

## REVIEW ARTICLE

## MEDICAL PROGRESS

## SUPRAVENTRICULAR TACHYCARDIA

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**S**UPRAVENTRICULAR tachycardia is any tachyarrhythmia that requires atrial or atrioventricular junctional tissue for its initiation and maintenance. Decades ago the term "paroxysmal atrial tachycardia" was coined to describe supraventricular tachycardia that began and ended abruptly. This term has become obsolete, however, since it is now clear that many such arrhythmias arise in the atrioventricular junction, not in atrial muscle itself. Much of our present knowledge about the sites of origin and mechanisms of supraventricular tachycardia has been derived from cellular electrophysiologic studies and from clinical electrophysiologic studies using the technique of programmed electrical stimulation.<sup>1,2</sup> We have learned that such arrhythmias may arise from reentry caused by unidirectional conduction block in one region of the heart and slow conduction in another, from enhanced automaticity akin to that seen in normal pacemaker cells of the sinus node and in latent pacemaker cells elsewhere in the heart, or from triggered activity, a novel type of abnormally enhanced impulse initiation caused by membrane currents that can be activated and inactivated by premature stimulation or rapid pacing.<sup>1</sup>

This article will focus on recent advances in our understanding of supraventricular tachycardias and their management. Although atrial fibrillation and atrial flutter can properly be considered supraventricular tachycardias, these related arrhythmias are sufficiently distinct in terms of their electrophysiology and management that they will not be considered in this article.<sup>3</sup>

## MECHANISMS OF SUPRAVENTRICULAR TACHYCARDIA

## Atrioventricular Nodal Reentrant Tachycardia

Atrioventricular nodal reentry is probably the most common cause of paroxysmal regular supraventricular tachycardia, accounting for half or more of the cases of supraventricular tachycardia that are referred for diagnostic electrophysiologic study.<sup>4,5</sup> Although atrioventricular nodal reentrant tachycardia may appear at any

age, most patients first seek medical attention during the fourth or fifth decade of life. Seventy percent of patients with this arrhythmia are women.<sup>4,6</sup>

In patients with atrioventricular nodal reentrant tachycardia at least two functionally distinct conduction pathways are demonstrable within the atrioventricular node during electrophysiologic study. One pathway, referred to as the fast pathway, is characterized by rapid conduction velocity and a relatively long refractory period. The second, or slow, pathway typically has slow conduction velocity and a short refractory period. During sinus rhythm, conduction through the atrioventricular node to the ventricles occurs over the fast pathway (Fig. 1). Atrioventricular nodal reentry is typically initiated by an atrial premature depolarization that blocks anterogradely in the fast pathway (because of its longer refractory period) while conducting slowly in the anterograde direction over the slow pathway. If anterograde conduction over the slow pathway is slow enough, the refractory period in the fast pathway has enough time to end. This enables the impulse to propagate retrogradely over the fast pathway back to the atrium, thereby completing the reentrant circuit (Fig. 1).

Common atrioventricular nodal reentrant tachycardia, as just described, is due to a reentrant circuit in which there is anterograde conduction over the slow atrioventricular nodal pathway and retrograde conduction over the fast pathway. Since anterograde conduction over the His bundle and bundle branches to the ventricles occurs at the same time as retrograde conduction over the fast pathway to the atria, there is simultaneous or nearly simultaneous inscription of the P waves and QRS complexes on the electrocardiogram (Fig. 1). The P waves are negative in surface electrocardiographic leads II, III, and aVF because the atria are activated in a caudocranial direction, but they are rarely visible because they are superimposed on the QRS complexes. In 10 percent of patients the reentry circuit is reversed, with anterograde conduction over the fast pathway and retrograde conduction over the slow pathway.<sup>4</sup> This type of tachycardia, referred to as uncommon atrioventricular nodal reentrant tachycardia, is characterized by clearly visible P waves that are inverted in surface electrocardiographic leads II, III, and aVF and an RP interval during tachycardia that is much longer than the PR interval (Fig. 1). Uncommon atrioventricular nodal reentry is usually initiated by a ventricular premature depolarization and is rarely sustained.<sup>7</sup>

## Supraventricular Tachycardia Mediated by Accessory Pathways

Normally, the atrioventricular node, His bundle, and bundle branches provide the only conduit for the transmission of impulses between the atria and the ventri-

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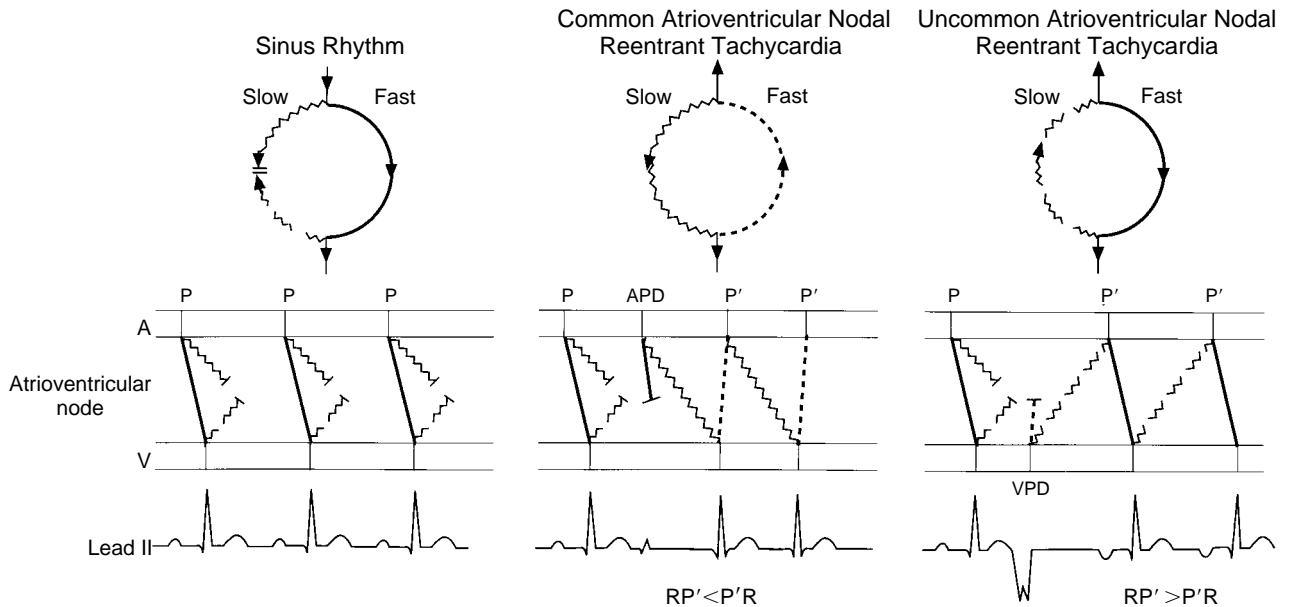


Figure 1. Mechanism of Atrioventricular Nodal Reentrant Tachycardia.

