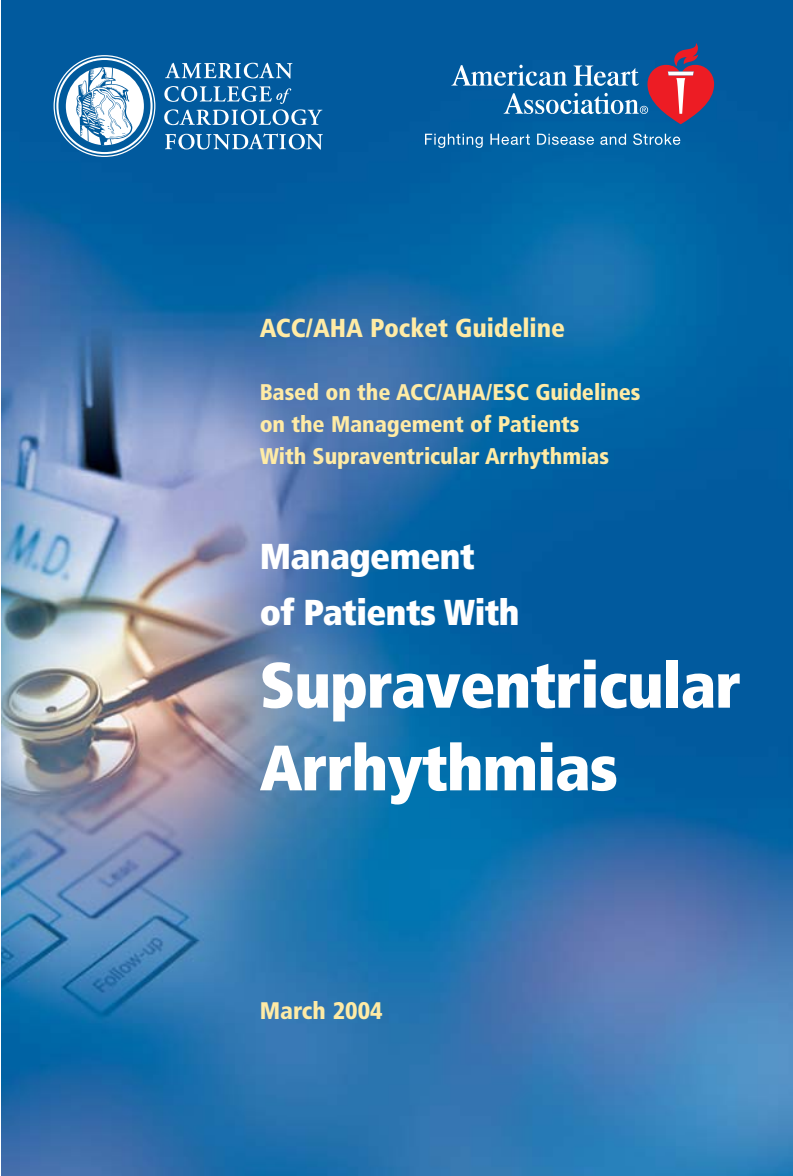
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American Heart
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Fighting Heart Disease and Stroke
ACC/AHA Pocket Guideline
Based on the ACC/AHA/ESC Guidelines
on the Management of Patients
With Supraventricular Arrhythmias
Management
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Follow-up
March 2004

Management of Patients With Supraventricular Arrhythmias

March 2004

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Contents

I. Introduction

Supraventricular arrhythmias (SVAs) include rhythms emanating from the sinus node, atrial tissue [atrial tachycardias (ATs), atrial flutter], and junctional tissue [atrioventricular nodal reciprocating tachycardia (AVNRT)]. Accessory pathway-mediated and atrioventricular reciprocating tachycardia (AVRT) are also included. SVA occurs in all age groups and may be associated with minimal symptoms, such as palpitations, or may present with syncope. In some conditions (i.e., those associated with bypass tracts), arrhythmias may be life-threatening. The prevalence of paroxysmal supraventricular tachycardia (PSVT) is 2 to 3 per 1,000. Over the past decade, impressive advances in curative treatment modes (catheter ablation) have become available. The purpose of this booklet is to summarize guidelines for use of drug and ablative procedures for patients with supraventricular tachycardia (SVT). Guidelines for treatment of atrial fibrillation were recently published; hence, this subject is excluded in the present booklet. In addition, SVT in the pediatric population is excluded. The *ACC/AHA/ESC Pocket Guidelines for the Management of Patients With Atrial Fibrillation* discusses antiarrhythmic drug doses and adverse effects, and therefore, this will not be repeated.

The guidelines outlined come from an expert committee selected by the European Society of Cardiology, American College of Cardiology, and American Heart Association. The ultimate judgment regarding care of a particular patient must be made by the physician and patient in light of all of the circumstances presented by that patient. In some circumstances, deviations from these guidelines may be appropriate. Recommendations are provided in tables and use the following classification outline, summarizing both the evidence and expert opinion:

Class I	Conditions for which there is evidence and/or general agreement that the procedure or treatment is useful and effective.
Class II	Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment. **Class IIa** The weight of evidence or opinion is in favor of the procedure or treatment. **Class IIb** Usefulness/efficacy is less well established by evidence or opinion.
Class III	Conditions for which there is evidence and/or general agreement that the procedure or treatment is not useful/effective and in some cases may be harmful.

II. General Evaluation and Management

A. Patients Without Documented Arrhythmia (Figure 1)

Clinical History

Distinguish whether palpitations are regular or irregular.

