Tachycardias

Tachycardias refer to arrhythmias with three or more complexes at rates exceeding 100 beats/min; they occur more often in structurally diseased than in normal hearts. Those paroxysmal tachycardias that are initiated by APCs or VPCs are considered to be due to reentry, except some of the digitalis-induced tachyarrhythmias, which are probably due to triggered activity (see below).

If the patient is hemodynamically stable, an attempt should be made to determine the mechanism and origin of the tachycardia, since this will usually lead to an appropriate therapeutic decision. Information to be obtained from the ECG includes (1) the presence, frequency, morphology, and regularity of P waves and QRS complexes; (2) the relationship between atrial and ventricular activity; (3) a comparison of the QRS morphology during sinus rhythm and during the tachycardia; and (4) the response to carotid sinus massage or other vagal maneuvers. It is useful first to compare a 12-lead ECG during the tachycardia with one recorded during sinus rhythm. One can also utilize the electrodes situated at the end of a flexible pacing catheter inserted into the esophagus behind the left atrium to record atrial activity.

Observation of the jugular venous pulse can provide clues to the presence of atrial activity and its relationship to ventricular ectopy. Intermittent cannon *a* waves suggest AV dissociation, while persistent cannon *a* waves suggest 1:1 VA conduction. Flutter waves may be seen or no atrial activity may be apparent, as in the presence of atrial flutter and atrial fibrillation (AF), respectively. The arterial pulse may also manifest AV dissociation or AF by demonstrating variations in amplitude. A first heart sound of variable intensity during a regular rhythm also suggests AV dissociation or AF.

Carotid sinus pressure should be applied only while the patient is electrocardiographically monitored with resuscitative equipment available to manage the rare episode of asystole and/or VF associated with this procedure. Carotid sinus massage should not be performed in patients with carotid arterial bruits. The patient should be positioned flat with the neck extended. Massage of one carotid bulb at a time should be performed by applying firm pressure just underneath the angle of the jaw for up to 5 s. Alternative vagomimetic maneuvers include the Valsalva maneuver, immersion of the face in cold water, and administration of 5 to 10 mg edrophonium.

SINUS TACHYCARDIA

In the adult, sinus tachycardia is said to be present when the heart rate exceeds 100 beats/min. Sinus tachycardia rarely exceeds 200 beats/min and is not a primary arrhythmia; instead, it represents a physiologic response to a variety of stresses, such as fever, volume depletion, anxiety, exercise, thyrotoxicosis, hypoxemia, hypotension, or CHF. Sinus tachycardia has a gradual onset and offset. The ECG demonstrates P waves with sinus contour preceding each QRS complex. Carotid sinus pressure usually produces modest slowing with a gradual return to the previous rate upon cessation. This contrasts with the response of PSVTs, which may slow slightly and terminate abruptly.

TREATMENT

Sinus tachycardia should not be treated as a primary arrhythmia, since it is almost always a physiologic response to a demand placed on the heart. As such, therapy should be directed to the primary disorder. However, in the setting of CHF, enhanced sympathetic activity has detrimental effects on myocardial function and merits treatment. Use of beta blockers in this situation decreases the effects of neurohormonal activation that leads to worsening CHF. Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers are additional drugs affecting neurohormonal activation in heart failure whose use improves outcomes. The other situations in which sinus tachycardia is a consequence (listed above) can all be readily treated.

ATRIAL FIBRILLATION

AF is a common arrhythmia that may occur in paroxysmal and persistent forms. It may be seen in normal individuals, particularly during emotional stress or following surgery, exercise, acute alcoholic intoxication, or a prominent surge of vagal tone (i.e., vasovagal response). It may also occur in patients with heart or lung disease who develop acute hypoxia, hypercapnia, or metabolic or hemodynamic derangements. Persistent AF usually occurs in patients with cardiovascular disease, most commonly rheumatic heart disease, nonrheumatic mitral valve disease, hypertensive cardiovascular disease, chronic lung disease, atrial septal defect, and a variety of miscellaneous cardiac abnormalities. AF may be the presenting finding in thyrotoxicosis. So-called lone AF, which occurs in patients without underlying heart disease, often represents the tachycardia phase of the tachycardia-bradycardia syndrome.

