation.

Circus Movement Tachycardia

Programmed electrical stimulation of the heart has made it possible to demonstrate and understand how a circus movement tachycardia can be initiated and terminated in a patient having two connections between the atrium and the ventricle instead of one. As shown in Figure 93.4, a critically timed premature atrial beat that finds the accessory pathway refractory is conducted from the atrium to the ventricle over the AV node–His pathway only, and returns from the ventricle to the atrium over the accessory pathway. Perpetuation of this type of conduction results in a circus movement tachycardia (CMT), also called an atrioventricular reentrant tachycardia (AVRT). A CMT with AV conduction over the AV node and ventriculoatrial conduction over the accessory AV pathway is called an orthodromic CMT (Fig. 93.4A). A tachycardia running in the reverse direction (AV conduction over the accessory AV pathway and ventriculoatrial conduction over the His-AV node) is called an antidromic CMT (Fig. 93.4B).

Orthodromic tachycardia is 10 to 15 times more common than the antidromic type. Programmed stimulation of the heart has shown that there are many ways to initiate a CMT. However, they all have in common the creation of a unidirectional block in one of the two AV pathways. This can occur by a critically timed atrial or ventricular premature beat, by reaching a critical sinus rate, or after administration of a drug-creating block in one of the two pathways.

Intracardiac recordings during CMT and observations during surgery and catheter ablation have shown that the most common location of an accessory AV pathway is the one connecting the left atrium with the left ventricle (50%). A posteroseptal, right ventricular, or anteroseptal insertion of the AV pathway is found in approximately 30%, 13%, and 7% of patients, respectively (Fig. 93.5).
Circus movement tachycardia is the most common type of tachycardia in the patient with an accessory AV pathway. As discussed in Chapter 91, intracardiac recordings during supraventricular tachycardia showed that an accessory AV pathway that can conduct only retrogradely, a so-called concealed accessory pathway, is often incorporated into the tachycardia circuit. These patients never show ventricular preexcitation during sinus rhythm but do have an accessory connection between the ventricle and the atrium that plays an essential role in the tachycardia mechanism.

Atrial Fibrillation

In the human heart, the ventricles are protected by the refractory period of the AV node against a high ventricular rate during a rapid atrial rhythm. In patients having a rapidly conducting accessory pathway, atrial fibrillation can be an extremely dangerous arrhythmia if the accessory connection has a short anterograde refractory period. An example is shown in Figure 93.6. In that situation, the occurrence of ventricular fibrillation has been documented. In patients with the WPW syndrome, the ventricular rate during fibrillation is determined not only by the duration of the refractory period of the accessory pathway in anterograde direction, but also by factors such as the length of the refractory period of the AV node and ventricle and concealed anterograde and retrograde penetration into the accessory pathway and into the AV node.17

The duration of the anterograde refractory period of the accessory pathway is influenced by the autonomic nervous system. Sympathetic discharge induced by the fall in blood pressure and the anxiety that accompany atrial fibrillation may lead to abbreviation of the refractory period of the accessory pathway and a further increase in ventricular rate18 and induction of ventricular fibrillation (Fig. 93.7).

FIGURE 93.6. The two most common types of tachycardia in the Wolff-Parkinson-White syndrome. (A) An orthodromic circus movement tachycardia with AV conduction over the AV node–His pathway and ventriculoatrial conduction over the accessory pathway. (B) Atrial fibrillation with a shortest RR interval of 200 ms. (C) The ECG during sinus rhythm. This is the same patient as in Figure 93.8B. This example is chosen to stress that maximal preexcitation and high ventricular rates can occur during atrial fibrillation in patients showing little ventricular preexcitation during sinus rhythm.

