V SUPRAVENTRICULAR TACHYCARDIA

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Over the past decade, enormous strides have been made in the treatment of patients with supraventricular tachycardia (SVT). Although acute therapy for SVT continues to require drugs or cardioversion, advances in the understanding of the mechanisms of SVT have led to the development of catheter ablation procedures for most forms of SVT.[1] These procedures often cure the condition, freeing the patient from the need for lifelong drug therapy. This chapter focuses on the most common forms of SVT—excluding atrial fibrillation, which is discussed in detail elsewhere [*see 1:IV Atrial Fibrillation*].

Classification

SVT is often paroxysmal (PSVT). Clinically, PSVT is marked by palpitations, occurring in episodes that start and end abruptly. During these episodes, the 12-lead ECG shows a heart rate greater than 100 beats/min and, typically, narrow QRS complexes. For almost all patients with PSVT, the underlying mechanism of the tachycardia is atrioventricular node reentry (AVNRT), reentry involving an accessory pathway (AVRT), or atrial tachycardia. AVNRT and AVRT are the most common and the second most common causes of PSVT, respectively. Atrial flutter also presents as a rapid regular tachycardia, but this arrhythmia usually does not begin and end abruptly.

The clinician has a variety of tools to distinguish the various mechanisms of SVT [*see Figure 1*]. The use of carotid massage[2] or intravenous adenosine[3] [*see Figure 2*] may be diagnostic, therapeutic, or both. If vagal maneuvers terminate the arrhythmia acutely or produce no effect, the patient probably has AVNRT or AVRT. In patients with atrial tachycardia, these maneuvers will frequently result in transient AV block. Perpetuation of the arrhythmia in the face of AV block strongly suggests atrial tachycardia or atrial flutter.[3] Intravenous adenosine will almost always terminate tachycardia from AVNRT or AVRT, but focal atrial tachycardia may also terminate abruptly after adenosine. Hence, the use of adenosine does not reliably distinguish those disorders from atrial tachycardia unless it produces AV block.[3]

Paying careful attention to the relationship between the P wave and the QRS complex during tachycardia is also very helpful in distinguishing tachycardia mechanisms[4] [*see Figure 1*]. If the retrograde P wave falls within or just after the QRS, the most likely diagnosis is AVNRT [*see Figure 3*]. If the tachycardia shows a retrograde P wave in the ST segment [*see Figure 4*], AVRT is

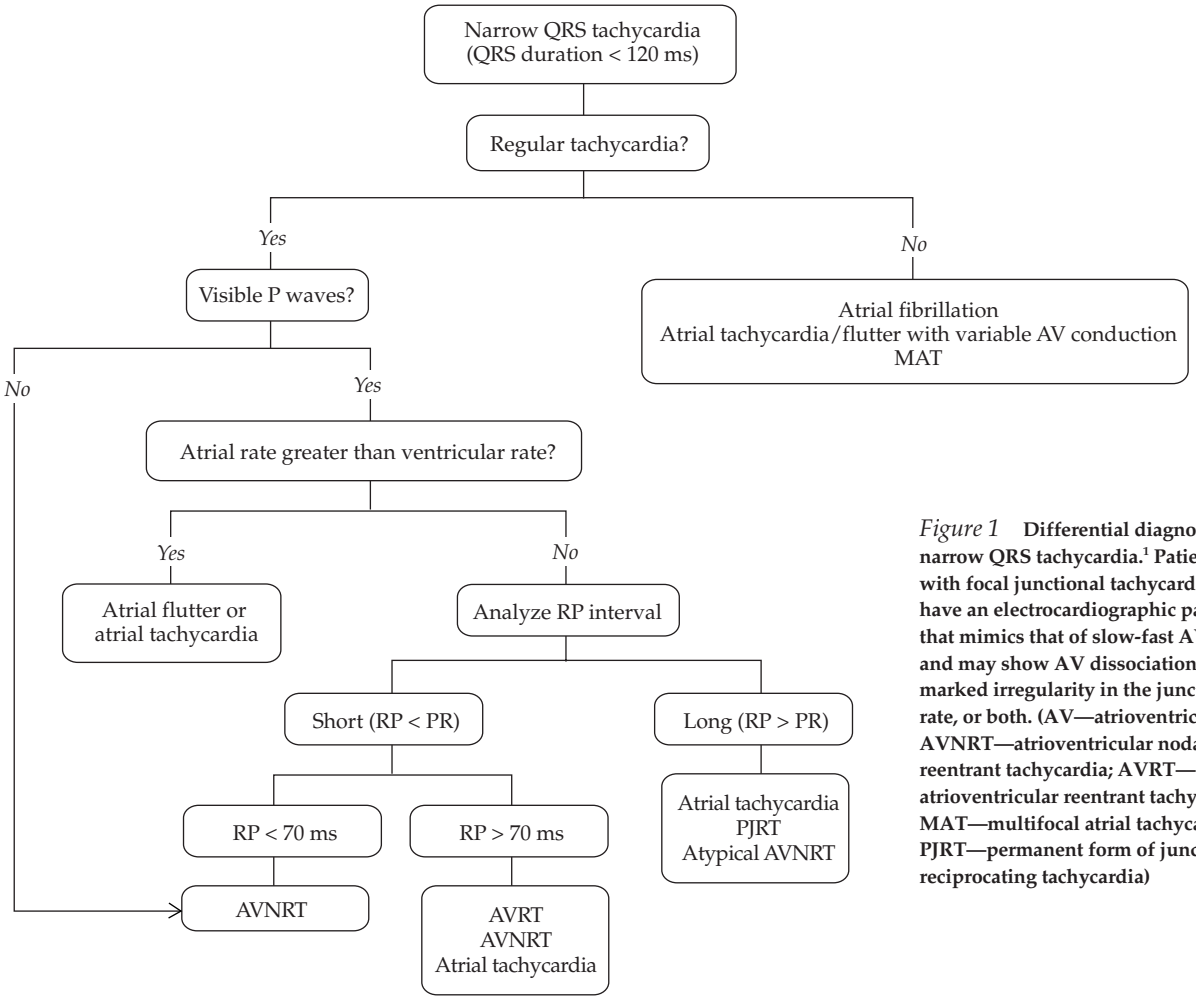

Figure 1 **Differential diagnosis for narrow QRS tachycardia.[1] Patients with focal junctional tachycardia may have an electrocardiographic pattern that mimics that of slow-fast AVNRT and may show AV dissociation, marked irregularity in the junctional rate, or both. (AV—atrioventricular; AVNRT—atrioventricular nodal reentrant tachycardia; AVRT—atrioventricular reentrant tachycardia; MAT—multifocal atrial tachycardia; PJRT—permanent form of junctional reciprocating tachycardia)**

