

# Supraventricular tachycardia: Implications for the intensivist

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**The critical care physician must have a keen awareness of supraventricular tachycardia patterns, mechanisms, precipitants, and treatment. Although long-term management of most forms of supraventricular tachycardia lies primarily in the realm of the cardiac electrophysiologist, the intensivist must be proficient at acute arrhythmia therapy. Expertise in electrocardiography, phar-**

**macokinetics, and pharmacodynamics is essential. Careful assessment of hemodynamics and prudent bedside clinical acumen help assure optimal patient outcomes. (Crit Care Med 2000; 28[Suppl.]:N129–N135)**

**KEY WORDS: supraventricular tachycardia; pharmacotherapy; atrial fibrillation**

**D**uring the last 10–15 yrs, dramatic advances in radiofrequency catheter ablation have placed long-term management of supraventricular tachycardias squarely in the hands of the cardiac electrophysiologist. Paroxysmal supraventricular tachycardias and typical atrial flutter are curable in >90% of patients who suffer from these maladies. Ablation of atypical atrial flutter is also possible (1) and certain forms of paroxysmal atrial fibrillation are responsive to focal application of radiofrequency energy (2–4).

The substrate for most types of supraventricular tachycardia is present before admission to an intensive care unit. Notable exceptions include atrial fibrillation (and flutter) after open-heart surgery and multifocal atrial tachycardia, each of which may be transient, requiring no chronic therapy. Conditions such as hypoxemia, electrolyte imbalance, catecholamine excess (endogenous and exogenous), and other metabolic disturbances predispose patients (with and without preexisting arrhythmic substrates) to tachyarrhythmias. The nonelectrophysiologist must be prepared for acute management of supraventricular tachycardia. To accomplish this, an intensivist must have an understanding of basic arrhythmia mechanisms, appropriate choices for acute pharmacotherapy, and indications

for urgent or emergent direct current cardioversion.

## **PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA**

There are five types of paroxysmal supraventricular tachycardia (PSVT). Atrioventricular (AV) nodal reentry tachycardia (AVNRT) is by far the most common and accounts for 50% to 60% of PSVT evaluated at referral centers (5). Although the precise reentrant circuit is not well defined, it is clear that the anterior and posterior AV nodal approaches and the perinodal atrial tissue are involved.

AVNRT is uncommon in childhood, although one tertiary center reported that 9% of the patients referred to them for ablation of AVNRT were  $\leq 16$  yrs old (6), and usually presents after the age of 20. It is more common in women than men. The typical heart rate in AVNRT ranges from 150 beats/min to 250 beats/min. Palpitations, lightheadedness, and near-syncope may accompany an episode. True syncope is unusual. Neck pounding is virtually pathognomonic (7). Its absence does not exclude AVNRT.

In 76% to 90% of cases, antegrade conduction proceeds via the posterior (slow) AV nodal approach (pathway) and retrograde conduction via the anterior (fast) AV nodal pathway (5, 6). This is slow-fast AVNRT. Because retrograde conduction is so rapid, atrial and ventricular activation are virtually simultaneous. P waves may not be visible on the surface electrocardiogram (ECG) or appear in the terminal portion of the QRS complex (Fig. 1). Atrial contraction on a closed AV valve may produce neck pound-

ing. Unusual variants of AV nodal reentry also exist. There are fast-slow, slow-slow, and slow-sort of slow circuits.

Atrioventricular reentry (AVRT) is the next most common PSVT, accounting for 30% of PSVT. Atrioventricular reentry, also commonly referred to as orthodromic tachycardia, presents on average at a somewhat earlier age than AVNRT. The antegrade limb of the circuit proceeds down the normal AV nodal-His-Purkinje system. The retrograde limb uses an accessory pathway that usually is located along the mitral valve annulus. Because the accessory pathway conducts only retrograde, it is not seen (even in sinus rhythm) on surface electrocardiogram and is, therefore, said to be concealed.