FIGURE 93.7. Example of deterioration of atrial fibrillation into ventricular fibrillation in a patient with a posteroseptal accessory AV pathway. Twelve ECG leads were recorded simultaneously. The left panel shows the ECG during sinus rhythm.
The Electrocardiogram in Locating the Accessory Pathway

Over the years different ECG schemes have been presented to identify the location of the accessory pathway. They are based on the delta wave axis in the frontal and horizontal plane. Sufficient preexcitation should be present to locate the ventricular insertion of the accessory pathway. Accessory pathways may be located around the two atrioventricular rings. The most common location is a connection between the left atrium and the left ventricle on the free wall. As shown in Figure 93.8, during sinus rhythm, ventricular activation starts in the lateral upper corner of the left ventricle moving away from lead aVL, resulting in a negative delta wave in that lead. In the frontal plane ventricular activation thereafter spreads toward the apex and the right ventricle, resulting in a positive delta wave in leads II and III. In the horizontal plane, the delta and QRS axis depend on a left posterior or left lateral location of the accessory pathway. If the ventricular end is located left posteriorly, all precordial leads show a positive delta wave and a positive QRS complex (a concordant pattern). If the ventricular end is located more left laterally, an R/S ratio of around 1 is found in lead V6, while a completely positive QRS complex is seen in the precordial leads V1 to V6.

The atrial end of the accessory pathway is located in the lateral free wall of the left atrium. That location leads to a characteristic P-wave configuration during a CMT using a left-sided accessory pathway. As shown in Figure 93.8, during CMT, atrial activation moves away from lead aVL and I, in the direction of lead III, resulting in a negative P wave in leads I and aVL and a positive P wave in lead III.

In a posteroseptal accessory pathway (Fig. 93.9), ventricular activation by way of the accessory pathway during sinus rhythm, results in the earliest ventricular activation in the posteroinferior area of the ventricle, resulting in the frontal plane in negative delta waves in leads II, III, and aVF.

During CMT, atrial activation starts in the posteroinferior part of the atrium close to the midline, resulting in negative P waves in leads II, III, and aVF, and a positive P wave in aVR and aVL. The precordial leads demonstrate whether the ventricular end of the accessory pathway inserts on the right or the left side of the septum. Insertion on the right side leads to a negative or isoelectric delta wave in lead V1 and a negative QRS complex. A left-sided insertion results in a positive delta wave and QRS complex in lead V1.

In a right free wall location of the accessory pathway (Fig. 93.10), ventricular activation over the accessory pathway during sinus rhythm starts in the free wall of the right ventricle, resulting in ventricular activation in the frontal plane away from lead III in the direction of lead I and aVL, which results in a negative delta wave in lead III and a positive delta wave in I and aVL. Of interest is the finding that in a right-sided accessory pathway, because of close proximity of the accessory pathway to the sinus node, the delta wave begins before the P wave is completed. This is in contrast to a left free wall accessory pathway, where the delta wave starts after the P wave has been completed. When a CMT occurs in a patient with a right free wall accessory pathway, atrial activation starts laterally low in the right atrium, resulting in the P wave being negative in lead III and positive in lead aVL.

**FIGURE 93.8.** An ECG during sinus rhythm and circus movement tachycardia in a patient with a left free wall accessory AV pathway. As discussed in the text, in the presence of sufficient ventricular preexcitation, the delta wave axis during sinus rhythm indicates the location of the ventricular end of the accessory pathway, while the P-wave axis during circus movement tachycardia indicates the location of the atrial end.
FIGURE 93.9. An ECG during sinus rhythm and circus movement tachycardia in a patient with a posteroseptal accessory AV pathway. The drawing on the left illustrates that during sinus rhythm ventricular activation over the accessory pathway starts in the posteroinferior area, resulting in negative delta waves in leads II and III. During circus movement tachycardia atrial activation begins in the posteroinferior part of the atrium, leading to negative P waves in leads II and III.

In an anteroseptal location of the accessory pathway (Fig. 93.11), ventricular activation over the accessory pathway during sinus rhythm starts in the anterosuperior part of the ventricle. The QRS is rather narrow because of more simultaneous activation of both ventricles, and the delta wave is positive in leads II and III. During CMT, atrial activation starts in the anterosuperior part of the atrium close to the midline, resulting in a positive P wave in leads II and III.

FIGURE 93.10. An ECG during sinus rhythm and circus movement tachycardia in a patient with a right free wall accessory AV pathway. See text.
Noninvasive Tests to Recognize the Low-Risk Patient

Risk for Sudden Death

The presence of an accessory AV pathway with a short anterograde refractory period may lead to life-threatening high ventricular rates if atrial fibrillation supervenes. Is it possible to identify such risk in the patient with an ECG showing preexcitation? As shown in Table 93.2 several factors have been associated with an increased risk of sudden death in the WPW patient. Some are accepted and some are controversial.