Each panel shows the atrioventricular node (top), a Lewis diagram (middle), and a surface electrocardiographic lead (bottom). Solid lines indicate anterograde atrioventricular nodal conduction, and broken lines retrograde conduction; straight lines indicate conduction over the fast pathway, and wavy lines conduction over the slow pathway. P denotes sinus P waves, P' atrial echoes resulting from atrioventricular nodal reentry, APD atrial premature depolarization, VPD ventricular premature depolarization, A atrial, V ventricular, and R R waves. During sinus rhythm the presence of the slow pathway is concealed because the impulse traveling over the fast pathway turns around after traversing the atrioventricular node and retrogradely penetrates the slow pathway, colliding with the oncoming impulse moving anterogradely over the slow pathway. Note the simultaneous registration of P' waves and QRS complexes during common atrioventricular nodal reentrant tachycardia, with  $RP' < P'R$ . Retrograde P' waves result in the appearance of pseudo-S waves in the inferior electrocardiographic leads. During uncommon atrioventricular nodal reentrant tachycardia, inverted P' waves are visible, with  $RP' > P'R$ .

cles. Accessory pathways are anomalous bands of conducting tissue that form a connection between the atrium and ventricle in addition to the normal atrioventricular conducting system. Most accessory pathways are characterized by rapid nondecremental conduction and can conduct anterogradely from atrium to ventricle as well as retrogradely in the opposite direction.<sup>8</sup> When there is anterograde accessory-pathway conduction during sinus rhythm, ventricular preexcitation occurs, because of early activation of a part of the ventricles over the accessory pathway. This results in the combination of a short PR interval and a delta wave, the electrocardiographic signature of the Wolff-Parkinson-White syndrome (Fig. 2).<sup>9</sup> Accessory pathways do not always result in ventricular preexcitation. Nearly a quarter of accessory pathways are capable of only retrograde ventriculoatrial conduction.<sup>10</sup> Such accessory pathways, called concealed bypass tracts because their presence is not evident on the surface electrocardiogram during sinus rhythm, may still cause episodes of supraventricular tachycardia (see below).

The most common supraventricular tachycardia in patients with the Wolff-Parkinson-White syndrome is orthodromic atrioventricular reentrant tachycardia. Orthodromic tachycardia typically begins with a spontaneous atrial or ventricular premature depolarization

that initiates a reentrant circuit in which the circulating impulse travels anterogradely over the atrioventricular node, His bundle, and bundle branches to the ventricles and then retrogradely over the accessory pathway to the atrium (Fig. 2). Since during orthodromic tachycardia the ventricles are depolarized over the normal atrioventricular conduction system, no delta wave is seen during tachycardia. Orthodromic tachycardia also occurs in patients with concealed accessory pathways. In fact, approximately 30 percent of patients referred for electrophysiologic study because of supraventricular tachycardia without evidence of preexcitation during sinus rhythm are discovered to have orthodromic atrioventricular reentrant tachycardia, making this the most common cause of supraventricular tachycardia after atrioventricular nodal reentry.<sup>5</sup>

In some patients with concealed accessory pathways, the accessory pathway is characterized by very slow decremental retrograde conduction.<sup>11-13</sup> During orthodromic tachycardia in such patients, the retrograde P waves are inscribed long after the QRS complex, an electrocardiographic pattern described as an RP interval that is much longer than the PR interval during tachycardia. This type of orthodromic tachycardia may begin spontaneously during sinus rhythm, is typically incessant,<sup>7,13</sup> and has been called the "permanent form

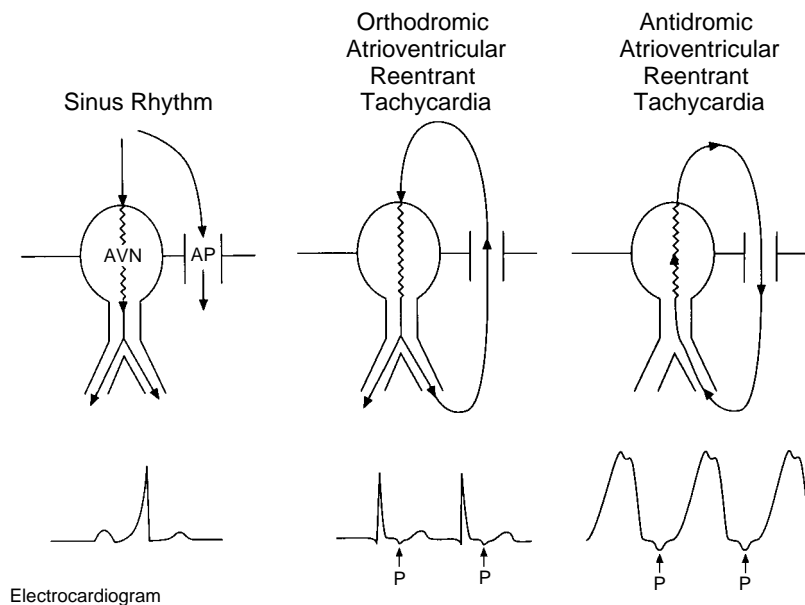


Figure 2. Mechanism of Atrioventricular Reentrant Tachycardia in Patients with the Wolff-Parkinson-White Syndrome.