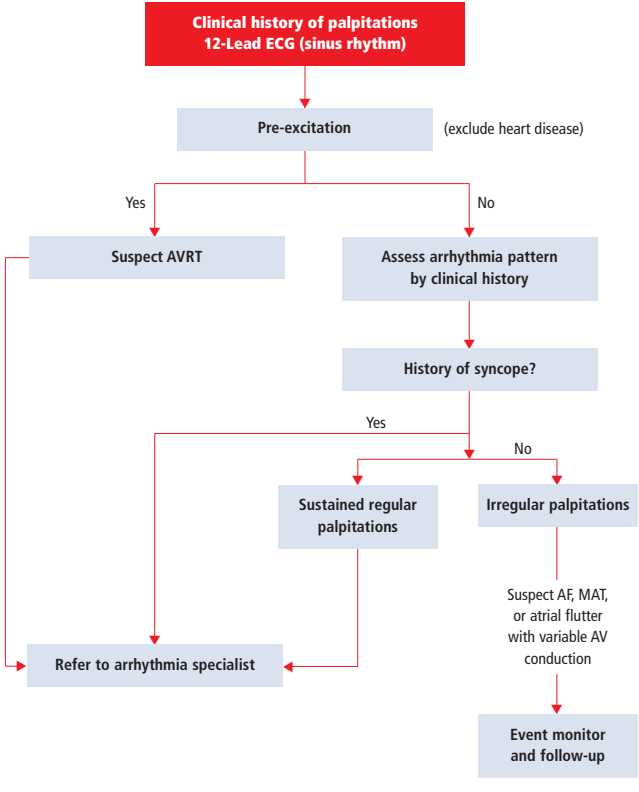
- Pauses or dropped beats followed by a sensation of a strong heartbeat support presence of premature beats.
- Irregular palpitations may be due to premature extra beats, atrial fibrillation, or multifocal AT.
- Regular and recurrent palpitations with abrupt onset and termination are designated as paroxysmal (also referred to as PSVT). Termination by vagal maneuvers suggests a re-entrant tachycardia involving atrioventricular nodal tissue (e.g., AVNRT, AVRT).
- Sinus tachycardia is nonparoxysmal and accelerates and terminates gradually.

B. Patients With Documented Arrhythmia

1. Narrow QRS-Complex Tachycardia

If the ventricular action on ECG (QRS) is narrow [less than 120 milliseconds (ms)], then the tachycardia is

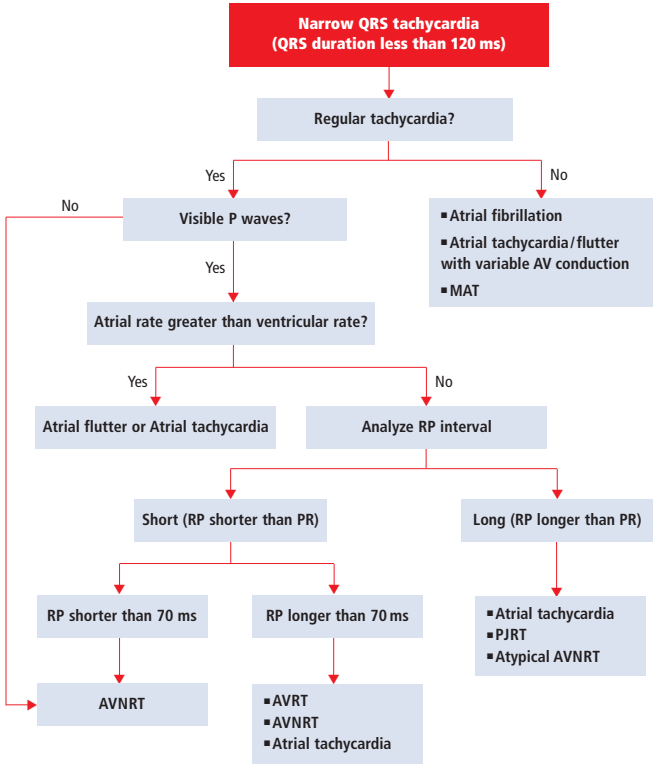
Figure 1



Initial evaluation of patients with suspected tachycardia.

AF indicates atrial fibrillation; **AV,** atrioventricular; **AVRT,** atrioventricular reciprocating tachycardia; **ECG,** electrocardiogram; **MAT,** multifocal atrial tachycardia.

Figure 2



Differential diagnosis for narrow QRS-complex tachycardia.

Patients with focal junctional tachycardia may mimic the pattern of slow-fast AVNRT and may show AV dissociation and/or marked irregularity in the junctional rate.

AV indicates atrioventricular; **AVNRT,** atrioventricular nodal reciprocating tachycardia; **AVRT,** atrioventricular reciprocating tachycardia; **MAT,** multifocal atrial tachycardia; **ms,** milliseconds; **PJRT,** permanent form of junctional reciprocating tachycardia; **QRS,** ventricular activation on electrocardiogram.

almost always supraventricular, and the differential diagnosis relates to its mechanism *(Figure 2)*. The clinician must determine the relationship of the P waves to the ventricular complex *(Figure 3)*. Responses of narrow QRS-complex tachycardias to adenosine *(Figure 4)* or carotid massage may aid in the differential diagnosis.

2. Wide QRS-Complex Tachycardia

At times, the patient will present with rapid wide-complex (greater than 120 ms) tachycardia, and the clinician must decide whether the patient has

a) SVT with bundle-branch block (BBB) (or aberration),

b) SVT with atrioventricular (AV) conduction over an accessory pathway, or

c) ventricular tachycardia (VT).

This categorization depends not only on the relation of the P wave to the QRS but also on specific morphologic findings, especially in the precordial leads *(Figure 5)*.

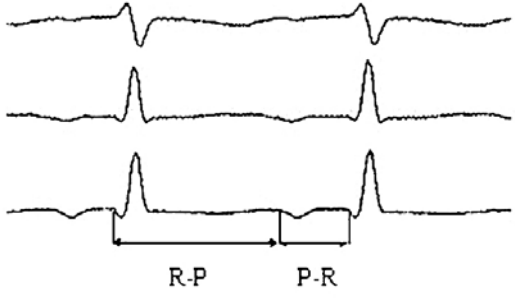
3. Management

If the diagnosis of SVT cannot be proven, the patient should be treated as if VT were present. Medications for SVT (verapamil or diltiazem) may precipitate hemodynamic collapse in a patient with VT. Special circumstances (i.e., pre-excited tachycardias and VT due to digitalis toxicity) may require alternative therapy. Immediate direct-current (DC) cardioversion is the treatment of choice for any hemodynamically unstable tachycardia.

Indications for referral to a cardiac arrhythmia specialist:

- All patients with Wolff-Parkinson-White (WPW) syndrome (pre-excitation plus arrhythmias).
- All patients with severe symptoms during palpitations, such as syncope or dyspnea.
- Wide QRS-complex tachycardia of unknown origin.
- Narrow QRS-complex tachycardias with drug resistance, drug intolerance, or desire to be free of drug therapy.