Editors' Choice: See related study. [PMID: 16790700]

The morbidity associated with AF is related to (1) excessive ventricular rate, which in turn may lead to hypotension, pulmonary congestion, or angina pectoris in susceptible individuals, and in some patients can produce a tachycardia-mediated cardiomyopathy; (2) the pause following cessation of AF, which can cause syncope; (3) systemic embolization, which occurs most commonly in patients with rheumatic heart disease (Table 214-1); (4) loss of the contribution of atrial contraction to cardiac output, which may cause fatigue; and (5) anxiety secondary to palpitations. In patients with severe cardiac dysfunction, particularly those with hypertrophied, noncompliant ventricles, the combination of the loss of the atrial contribution to ventricular filling and the abbreviated filling period due to the rapid ventricular rate in AF can produce marked hemodynamic instability, resulting in hypotension, syncope, or heart failure. In patients with mitral stenosis, in whom ventricular filling time is critical, development of AF with a rapid ventricular rate may precipitate pulmonary edema (Chap. 219). AF may also cause a cardiomyopathy related to persistent rapid rates (so-called tachycardia-induced cardiomyopathy).

Table 214–1. Factors Associated with High Risk of Stroke in Patients with Atrial Fibrillation

1. Age >65
2. Hypertension
3. Rheumatic heart disease
4. Prior stroke or transient ischemic attack
5. Diabetes mellitus
6. Congestive heart failure
7. Transesophageal echocardiographic characteristics

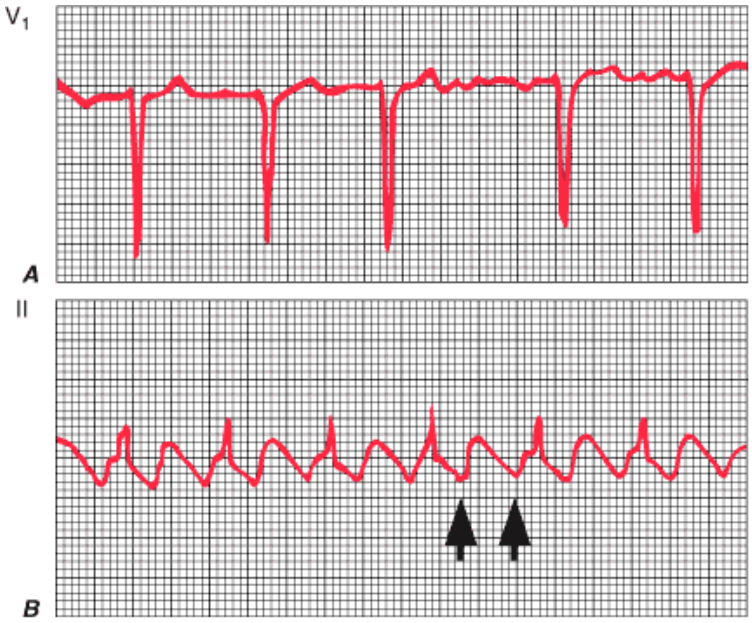
   Spontaneous echo contrast in left atrium

   Left atrial appendage velocity <20 cm/s

   Complex aortic atheroma

AF is characterized by disorganized atrial activity without discrete P waves on the surface ECG (Fig. 214-5*A*). Atrial activation is manifested by an undulating baseline or by more sharply inscribed atrial deflections of varying amplitude and frequency ranging from 350 to 600 beats/min. The ventricular response is irregularly irregular. This results from the large number of atrial impulses that penetrate the AV node, making it partially refractory to subsequent impulses. This effect of nonconducted atrial impulses to influence the response to subsequent atrial impulses is termed *concealed conduction*. As a result, the ventricular response is relatively slow, considering the actual atrial rate. AF may convert to atrial flutter, especially in response to antiarrhythmic drugs such as quinidine or flecainide. If AF converts to atrial flutter, which has a slower atrial rate, the effect of concealed conduction may be diminished, and a paradoxical increase in the ventricular response may occur. The main factor determining the rate of the ventricular response is the functional refractory period of the AV node or the most rapid paced rate at which 1:1 conduction through the AV node can be observed.