During sinus rhythm the slurred initial portion of the QRS or delta wave is due to early activation of part of the ventricles through rapid anterograde conduction over the accessory pathway (AP). During orthodromic atrioventricular reentrant tachycardia no delta wave is seen because all anterograde conduction is over the atrioventricular node (AVN) and through the normal His-Purkinje system. Retrograde P waves are visible shortly after each QRS. During antidromic atrioventricular reentrant tachycardia there is maximal preexcitation with wide, bizarre QRS complexes, because ventricular activation results entirely from anterograde conduction over the accessory pathway.

of junctional reciprocating tachycardia.<sup>9,11,12</sup> Left untreated, it may lead to tachycardia-related cardiomyopathy.<sup>14</sup>

In approximately 10 percent of patients with the Wolff-Parkinson-White syndrome and atrioventricular reentrant tachycardia, the reentrant circuit travels in the opposite direction, anterogradely from atrium to ventricle over the accessory pathway and retrogradely up the bundle branches and His bundle and through the atrioventricular node back to the atrium.<sup>15,16</sup> This type of tachycardia, referred to as antidromic atrioventricular reentrant tachycardia, is characterized by a wide QRS configuration that is an exaggeration of the delta wave seen during sinus rhythm (Fig. 2). Antidromic tachycardia is more common among patients with multiple accessory pathways.<sup>16</sup>

Atrial fibrillation and atrial flutter are frequently seen in patients with the Wolff-Parkinson-White syndrome.<sup>17</sup> These arrhythmias are usually precipitated by an episode of atrioventricular reentrant tachycardia, but they may also occur alone.<sup>18,19</sup> Atrial fibrillation and atrial flutter are particularly hazardous in patients with the Wolff-Parkinson-White syndrome because most accessory pathways have rapid nondecremental conduction. These patients may achieve ventricular rates that approach or exceed 300 beats per minute during atrial fibrillation or atrial flutter. Ven-

tricular fibrillation can occur under such circumstances; it may be a particular risk in patients with multiple accessory pathways.<sup>20</sup> Sudden death, though unusual, may even be the first clinical manifestation of the Wolff-Parkinson-White syndrome.<sup>20,21</sup>

The anterograde effective refractory period of the accessory pathway is probably the most important factor in determining the risk of ventricular fibrillation during atrial fibrillation.<sup>20,22</sup> A value below 250 msec has been used to segregate patients who are at particular risk.<sup>20</sup> Evidence of intermittent preexcitation on the resting electrocardiogram,<sup>23</sup> the sudden disappearance of the delta wave after the intravenous injection of a high dose of procainamide<sup>24</sup> or ajmaline,<sup>25</sup> and the sudden disappearance of the delta wave at a relatively slow heart rate during exercise<sup>26</sup> have all been used as noninvasive markers of a long refractory period and therefore a low risk. Electrophysiologic study, however, is the most reliable method of determining the refractory period of the accessory pathway and the number of pathways present. Such a

study is recommended as part of the routine evaluation of all symptomatic patients with the Wolff-Parkinson-White syndrome, even in the absence of documented tachycardia.

#### Nonparoxysmal Junctional Tachycardia

Nonparoxysmal junctional tachycardia, caused by enhanced impulse initiation within the atrioventricular junction rather than by reentry, is a very rare cause of supraventricular tachycardia in adults. The putative mechanism in most circumstances is enhanced automaticity or triggered activity.<sup>27</sup> It may be seen in circumstances that involve inflammation near the atrioventricular junction, such as recent mitral- or aortic-valve surgery, acute myocardial infarction, or digitalis toxicity.<sup>28,29</sup> Electrocardiographically, this arrhythmia appears as a regular narrow-QRS-complex tachycardia, with either atrioventricular dissociation or 1:1 ventriculoatrial activation.

#### Sinus-Node Reentrant Tachycardia

Sinus-node reentrant tachycardia is due to a reentry circuit that incorporates the sinoatrial node.<sup>30,31</sup> Among symptomatic patients with supraventricular tachycardia who are referred for electrophysiologic study, fewer than 5 percent have sinus-node reentry.<sup>5,32</sup> Since the tachycardia originates in the sinus node, the P waves

during tachycardia are identical to sinus P waves. Unlike sinus tachycardia, sinus-node reentrant tachycardia begins and ends abruptly. The PR interval during such a tachycardia is directly proportional to the atrial rate, but the pattern of an RP interval that is greater than the PR interval is usually present.

#### **Unifocal Atrial Tachycardia**

Atrial tachycardia is a supraventricular tachycardia arising from atrial muscle that does not include the sinus node. Unifocal atrial tachycardia is characterized by a single P-wave morphologic pattern, the configuration of which depends entirely on the atrial site from which the tachycardia originates. In unifocal atrial tachycardia the atrial rate is usually less than 250 beats per minute, which helps distinguish this tachycardia from atrial flutter. However, some unifocal atrial tachycardias may exceed 250 beats per minute, and the rate of atrial flutter in some patients with dilated atria or depressed atrial conduction may be slower than 250 beats per minute. Thus, distinguishing between the two arrhythmias on the basis of rate alone is sometimes impossible.

Unifocal atrial tachycardia is a rare cause of supraventricular tachycardia, accounting for fewer than 15 percent of patients who seek attention because of symptomatic arrhythmias.<sup>5</sup> In some patients unifocal atrial tachycardia is due to reentry.<sup>31,33</sup> These patients frequently have structural heart disease leading to abnormalities of atrial refractoriness and conduction, the substrate on which reentry depends.<sup>33</sup> Reentrant atrial tachycardias are usually paroxysmal. Unifocal atrial tachycardia may also result from enhanced automaticity or triggered activity within an atrial focus.<sup>34,35</sup> Such a tachycardia may occur in patients with or without underlying structural heart disease, tends to be incessant rather than paroxysmal, and often involves wide variations in the atrial rate in response to fluctuations in autonomic tone. The tachycardia may exceed 250 beats per minute during periods of heightened sympathetic activity and, left untreated, may cause tachycardia-related cardiomyopathy.<sup>36</sup> An infrequent mechanism of unifocal atrial tachycardia is triggered activity due to digitalis intoxication.<sup>37</sup> In such cases one typically sees 2:1, 3:1, or variable atrioventricular conduction, so-called paroxysmal atrial tachycardia with block, which is due to the depression of atrioventricular nodal conduction induced by digitalis.