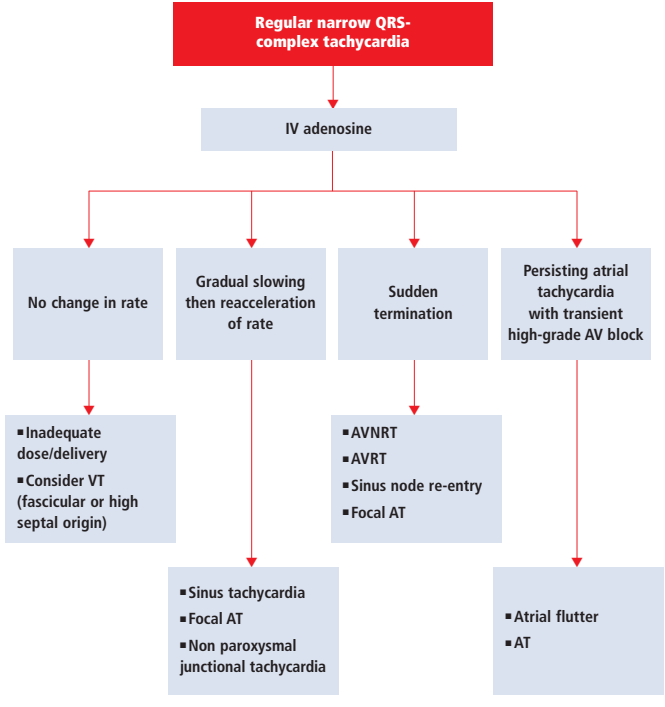
Figure 3



Electrocardiograph tracing with limb leads I, II, and III, showing an RP (initial R to initial P) interval longer than the PR interval.

The P wave differs from the sinus P wave.

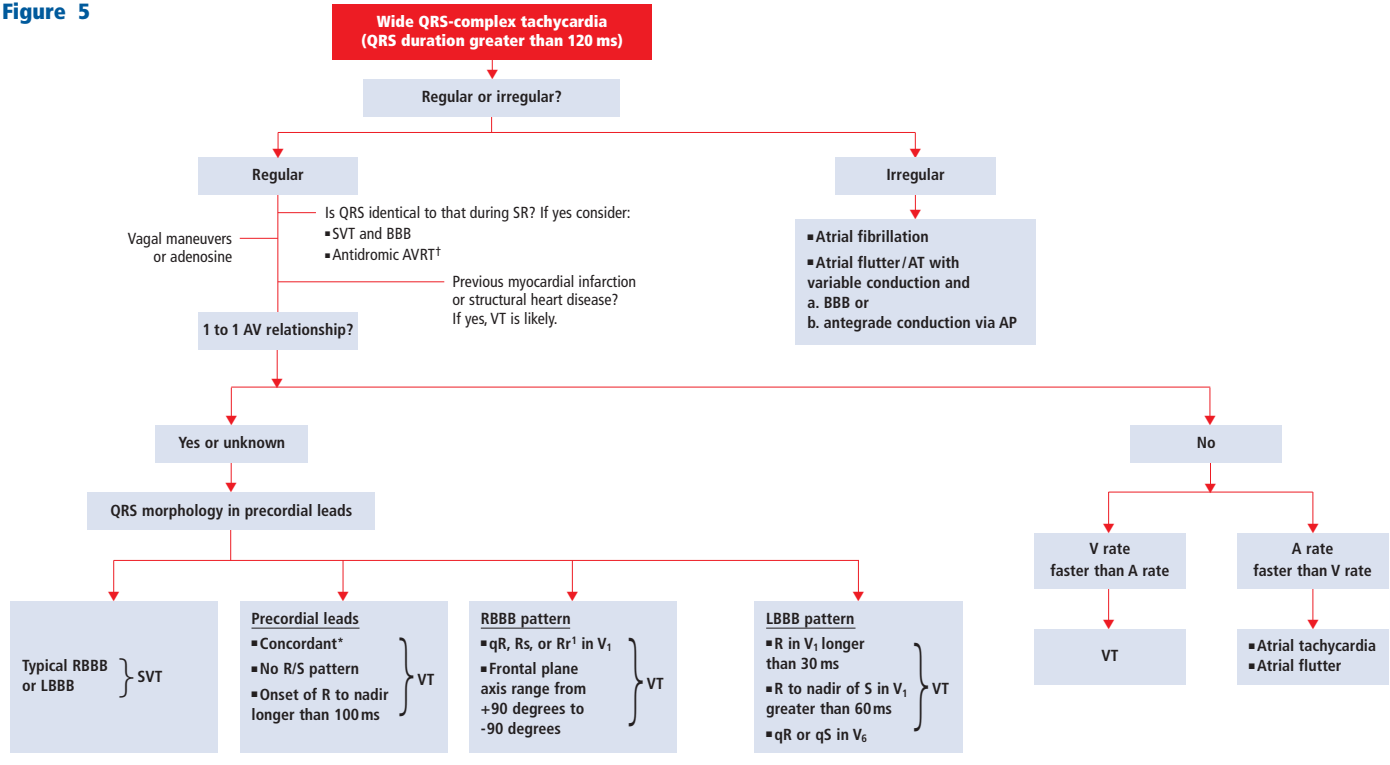
Figure 4



Responses of narrow-complex tachycardias to adenosine.

AT indicates atrial tachycardia; **AV,** atrioventricular; **AVNRT,** atrioventricular nodal reciprocating tachycardia; **AVRT,** atrioventricular reciprocating tachycardia; **IV,** intravenous; **QRS,** ventricular activation on electrocardiogram; **VT,** ventricular tachycardia.

Figure 5



Differential diagnosis for wide QRS-complex tachycardia (greater than 120 ms).