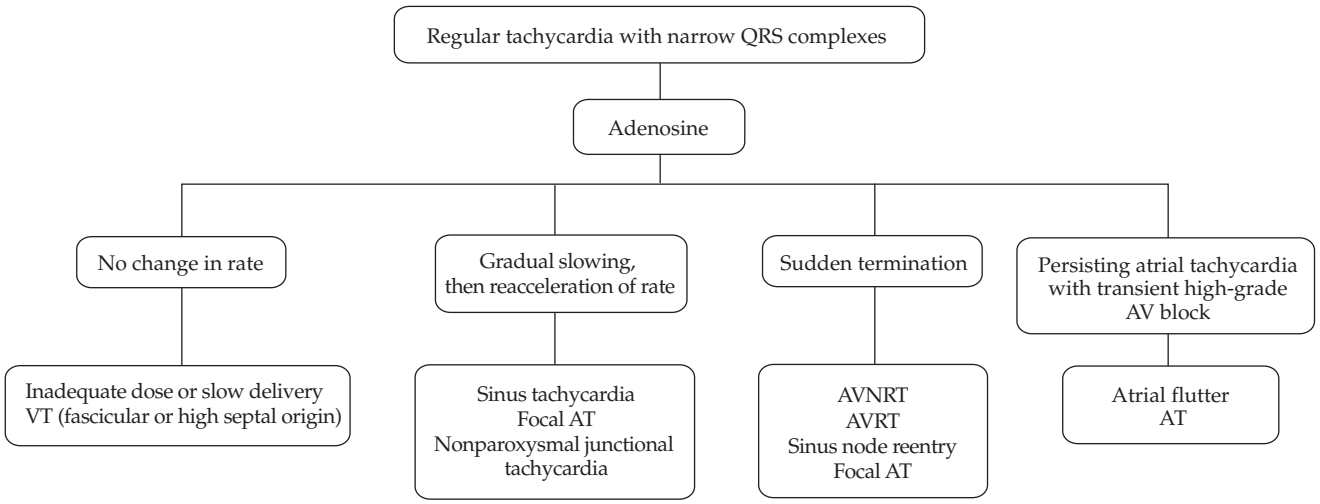

Figure 2 **The response to intravenous adenosine can be useful in determining the cause of tachycardia.[1] (AT—atrial tachycardia; AV—atrioventricular; AVNRT—atrioventricular nodal reentrant tachycardia; AVRT—atrioventricular reentrant tachycardia; VT—ventricular tachycardia)**

most likely. Finally, atrial tachycardia is characterized by the presence of P waves immediately in front of the QRS (long RP tachycardia) [*see Figure 5*].

Although the QRS complex is usually narrow in SVT, it may be broad (> 120 ms) in patients who have either bundle branch block or aberrant conduction. A number of ECG findings have been found very helpful in distinguishing SVT with a broad QRS complex from ventricular tachycardia (VT).[5,6] For example, AV dissociation (i.e., independent atrial activity during tachycardia), fusion beats, or capture beats prove the presence of VT. Unfortunately, AV dissociation is not apparent in 80% to 85% of patients with rapid VT, because the P wave is obscured by the QRS complex and T waves.[6] In this setting, morphologic criteria may be very helpful in distinguishing SVT from VT.

Use of morphologic criteria begins with careful attention to the precordial leads [*see Figure 6*]. Any of the following features in the precordial tracings will favor the diagnosis of VT: (1) Concordance of all the precordial leads (i.e., all are positive or all are negative); (2) absence of an initial positive deflection (r wave) followed by a negative deflection (s wave; recall that in ECG nomenclature, upper-case letters denote dominance; small waves are designated by lowercase letters); (3) an r/s pattern is present but the time from the initial r to the nadir of the s wave is greater than 60 ms; (4) presence of a right bundle branch pattern in lead V1, with an r greater than s or a qr pattern, where q indicates the initial negative deflection; (5) presence of a left bundle branch pattern in V1, with a broad r wave (> 30 ms) or an interval of greater than 60 ms from the onset of the r wave to the nadir of the s wave; (6) extreme left axis deviation; or (7) very broad QRS complexes (> 160 ms).

Atrioventricular Nodal Reentry Tachycardia

PATHOGENESIS

Normally, sinus impulses are discharged into the surrounding atria and directed to the region of the node that resides in the atrial septum. The AV nodal impulses then propagate through the ventricles over the His-Purkinje system. The normal AV node has a single transmission pathway. In two to three persons

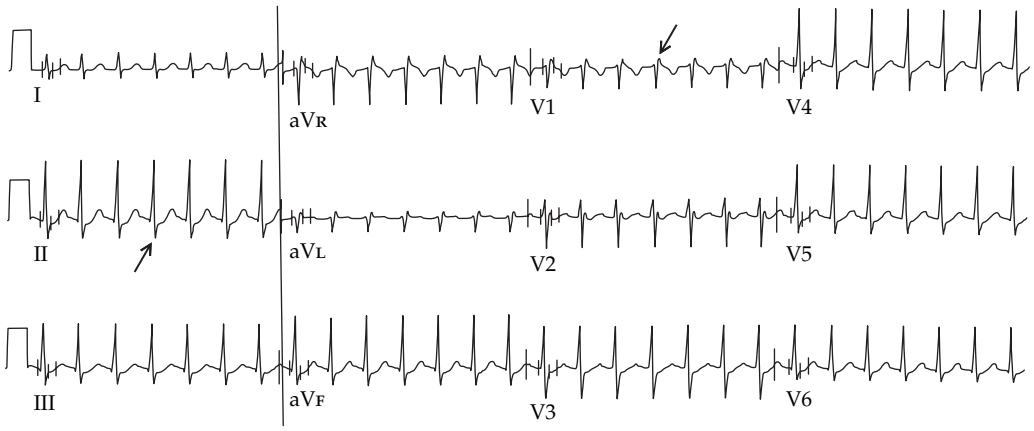

Figure 3 **A 12-lead ECG shows paroxysmal supraventricular tachycardia from AV nodal reentry (AVNRT). The arrows point to a pseudo r[1] in lead V1 and S waves in the inferior leads (II, III, and aVF), which disappeared with conversion to sinus rhythm.**