#### **Multifocal Atrial Tachycardia**

The diagnosis of multifocal atrial tachycardia is based on electrocardiographic criteria requiring three or more different morphologic patterns of P waves and an irregular atrial rate averaging 100 beats per minute or more. Variation in the PR intervals and varying degrees of atrioventricular block are common. Isoelectric periods between adjacent P waves help distinguish this arrhythmia from atrial fibrillation. The putative mechanism is enhanced automaticity, although triggered ac-

tivity is also a possibility.<sup>38</sup> Multifocal atrial tachycardia is most common in acutely ill elderly patients. Sixty percent of patients with this arrhythmia have pulmonary disease.<sup>38</sup>

### **INITIAL MANAGEMENT**

#### **Differential Diagnosis**

The symptoms associated with supraventricular tachycardia can be so subtle that the affected person does not seek medical attention. Others may have palpitations, lightheadedness, dyspnea, diaphoresis, angina, or vague chest discomfort. These symptoms may be more common in patients with underlying heart disease. Serious sequelae, such as overt heart failure, myocardial infarction, and syncope, are uncommon; sudden death due to supraventricular tachycardia is quite rare.

In patients with sustained supraventricular tachycardia who do not have serious hemodynamic compromise, one should attempt to determine the mechanism of the tachycardia before initiating treatment. The history and physical examination are rarely helpful, except in atrioventricular nodal reentrant tachycardia, in which simultaneous activation of the atria and ventricles results in atrial contraction against closed atrioventricular valves. Patients with this condition typically have prominent jugular venous pulsations that match the rate of the tachycardia, a feature that has been referred to as the frog sign.<sup>39,40</sup> Clues to the correct diagnosis may be found in the manner in which the tachycardia occurs and by a careful inspection of the 12-lead electrocardiogram (Table 1).

Despite careful analysis of the surface electrocardiogram, an incorrect diagnosis may still be made in as many as 20 percent of cases.<sup>41</sup> Diagnostic accuracy may be improved with vagal maneuvers designed to block atrioventricular nodal conduction, such as carotid-sinus massage or the Valsalva maneuver, or the intravenous administration of atrioventricular nodal blockers (see below). Supraventricular tachycardias that continue in the presence of atrioventricular block are thus unmasked as atrial (Fig. 3). Conversely, a tachycardia that terminates with a nonconducted P wave is most likely to be atrioventricular reentrant tachycardia or atrioventricular nodal reentrant tachycardia (Fig. 3). Finally, one should be aware that some tachycardias may be conducted aberrantly, giving rise to a wide-QRS-complex tachycardia suggestive of ventricular tachycardia. A number of electrocardiographic clues to aid in the differential diagnosis have been described.<sup>42,43</sup>

#### **Therapy**

If vagal maneuvers do not terminate a regular supraventricular tachycardia of recent onset,<sup>44</sup> initial pharmacologic therapy should be directed toward blocking atrioventricular nodal conduction. Adenosine, an A<sub>1</sub>-receptor agonist with a rapid onset and brief duration of action, is the drug of choice.<sup>45,46</sup> Adenosine is

Table 1. Clinical Clues to the Differential Diagnosis of Supraventricular Tachycardia.

TACHYCARDIA	PREVALENCE	USUAL PRESENTATION	ELECTROCARDIOGRAPHIC CHARACTERISTICS
Atrioventricular nodal reentrant			
Common	Common	Paroxysmal	P waves hidden, pseudo-R in V <sub>1</sub> , pseudo-S in II or III
Uncommon	Uncommon	Paroxysmal	Inverted P waves, RP>PR
Accessory-pathway-mediated supraventricular			
Orthodromic atrioventricular reentrant	Common	Paroxysmal	Inverted P waves,* RP<PR, QRS alternans
Atrial fibrillation (Wolff-Parkinson-White)	Common	Paroxysmal	Irregularly irregular, variable QRS configuration
Antidromic atrioventricular reentrant	Rare	Paroxysmal	Inverted P waves, wide and bizarre QRS
Permanent junctional reciprocating	Rare	Incessant	Inverted P waves,* RP>PR
Sinus-node reentrant	Uncommon	Paroxysmal	Upright P waves, RP>PR
Unifocal atrial			
Reentrant	Uncommon	Paroxysmal	Upright, biphasic, or inverted P waves; RP>PR
Automatic	Rare	Incessant	Upright, biphasic, or inverted P waves; RP>PR; variable atrial rate
Multifocal atrial	Common	Incessant	Variable P waves, variable rate, variable PR intervals

\*The electrocardiographic lead or leads showing inverted P waves are related to the site of the earliest atrial activation during tachycardia.

administered intravenously, exerting effects that are apparent within 15 to 30 seconds. Doses that effectively terminate tachycardia frequently cause transient side effects such as facial flushing, chest pain, and dyspnea. However, because of the extremely short elimination half-life of adenosine, its effects typically disappear within 10 to 20 seconds.<sup>45</sup> Care should be taken in some circumstances. Dipyridamole potentiates the effect of adenosine.<sup>47</sup> Moreover, heart-transplant recipients seem to have a denervation supersensitivity to adenosine.<sup>48</sup> In patients with asthma, bronchospasm triggered by intravenous adenosine remains a theoretical but clinically undocumented complication.<sup>49,50</sup>

Ninety percent or more of tachycardias due to atrioventricular nodal reentry or atrioventricular reentry are terminated by a 12-mg dose of adenosine (Fig. 3).<sup>51</sup> Adenosine also frequently terminates sinus-node reentrant tachycardia,<sup>32,52</sup> but the drug only occasionally terminates unifocal atrial tachycardia.<sup>53,54</sup> Multifocal atrial tachycardia and atrial flutter or fibrillation almost always persist (Fig. 3).<sup>53</sup> The calcium-channel blockers verapamil<sup>55</sup> and diltiazem,<sup>56</sup> when given intravenously, are also useful for terminating supraventricular tachycardia. When compared in a randomized, double-blind study, intravenous verapamil and adenosine were equally effective in terminating episodes of supraventricular tachycardia.<sup>51</sup> Calcium-channel blockers have peripheral vasodilating and negative inotropic effects that may be exaggerated in patients already taking a beta-blocker. Verapamil has a longer duration of action than adenosine, which may be an advantage in preventing the immediate recurrence of tachycardia but a disadvantage if verapamil causes hypotension.