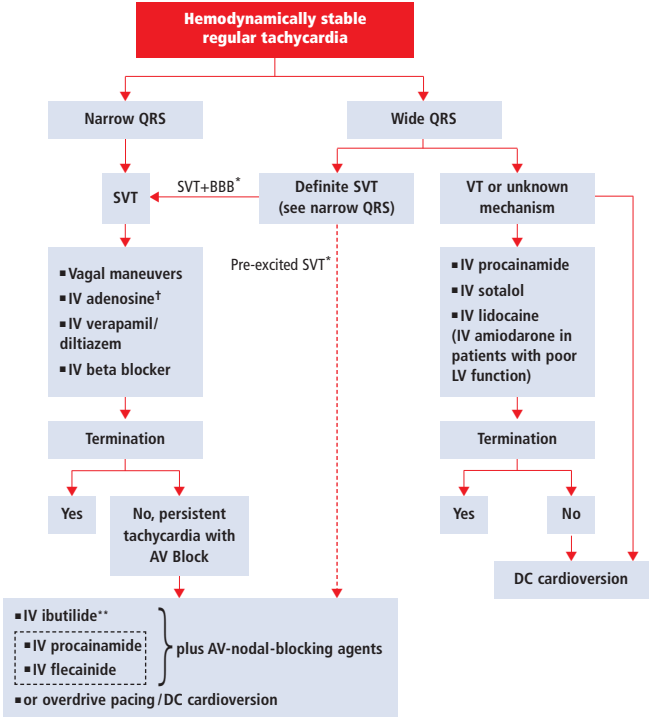
A QRS conduction delay during sinus rhythm, when available for comparison, reduces the value of QRS morphology analysis. Adenosine should be used with caution when the diagnosis is unclear because it may produce VF in patients with coronary artery disease and AF with a rapid ventricular rate in pre-excited tachycardias. Various adenosine responses are shown in Figure 6.

* Concordant indicates that all precordial leads show either positive or negative deflections. Fusion complexes are diagnostic of VT.

† In pre-excited tachycardias, the QRS is generally wider (i.e., more pre-excited) than in sinus rhythm.

A indicates atrial; **AF,** atrial fibrillation; **AP,** accessory pathway; **AT,** atrial tachycardia; **AV,** atrioventricular; **AVRT,** atrioventricular reciprocating tachycardia; **BBB,** bundle-branch block; **LBBB,** left bundle-branch block; **ms,** milliseconds; **QRS,** ventricular activation on ECG; **RBBB,** right bundle-branch block; **SR,** sinus rhythm; **SVT,** supraventricular tachycardias; **V,** ventricular; **VF,** ventricular fibrillation; **VT,** ventricular tachycardia.

Figure 6



Acute management of patients with hemodynamically stable and regular tachycardia.

* A 12-lead ECG during sinus rhythm must be available for diagnosis.

† Adenosine should be used with caution in patients with severe coronary artery disease and may produce AF, which may result in rapid ventricular rates for patients with pre-excitation.

** Ibutilide is especially effective for patients with atrial flutter but should not be used in patients with an ejection fraction less than 30% because of increased risk of polymorphic VT.

AF indicates atrial fibrillation; **AV,** atrioventricular; **BBB,** bundle-branch block; **DC,** direct current; **ECG,** electrocardiogram; **IV,** intravenous; **LV,** left ventricular; **QRS,** ventricular activation on ECG; **SVT,** supraventricular tachycardia; **VT,** ventricular tachycardia.

Recommendations for Acute Management of Hemodynamically Stable and Regular Tachycardia

ECG	Recommendation*	Class	Evidence
Narrow QRS-complex tachycardia (SVT)	Vagal maneuvers	I	B
	Adenosine	I	A
	Verapamil, diltiazem	I	A
	Beta blockers	IIb	C
	Amiodarone	IIb	C
	Digoxin	IIb	C
Wide QRS-complex tachycardia			
■ **SVT and BBB**	See above		
■ **Pre-excited SVT/AF†**	Flecainide‡	I	B
	Ibutilide‡	I	B
	Procainamide,‡	I	B
	DC cardioversion	I	C
■ **Wide QRS-complex tachycardia of unknown origin**	Procainamide‡	I	B
	Sotalol‡	I	B
	Amiodarone	I	B
	DC cardioversion	I	B
	Lidocaine	IIb	B
	Adenosine§	IIb	C
	Beta blockers††	III	C
	Verapamil**	III	B
Wide QRS-complex tachycardia of unknown origin in patients with poor LV function	Amiodarone	I	B
	DC cardioversion, lidocaine	I	B

The order in which treatment recommendations appear in this table within each class of recommendation does not necessarily reflect a preferred sequence of administration. Please refer to text for details. For pertinent drug dosing information, please refer to the *ACC/AHA/ESC Guidelines for the Management of Patients With Atrial Fibrillation.*

* All listed drugs are administered intravenously.

† See Section III-D.

‡ Should not be taken by patients with reduced LV function.

§ Adenosine should be used with caution in patients with severe coronary artery disease because vasodilation of normal coronary vessels may produce ischemia in vulnerable territory. It should be used only with full resuscitative equipment available.

†† Beta blockers may be used as first-line therapy for those with catecholamine-sensitive tachycardias, such as right ventricular outflow tachycardia.

** Verapamil may be used as first-line therapy for those with LV fascicular VT.

AF indicates atrial fibrillation; **BBB**, bundle-branch block; **DC**, direct current; **ECG,** electrocardiogram; **LV**, left ventricular; **QRS,** ventricular activation on ECG; **SVT,** supraventricular tachycardia.

Evaluation/Management

III. Specific Arrhythmias

A. Inappropriate Sinus Tachycardia

Inappropriate sinus tachycardia refers to a persistent increase in resting heart rate unrelated to the level of physical, emotional, pathological, or pharmacological stress. Approximately 90% of patients are female. The degree of disability can vary from asymptomatic to individuals who are totally incapacitated.

The diagnosis is based on the following criteria:

- Persistent sinus tachycardia (heart rate greater than 100 bpm) during the day with excessive rate increase in response to activity and nocturnal normalization of rate as confirmed by a 24-hour Holter recording.
- The tachycardia and its symptoms are not paroxysmal.
- P-wave morphology is identical to sinus rhythm.
- Exclusion of a secondary systemic cause (hyperthyroidism, pheochromocytoma, physical deconditioning).

Treatment

The treatment is predominantly symptom driven. The long-term success rate of sinus node modification by catheter ablation has been reported to be approximately 66%. The diagnosis of postural orthostatic tachycardia syndrome must be excluded before ablation is considered.

Recommendations for Treatment of Inappropriate Sinus Tachycardia

Treatment	Recommendation	Class	Evidence
Medical	Beta blockers	I	C
	Verapamil, diltiazem	IIa	C
Interventional	Catheter ablation-sinus node modification/elimination*	IIb	C

The order in which treatment recommendations appear in this table within each class of recommendation does not necessarily reflect a preferred sequence of administration. Please refer to text for details. For pertinent drug dosing information, please refer to the *ACC/AHA/ESC Guidelines for the Management of Patients With Atrial Fibrillation.*

* Used as a last resort.