Information about the exact value of the anterograde refractory period of the accessory pathway can be obtained during intracardiac and intraesophageal electrical stimulation.

There are three noninvasive techniques that can facilitate determining the approximate length of the refractory period of the accessory pathway in an anterograde direction. First, the finding of intermittent preexcitation during sinus rhythm (Fig. 93.12) indicates a long anterograde refractory period of the accessory pathway. Second, as first shown by Levy et al., sudden disappearance of preexcitation during exercise points to a long anterograde refractory period of the accessory pathway (Fig. 93.13). One should be careful in interpretation, however, because sympathetic stimulation during exercise speeds up trans–AV nodal conduction and might thereby diminish the area of the ventricles preexcited over the accessory pathway. Several ECG leads, therefore, should be taken simultaneously, and special attention should be given not only to the sudden disappearance of preexcitation during exercise but also to the sudden reappearance of preexcitation after exercise when a sudden marked change in the ECG takes place of resumption over the accessory connection.

Third, failure to produce a complete block in the accessory pathway by intravenous injection of procainamide (10mg/kg body weight over a 5-minute period) strongly suggests a short anterograde refractory period of the accessory pathway (<270msec) (Fig. 93.14). Because procainamide also prolongs the refractory period of the His-Purkinje system, the tests should be done in surroundings in which complete heart block can be appropriately managed.

By using these tests, preexcitation patients who are at low risk for a high ventricular rate can be identified, the best test being sudden block in the accessory pathway during exercise because that finding indicates that also during maximum sympathetic stimulation, no dangerously short

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**TABLE 93.2. Factors associated with sudden cardiac death in Wolff-Parkinson-White syndrome**

<table>
<thead>
<tr>
<th>Accepted</th>
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<tbody>
<tr>
<td>Male (85%)</td>
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<tr>
<td>Short anterograde refractory period of the accessory pathway</td>
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<tr>
<td>Shortest RR during atrial fibrillation</td>
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<tr>
<td>High adrenergic state</td>
<td></td>
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<tr>
<td>Controversial</td>
<td></td>
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<tr>
<td>Multiple accessory pathways</td>
<td></td>
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<tr>
<td>Septal location of the accessory pathway</td>
<td></td>
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<tr>
<td>Age</td>
<td></td>
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<tr>
<td>Presence of circus movement tachycardia</td>
<td></td>
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<tr>
<td>Presence of digitalis</td>
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</tbody>
</table>
FIGURE 93.12. Intermittent preexcitation. The ECG shows alternating conduction over both the accessory pathway and the AV node and AV conduction over the AV node only.

FIGURE 93.13. Effect of exercise in a patient with the Wolff-Parkinson-White (WPW) syndrome. On reaching a critical sinus rate, 1:1 conduction over the accessory pathway disappears. Thereafter, only every third sinus beat is conducted over the accessory pathway until complete block in the accessory pathway occurs.

FIGURE 93.14. Example of the occurrence of complete block in the accessory AV pathway after the IV administration of procainamide (600 mg). This finding indicates a long anterograde refractory period of the accessory pathway.

anterograde refractory period of the accessory pathway is present.

**Treatment**

**Drugs**

Circus movement tachycardias in preexcitation have an initiating and a perpetuating mechanism. A premature beat exposing the different properties of the two AV connections is usually the initiating mechanism. Perpetuation of tachycardia is determined by the electrophysiologic properties
within the tachycardia circuit. Different drugs may affect the properties of the AV node–His pathway and the accessory pathway [Fig. 93.15]. Before discussing drugs in the treatment of CMT, emphasis should be placed on the effectiveness of vagal maneuvers that block conduction in the AV node in the patient with a CMT [Table 93.3]. These maneuvers should be performed as soon as possible after onset of the tachycardia. The longer one waits, the higher the sympathetic tone and the less likely that the maneuvers will be successful. If vagal maneuvers are unsuccessful, the intravenous injection of a drug that suddenly prolongs the refractory period of the AV node [adenosine or verapamil] or produces lengthening of the refractory period of the accessory AV pathway [procainamide] usually breaks the CMT [Table 93.4]. Pacing or cardioversion is rarely required to interrupt CMT. To prevent CMT, amiodarone in small doses [100 to 200mg/day], or drugs such as the class IC agents flecainide, propafenone, or sotalol, a beta-blocking agent with class III effects, are usually effective. Controlled studies comparing the efficacy of the different drugs are not available. It is clear, however, that there is no “magic” drug. Physicians, therefore, should prescribe a drug with which they are familiar.