Adenosine and calcium-channel blockers should not be given to patients who have atrial fibrillation with an anterogradely conducting accessory pathway, because blocking atrioventricular nodal conduction may provoke conduction down the accessory pathway, leading to an increase rather than a decrease in the ventricular rate and to hemodynamic collapse.<sup>57,58</sup> The ideal emergency treatment for such patients is intravenous procainamide, which will slow the ventricular rate by blocking conduction over the accessory pathway. Electrical cardioversion may also be used when necessary.

Hemodynamic collapse may also result when calcium-channel blockers are administered to patients whose ventricular tachycardia is mistakenly believed to be supraventricular tachycardia with aberrant intraventricular conduction.<sup>59</sup> Such adverse effects can theoretically occur after the administration of adenosine as well, but the extremely brief duration of action of adenosine makes them very unlikely.<sup>60-63</sup>

Calcium-channel blockers are preferable in patients with atrial tachycardia, which is rarely terminated by adenosine. Administered as a constant intravenous infusion, a calcium-channel blocker can block atrioventricular nodal conduction, reduce the ventricular rate, and thereby help relieve symptoms even though the tachycardia persists. The longer duration of action of the calcium-channel blockers is also advantageous in patients with concealed slowly conducting accessory pathways and incessant atrioventricular reentrant tachycardia. Intravenous digoxin, because of its delayed onset of action, and intravenous beta-blockers, because of their negative inotropic and bronchoconstrictive effects, offer no real advantage over adenosine or calcium-channel blockers, nor are they as effective. Magnesium,<sup>64</sup> disopyramide,<sup>65</sup> and flecainide<sup>66</sup> have been used to terminate various types of supraventricular tachycardia, but the limited experience with these therapies makes them inappropriate in most circumstances.

#### LONG-TERM MEDICAL THERAPY

Not all patients with supraventricular tachycardia require long-term (maintenance) medical treatment. To decide whether such treatment is necessary, one must weigh carefully the risk of the arrhythmia to the patient if left untreated, the risk of the therapy being contemplated, the severity of symptoms, and the frequency of the episodes of tachycardia. Frequency is best assessed with Holter monitoring and electrocardiographic recording of events.

Patients with atrioventricular nodal reentry or orthodromic atrioventricular reentry due to a concealed accessory pathway in whom tachycardias are frequent and refractory to vagal maneuvers should first be given a drug that blocks atrioventricular nodal conduction (Table 2). Verapamil is probably the most widely pre-

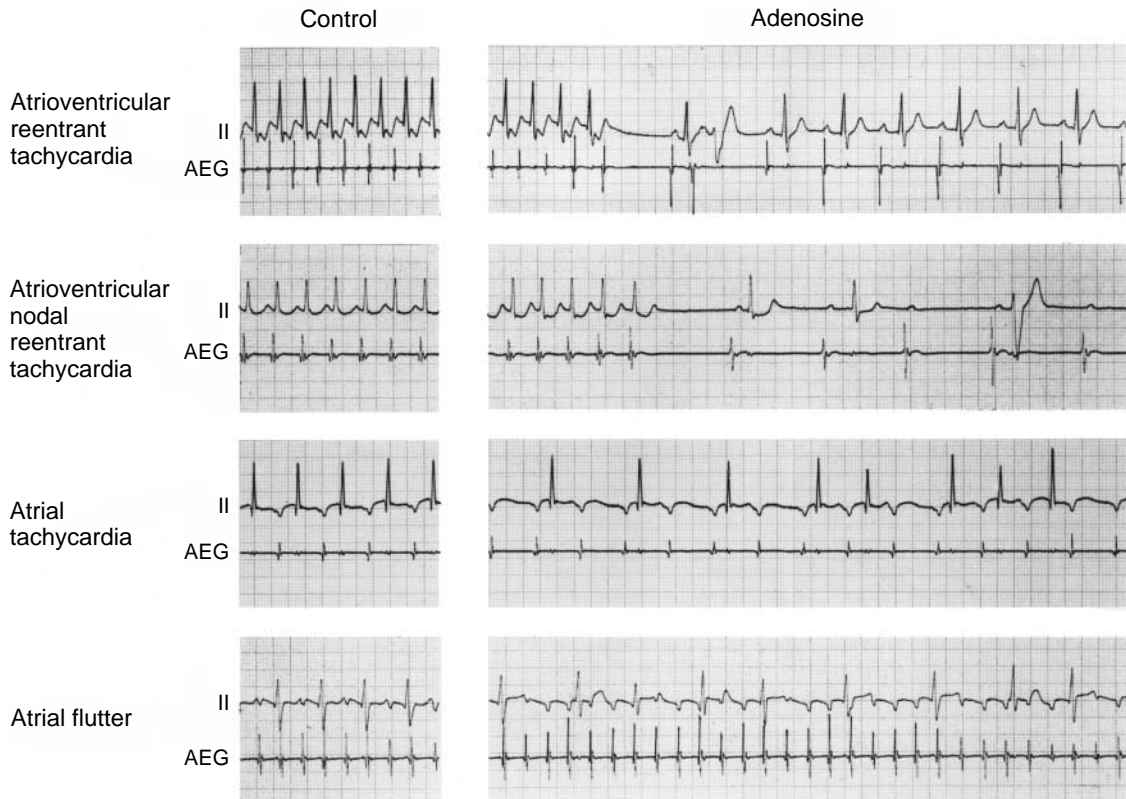


Figure 3. Effect of Adenosine on Atrioventricular Reentrant Tachycardia, Atrioventricular Nodal Reentrant Tachycardia, Atrial Tachycardia, and Atrial Flutter.