B. Atrioventricular Nodal Reciprocating Tachycardia (AVNRT)

AVNRT is a re-entry tachycardia that involves the AV node and perinodal atrial tissue. One pathway (fast) is located near the superior portion of the AV node and the other (slow) along the septal margin of the tricuspid annulus. During typical AVNRT (85-90%), antegrade conduction occurs over the slow pathway, with a turnaround point in the AV junction, and retrograde conduction occurs over the fast pathway. The converse is found during atypical AVNRT, resulting in a long R-P tachycardia with negative P waves in III and AVF inscribed before the QRS.

Recommendations for Long-Term Treatment of Patients With Recurrent AVNRT

Clinical Presentation	Intervention	Class	Evidence
Poorly tolerated AVNRT with hemodynamic intolerance	Catheter ablation	I	B
	Verapamil, diltiazem, beta blockers, sotalol, amiodarone	IIa	C
	Flecainide,* propafenone*	IIa	C
Recurrent symptomatic AVNRT	Catheter ablation	I	B
	Verapamil	I	B
	Diltiazem, beta blockers	I	C
	Digoxin†	IIb	C
Recurrent AVNRT unresponsive to beta blockade or calcium channel blocker and patient not desiring RF ablation	Flecainide,* propafenone,* sotalol	IIa	B
	Amiodarone	IIb	C
AVNRT with infrequent or single episode in patients who desire complete control of arrhythmia	Catheter ablation	I	B
Documented PSVT with only dual AV-nodal pathways or single echo beats demonstrated during electrophysiological study and no other identified cause of arrhythmia	Verapamil, diltiazem, beta blockers, flecainide,* propafenone*	I	C
	Catheter ablation‡	I	B
Infrequent, well-tolerated AVNRT	No therapy	I	C
	Vagal maneuvers	I	B
	"Pill-in-the-pocket"	I	B
	Verapamil, diltiazem, beta blockers	I	B
	Catheter ablation	I	B

The order in which treatment recommendations appear in this table within each class of recommendation does not necessarily reflect a preferred sequence of administration. Please refer to text for details. For pertinent drug dosing information, please refer to the *ACC/AHA/ESC Guidelines for the Management of Patients With Atrial Fibrillation.*

* Relatively contraindicated for patients with coronary artery disease, left ventricular dysfunction, or other significant heart disease.

† Often ineffective because pharmacological effects can be overridden by enhanced sympathetic tone.

‡ Decision depends on symptoms.

AV indicates atrioventricular; **AVNRT,** atrioventricular nodal reciprocating tachycardia; **LV,** left ventricular; **PSVT,** paroxysmal supraventricular tachycardia; **RF,** radiofrequency.

Treatment

Standard treatment is use of drugs that primarily block AV nodal conduction (beta blockers, calcium channel blockers, adenosine). Another treatment option that has been shown to be effective and safe involves catheter ablation to destroy the slow pathway. Indications for ablation depend on clinical judgment and are often predicated on patient preference. Factors that contribute to the decision include tachycardia frequency, tolerance of symptoms, and patient inclination relative to chronic drug therapy vs. ablation. The patient must accept the risk, albeit small (less than 1%), of AV block and pacemaker insertion.

C. Focal and Nonparoxysmal Junctional Tachycardia

1. Focal Junctional Tachycardia

The unifying feature of focal junctional tachycardias, also known as automatic or junctional ectopic tachycardia, is their origin from the AV node or His bundle. The ECG features of focal junctional tachycardia include heart rates of 110 to 250 bpm and a narrow complex or typical BBB conduction pattern with AV dissociation. Occasionally the junctional rhythm is quite erratic, suggesting AF. This is a rare arrhythmia seen in young adults, and if persistent, it may produce congestive heart failure. Drug therapy has been associated with only variable success, and catheter ablative procedures are associated with a 5% to 10% risk of AV block.

2. Nonparoxysmal Junctional Tachycardia

Nonparoxysmal junctional tachycardia is a benign arrhythmia that is characterized by a narrow-complex tachycardia with rates of 70 to 120 bpm. The arrhythmia is thought to be due to abnormal automaticity or triggered rhythms and serves as a marker for underlying problems including digitalis toxicity, postcardiac surgery, hypokalemia, and myocardial ischemia. Treatment is most often directed at the underlying condition.

Recommendations for Treatment of Focal and Nonparoxysmal Junctional Tachycardia Syndromes

Clinical Presentation	Recommendation	Class	Evidence
Focal junctional tachycardia	Beta blockers	IIa	C
	Flecainide	IIa	C
	Propafenone*	IIa	C
	Sotalol*	IIa	C
	Amiodarone*	IIa	C
	Catheter ablation	IIa	C
Nonparoxysmal junctional tachycardia	Reverse digitalis toxicity	I	C
	Correct hypokalemia	I	C
	Treat myocardial ischemia	I	C
	Beta blockers, calcium-channel blockers	IIa	C

The order in which treatment recommendations appear in this table within each class of recommendation does not necessarily reflect a preferred sequence of administration. Please refer to text for details. For pertinent drug dosing information, please refer to the *ACC/AHA/ESC Guidelines for the Management of Patients With Atrial Fibrillation.*

* Data available for pediatric patients only.

D. Atrioventricular Reciprocating Re-entry Tachycardia (Extranodal Accessory Pathways)

Typical accessory pathways are extranodal pathways that connect the myocardium of the atrium and the ventricle across the AV groove. Accessory pathways that are capable of only retrograde conduction are referred to as "concealed," whereas those capable of anterograde conduction are "manifest," demonstrating pre-excitation on a standard ECG. The term WPW syndrome is reserved for patients who have both pre-excitation and tachyarrhythmias.

Several forms of tachycardias may occur:

- Orthodromic AVRT (most common, 95%) involves anterograde conduction over the AV node and retrograde conduction over the accessory pathway.
- Antidromic AVRT anterograde conduction over the accessory pathway and retrograde conduction over the AV node (or rarely, over a second accessory pathway) resulting in pre-excited QRS complexes during tachycardia.
- Pre-excited tachycardias in patients with AT or atrial flutter with a bystander (not a critical part of the tachycardia circuit) accessory pathway.
- Pre-excited atrial fibrillation, the most feared arrhythmia, occurs in 30% of patients with the WPW syndrome.
- PJRT (permanent form of junctional reciprocating tachycardia): a rare clinical syndrome with a slowly conducting concealed posteroseptal accessory pathway characterized by an incessant, long RP tachycardia with negative P waves in leads II, III, and aVF.