Because AVRT normally proceeds antegrade, the QRS complex is generally narrow. A short RP interval and longer PR interval is typical. Because AVNRT and AVRT activate the periannular atrial tissue first, P waves (if visible on surface electrocardiogram) will be negative in the inferior leads. Upright P waves in these leads indicates atrial (or sinus) tachycardia. AVRT tends to go faster than AVNRT and is more prone to present with QRS alternans on surface electrocardiogram. Slowing of tachycardia rate on development of bundle branch block ipsilateral to the pathway is characteristic of AVRT. AV block is unusual during AVNRT and excludes the diagnosis of AVRT, which requires both atrial and ventricular participation. The presence of AV block strongly suggests the diagnosis of atrial tachycardia.

Intra-atrial reentry, automatic atrial tachycardia, and sinus nodal reentry account for the other 8% to 10% of PSVT.

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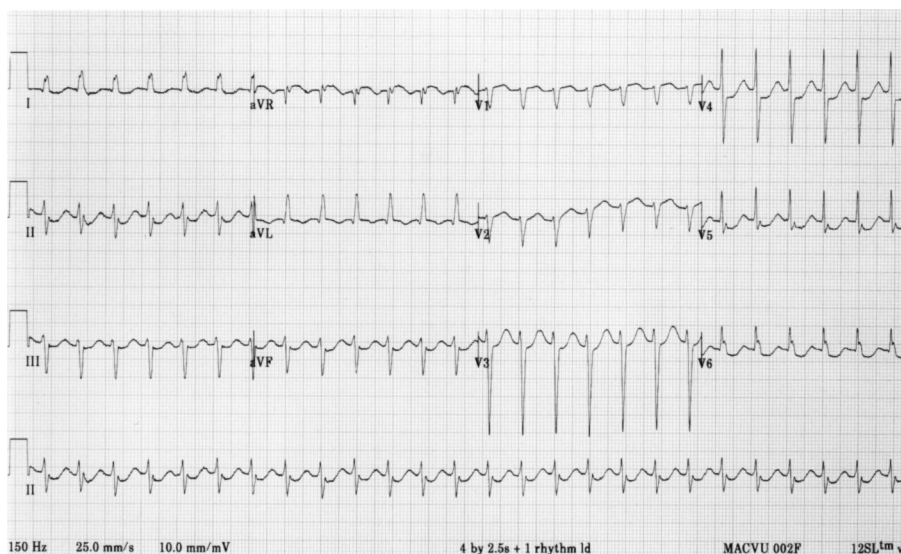


Figure 1. A narrow-complex tachycardia is present. Although there is some distortion of the terminal portion of the QRS complexes, clear P waves are not discernible. This is compatible with typical (slow-fast) AV nodal reentry. I, II, III, aVR, aVL, aVF, V<sub>1</sub>-V<sub>6</sub>, standard ECG leads.

Although sinus node reentry is said to account for 3% of PSVT, it rarely occurs as an isolated phenomenon (8).

Intra-atrial reentry requires a circuit with unidirectional block and a zone of slow conduction. Approximately 50% of patients with intra-atrial reentry have evidence of structural heart disease (9). This tachycardia is particularly prone to develop after surgery for congenital cardiac anomalies. Reentry occurs around structural barriers, such as suture lines. In patients without clear-cut structural disease, subtle changes such as scarring and fibrosis provide the substrate for reentry.

Automatic atrial tachycardias occur along the crista terminalis, near the ostium of the coronary sinus, along the tricuspid and mitral annuli, in both atrial appendages, and at the ostia of the pulmonary veins. They are exquisitely sensitive to catecholamines. Although these tachycardias may present in the absence of structural heart disease or obvious precipitants, they are also commonly associated with chronic lung disease, pneumonia, myocardial (atrial) infarction, and acute alcoholic binges. Amphetamine or cocaine abuse may precipitate these tachyarrhythmias.

Reentrant atrial tachycardia tends to be paroxysmal, whereas automatic forms are more likely to be incessant. Both tend to have atrial rates <200 beats/min. When the ventricular rate exceeds 120 beats/min >75% of the time, tachycardia-mediated cardiomyopathy may ensue.

The presence of atrioventricular block during tachycardia provides strong evidence that the rhythm disturbance is atrial in origin. Negative P waves are not helpful in differentiating atrial tachycardia from AVNRT or AVRT. An inferior P wave axis with a negative P in lead I is diagnostic of left atrial tachycardia. An inferior P axis, a positive P in lead I, and a P wave morphology different from sinus rhythm can only result from atrial tachycardia.