Each panel shows the tracings for surface electrocardiographic lead II and an intracardiac bipolar high right atrial electrogram (AEG) that shows the position of the P waves. Retrograde P waves and QRS complexes are registered simultaneously during atrioventricular nodal reentrant tachycardia, whereas retrograde P waves are registered shortly after each QRS during atrioventricular reentrant tachycardia but with  $RP < PR$ . In both atrioventricular reentrant tachycardia and atrioventricular nodal reentrant tachycardia, adenosine blocks anterograde conduction in the atrioventricular node, causing termination of the tachycardia after a retrograde P wave. During atrial tachycardia, the RP interval is greater than the PR interval. Adenosine causes transient 2:1 atrioventricular conduction without affecting the atrial rate or interrupting the tachycardia, thus ruling out an accessory pathway as part of the mechanism of the tachycardia. During atrial flutter there is 2:1 atrioventricular conduction, but only alternate P waves are visible on the surface electrocardiogram, with the RP interval apparently greater than the PR interval. Adenosine causes transient atrioventricular block, revealing typical flutter waves.

scribed drug for this purpose, although a randomized, double-blind comparison of verapamil with digoxin and propranolol failed to demonstrate the superiority of one drug over the others.<sup>67</sup> The effectiveness of single large doses of atrioventricular nodal blockers administered as a "cocktail" to abort episodes of arrhythmia has not been established in careful clinical trials.<sup>68</sup> Side effects often limit the long-term use of calcium-channel blockers or beta-blockers, whereas digoxin is almost always well tolerated.<sup>69</sup>

Patients with atrioventricular nodal reentry and orthodromic atrioventricular reentry due to a concealed accessory pathway who cannot tolerate or do not respond to atrioventricular nodal blockers may be candidates for long-term therapy with a class I antiarrhythmic drug (Table 2). These drugs typically block accessory-pathway and retrograde fast atrioventricular nodal conduction. The class IA drugs procainamide,<sup>70</sup> quinidine,<sup>71</sup> and disopyramide<sup>65</sup> have been effective in some patients. Side effects limit the usefulness of these

drugs; the risk of a ventricular proarrhythmia such as drug-induced torsade de pointes is of particular concern. Flecainide<sup>72</sup> and propafenone,<sup>73</sup> which are class IC agents, have both been shown to be effective in prospective, double-blind, placebo-controlled clinical trials. When used to treat supraventricular tachycardia in patients without associated structural or ischemic heart disease, class IC agents do not appear to increase the risk of death, as they do in patients with ventricular arrhythmias and recent myocardial infarctions.<sup>74-76</sup> Nevertheless, one should consult a cardiologist with particular expertise in managing arrhythmias before using class IA or IC drugs to treat patients with supraventricular tachycardia.

The choice of maintenance therapy for patients with manifest ventricular preexcitation during sinus rhythm should be made only after electrophysiologic study to assess the anterograde refractory period of the accessory pathway or pathways and the risk of hazardous ventricular rates during atrial fibrillation. Drugs that block

Table 2. Long-Term Antiarrhythmic-Drug Therapy for Supraventricular Tachycardia.

TACHYCARDIA	FIRST CHOICE	SECOND CHOICE	THIRD CHOICE
Atrioventricular nodal reentrant and atrioventricular reentrant (concealed by-pass tract)	Calcium-channel blockers, beta-blockers, digoxin	Flecainide, propafenone*† Quinidine, procainamide, disopyramide‡‡	Amiodarone
Wolff–Parkinson–White syndrome	Flecainide, propafenone* Quinidine, procainamide, disopyramide‡‡	Beta-blockers, calcium-channel blockers†	Amiodarone, sotalol
Sinus-node reentrant	Calcium-channel blockers, beta-blockers, digoxin	Flecainide, propafenone*† Quinidine, procainamide, disopyramide‡‡	—
Unifocal atrial Reentrant	Flecainide, propafenone* Quinidine, procainamide, disopyramide‡‡	Amiodarone, sotalol	—
Automatic	Beta-blockers, calcium-channel blockers, digoxin	Flecainide, moricizine†	Amiodarone
Multifocal atrial	Magnesium and potassium supplements	Metoprolol, verapamil†	—

\*For patients with no associated heart disease.

†To be used in combination with a first-choice drug.

‡‡For patients with associated heart disease.

atrioventricular nodal conduction should not be used alone in patients with short accessory-pathway refractory periods and rapid ventricular rates during atrial fibrillation.<sup>57,77</sup> In such patients one should rely on a class I antiarrhythmic agent that lengthens the refractory period of the accessory pathway and reduces ventricular rates during atrial fibrillation (Table 2). The class I drugs may be used alone or in combination with an atrioventricular nodal blocker. Sotalol<sup>78,79</sup> and amiodarone,<sup>80-82</sup> although not approved by the Food and Drug Administration for use in patients with the Wolff–Parkinson–White syndrome, have been used in high-risk symptomatic patients, but the risk of ventricular proarrhythmia (with sotalol) or intolerable side effects (with amiodarone) limits their usefulness.

In cases of unifocal atrial tachycardias due to reentry, class IA, IC, and III drugs have been used, although prospective, placebo-controlled trials demonstrating their efficacy with oral therapy are lacking (Table 2). Atrial tachycardias due to abnormal automaticity or triggered activity are particularly difficult to abolish with antiarrhythmic drugs. Flecainide,<sup>83</sup> moricizine,<sup>84</sup> verapamil,<sup>85</sup> sotalol,<sup>86</sup> and amiodarone<sup>87</sup> are only rarely effective. Often the most easily achievable goal is to block atrioventricular nodal conduction, thereby reducing the ventricular rate despite the persistence of tachycardia at the atrial level. Patients frequently require surgical<sup>88</sup> or catheter<sup>89</sup> ablation of the abnormal atrial focus to prevent tachycardia-related cardiomyopathy. The management of multifocal atrial tachycardia begins with the correction of electrolyte abnormalities<sup>90</sup> and improvement of the underlying metabolic and pulmonary derangements that typically precipitate this arrhythmia. Metoprolol<sup>91,92</sup> may be effective in some cases. In patients without severe left ventricular dysfunction, verapamil<sup>92-94</sup> can be used to reduce atrial firing rates as well as to slow the ventricular rate.

#### CATHETER ABLATION

In 1968 the first successful surgical ablation of an accessory pathway and the resulting cure of a patient

with the Wolff–Parkinson–White syndrome were reported.<sup>95</sup> This landmark case and the remarkable success of surgical treatment of tachycardias mediated by accessory pathways and other supraventricular tachycardias<sup>96-98</sup> sparked interest in the development of catheter-based techniques for the nonsurgical cure of patients with supraventricular tachycardia. A variety of techniques for catheter ablation have been used clinically or are under development.<sup>99,100</sup> Ablation with radiofrequency energy has emerged as the method of choice.