The choice of treatment of atrial fibrillation is influenced by the ventricular rate and hemodynamic consequences of the arrhythmia. Cardioversion should be done immediately, if a rapid ventricular rhythm during atrial fibrillation leads to severe circulatory impairment. If the arrhythmia is better tolerated, drugs that prolong the anterograde refractory period of the accessory pathway should be given. In the patients with paroxysmal atrial fibrillation to prevent recurrences of the arrhythmia, drugs that lengthen the anterograde refractory period of the accessory pathway, as well as a beta-blocking agent, should be prescribed. The increased sympathetic tone, after onset of atrial fibrillation, tends to decrease the anterograde refractory period of the accessory pathway, leading to an increase in ventricular rate. Digitalis and verapamil should not be given to the patient with preexcitation. Both drugs may shorten the anterograde refractory period of an accessory pathway, thereby leading to an increase in ventricular rate during atrial fibrillation.

**Nonpharmacologic Treatment**

Initially, surgical interruption of the accessory pathway was the only option in patients requiring nonpharmacologic treatment of arrhythmias in preexcitation,25 but this has now been replaced by catheter ablation of the accessory pathway.26,27 This patient-friendly method, which is performed during a cardiac catheterization, is associated with a low risk and a high success rate. This has resulted in increasing use of catheter ablation treatment in patients with the WPW syndrome and certainly in patients suffering from tachycardias.28 However, as will be discussed below, there is controversy over catheter ablation in asymptomatic patients having a short anterograde refractory period of their accessory pathway.

**The Practical Approach to the Patient**

The Patient with an Arrhythmia

When the patient comes for treatment of an arrhythmia, the important questions relate to the following: (1) the type of arrhythmia (CMT, atrial fibrillation, or both); (2) its

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**TABLE 93.3. Maneuvers used to interrupt a supraventricular tachycardia**

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<thead>
<tr>
<th>Maneuver</th>
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<tr>
<td>Valsalva</td>
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<tr>
<td>Squatting [Valsalva maneuver]</td>
</tr>
<tr>
<td>“Gag” reflex [finger in the throat]</td>
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<tr>
<td>“Dive reflex” (immersion of the face in cold water)</td>
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<tr>
<td>“Upside-down position” [legs against the wall]</td>
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incidence, [3] its symptoms and the patient’s tolerance; [4] its tachycardia initiating events; and [5] the presence of additional cardiac disease. Careful attention, therefore, should be paid to the history and circumstances provoking arrhythmias, so that they can be identified. They preferably should be documented by long-term ECG recordings. The necessity and mode of treatment depend on the incidence and severity of the attacks of tachycardia. If the problem is a rare CMT without serious hemodynamic consequences, the patient should try the vagal maneuvers described previously. If they fail, the patient should take oral medication such as flecainide or quinidine, and wait for the arrhythmia to subside. This “cocktail” approach is preferred in the patient with rare well-tolerated tachycardia. When tachycardias occur more frequently and especially when a short anterograde refractory period of the accessory pathway is present, the patient should be referred to a center with experience in catheter ablation of accessory pathways.

The Patient Without an Arrhythmia

When treating an asymptomatic patient with a WPW ECG, the first step is to perform the noninvasive tests outlined above, to determine if the patient is at low risk. In that situation, no measures are advised other than an explanation to the patient of the ECG findings. It is advisable to give the patient a copy of the ECG and a short note about the fact that the WPW syndrome is present, to prevent the misdiagnosis of myocardial infarction and to explain the basis of cardiac arrhythmias in case they develop later.