Figure 214-5



Atrial fibrillation and atrial flutter. *A*. Lead V_1 demonstrating an irregular ventricular rhythm associated with poorly defined irregular atrial activity consistent with atrial fibrillation. *B*. Lead II demonstrates atrial flutter, identified by the regular "sawtooth-like" activity (*arrows*) at an atrial rate of 300 beats/min with 2:1 ventricular response.

If, in the presence of AF, the ventricular rhythm becomes regular and slow (e.g., 30 to 60 beats/min), complete heart block is suggested, and if the ventricular rhythm is regular and rapid (e.g., ≥100 beats/min), a tachycardia arising in the AV junction or ventricle should be suspected. Digitalis intoxication is a common cause of both phenomena.

Patients with AF exhibit a loss of *a* waves in the jugular venous pulse and variable pulse pressures in the carotid arterial pulse. The first heart sound usually varies in intensity. On echocardiography, the left atrium is frequently enlarged, and in patients in whom the left atrial diameter >4.5 cm, it may be difficult to convert AF to sinus rhythm and/or maintain the latter, despite therapy.

TREATMENT

In acute AF, a precipitating factor such as fever, pneumonia, alcoholic intoxication, thyrotoxicosis, pulmonary emboli, CHF, or pericarditis should be sought. When such a factor is present, therapy should be directed toward the primary abnormality. If the patient's clinical status is severely compromised, electrical cardioversion is the treatment of choice. In the absence of severe cardiovascular compromise, slowing of the ventricular rate becomes the initial therapeutic goal. This may be most rapidly accomplished with β-adrenergic blockers and/or calcium channel antagonists. Both prolong the refractory period of the AV node and slow conduction within it. When catecholamine levels or sympathetic nervous system tone are likely to be elevated, beta blockers may be favored. Digitalis preparations are less effective, take longer to act, and are associated with more toxicity. Conversion to sinus rhythm may then be attempted. Prior to cardioversion, precautions must be taken to reduce the risk of systemic embolization. Patients should be anticoagulated to an INR of at least 1.8 for the prior 3 consecutive weeks or have had AF for <48 h. Alternatively, for those patients with AF for >48 h who are not anticoagulated, a transesophageal echocardiogram can exclude the presence of left atrial thrombus and allow safe cardioversion. Following cardioversion, anticoagulation must be maintained for at least 4 weeks until atrial mechanical function returns to normal.

Antiarrhythmic medications in either oral or intravenous form may be employed but are only modestly effective in restoring sinus rhythm. When antiarrhythmic agents such as the quinidine-like drugs (class IA) or the flecainide-like agents (class IC) are used (Table 214-2), it is important to increase AV node refractoriness prior to administering such drugs because their vagolytic effect and/or their ability to convert AF to atrial flutter may reduce the concealed conduction in the AV node and lead to an excessively rapid ventricular response. β-Adrenergic blockers are especially useful in this regard. Intravenous ibutilide, a class III agent, is reasonably effective in converting new-onset AF to sinus rhythm.

Table 214–2. Classification of Antiarrhythmic Drugs

Class I	Drugs that reduce maximal velocity of phase of depolarization (V_{max}) due to block of inward Na^+ current in tissue with fast esponse action potentials

IA	↓ V_{max} at all heart rates and ↑ action potential duration, e.g., quinidine, procainamide, disopyramide
IB	Little effect at slow rates on V_{max} in normal tissue; ↓ V_{max} in partially depolarized cells with fast response action potentials
	Effects increased at faster rates
	No change or ↓ in action potential duration, e.g., lidocaine, phenytoin, tocainide, mexiletine
IC	↓ V_{max} at normal rates in normal tissue
	Minimal effect on action potential duration, e.g., flecainide, propafenone, moricizine
Class II	Antisympathetic agents, e.g., propranolol and other β-adrenergic blockers: ↓ SA nodal automaticity, ↑ AV nodal refractoriness, and ↓ AV nodal conduction velocity
Class III	Agents that prolong action potential duration in tissue with fast-response action potentials, e.g., bretylium, amiodarone, sotalol, ibutilide, dofetilide
Class IV	Calcium (slow) channel blocking agents: ↓ conduction velocity and ↑ refractoriness in tissue with slow-response action potentials, e.g., verapamil, diltiazem
Drugs that cannot be classified by this schema: Digitalis, Adenosine	