In patients with manifest preexcitation and tachycardia mediated by accessory pathways, radiofrequency ablation of the accessory pathways results in the prompt disappearance of the delta wave and prevents further episodes of tachycardia (Fig. 4). In experienced laboratories, radiofrequency ablation of accessory pathways can be accomplished successfully in at least 90 percent of patients,<sup>101-109</sup> thus rendering surgical treatment of tachycardia mediated by such pathways virtually obsolete. In approximately 10 percent of patients, accessory-pathway conduction will return within two months after apparently successful ablation, requiring a second procedure.<sup>108,110</sup> In procedures performed under the supervision of experienced electrophysiologists in tertiary care centers, procedure-related complications of radiofrequency ablation have been rare (Table 3). Complete atrioventricular block requiring a permanent pacemaker may occur in 0.3 percent of patients, all of whom have septal accessory pathways.

Radiofrequency ablation has also been used to cure patients with atrioventricular nodal reentrant tachycardia.<sup>6,102,108,109,111-116</sup> Ablation of the fast pathway interrupts the retrograde limb of the reentry circuit in the common form of atrioventricular nodal reentrant tachycardia, whereas ablation of the slow pathway interrupts the anterograde limb of the reentry circuit (Fig. 5). Successful ablation can be achieved in 90 percent or more of patients with either technique.<sup>108,109</sup> In 5 to 10 percent of patients, tachycardia recurs after an apparently successful procedure, requiring a second ablation procedure.<sup>108</sup> Like ablation of tachycardia mediated by

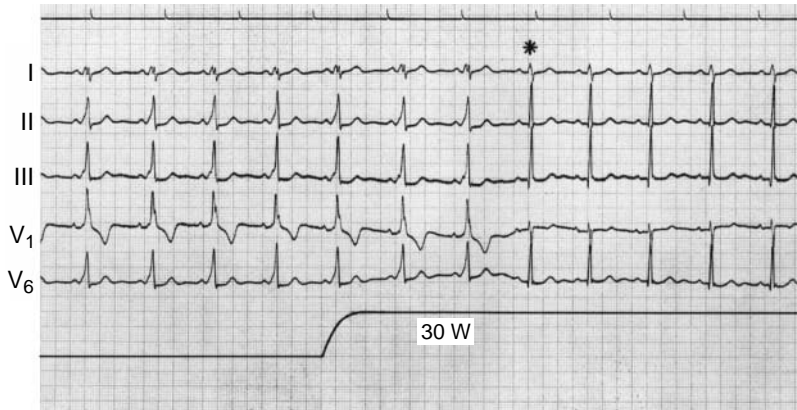


Figure 4. Radiofrequency Ablation of the Accessory Pathway in a Patient with the Wolff–Parkinson–White Syndrome.

The top tracing shows one-second intervals. Three seconds after the onset of radiofrequency current, the delta wave suddenly disappears and the PR interval lengthens to normal (asterisk), indicating successful ablation of the accessory pathway.

accessory pathways, ablation of atrioventricular nodal reentry has been accompanied by relatively few serious complications, although the incidence of inadvertent complete atrioventricular block necessitating the implantation of a permanent pacemaker increases slightly (Table 3). A recent study in which ablation of the fast or slow pathway was performed in patients assigned to one of the treatments in random fashion suggested no significant difference in the incidence of complete atrioventricular block between the two approaches.<sup>117</sup> In the most recent and largest series reported,<sup>108</sup> the incidence of complete atrioventricular block was only 1.2 percent, a figure much lower than in earlier reports. This suggests that experience on the part of the operator may help to reduce the incidence of this complication.

Sinus-node reentrant tachycardia<sup>32,118</sup> and atrial tachycardia<sup>89,108,118-123</sup> have also been the targets of ablation, although there is much less published experience with these arrhythmias, probably because of their relative rarity. Although success rates for radiofrequency ablation of unifocal atrial tachycardia have ranged from 70 to 90 percent in most series, all have been small series, and follow-up has been relatively brief. Success rates are lower when the atrial tachycardia arises from two distinctly different sites.<sup>120</sup> With more than two atrial sites, the results are very poor. Radiofrequency ablation is therefore not an appropriate treatment for patients with multifocal atrial tachycardia. However, if atrial or ventricular rates cannot be controlled pharmacologically in these patients, radiofrequency ablation of the atrioventricular node followed by implantation of a permanent pacemaker should be considered.

Radiofrequency ablation can usually be performed with only a brief hospitalization or even on an outpatient basis,<sup>124,125</sup> causes little discomfort, and is probably more cost effective than drug therapy, at least for patients with frequent, symptomatic episodes of su-

praventricular tachycardia.<sup>126</sup> Because ablation is such a new therapy, data on long-term follow-up are not yet available. However, intermediate-term follow-up, including coronary angiography and electrophysiologic testing, suggests that there is no long-term damage to the coronary arteries and that the radiofrequency lesions are not arrhythmogenic.<sup>127</sup> Except in unusual cases, most experienced laboratories require less than 30 minutes of fluoroscopy time to ablate a single accessory pathway or modify the atrioventricular node. This degree of exposure to radiation probably poses a lifetime risk of fatal cancer of less than 0.1 percent and a risk of genetic defects in fewer than 5 to 20 per million births.<sup>128</sup>

The risk of death as a direct consequence of an ablation procedure is extremely difficult to estimate. To date, more than 5000 patients with supraventricular tachycardia managed by radiofrequency ablation have been reported in the literature. Only seven patients were thought to have died as a direct consequence of the ablation,<sup>129-132</sup> suggesting a procedure-related mortality rate on the order of 0.1 percent. Since published data come from large centers with the most experience and the best outcomes, this estimate of the risk of death from radiofrequency ablation should be viewed as a minimum. Similarly, published estimates of other serious complications, such as arterial embolization dur-

Table 3. Results of Radiofrequency Ablation in Patients with Supraventricular Tachycardia.\*