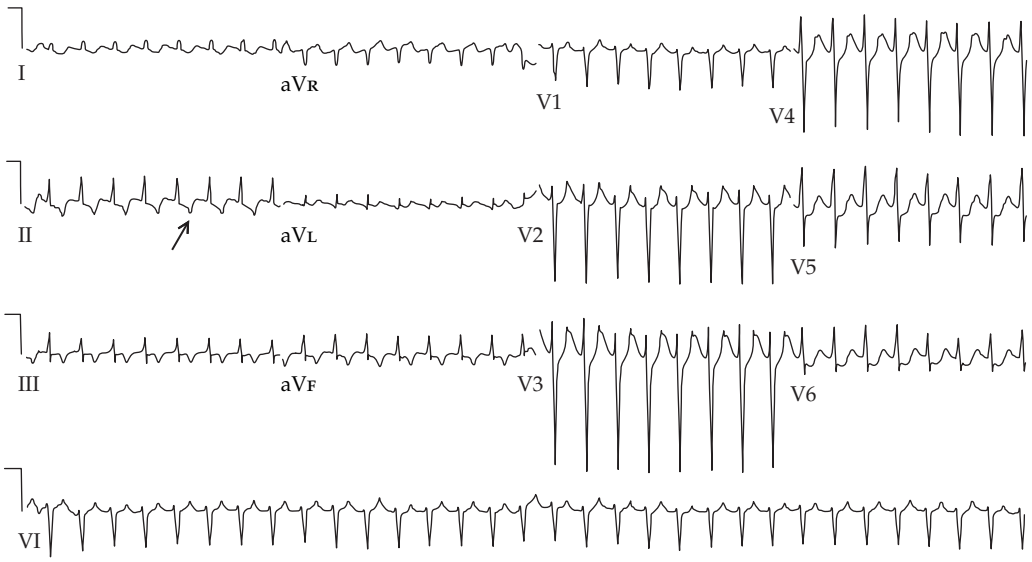

Figure 4 **A 12-lead ECG showing narrow complex tachycardia with P waves inscribed well after the QRS, taken from a patient who had paroxysmal supraventricular tachycardia supported by an accessory pathway.**

per 1,000 population, however, the AV node has both a normal (fast) pathway and a second, slow pathway.[7,8] In such persons, the sinus impulse is ordinarily transmitted over the fast pathway to the ventricle, and slow pathway conduction is preempted. However, if an atrial premature complex (APC) occurs at a critical point in the conduction cycle, the impulse can block in the fast pathway, thus allowing for anterograde (forward) conduction over the slow pathway and retrograde (backward) conduction over the fast pathway [*see Figure 7*]. The latter situation may produce a single echo beat (a beat that returns to the chamber of origin) or stabilize into a circus-movement tachycardia.

DIAGNOSIS

The diagnosis of AVNRT can usually be made by careful analysis of the 12-lead ECG.[4] Because retrograde conduction over the AV node is occurring more or less simultaneously with anterograde conduction to the ventricles, the P wave is either buried within the QRS complex or inscribed just after the QRS. The P wave inscribed by retroconduction over the AV node will be negative in the inferior leads and positive in lead V1; therefore, PSVT from AVNRT may manifest as small negative deflections in the inferior leads and a small positive deflection in V1—the so-called pseudo r[1] pattern[5] [*see Figure 3*].

MANAGEMENT

Acute Therapy

AVNRT may respond to carotid sinus massage[2] but is highly responsive to intravenous adenosine,[3] beta blockers,[9] or calcium channel blockers[10] [*see Table 1*].

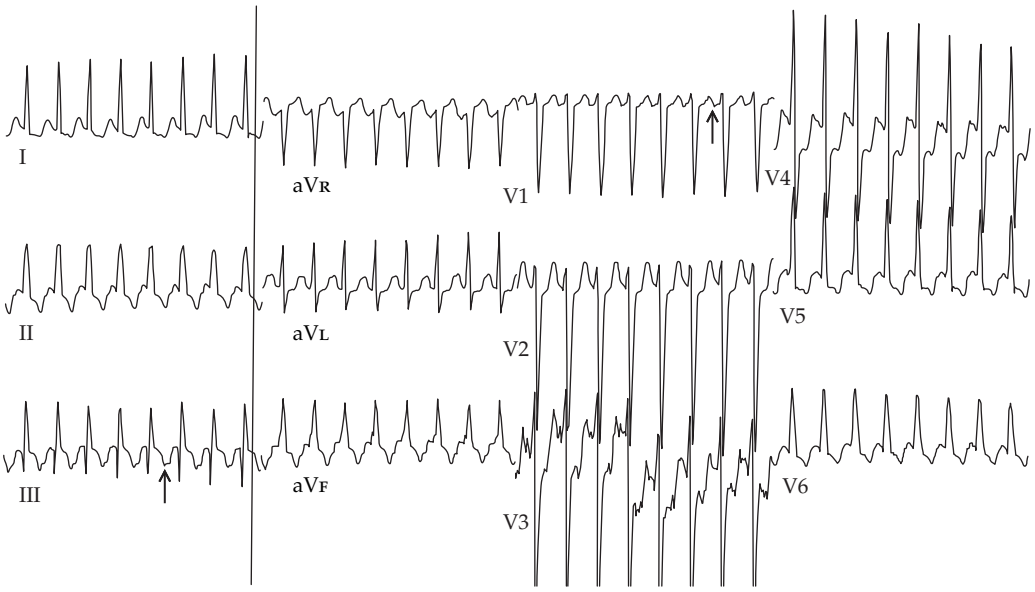

Figure 5 **A 12-lead ECG shows tachycardia with P waves (arrow) just preceding the QRS complex. The patient in this case had a focal atrial tachycardia emanating from the lateral tricuspid annulus.**