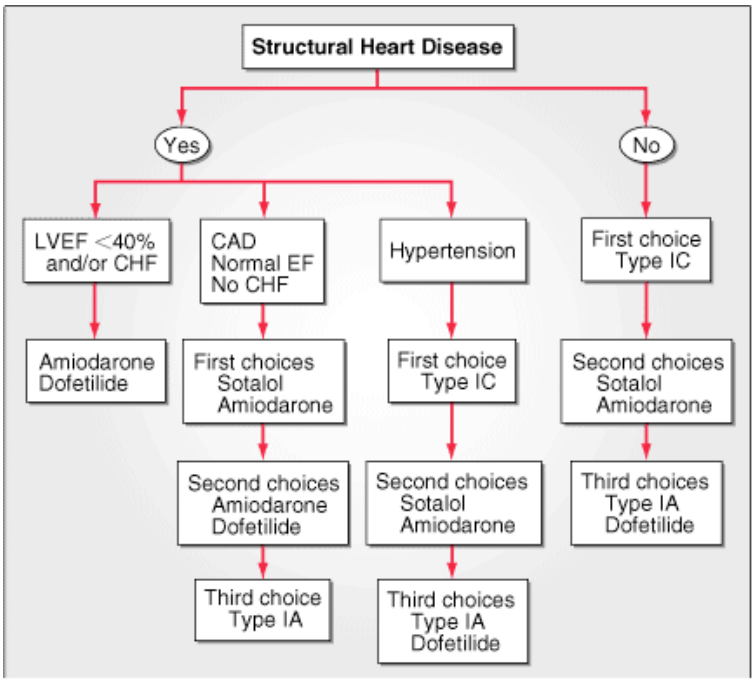
Note: SA, sinoatrial; AV, atrioventricular.

Direct-current (DC) electrical cardioversion is a highly effective method to restore sinus rhythm, either as a primary method of therapy or following the failure of antiarrhythmic medications. DC cardioversion is accomplished through the delivery of at least 200 W·s of energy between electrodes placed to the right of the sternum and the cardiac apex or to the left of the scapula. New methods of cardioversion using biphasic waveforms have increased the efficacy of transthoracic cardioversion to >90%. If external cardioversion is unsuccessful, internal cardioversion with energy delivered between two catheters inside the heart or one inside and a patch outside the heart may prove effective. Recent studies suggest pretreatment with ibutilide can facilitate cardioversion.

It is unlikely that patients with chronic AF will convert to and remain in sinus rhythm in the presence of long-standing rheumatic heart disease and/or when the atria are markedly enlarged. The goal of therapy in patients in whom AF cannot be converted to sinus rhythm is control of the ventricular response. This can usually be accomplished by beta blockers, calcium channel blockers, or digitalis, singly or in combination. In occasional patients, the ventricular response cannot be controlled by pharmacologic therapy alone. In such patients, the creation of complete heart block by radiofrequency catheter ablation of the AV junction followed by permanent pacemaker implantation is appropriate.

If sinus rhythm is restored electrically or pharmacologically, quinidine or related agents as well as the class IC agents (e.g., flecainide or propafenone), sotalol, dofetilide, or amiodarone may be used to prevent recurrence. In patients in whom cardioversion is unsuccessful or in whom AF has recurred or is likely to recur despite antiarrhythmic therapy, it is probably wisest to allow the patient to remain in AF and to control the ventricular response with calcium antagonists, β-adrenergic blockers, or digitalis glycosides. Since such patients are always at risk of systemic embolization, particularly in the presence of organic heart disease, chronic anticoagulation must be considered (Table 214-3). Chronic anticoagulation is particularly important in the elderly, where the attributable risk of AF for stroke approaches 30%. Several studies have now demonstrated conclusively that the incidence of embolization in patients with AF not associated with valvular heart disease is reduced by chronic anticoagulation with warfarin-like agents. Recommendation for chronic anticoagulation should be instituted based on clinical risk factors for stroke regardless of whether or not an antiarrhythmic medication is utilized. Studies have demonstrated that antiarrhythmic drugs are not associated with stroke reduction perhaps because of asymptomatic (unrecognized) recurrences. Aspirin also may be effective for this purpose in patients who are not at high risk for stroke. Although anticoagulation may be associated with hemorrhagic complications, the risk is largely associated with INRs above the recommended range of 2.0 to 3.0. Recommendations for the selection of antiarrhythmic medications to prevent the recurrence of AF are shown in Fig. 214-6.