Sudden Death in WPW Syndrome and Risk Stratification

Markers that identify patients at increased risk include: 1) a shortest pre-excited R-R interval less than 250 ms during AF, 2) a history of symptomatic tachycardia, 3) multiple accessory pathways, and 4) Ebstein's anomaly. The risk for sudden cardiac death is estimated at between 0.15% and 0.39% of patients with WPW syndrome over 3- to 10-year follow-up.

Asymptomatic Patients With Accessory Pathways

The positive predictive value of invasive electrophysiological testing is too low to justify routine use in asymptomatic patients. The decision to ablate pathways in individuals with high-risk occupations, such as school bus drivers, pilots, and athletes, is made on individual clinical considerations.

Treatment

Acute Treatment of Patients With Pre-excited Tachycardias

AV nodal-blocking agents are not effective, and adenosine may produce AF with a rapid ventricular rate. Antiarrhythmic drugs that prevent rapid conduction through the pathway (flecainide, procainamide, or ibutilide), are preferable, even if they may not convert the atrial arrhythmia.

Long-Term Therapy

Antiarrhythmic drugs represent one therapeutic option for management of patients with accessory pathway–mediated arrhythmias, but they have been increasingly replaced by catheter ablation. A regimen designed for use of drug(s) at the onset of an episode should only be used for patients with infrequent, well-tolerated episodes.

Some patients with infrequent episodes of tachycardia may be managed with the single-dose "pill-in-the-pocket" approach: taking an antiarrhythmic drug only at the onset of a tachycardia episode. This approach to treatment is reserved for patients without pre-excitation and with uncommon and hemodynamically tolerated tachycardia.

Catheter ablative techniques are successful in approximately 95% of cases and have sufficient efficacy and low risk to be used for symptomatic patients, either as initial therapy or for patients experiencing side effects or arrhythmia recurrence during drug therapy. The type of possible complications varies depending on the site of the pathway. The incidence of inadvertent complete AV block ranges from 0.17% to 1.0% and relates to septal and posteroseptal accessory pathways. Significant adverse effects range from 1.8% to 4%, including a 0.08% to 0.13% risk of death.

Recommendations for Long-Term Therapy of Accessory Pathway-Mediated Arrhythmias

Arrhythmia	Recommendation	Class	Evidence
WPW syndrome (pre-excitation and symptomatic arrhythmias), well tolerated	Catheter ablation	I	B
	Flecainide, propafenone	IIa	C
	Sotalol, amiodarone, beta blockers	IIa	C
	Verapamil, diltiazem, digoxin	III	C
WPW syndrome (with AF and rapid-conduction or poorly tolerated AVRT)	Catheter ablation	I	B
AVRT, poorly tolerated (no pre-excitation)	Catheter ablation	I	B
	Flecainide, propafenone	IIa	C
	Sotalol, amiodarone	IIa	C
	Beta blockers	IIb	C
	Verapamil, diltiazem, digoxin	III	C
Single or infrequent AVRT episode(s) (no pre-excitation)	None	I	C
	Vagal maneuvers	I	B
	"Pill-in-the-pocket"—verapamil, diltiazem, beta blockers	I	B
	Catheter ablation	IIa	B
	Sotalol, amiodarone	IIb	B
	Flecainide, propafenone	IIb	C
	Digoxin	III	C
Pre-excitation, asymptomatic	None	I	C
	Catheter ablation	IIa	B

The order in which treatment recommendations appear in this table within each class of recommendation does not necessarily reflect a preferred sequence of administration. Please refer to text for details. For pertinent drug dosing information, please refer to the *ACC/AHA/ESC Guidelines for the Management of Patients With Atrial Fibrillation.*

AF indicates atrial fibrillation; **AVRT,** atrioventricular reciprocating tachycardia; **WPW,** Wolff-Parkinson-White.

E. Focal Atrial Tachycardia

Focal ATs are characterized by radial spread of activation from a focus, with endocardial activation not extending through the entire atrial cycle. They are usually manifest by atrial rates between 100 and 250 bpm (rarely at 300 bpm). The mechanism has been attributed to abnormal or enhanced automaticity, triggered activity (due to delayed after-depolarization), or microre-entry. A progressive increase in atrial rate with tachycardia onset ("warm-up") or progressive decrease before tachycardia termination ("cool-down") suggests an automatic mechanism. Approximately 10% of patients have multiple foci. Focal AT may be incessant, leading to tachycardia-induced cardiomyopathy.

Treatment

Therapeutic options include use of drugs for rate control (beta blockers, calcium-channel blockers, or digoxin) or for suppression of the arrhythmic focus. In addition, class Ia or Ic (flecainide and propafenone) drugs may prove effective.

The available studies suggest use of IV adenosine, beta blockers, or calcium-channel blockers either for acute termination (unusual) or more frequently to achieve rate control. Adenosine will terminate focal AT in a significant number of patients. DC

Recommendations for Treatment of Focal Atrial Tachycardias*

Clinical Situation	Recommendation	Class	Evidence
Acute treatment†			
A. Conversion			
Hemodynamically unstable patient	DC Cardioversion	I	B
Hemodynamically stable patient	Adenosine	IIa	C
	Beta blockers	IIa	C
	Verapamil, diltiazem	IIa	C
	Procainamide	IIa	C
	Flecainide, propafenone	IIa	C
	Amiodarone, sotalol	IIa	C
B. Rate regulation (in absence of digitalis therapy)	Beta blockers	I	C
	Verapamil, diltiazem	I	C
	Digoxin	IIb	C
Prophylactic therapy			
Recurrent symptomatic AT	Catheter ablation	I	B
	Beta blockers, calcium-channel blockers	I	C
	Disopyramide‡	IIa	C
	Flecainide, propafenone‡	IIa	C
	Sotalol, amiodarone	IIa	C
Asymptomatic or symptomatic incessant ATs	Catheter ablation	I	B
Nonsustained and asymptomatic ATs	No therapy	I	C
	Catheter ablation	III	C

The order in which treatment recommendations appear in this table within each class of recommendation does not necessarily reflect a preferred sequence of administration. Please refer to text for details. For pertinent drug dosing information, please refer to the *ACC/AHA/ESC Guidelines for the Management of Patients With Atrial Fibrillation.*

*Excluded are patients with multifocal AT in whom beta blockers and sotalol are often contraindicated because of pulmonary disease.