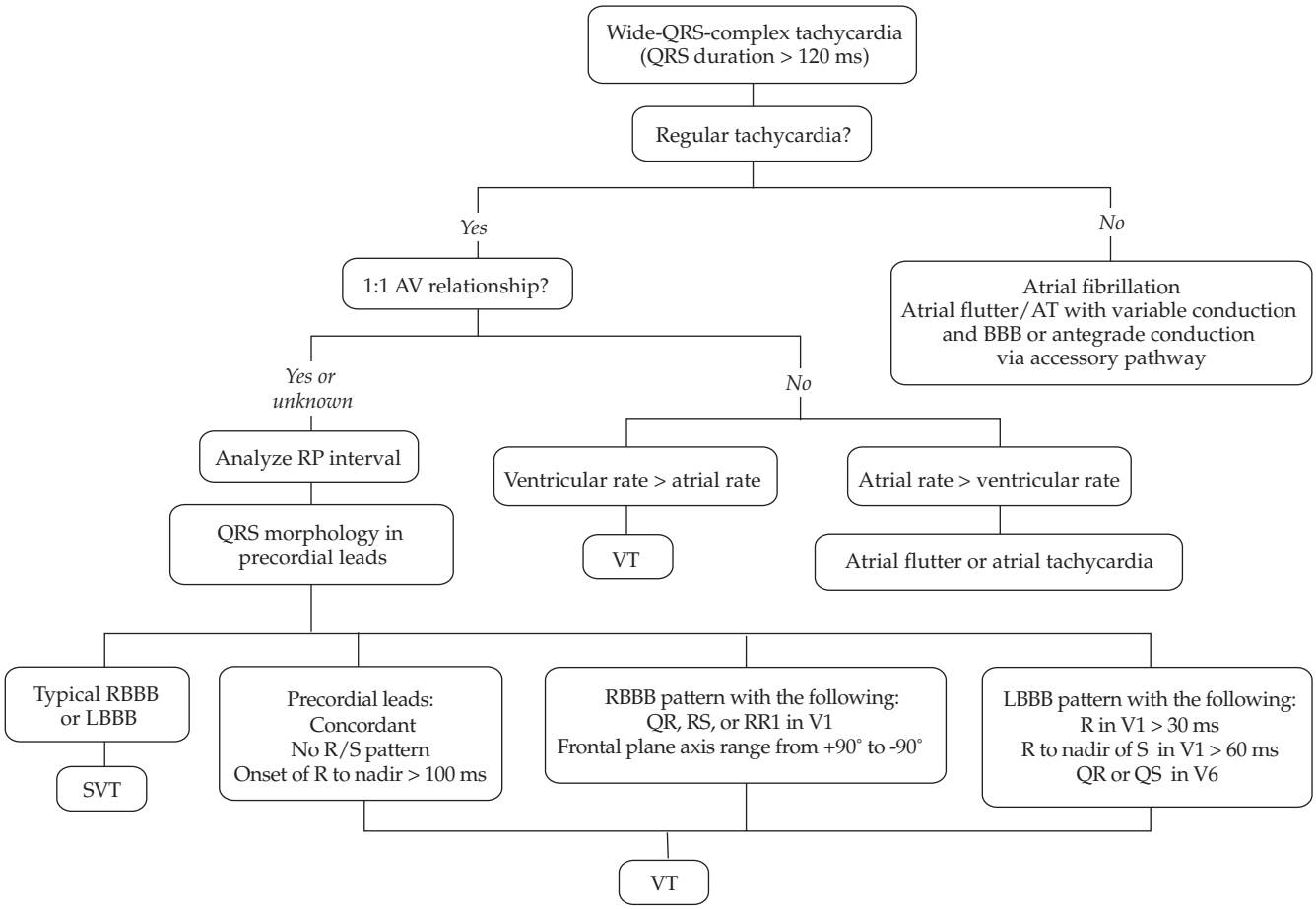

Figure 6 **Differential diagnosis for wide (> 120 ms) QRS complex tachycardia.[1] If the tachycardia is regular and comparison with a baseline electrocardiogram shows that the QRS complex is identical to that during sinus rhythm, the patient may have supraventricular tachycardia (SVT) with bundle branch block (BBB) or antidromic atrioventricular reentrant tachycardia (AVRT). If the patient has a history of myocardial infarction or has structural heart disease, ventricular tachycardia (VT) is likely. Vagal maneuvers or adenosine may convert regular tachycardia, although adenosine should be used with caution when the diagnosis is unclear, because this drug may produce ventricular fibrillation (VF) in patients with coronary artery disease and patients with alternative pathways who have atrial fibrillation with a rapid ventricular rate. Precordial leads are concordant when all show either positive or negative deflections. Fusion complexes are diagnostic of VT. In preexcited tachycardias, the QRS is generally wider (i.e., more preexcited) than during sinus rhythm. (AT—atrial tachycardia; AV—atrioventricular; LBBB—left bundle branch block; RBBB—right bundle branch block)**

Adenosine If carotid massage fails to convert SVT, the drug of choice is intravenous adenosine, which is effective in 95% of cases.[10,11] The initial dose is given as a rapid bolus infusion of 6 mg, followed by 12 mg and finally 18 mg if necessary. The bolus must be given rapidly and then followed by a saline flush. If administration is too slow, the adenosine may be metabolized before it reaches the AV node. Possible adverse effects include headache, wheezing, and flushing. These effects disappear within 45 to 60 seconds. It is important to note that atrial, ventricular, and junctional premature beats are commonly observed after adenosine. In 3% to 5% of cases, the APCs trigger atrial fibrillation,[3] which may result in serious problems for patients with accessory pathways (see below). If possible, an external defibrillator should be readily available when adenosine is administered.

The most common reason for failure to respond to adenosine is that multiple premature beats are retriggering the tachycardia. In this setting, a longer-acting intravenous preparation (i.e., 5 mg of metoprolol or 0.1 mg/kg of verapamil) is indicated. Agents that more selectively block purogenic receptors have been

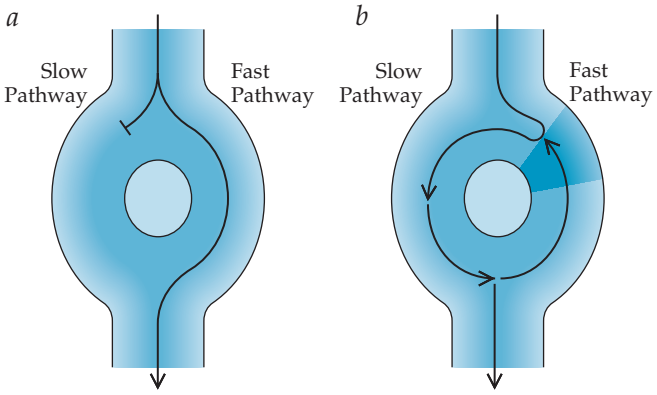

Figure 7 **In persons with dual pathways in the AV node, the sinus impulse is normally transmitted over the fast pathway to the ventricle and slow pathway conduction is preempted (*a*). However, if an atrial premature complex occurs during the fast pathway's refractory period, the impulse can block in the fast pathway. This may allow for anterograde (forward) conduction over the slow pathway and retrograde (backward) conduction over the fast pathway (*b*).**

Table 1 Drugs Used to Maintain Sinus Rhythm in Patients with Supraventricular Tachycardia[55]