Figure 214-6



Recommendations for the selection of antiarrhythmic medications to prevent the recurrence of atrial fibrillation. See Tables 214-2 and 214-4 for definition of class IA and IC drugs. An atrioventricular nodal blocking agent (i.e., beta blocker, calcium channel blocker, or digoxin) should be added to all class IC and IA agents as well as to dofetilide. LVEF, left ventricular ejection fraction; CHF, congestive heart failure; CAD, coronary artery disease; EF, ejection fraction.

Table 214–3. Recommendations for Long-Term Anticoagulation in Patients with Chronic Atrial Fibrillation

Age, years	Risk Factors[a]	Recommendations
<65	Absent	Aspirin
	Present	Warfarin[target INR 2.5 (range 2.0–3.0)]
65–75	Absent	Aspirin or warfarin
	Present	Warfarin[target INR 2.5 (range 2.0–3.0)]
>75	All patients	Warfarin[target INR 2.5 (range 2.5–3.0)]

[a]Risk factors are prior transient ischemic attack, systemic embolus or stroke, hypertension, poor left ventricular function, rheumatic mitral valve disease, prosthetic heart valve, congestive heart failure.

V Fuster et al: ACC/AHA/ESC guidelines for the management of patients with atrial fibrillation: executive summary. A Report of the American College of Cardiology/ American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines and Policy Conferences (Committee to Develop Guidelines for the Management of Patients With Atrial Fibrillation): developed in Collaboration With the North American Society of Pacing and Electrophysiology. J Am Coll Cardiol. 2001 Oct;38(4):1231-66.

Ablation therapy for cure of AF is an active area of investigation. This therapy is generally employed for patients with paroxysmal AF. This type of AF is often triggered by automatic foci located in the pulmonary veins. Ablation around the pulmonary veins to prevent electrical transit of impulses from the pulmonary veins to and from the left atrium may be curative. While ablation or isolation of these foci is possible, the procedure can result in pulmonary vein stenosis, pulmonary hypertension, and stroke. The MAZE procedure is a surgical approach to cure AF through the creation of multiple scars in the right and left atria to compartmentalize the electrical conduction in these chambers and disallow the propagation of fibrillatory waves. The morbidity, mortality, and success rate of such catheter-based procedures render them experimental at this time.

ATRIAL FLUTTER

This arrhythmia occurs most often in patients with organic heart disease. Flutter may be paroxysmal, in which case there is usually a precipitating factor, such as pericarditis or acute respiratory failure, or it may be persistent. Atrial flutter (as well as AF) is very common during the first week following open-heart surgery. Atrial flutter is usually less long-lived than is AF, although on occasion it may persist for months to years. Often, if it lasts for more than a week, atrial flutter will convert to AF. Systemic embolization is less common in atrial flutter than in AF.

Atrial flutter is characterized by an atrial rate between 250 and 350 beats/min. Typically, the ventricular rate is half the atrial rate, i.e., ~150 beats/min because of 2:1 block in the AV node. If the atrial rate is slowed to <220 beats/min by antiarrhythmic agents such as quinidine, which also possess vagolytic properties, the ventricular rate may rise suddenly because of the development of 1:1 AV conduction. Classically, flutter waves are seen as regular sawtooth-like atrial activity, most prominent in the inferior leads (Fig. 214-5*B*). When the ventricular response is regular and not a simple fraction of the atrial rate, complete AV block is present, which may be a manifestation of digitalis toxicity. Activation mapping suggests that atrial flutter is a form of atrial reentry localized to the right atrium.