† All listed drugs for acute treatment are taken intravenously.

‡ Flecainide, propafenone, and disopyramide should not be used unless they are combined with an AV-nodal–blocking agent.

AT indicates atrial tachycardia; **DC,** direct current.

cardioversion seldom terminates automatic ATs but may be successful for ATs based on microre-entry or triggered automaticity and should be attempted in patients with drug-resistant arrhythmia.

Chronic control involves initial use of AV-nodal–blocking drugs because they may prove effective and they have minimal side effects. Other, more potent agents should be reserved for after failure of an AV-nodal blocker. Focal AT is ablated by targeting the site of origin of the AT. Catheter ablation has a success rate of 80% to 90% for right atrial foci and 70% to 80% for left atrial foci. The incidence of significant complications is low (1% to 2%). Ablation of AT from the atrial septum or Koch's triangle may produce AV block.

Multifocal Atrial Tachycardia

This tachycardia is characterized by finding three or more different P-wave morphologies at different rates. The rhythm is always irregular and frequently confused with AF. It is most commonly associated with underlying pulmonary disease but may result from metabolic or electrolyte derangements. Therapy includes correction of underlying abnormalities but often requires use of calcium-channel blockers, because there is no role for DC cardioversion, antiarrhythmic drugs, or ablation.

F. Macro–Re-entrant Atrial Tachycardia

Atrial flutter is defined as an organized rapid (250 to 350 bpm) macro–re-entrant atrial rhythm. The most common forms relate to re-entrant rhythms that circulate around the tricuspid annulus. Isthmus-dependent flutter refers to circuits in which the arrhythmia involves the cavotricuspid isthmus (CTI). They are most frequently manifest as counterclockwise (negative flutter deflections in inferior leads) but can be clockwise (positive deflections in the inferior leads).

Non–isthmus-dependent atrial flutter is less frequent and is often caused by surgical scars that produce a central obstacle for re-entry. For patients with non–isthmus-dependent flutter, large areas of atrial scar are found (with cardiac mapping) and are often associated with multiple re-entrant circuits. Atrial flutter may cause insidious symptoms, such as exercise-induced fatigue, worsening heart failure, or pulmonary disease. Patients often present with a 2:1 AV conduction, which, if left untreated, may promote cardiomyopathy.

Treatment

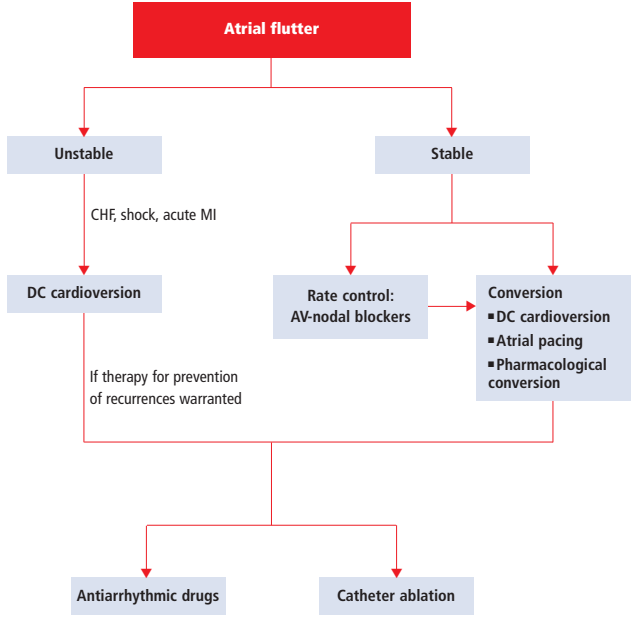
Acute therapy depends on the clinical status of the patient and underlying cardiorespiratory problems. If the arrhythmia is attended by heart failure, shock, or myocardial ischemia, then prompt DC cardioversion is in order. Rapid atrial (or esophageal pacing) and low-energy DC cardioversion are very effective in termination of atrial flutter. In most instances, however, patients with flutter are stable, and trials of AV-nodal–blocking drugs for rate control are in order. This is especially important if the subsequent use of antiarrhythmic drugs is planned, because slowing of the flutter rate by antiarrhythmic drugs (especially class Ic drugs) may result in a paradoxical increase in the ventricular rate. If the atrial flutter persists longer than 48 hours, then either a 3- to 4-week course of anticoagulant therapy or a negative transesophageal echocardiogram (absence of clots) is

advisable before electrical or drug conversion is attempted. These recommendations are identical to those used for management of atrial fibrillation. Neither AV-nodal drugs nor amiodarone is effective for conversion of atrial flutter. Intravenous ibutilide appears to be the most effective agent for acute drug termination of flutter, with an efficacy between 38% and 76%, and is more effective than intravenous Class Ic agents.

Class III drugs, especially dofetilide, appear to be quite effective chronic therapy for patients with flutter (73% response rate). Chronic therapy is usually not required after sinus rhythm is restored if atrial flutter occurs as part of an acute disease process.

Catheter ablation of the CTI is a safe and effective cure for patients with CTI-dependent flutter. For those patients with non–isthmus-dependent flutter, referral to a specialized center is in order because multiple complex circuits are frequently found. Success rates vary from 50% to 88% depending on lesion complexity.

Figure 7



Management of atrial flutter depending on hemodynamic stability.

Attempts to electively revert atrial flutter to sinus rhythm should be preceded and followed by anticoagulant precautions, as per atrial fibrillation.

AV, atrioventricular; **CHF,** congestive heart failure; **DC,** direct current; **MI,** myocardial infarction.

Recommendations for Acute Management of Atrial Flutter

Clinical Status/ Proposed Therapy	Recommendation*	Class	Evidence
Poorly tolerated			
Conversion	DC cardioversion	I	C
Rate control	Beta blockers	IIa	C
	Verapamil, diltiazem	IIa	C
	Digitalis†	IIb	C
	Amiodarone	IIb	C
Stable flutter			
Conversion	Atrial or transesophageal pacing	I	A
	DC cardioversion	I	C
	Ibutilide‡	IIa	A
	Flecainide§	IIb	A
	Propafenone§	IIb	A
	Sotalol	IIb	C
	Procainamide§	IIb	A
	Amiodarone	IIb	C
Rate control	Diltiazem, verapamil	I	A
	Beta blockers	I	C
	Digitalis†	IIb	C
	Amiodarone	IIb	C

The order in which treatment recommendations appear in this table within each class of recommendation does not necessarily reflect a preferred sequence of administration. Please refer to text for details. For pertinent drug dosing information, please refer to the *ACC/AHA/ESC Guidelines for the Management of Patients With Atrial Fibrillation.*

Cardioversion should be considered only if the patient is anticoagulated (international normalized ratio equals 2 to 3), the arrhythmia is less than 48 hours in duration, or the transesophageal echocardiogram shows no atrial clots.