Drug	*Typical Daily Dose*	*Potential Adverse Effects*
Amiodarone	100–400 mg*	Photosensitivity, pulmonary toxicity, polyneuropathy, GI upset, bradycardia, torsade de pointes (rare), hepatic toxicity, thyroid dysfunction
Disopyramide	400–750 mg	Torsade de pointes, heart failure, glaucoma, urinary retention, dry mouth
Dofetilide	500–1,000 μg	Torsade de pointes
Flecainide	200–300 mg	Ventricular tachycardia, heart failure, enchanced AV nodal conduction (conversion to atrial flutter)
Procainamide	1,000–4,000 mg	Torsade de pointes, lupuslike syndrome, GI symptoms
Propafenone	450–900 mg	Ventricular tachycardia, heart failure, enhanced AV nodal conduction (conversion to atrial flutter)
Quinidine	600–1,500 mg	Torsade de pointes, GI upset, enhanced AV nodal conduction
Sotalol	240–320 mg†	Torsade de pointes, heart failure, bradycardia, exacerbation of chronic obstructive or bronchospastic lung disease

*A loading dose of 600 mg/day is usually given for 1 month, or a dose of 1,000 mg/day is given for 1 week.
†Adjust dose for renal function and QT-interval response during in-hospital initiation phase
AV—atrioventricular GI—gastrointestinal

shown to be very effective and associated with fewer side effects than older agents. Selective purogenic blockers are currently under investigation.

Long-term Therapy

A wide variety of drugs have proved effective for controlling episodes of AVNRT, including beta blockers,[9] calcium channel blockers,[12] and digoxin[13] [*see Table 1*]. Long-term drug therapy is associated with frequent recurrences and adverse effects, however. In patients without structural cardiac disease, class IC antiarrhythmic agents (e.g., flecainide, propafenone) are more effective than drugs that act by blocking AV nodal conduction, but recurrence rates nevertheless range from 25% to 35%.[14-16] For patients who have episodes infrequently and tolerate them well, some cardiologists will prescribe medication for use as needed—the "pill in the pocket" approach. For example, single-dose diltiazem (120 mg) and propranolol (80 mg) have been shown to be more effective than placebo or flecainide in patients with PSVT.[17]

Catheter Ablation

Current catheter ablative techniques involve placement of an electrode catheter between the tricuspid annulus and coronary sinus in the so-called slow pathway region.[18] One or more applications of radiofrequency energy are delivered through the catheter to destroy or attenuate the slow pathway. The success rate of ablation is over 96%, and the only significant complication is AV block, which occurs in approximately 1% of patients.[19]

Catheter ablation for AVNRT has proved so safe and effective that it is clearly the procedure of choice for patients in whom drug therapy fails. Moreover, it can be offered to those with milder symptoms who prefer to avoid long-term drug therapy. Precise recommendations for drug therapy versus ablative therapy are provided in the American College of Cardiology/American Heart Association/European Society of Cardiology guidelines.[1]

Atrioventricular Reentry Tachycardia

PATHOGENESIS

The normal conduction system of the heart limits the propagation of electrical impulses from the atria to a single pathway through the AV node and the His-Purkinje system. This limitation delays ventricular activation and thus optimizes mechanical function. The presence of an alternative pathway of atrioventricular conduction creates the potential for reentrant tachycardia.

The most prominent manifestation of accessory atrioventricular pathways is the Wolff-Parkinson-White (WPW) syndrome. In this syndrome, the accessory pathway can be located at various regions around the tricuspid and the mitral atrioventricular rings, but it is most commonly sited at the left free wall of the mitral annulus. The next most common pathway sites are the posteroseptal and right free wall areas. Pathways in the anteroseptal and the midseptal regions are relatively rare. Occasionally, posteroseptal pathways can be associated with a branching vein from the coronary sinus. On occasion, a patient will have more than one accessory pathway.

The basic mechanism of tachycardia in AVRT is similar to that of AVNRT. Electrical impulses can travel down both the AV node and the accessory pathway to activate the ventricles, with ventricular activation occurring earlier at sites near the accessory pathway than at sites activated normally (i.e., ventricular preexcitation). An APC may block in the accessory pathway but conduct over the normal pathway to activate the ventricle. After ventricular depolarization, the impulse may return to the atrium via retrograde conduction over the accessory pathway, leading to a sustained tachycardia.[20]

The most feared arrhythmia in the WPW syndrome involves atrial fibrillation with dominant conduction over an accessory pathway that has rapid conduction properties[21,22] [*see Figure 8*]. These patients may experience extraordinarily rapid ventricular rates and are at risk for sudden cardiac death from ventricular fibrillation.[23] In one large series, atrial fibrillation developed in 30% of patients with the WPW syndrome.[24]

DIAGNOSIS

Clinical Presentation

Symptomatic tachyarrhythmias associated with the WPW syndrome generally begin in the teenage years or during early adulthood. Pregnancy may produce an initial attack in some women. Pregnancy can also be associated with an increasing frequency of attacks and more symptomatic episodes. Symptoms are generally paroxysmal palpitations with or without dizziness, syncope, shortness of breath, weakness, or chest pain. Diuresis is another frequently described symptom; it occurs 30 minutes to an hour after onset of tachycardia and may be related to production of atrial natriuretic factor during the arrhythmia.

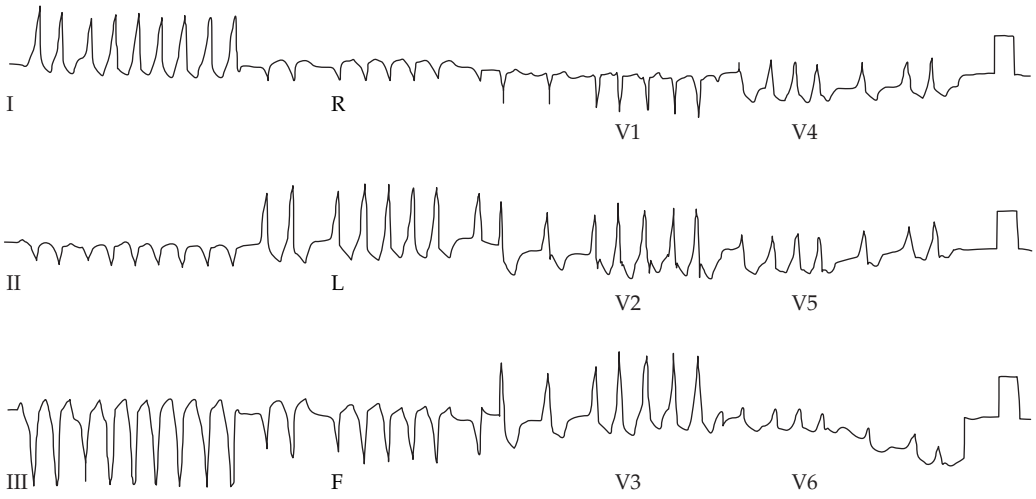

Figure 8 **A 12-lead ECG in a patient with Wolff-Parkinson-White syndrome shows the rapid, irregularly irregular ventricular rate and wide QRS complexes of atrial fibrillation with a very short refractory period. This is an especially dangerous arrhythmia.**