TREATMENT

The most effective treatment of atrial flutter is DC cardioversion, which can be accomplished at low energy (25 to 50 W·s) under mild sedation. Higher energies (100 to 200 W·s) are often used because they are less likely to cause AF, which not infrequently occurs following lower energy delivery. Although atrial flutter is associated with a slightly lower risk of embolization than AF, the same precautions should be followed in regard to anticoagulation as are used with AF. The reason for the increased risk of emboli in atrial flutter is uncertain, but the coexistence of AF is common. In patients who develop atrial flutter following open-heart surgery or recurrent flutter in the setting of acute myocardial infarction, particularly if they are being treated with digitalis, atrial pacing (using temporary pacing wires implanted at the time of operation or a pacing lead inserted into the atrium pervenously) at rates of 115 to 130% of the atrial flutter rate can usually convert the atrial flutter to sinus rhythm. Atrial pacing may also result in the conversion of atrial flutter to AF, which allows for easier control of the ventricular response.

If immediate conversion of atrial flutter is not mandated by the patient's clinical status, the ventricular response should first be slowed by blocking the AV node with a beta blocker, calcium antagonist, or digitalis. Digitalis is the least effective and occasionally converts atrial flutter into AF. Once AV nodal conduction is slowed with any of these drugs, an attempt to convert flutter to sinus rhythm using a class I (A or C) agent or amiodarone should be made. Increasing doses of the drug selected are administered until the rhythm converts or side effects occur. Ibutilide is a new antiarrhythmic agent that is administered intravenously and appears to be particularly effective for conversion of atrial flutter to sinus rhythm.

Quinidine, other class IA drugs, flecainide, propafenone, sotalol, dofetilide, and amiodarone (Table 214-4) may be useful in preventing recurrences of atrial flutter. Radiofrequency ablation is a highly effective treatment for patients with the most typical forms of atrial flutter, which are due to reentry around the tricuspid valve in a counterclockwise or clockwise fashion. The coronary sinus and inferior vena cava cause the wavefront of activation to pass between them and the tricuspid valve. Ablation of the narrowed isthmus using radiofrequency energy can cure flutter in >85% of cases. This is far more successful than the response to drugs. As such, ablation is considered by many to be the therapy of choice for recurrent atrial flutter.

Table 214–4. Drugs Used to Treat Cardiac Tachyarrhythmias

Drug	Mode of Administration	$t_{1/2}$ (oral), h	Route of Metabolism	Clinical Effects and/or Indications for Use
Digoxin	IV, 0.25–1.5 mg Oral, 0.75–1.5 mg loading dose over 12–24 h Maintenance, 0.23–0.50 mg/kg	36	Renal	Slowing of ventricular rate during AF, flutter, and other atrial tachycardias in the absence of preexcitation; slowing, termination, and/or prevention of SVT due to AV nodal reentry and AV reentry utilizing bypass tracts; may terminate or prevent intraatrial reentrant tachycardias; ineffective in prevention of automatic atrial tachycardias
Adenosine	IV bolus, 6–12 mg	<10s		Acute termination of regular reentrant SVT involving the AV node
Quinidine (class IA)	Oral, 200–400 mg q6h	8–9	Hepatic, 80% Renal, 20%	Atrial and ventricular tachyarrhythmias; all types of SVT; control of ventricular rate in patients with preexcitation and AF and flutter
Procainamide (class IA)	IV, 40–50 mg/min to total of 10–20 mg/kg	3–5	Hepatic, 50%	Same as quinidine