If noninvasive testing suggests a short anterograde refractory period of the accessory pathway, most arrhythmologists feel that the decision to recommend an invasive study and catheter ablation of the accessory pathway should be made individually, depending on the age of the patient, the location of the accessory pathway, and the patient’s social and professional factors. If catheter ablation were a totally risk-free procedure, one would logically recommend such a procedure to the asymptomatic WPW patient with a short anterograde refractory period of the accessory pathway. However, there are certain risks associated with catheter ablation. They include the general risks of a cardiac catheterization, such as thromboembolic complications, infection, bleeding, cardiac perforation with or without cardiac tamponade, valvular damage, and radiation damage. In addition there are specific risks in patients with accessory pathways. In small children, the catheter ablation lesion is relatively large because of their heart size. Risk is also related to the location of the accessory pathway. Ablation of a para-Hisian accessory pathway carries the risk of complete AV block. In epicardially located posteroseptal or left posterior accessory pathways, where ablation has to be performed in the coronary venous system, there is the risk of damage to the circumflex coronary artery or perforation of the venous system, leading to cardiac tamponade. In right free wall accessory pathways, the right coronary artery may be damaged during catheter ablation.

Three registry studies reported on the complications of radiofrequency ablation in symptomatic patients with accessory pathways. Although rare, deaths were reported in all three studies, along with other complications, such as complete AV block, cardiac perforation with and without tamponade, and cardiovascular accidents. These studies were done in the early 1990s in symptomatic patients. Unfortunately, we do not have more recent information about the current complication rate of catheter ablation in patients with accessory pathways with the exception of the pediatric age group. Kuessler et al. compared 1991–1995 with 1996–1999 and found an increased success rate of catheter ablation of accessory pathways but no significant change in complications.

The Role of Invasive Testing

Recent publications by Pappone and coworkers suggest that the ability to induce a sustained arrhythmia [supraventricular tachyarrhythmia (SVT) or atrial fibrillation] in the asymptomatic patient with a WPW ECG is a marker of risk and should be followed by catheter ablation of the accessory pathway. They put less emphasis on the duration of the anterograde refractory period of the AP as a risk factor, although in asymptomatic WPW patients, a short duration identifies the risk for a high ventricular rate for atrial fibrillation. In addition, another problem is that the electrophysiologic properties of both the accessory pathway and the normal AV conduction system may change over time, often in an unpredictable way. In patients with a short anterograde refractory period of their accessory pathway (<260ms), it is rare to see marked lengthening of that value during a repeat study much later, while such lengthening, even complete anterograde block in the accessory pathway, is not unusual in patients with a relatively long anterograde refractory period of the accessory pathway. Another point is that the inability to induce an arrhythmia now is no guarantee that it will not be possible much later. For example, in the patient with a short anterograde refractory period of the accessory pathway, no arrhythmia may be induced at a young age, but atrial fibrillation may develop when the patient is in his sixties.

A major problem in the studies by Pappone et al. is the much higher incidence of [serious] arrhythmias than in previous natural history publications. Also, it may not be easy to identify the asymptomatic WPW patient. This seems to be especially difficult in asymptomatic children where it is unusual to make a routine ECG recording.

Current Guidelines

The most recent guidelines of the American College of Cardiology and the European Society of Cardiology about the management of asymptomatic WPW patients, suggest restricting catheter ablation of accessory pathways to patients in high-risk occupations and professional athletes, in other words, to advise on the basis of individual considerations. Catheter ablation in asymptomatic preexcitation was classified as a IIA indication with a B level of evidence. According to the North American Society for Pacing and Electrophysiology (NASPE) Expert Consensus Conference, an asymptomatic WPW is a class IIB indication for catheter ablation in children above 5 years of age and a class III indication in younger children.
Summary

While there is no difference in opinion about the management of the WPW patient suffering from cardiac arrhythmias related to the presence of an accessory AV pathway, there is controversy about how to manage an asymptomatic WPW patient. In view of the risks of invasive studies and catheter ablation, one should start with noninvasive studies (exercise, long-term ECG recordings, effects of an intravenous pharmacologic intervention) to identify the low-risk patient with a long anterograde refractory period of the accessory pathway. In patients not showing block in their accessory pathway during these noninvasive studies, esophageal pacing can be performed to determine the anterograde refractory period of the accessory pathway and the ability to initiate sustained arrhythmias. If the latter is the case, benefit and risk of an invasive investigation and catheter ablation should be based on individual considerations, such as age, gender, occupation, and athletic participation. This should be discussed with the patient and, in the case of a child, also with the parents. Because knowledge about the success and complication rate at a certain electrophysiologic center plays a major role in decision making, that information should be made available to help select the appropriate center for invasive diagnosis and treatment.

References