Electrocardiographic Findings

Ventricular preexcitation may be evident on a baseline ECG as fusion complexes (WPW pattern). The WPW pattern comprises a short PR interval and an earlier than normal deflection on the QRS complex (delta wave).[25] The ECG during AVRT will usually show a narrow complex with the retrograde P wave falling in the ST segment because atrial activation occurs well after ventricular depolarization[4] [*see Figure 4*]. Of interest is that a subset of patients with AVRT never show manifest anterograde conduction over the accessory pathway yet still have this form of tachycardia.[20] The only evidence that the tachycardia is supported by an accessory pathway is that the retrograde P wave clearly occurs after the QRS during tachycardia. On rare occasions, patients may have slowly conducting retrograde pathways[26]; their ECG will show a long RP–short PR relationship. These patients tend to have persistent tachycardias that have been referred to as the permanent form of junctional tachycardia (PJRT). In addition, approximately 5% of patients with the WPW syndrome (WPW pattern and arrhythmias) will show anterograde conduction over the accessory pathway with retrograde conduction through the AV node or over a separate accessory pathway. The ECG in these patients will show a wide-complex tachycardia with retrograde P waves preceding the QRS complex.

MANAGEMENT

Acute Therapy

Acute management of AVRT is similar to that for AVNRT: adenosine is the drug of choice,[11] but calcium channel blockers[13] or beta blockers[9] are also effective. Again, because adenosine usually provokes APCs and thus may in rare instances precipitate atrial fibrillation,[3] it is advisable to have ready access to an external defibrillator when using this agent.

Long-term Therapy

Long-term therapy for AVRT may be directed at interfering with conduction either through the AV node (i.e., with beta blockers or calcium channel blockers[27]) or through the accessory pathway (i.e., with class IC or class III antiarrhythmic agents[28-32]). Oral digitalis therapy is contraindicated because very rapid ventricular rates may occur if atrial fibrillation develops. Class IC agents appear to be more effective than AV nodal blockers, but their use is restricted to patients who do not have significant cardiac disease. Class III agents (particularly amiodarone) are limited by long-term systemic toxicity and modest efficacy for patients with WPW and atrial fibrillation.[30] In general, drug therapy is attended by a significant risk of arrhythmic recurrence and adverse drug effects.

Treatment of WPW and Atrial Fibrillation

The treatment of WPW and atrial fibrillation is different from the treatment of AVRT. Because atrial fibrillation may precipitate a life-threatening arrhythmia, urgent therapy is required. If the patient presents with hemodynamic collapse, emergency direct current (DC) cardioversion is the first step. If the patient is less ill, trials of intravenous drug therapy are in order.[33] The drug of choice is procainamide, 50 mg/min to a total of 1 g, or ibutilide, 2 mg infused over 15 minutes. Ibutilide is very effective but should be used only in patients without significant structural cardiac disease. Intravenous digoxin or calcium channel blockers may result in an inordinate increase in heart rate and so should be avoided. Beta blockers, lidocaine, and adenosine are not likely to be effective, and their use will tend only to delay effective therapy.

Catheter Ablation

Reports from both single centers[34,35] and multicenter prospective registries[36,37] have documented the efficacy and possible adverse effects of ablative therapy in AVRT. Current techniques allow for successful ablation of accessory pathways that traverse the AV annulus or the anterior or posteroseptal spaces. For pathways over the left AV groove, current ablation techniques involve use of either transseptal or retrograde aortic approaches.[38] The overall success rate for ablation is approximately 95%.

Complications of ablation are primarily related to the site of the accessory pathway. For example, patients with an anteroseptal accessory pathway are at risk for injury to the AV node (5%), whereas ablations of left-sided accessory pathways carry a risk of cerebrovascular accident, myocardial perforation, or coronary artery occlusion.[36,37] The overall incidence of significant adverse

effects varies from 2% to 4%. Death associated with ablative procedures is quite rare, occurring in 0.13% to 0.2% of cases.[36,37,39]

Treatment Selection

The remarkable efficacy and safety of ablation make this mode of therapy more attractive than long-term drug therapy for symptomatic patients. Drug therapy carries the possibility of recurrent arrhythmias, including atrial fibrillation. Hence, ablation is currently recommended for all patients with symptomatic WPW. Patients with mild symptoms and without manifest preexcitation can be managed with drug therapy, but even in these cases ablation would appear to be a favored approach. Some of these patients decline long-term drug therapy, leaving ablation as the only alternative.

Asymptomatic preexcitation The management of asymptomatic preexcitation remains controversial. The vast majority of these patients have an overall good prognosis; sudden cardiac death is a rare initial manifestation. Leitch and colleagues followed asymptomatic WPW subjects in whom atrial fibrillation was induced during invasive electrophysiologic study; although approximately 20% demonstrated the capacity for rapid ventricular conduction, on follow-up few became symptomatic and none died suddenly.[39] A later study, however, emphasized findings on electrophysiologic testing (e.g., inducible AVRT, atrial fibrillation, and multiple pathways) that indicated increased risk for subsequent spontaneous development of atrial fibrillation or even sudden death.[40] Whether to treat an asymptomatic patient can also be decided on an individual basis[1]; for example, patients judged to be in high-risk occupations (e.g., airplane pilots, bus drivers) might well be considered for ablative therapy.

Focal Atrial Tachycardia

Regular tachycardias emanating in an atrial area and showing a centripetal pattern of spread are designated as focal atrial tachycardias (FATs). These arrhythmias are the least common cause of PSVT but nevertheless can cause significant morbidity. This is particularly true if the arrhythmia is incessant, which can result in the development of so-called tachycardia myopathy.