(class IA)	total of 10–20 mg/kg Oral, 500–1000 mg q6h (sustained-release forms)		Renal, 50%	
Disopyramide (class IA)	Oral, 100–300 mg q6h	8–9	Hepatic, 50% Renal, 50%	Same as quinidine
Lidocaine (class IB)	IV, 20–50 mg/min to total of 5 mg/kg loading dose, followed by 1–4 mg/kg	1–2	Hepatic	VT and VF, especially during acute ischemia and myocardial infarction
Phenytoin (class IB)	IV, 20 mg/kg to total dose of 1000 mg Oral, 1000-mg loading dose over 24 h Maintenance, 100–400 mg/d	18–36	Hepatic	Tachyarrhythmias induced by digitalis; occasionally effective for ventricular tachyarrhythmias not induced by digitalis alone or in combination with other antiarrhythmic agents; polymorphic VT associated with increased QT interval
Mexiletine (class IB)	Oral, 100–300 mg q6–8h	9–12	Hepatic	Ventricular tachyarrhythmias; secondary agent in combination with other class I medication
Flecainide (class IC)	Oral, begin at 50–100 mg bid, increase by ≤50 mg in 4-day intervals to a maximum of mg daily	7–23	Hepatic, 75% Renal, 25%	Supraventricular tachyarrhythmias including atrial fibrillation and flutter; also ventricular arrhythmias refractory to other medications or radiofrequency ablation
Propafenone (class IC)	Oral, 150–300 mg q8h	5–8	Hepatic	Same as flecainide
Moricizine (class IC)	Oral, 200–400 mg q8h	2–6	Hepatic	Same as flecainide
Beta blockers (class II) e.g., metoprolol	IV, load with 5–10 mg q5min for 3 doses, then 3 mg q6h Oral, 25–100 mg bid	3–4	Hepatic	Slowing of ventricular rate during AF, atrial flutter, and other atrial tachyarrhythmias in the absence of preexcitation; SVT due to AV nodal reentry; reentry utilizing bypass tracts; arrhythmias (e.g., VT) induced by exercise or occurring in the presence of hyperthyroidism; polymorphic VT associated with congenital long QT syndrome
Bretylium (class III)	IV, 1–2 mg/kg per min to total load, 5–10 mg/kg Maintenance, 0.5–2 mg/kg	8–14	Renal	Refractory VT and VF, especially due to acute ischemia
Amiodarone (class III)	IV, 5–10 mg/kg load over 20 min, then 1 g/24 h		Hepatic	Sustained VT and VF
	Oral, load 800–1600 mg/d for 1 week, then 400–600 mg/d for 3 weeks, then 200–400 mg/d thereafter	13–103		AF and atrial flutter, other types of SVT, VT, VF
Sotalol (class III)	Oral, 80–320 mg q12h	10–20	Renal, 90% Hepatic, 10%	Atrial and ventricular tachyarrhythmias
Ibutilide (class III)	IV, <60 kg: 0.01 mg/kg over 10 min IV, ≥60 kg: 1 mg over 10 min, repeat after 10 min if no effect	2–6	Hepatic	AF, atrial flutter, and other SVTs including preexcitation tachycardias
Dofetilide	125–250 mg PO bid	8–12	Renal	AF, atrial flutter, and other SVTs

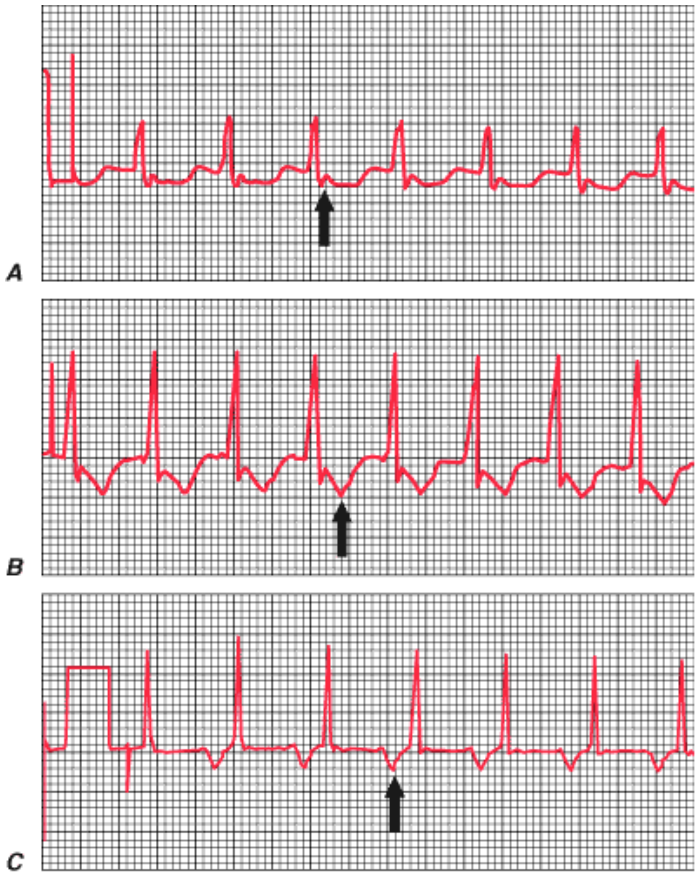
Calcium channel blockers (class IV) e.g., verapamil	IV, 2.5–10 mg over 1–2 min to total of 0.15 mg/kg Oral, 240–480 qd	6–24	Hepatic	Slowing of ventricular rate during AF and flutter, and other SVTs in the absence of preexcitation; idiopathic VT
Diltiazem	IV, load with 0.25 mg/kg over 2 min; if needed, repeat after 15 min with 0.35 mg over 2 min Maintenance, 10–15 mg/h		Hepatic	Same as verapamil

Note: AF, atrial fibrillation; AV, atrioventricular; SVT, supraventricular tachycardia; VF, ventricular fibrillation; VT, ventricular tachycardia.