VARIABLE	ATRIOVENTRICULAR NODAL REENTRANT TACHYCARDIA (N = 560)	ACCESSORY-PATHWAY-MEDIATED SUPRAVENTRICULAR TACHYCARDIA (N = 1150)
Success of procedure — %	92	93
Complications — no. (%)		
Tamponade or effusion	1 (0.2)	5 (0.4)
Atrioventricular block	21 (3.8)	4 (0.3)
Thromboembolic effects — no. (%)		3 (0.3)
Atrial thrombus	—	1 (0.1)
Pulmonary embolus	1 (0.2)	1 (0.1)
Microembolus to foot	—	1 (0.1)
Transient ischemic attack	—	1 (0.1)
Vascular thrombosis	1 (0.2)	—
Local hematoma — no. (%)	2 (0.4)	3 (0.3)
Femoral-artery pseudoaneurysm — no. (%)	—	2 (0.2)
Aortic-valve perforation — no. (%)	—	2 (0.2)
Coronary-artery spasm — no. (%)	—	1 (0.1)
Coronary-artery occlusion — no. (%)	—	1 (0.1)
Pneumothorax — no. (%)	—	1 (0.1)

\*Data are from Kay et al.<sup>108</sup> and Scheinman<sup>109</sup> and were compiled from the six North American centers with the largest published clinical experience.

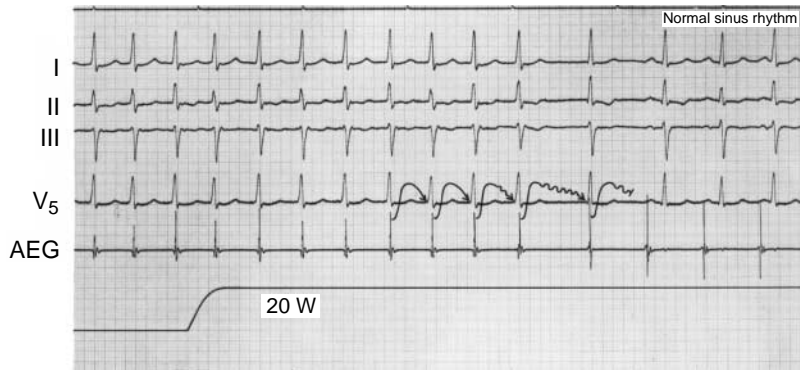


Figure 5. Radiofrequency Ablation of the Slow Atrioventricular Nodal Pathway during Atrioventricular Nodal Reentrant Tachycardia.

AEG denotes the intracardiac bipolar high right atrial electrogram, on which retrograde P waves and QRS complexes are registered simultaneously during atrioventricular nodal reentrant tachycardia. When radiofrequency current is turned on there is progressive delay (arrows) of anterograde atrioventricular nodal conduction and finally anterograde block in the slow pathway, resulting in the restoration of sinus rhythm. Note that retrograde conduction time over the fast atrioventricular nodal pathway during atrioventricular nodal reentrant tachycardia is unaffected while radiofrequency current is being applied.

ing the ablation of left-sided accessory pathways, should also be viewed as minimums. In order to ensure the best possible outcome with minimal risk, ablation should be performed only in carefully selected tertiary care centers with particular expertise in catheter ablation and a proved record of safety.

#### RECOMMENDATIONS FOR MANAGEMENT

Patients with atrioventricular nodal reentry who have come to medical attention because of single episodes of well-tolerated tachycardia might best be treated conservatively with observation alone, without drugs or ablation therapy. Such patients sometimes have arrhythmia-free intervals of many years. However, if patients have frequent episodes of tachycardia or if the first episode causes severe symptoms, long-term therapy with a calcium-channel blocker, a beta-blocker, or digoxin is appropriate. Such drugs can be used singly or in combination. If atrioventricular nodal blockers prove ineffective or produce intolerable side effects, we recommend radiofrequency catheter ablation rather than treatment with a class I or III antiarrhythmic drug as the next therapeutic step. Even in young patients the known risks of ablation, including the risk of needing a permanent pacemaker after such a procedure, are probably preferable to the cumulative risks of antiarrhythmic-drug use over many years of therapy.

We believe that radiofrequency ablation should be the treatment of choice for patients with the Wolff-Parkinson-White syndrome and symptomatic atrioventricular reentrant tachycardia. Calcium-channel blockers or digitalis are not appropriate as sole therapy for most such patients, beta-blockers are often poorly tolerated or ineffective, and the hazards of long-term treatment with a class I or III antiarrhythmic drug are probably greater than the risks of ablation. Since the risk of atrioventricular block after ablation of an acces-

sory pathway is very remote, the same recommendation can also be made for patients with concealed accessory pathways and symptomatic atrioventricular reentry, although an initial trial of an atrioventricular nodal blocker is reasonable. A particular challenge is the treatment of completely asymptomatic patients with the Wolff-Parkinson-White syndrome and no prior episode of tachycardia. Most studies point to an incidence of sudden death in such patients of about 0.1 percent per patient-year.<sup>133-137</sup> In many patients with electrocardiographic evidence of ventricular preexcitation early in life, anterograde conduction block develops over a period of years in the accessory pathway without any medical intervention whatsoever.<sup>138</sup>

Thus, neither electrophysiologic testing nor ablation is recommended in most asymptomatic persons. However, intervention may be appropriate for certain carefully selected asymptomatic persons in high-risk occupations, such as airline pilots, bus drivers, police officers, and firefighters, who may be prohibited from pursuing their chosen occupations because of the finding of ventricular preexcitation, and for young people who want to participate in highly competitive athletic activities. In such patients we frequently recommend electrophysiologic testing to determine the number of accessory pathways, their anatomical position, and the risk of life-threatening arrhythmia. Ablation can be recommended when this risk is high and the accessory pathway is in an easily accessible position, such as the left free wall, where the risk of causing atrioventricular block is negligible.

The question of how to treat patients with unifocal atrial tachycardia is still very much unsettled. In most patients a trial of a calcium-channel blocker or beta-blocker may be a reasonable first choice, although the likelihood that such therapy will abolish the tachycardia is remote. In patients who cannot tolerate or fail to respond to these agents, radiofrequency ablation offers an alternative to class I or III antiarrhythmic-drug therapy. As methods of localizing arrhythmogenic foci improve, catheter ablation will probably become the treatment of choice for most patients with atrial tachycardia as well as patients with the more common varieties of supraventricular tachycardia.

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