Atrial tachycardia may arise from sites in either the right or left atrium. The most common site of FAT is in the right atrium, with predilection for sites over the crista terminalis, tricuspid annulus, or coronary sinus.[41,42] In the left atrium, FAT is more apt to develop at the ostium of the pulmonary veins or over the mitral annulus.[42]

ELECTROCARDIOGRAPHIC DIAGNOSIS

In patients with FAT, the P wave may appear anywhere in the diastolic cycle but most often appears in front of the QRS (long RP tachycardia) [*see Figure 5*]. The ectopic P wave has a different shape than the sinus P wave unless the tachycardia originates from the high cristal or right pulmonary vein area. The P wave morphology gives excellent clues to tachycardia localization.[43,44] For example, P waves from left atrial foci will show negative deflection in leads I or aVL and positive deflections in the precordial leads. Right atrial foci tend to show negative P waves in lead V1 but positive or biphasic deflection in aVL. As a rule, foci from superior atrial sites generally produce strongly positive P waves in the inferior leads, whereas those arising from the inferior atrium produce negative P waves.

MANAGEMENT

Acute Therapy

Acute treatment of FAT attempts either to convert the arrhythmia or to slow the heart rate. Drugs used to slow the rate are the AV nodal blockers (i.e., digoxin, beta blockers, or calcium channel blockers). In contrast, class IC antiarrhythmic agents (e.g., flecainide, propafenone) or class III agents (e.g., amiodarone or sotalol) may terminate the tachycardia. Intravenous adenosine may be effective in terminating FAT and should be tried early. DC cardioversion may not be effective, particularly if the tachycardia results from an automatic mechanism.

Long-term Therapy

Long-term oral therapy for FAT is not well defined.[1] The general approach is empirical, with initial use of AV nodal blockers followed by class IC or III antiarrhythmic agents if the AV nodal blockers are ineffective.

Catheter Ablation

Catheter ablative procedures have been successfully applied to patients with FAT. Ablation has proved more effective for patients with right atrial foci (in whom the success rate is approximately 90%) than for those with left atrial foci (in whom the success rate is approximately 70%). A study of pooled data that included 514 patients showed an overall success rate of 86%, with an incidence of significant complications from 1% to 2%.[45]

Multifocal Atrial Tachycardia

Multifocal atrial tachycardia, generally regarded as automatic in origin, is characterized by atrial rates of 100 to 130 beats/min, three or more morphologically distinct (nonsinus) P waves, and variable AV conduction. It is commonly associated with respiratory disease and heart failure. Hypoxemia is a frequent finding. The arrhythmia may be exacerbated by digitalis excess, theophylline toxicity, or hypokalemia.

Treatment of multifocal atrial tachycardia is usually directed at the underlying precipitants. Metoprolol (used cautiously in patients with bronchospasm) or verapamil may slow atrial and ventricular rates and, occasionally, may restore sinus rhythm. Potassium and magnesium supplements may help suppress the arrhythmia. Amiodarone has also been useful in restoring sinus rhythm.

Atrial Flutter

Rapid reentrant atrial arrhythmias are referred to as atrial flutter. The most common circuit involves reentry around the tricuspid annulus. The reentrant circuit is usually counterclockwise (in the left anterior oblique projection) but may be clockwise.[46] Other circuits may involve the upper portion of the right atrium.[47] Less commonly, left atrial (LA) circuits are operative. LA circuits may involve the mitral annulus or scars around the posterior LA wall, pulmonary veins, or the foramen ovale.[47]

DIAGNOSIS

Clinical Presentation

Atrial flutter generally occurs in older patients who have associated cardiopulmonary disease. Atrial flutter may appear acutely during acute myocardial infarction, after cardiac surgery, or

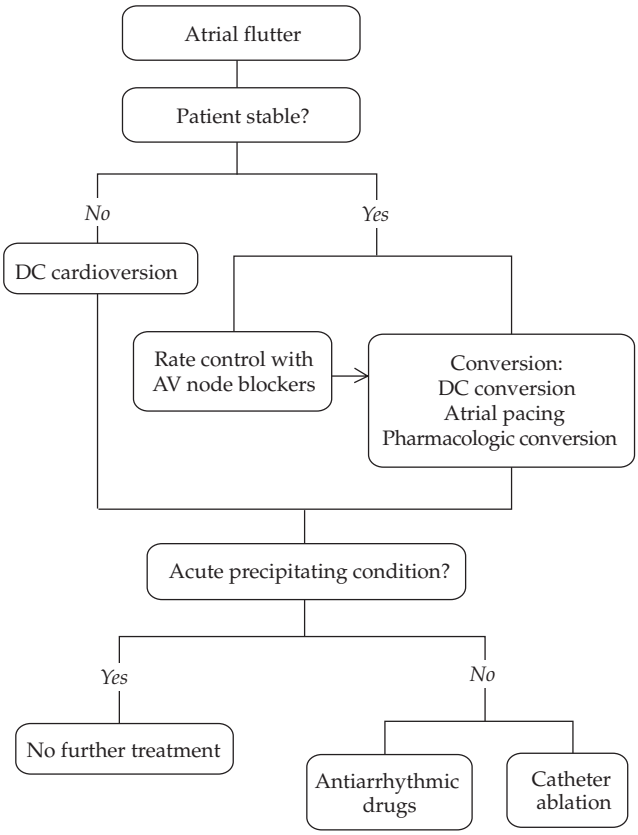

Figure 9 **Management of atrial flutter. With patients whose condition is unstable (e.g., because of heart failure, shock, or acute myocardial infarction), atrial flutter typically does not recur once the underlying disorder is resolved. Anticoagulant precautions, as per atrial fibrillation, should be taken in patients undergoing elective attempts to convert atrial flutter to sinus rhythm. (AV—atrioventricular; DC—direct current)**

with acute pulmonary insufficiency; in such cases, the arrhythmia usually does not recur once the inciting event has resolved. In contrast, atrial flutter in patients without concomitant acute illness tends to recur; like atrial fibrillation, it is usually a relapsing and remitting disease.

Electrocardiographic Findings

The ECG in patients with atrial flutter usually shows a flutter rate of 300 beats/min with 2:1 AV block. The most common atrial flutter pattern—a counterclockwise loop around the annulus—manifests as negative flutter waves in the inferior leads and positive waves in V1.[48] The ECG shows a continuous or so-called picket-fence appearance. In contrast, patients with a clockwise pattern will have positive flutter waves in the inferior leads and negative waves in V1. The ECG is much more variable for nonannular types of flutter circuits.[47]

MANAGEMENT

Acute Therapy

Treatment of atrial flutter is directed at attempts to convert the arrhythmia or use of AV nodal blockers to slow the ventricular response [*see Figure 9*]. Acute conversion of atrial flutter can be accomplished electrically by use of external DC shocks[49] or by pacing.[50,51] Atrial flutter is usually exquisitely responsive to a small "dose" of DC shock (i.e., 25 to 50 joules).[49] Atrial overdrive pacing is also quite effective for terminating flutter, especially when the patient has been pretreated with drugs (i.e., ibutilide or procainamide).[51] Overdrive pacing is particularly appropriate for atrial flutter that occurs after cardiac surgery, because atrial wires are routinely left in place postoperatively in such patients. Transesophageal pacing has also been used to terminate flutter,[52] but its popularity has been limited by the need for analgesics to alleviate the associated chest pain.