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIAS

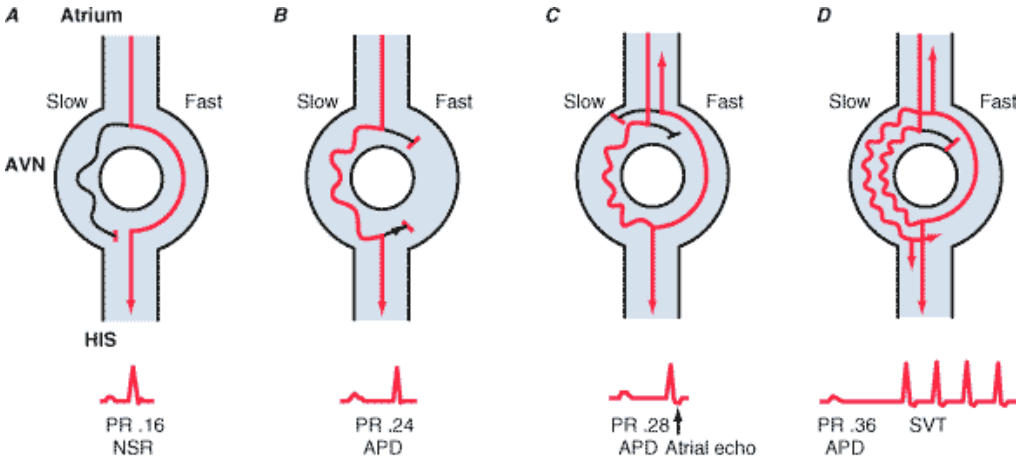
Fig. 214-7. In most cases, functional differences in conduction and refractoriness in the AV node or the presence of an AV bypass tract provide the substrate for the development of PSVT (previously termed *paroxysmal atrial tachycardia*). Electrophysiologic studies have demonstrated that reentry is responsible for the vast majority of cases of PSVT. Reentry has been localized to the sinus node, atrium, AV node, or a macroreentrant circuit involving conduction in the antegrade direction through the AV node and retrograde through an AV bypass tract (Fig. 214-8). Such a bypass tract may also conduct antegradely, in which case the Wolff-Parkinson-White (WPW) syndrome is said to be present. When the bypass tract manifests only retrograde conduction, it is termed a *concealed bypass tract* (Fig. 214-7*B*). In these cases, the QRS complex during sinus rhythm is normal. In the absence of the WPW syndrome, reentry through the AV node or through a concealed bypass tract makes up nearly 90% of all PSVTs. Atrial tachycardias due to automaticity are not paroxysmal and often present as an incessant arrhythmia.

Figure 214-7



Examples of supraventricular tachycardia (SVT). Arrows indicate P waves. *A*. AV nodal reentry. Upright P waves are visible at the end of the QRS complex. *B*. AV reentry using a concealed bypass tract. Inverted retrograde P waves are superimposed on the T waves. *C*. Automatic atrial tachycardia. Inverted P waves follow the T waves and precede the QRS complex.

Figure 214-8



Mechanism of AV nodal reentry: The atrium, AV node (AVN), and His bundle are shown schematically. The AV node is longitudinally dissociated into two pathways, slow and fast, with different functional properties (see text). In each panel of this diagram, red lines denote excitation in the AV node, which is manifest on the surface electrocardiogram (ECG), while black lines denote conduction that is concealed and not apparent on the surface electrocardiogram. *A*. During sinus rhythm (NSR) the impulse from the atrium conducts down both pathways. However, only conduction over the fast pathway is manifest on the surface ECG, producing a normal PR interval of 0.16 s. *B*. An atrial