*All listed drugs are taken intravenously.

† Digitalis may be especially useful for rate control in patients with heart failure.

‡ Ibutilide should not be used in patients with reduced left ventricular function.

§ Flecainide, propafenone, and procainamide should not be used unless they are combined with an atrioventricular-nodal–blocking agent.

DC indicates direct current.

Recommendations for Long-Term Management of Atrial Flutter

Clinical Status/ Proposed Therapy	Recommendation	Class	Evidence
First episode and well-tolerated atrial flutter	Cardioversion alone	I	B
	Catheter ablation*	IIa	B
Recurrent and well-tolerated atrial flutter	Catheter ablation*	I	B
	Dofetilide	IIa	C
	Amiodarone, sotalol, flecainide,†‡ quinidine,†‡ propafenone,†‡ procainamide,†‡ disopyramide†‡	IIb	C
Poorly tolerated atrial flutter	Catheter ablation*	I	B
Atrial flutter appearing after use of class Ic agents or amiodarone for treatment of AF	Catheter ablation*	I	B
	Stop current drug and use another	IIa	C
Symptomatic non–CTI-dependent flutter after failed antiarrhythmic drug therapy	Catheter ablation*	IIa	B

The order in which treatment recommendations appear in this table within each class of recommendation does not necessarily reflect a preferred sequence of administration. Please refer to text for details. For pertinent drug dosing information, please refer to the *ACC/AHA/ESC Guidelines for the Management of Patients With Atrial Fibrillation.*

* Catheter ablation of the AV junction and insertion of a pacemaker should be considered if catheter ablative cure is not possible and the patient fails drug therapy.

† These drugs should not be taken by patients with significant structural cardiac disease. Use of anticoagulants is identical to that described for patients with AF.

‡ Flecainide, propafenone, procainamide, quinidine, and disopyramide should not be used unless they are combined with an atrioventricular nodal–blocking agent.

AF indicates atrial fibrillation; **CTI,** cavotricuspid isthmus.

G. Special Circumstances

1. Pregnancy

SVA occurring during pregnancy may be a particularly difficult problem. There is concern about the hemodynamic effects on the mother and fetus and the possible adverse drug effects on the fetus. Certain principles should be emphasized.
1) Arrhythmias curable by ablation should be seriously considered before planned pregnancy.
2) Most arrhythmias consist of isolated atrial or ventricular premature beats and do not require therapy.
3) Acute therapy of arrhythmias should be directed at use of nonpharmacological approaches (i.e., vagal maneuvers). Intravenous adenosine and DC cardioversion have been shown to be safe. The major concern with antiarrhythmic drug treatment during pregnancy is the potential for adverse effects on the fetus. The first 8 weeks after conception are associated with the greatest teratogenic risk. Adverse effects on fetal growth/development are the major risks during the second and third trimesters. Antiarrhythmic drug therapy should only be used if symptoms are intolerable or if the tachycardia causes hemodynamic compromise.

Recommendations for Treatment Strategies for SVT During Pregnancy (PC1)

Treatment Strategy	Recommendation	Class	Evidence
Acute conversion of PSVT	Vagal maneuver	I	C
	Adenosine	I	C
	DC cardioversion	I	C
	Metoprolol, propranolol	IIa	C
	Verapamil	IIb	C
Prophylactic therapy	Digoxin	I	C
	Metoprolol*	I	B
	Propranolol*	IIa	B
	Sotalol,* flecainide†	IIa	C
	Procainamide	IIb	B
	Quinidine, propafenone,† verapamil	IIb	C
	Catheter ablation	IIb	C
	Atenolol‡	III	B
	Amiodarone	III	C

The order in which treatment recommendations appear in this table within each class of recommendation does not necessarily reflect a preferred sequence of administration. Please refer to text for details. For pertinent drug dosing information, please refer to the *ACC/AHA/ESC Guidelines for the Management of Patients With Atrial Fibrillation.*

* Beta-blocking agents should not be taken in the first trimester, if possible.

† Consider atrioventricular nodal–blocking agents in conjunction with flecainide and propafenone for certain tachycardias (see Section V).

‡ Atenolol is categorized in class C (drug classification for use during pregnancy) by legal authorities in some European countries.

DC indicates direct current; **PSVT,** paroxysmal supraventricular tachycardia.

Specific Arrhythmias

2. Adults With Congenital Heart Disease

The treatment of SVT in adult patients with repaired or unrepaired congenital heart disease is often complicated and should be managed at experienced centers. SVAs are an important cause of morbidity and, in some patients, mortality. These patients often have multiple atrial circuits or mechanisms responsible for arrhythmias. Atrial arrhythmias can indicate deteriorating hemodynamic function, which in some cases warrants specific investigation and operative treatment. Coexistent sinus node dysfunction is common, requiring pacemaker implantation to allow management of SVTs. Cardiac malformations often increase the difficulty of pacemaker implantation and catheter ablation procedures. In addition, arrhythmia therapy by either drugs or catheter ablation must be coordinated properly within the context of surgical repair.

Recommendations for Treatment of SVTs in Adults With Congenital Heart Disease

Condition	Recommendation	Class	Evidence
Failed antiarrhythmic drugs and symptomatic:			
■ **Repaired ASD**	Catheter ablation in an experienced center	I	C
■ **Mustard or Senning repair of transposition of the great vessels**	Catheter ablation in an experienced center	I	C
Unrepaired asymptomatic ASD that is not hemodynamically significant	Closure of the ASD for treatment of the arrhythmia	III	C
Unrepaired hemodynamically significant ASD with atrial flutter*	Closure of the ASD combined with ablation of the flutter isthmus	I	C
PSVT and Ebstein's anomaly with hemodynamic indications for surgical repair	Surgical ablation of accessory pathways at the time of operative repair of the malformation at an experienced center	I	C

* Conversion and antiarrhythmic drug therapy for initial management as described for atrial flutter.

ASD indicates atrial septal defect; **PSVT,** paroxysmal supraventricular tachycardia.