Ibutilide (a class III antiarrhythmic agent) may be used to convert atrial flutter to sinus rhythm. Randomized prospective studies have shown that ibutilide is approximately 70% effective for this purpose.[53] In addition, ibutilide has been shown to be far more effective than intravenous procainamide.[54] Ibutilide is given in 1 mg aliquots over 10 minutes separated by a 10-minute rest period. A total of 2 mg of the drug is used, and the patient must remain under telemetry monitoring for approximately 4 hours after drug delivery. Ibutilide should not be given to patients with severe structural cardiac disease (i.e., those with a left ventricular ejection fraction less than 30%) because the risk of torsade de pointes becomes significant in this setting.

Patients with atrial flutter are at risk for thromboembolism. The current recommendations for anticoagulant therapy are the same as those for patients with atrial fibrillation.[55] For example, if the flutter duration is less than 48 hours, the risk of left atrial clot is small, and one may proceed with chemical or electrical cardioversion without full anticoagulation. Anticoagulant therapy is still required for 4 to 6 weeks after conversion because of the increased risk of thromboembolism secondary to decreased left atrial flow velocity after conversion. If the flutter duration is greater than 48 hours, a transesophageal echocardiogram to exclude clot is recommended before cardioversion. Complete guidelines for antithrombotic therapy in patients with atrial flutter are described elsewhere [*see 1:IV Atrial Fibrillation*].

As an alternative to cardioversion, AV nodal blocking agents can be used to decrease the ventricular response in patients with flutter. Controlled trials have demonstrated the efficacy of intravenous calcium channel blockers (verapamil or diltiazem) in producing prompt decreases in heart rate.[56] Calcium channel blockers have been shown to reduce the heart rate below 100 beats/min more promptly than digoxin or amiodarone.

Long-term Therapy

Drug therapy for chronic atrial flutter is notoriously unreliable, and long-term rate control alone usually requires large doses of AV nodal blocking agents. A more effective intervention involves an ablative procedure in which radiofrequency lesions are applied in a line from the tricuspid annulus to the inferior vena cava.[57] This area is the critical isthmus for the usual type of atrial flutter circuit. Ablation of this area resulting in total conduction block of the isthmus is associated with a 90% to 100% cure rate in flutter.[58] Non–isthmus-dependent flutter circuits may involve either the right or the left atrium and usually require sophisticated mapping tools to determine the tachycardia circuit and the critical isthmus needed for curative ablation. These patients should be referred to experienced centers for evaluation.

Many patients have both atrial flutter and atrial fibrillation. For example, atrial flutter may deteriorate into atrial fibrillation, or bursts of atrial fibrillation may trigger atrial flutter. In addition, approximately 15% to 30% of patients treated with

class IC antiarrhythmics or amiodarone for atrial fibrillation will develop stable atrial flutter.[59] In these patients, radiofrequency ablation of the flutter circuit together with continuance of drug therapy is usually quite effective in controlling both atrial fibrillation and flutter. Ablation of the flutter usually does not cure the atrial fibrillation.[60]

Sinus Tachycardia

Sinus tachycardia is usually a normal reflex response to changes in physiologic, pharmacologic, or pathophysiologic stimuli, such as exercise, emotion (e.g., anxiety, anger), fever, hemodynamic or respiratory compromise, anemia, thyrotoxicosis, poor physical condition, sympathomimetic or vagolytic agents, and abnormal hemoglobins. Heart rate during sinus tachycardia generally does not exceed 180 beats/min, except perhaps in young persons, who may achieve sinus rates greater than 200 beats/min during vigorous exercise.

When sinus tachycardia is a reflex response to altered physiology, the resulting increase in cardiac output is usually beneficial. Tachycardia resolves when conditions return to normal.

Inappropriate Sinus Tachycardia

An infrequent but troublesome problem, inappropriate sinus tachycardia (IST) appears to be a true syndrome with cardiac, neurologic, and psychiatric components. It affects women more often than men. Structural heart disease is generally absent. In one series of 475 patients, IST was the indication for catheter ablation in 2.3%.[61]

DIAGNOSIS

Clinical Presentation

IST may be persistent or episodic. It is often precipitated by arising from a reclining or sitting position (postural orthostatic tachycardia).[62] Very rapid rates (> 170 beats/min) may be triggered by minimal exertion.

The tachycardia is frequently accompanied by symptoms of dizziness, near-syncope, or syncope. Fatigue and atypical chest pain may also accompany IST. Peculiar but inconsistent autonomic and hemodynamic findings may be seen in these patients. This suggests that the syndrome is not uniform in etiology.

Electrocardiographic Findings

Because tachycardia rates may arise from higher foci, the P waves seen during IST may differ slightly from those seen at rest.

MANAGEMENT

Drug Therapy

Beta blockers and calcium channel blockers (i.e., verapamil or diltiazem) may be used to alleviate tachycardia in IST. Unfortunately, these drugs are often not effective and tend to exacerbate the nonspecific symptoms that accompany this syndrome. Agents that alter sinus node automaticity, autonomic tone, or both, such as flecainide, propafenone, and amiodarone, may be tried in selected patients.[63]

Catheter Ablation

Radiofrequency ablation has been employed to ablate or modify the sinus node in IST. Large-tipped (8 to 10 mm) catheters are often required to create more sizable lesions. Successful modification or ablation has been achieved in 70% to 100% of patients.[64] Sinus nodal modification is associated with a 10% to 27% risk of sinus node damage necessitating permanent pacing.

Both intracardiac electrograms and intracardiac ultrasonography have been used to target lesion delivery. Intracardiac ultrasonography targets the fastest portions of the sinus node by ablating the uppermost portion of the crista terminalis. This approach seems to require fewer radiofrequency applications than do electrogram-guided approaches. It may also reduce the need for permanent pacing.

Long-term follow-up after radiofrequency modification has been less encouraging. Recurrence rates are high. Ablation to the extent that permanent pacing is required may be necessary for sustained success. Thus, patients require careful follow-up for recurrent tachycardia or progressive sinus node dysfunction. Surgical isolation of the sinus node for IST has also been followed by recurrent tachycardia at new foci.

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