Sinus node reentry may occur within the sinus node, the perinodal atrial tissue, or both. Although the mechanism may be difficult to prove clinically, most investigators agree that the P wave may be nearly identical to sinus rhythm, suggesting the reentrant exit point may differ slightly from sinus pacemaker beats. Average rates are generally 130–140 beats/min. (range, 80–200 beats/min).

*Approach to Paroxysmal Supraventricular Tachycardia Therapy.* Acute management of PSVT should begin with attempts to slow or (transiently) interrupt AV nodal conduction. Vagal maneuvers (such as carotid sinus massage or Valsalva) may be tried first. Adenosine is the initial drug of choice for acute management of PSVT. An initial intravenous dose of 6 mg may be followed 2 mins later by a second 6 mg (if necessary), and 12 mg may be given 2 mins later if 6 mg is unsuccessful.

Adenosine should terminate >90% of AVNRT and AVRT. It is also effective for sinus node reentry. Adenosine may also

terminate automatic atrial tachycardias, particularly those originating near the crista terminalis where vagal innervation is rich. Termination may be transient because of adenosine's short half-life (~10 secs).

Intravenous verapamil (5–10 mg) is usually effective for termination of PSVT when adenosine fails. AV block without arrhythmia termination again suggests the diagnosis of atrial tachycardia. Because of its longer half-life, verapamil may also be effective for prompt tachycardia recurrence after initial success with adenosine.

Automatic atrial tachycardia is difficult to manage with pharmacotherapy. Precipitants should be treated or eliminated whenever possible. Beta-blockers may slow atrial rate, but rarely restore sinus rhythm. Adenosine may produce sinus rhythm; however, tachycardia generally resumes as soon as the drug is metabolized (10). Vagal maneuvers may produce AV block but do not terminate these arrhythmias (11). Clinical successes have occurred with class IC agents and amiodarone. Flecainide should be avoided in patients with coronary artery disease and prescribed with great caution in patients with significant left ventricular dysfunction. In contrast to flecainide, amiodarone is available in the United States for intravenous administration. Intravenous amiodarone may result in hypotension (vasodilatation) and may have negative inotropic effects, particularly in the setting of preexisting left ventricular dysfunction.

Sinus node reentry may respond to vagal maneuvers, adenosine, verapamil and digitalis (relatively slow onset of action limits acute application). Acute management of intra-atrial reentry is similar to atrial fibrillation in the absence of antegrade accessory pathway conduction.

Emergent or urgent direct current cardioversion is indicated when PSVT results in angina pectoris, congestive heart failure, or hypotension. Techniques for, and limitations of, direct current cardioversion are discussed separately in this issue of *Critical Care Medicine*. Automatic tachycardias do not respond to direct current cardioversion.

## Wolff-Parkinson-White Syndrome and its Variants

The ECG pattern of Wolff-Parkinson-White (WPW) syndrome (Fig. 2), short PR interval, wide QRS complex with preexci-

tation (delta wave), has a reported prevalence of 0.1 to 0.3% in the general population. It is twice as common in males as females.

Classic WPW syndrome occurs when accessory pathway conduction is bidirectional (atrioventricular and ventriculoatrial). Symptomatic presentation usually occurs during teenage years or early adulthood. Pregnancy may exacerbate symptoms. The most common tachycardia is atrioventricular reentry antegrade down the AV node and His-Purkinje system, retrograde up the bypass tract, which is identical to AVRT involving a concealed bypass tract. Approximately 25% of patients with this ECG pattern are incapable of retrograde conduction and therefore do not have orthodromic AVRT. Asymptomatic patients generally have a benign prognosis; however, the initial presentation may be ventricular fibrillation (11).

Accessory pathways generally have conduction properties similar to myocardium. Decremental conduction (characteristic of the AV node) is uncommon. Pathways may, therefore, be capable of very rapid antegrade (AV) conduction. In these instances, atrial fibrillation may be associated with irregular wide QRS tachycardia and ventricular rates >300 beats/min (Fig. 3). Syncope or sudden cardiac death (as a result of degeneration into ventricular fibrillation) may ensue.

Regular wide QRS tachycardia in patients with WPW may have several mechanisms. Aberrancy resulting from right or left bundle branch block (fixed or functional) may occur during orthodromic AVRT. As noted, bundle branch block ipsilateral to the accessory pathway may slow tachycardia rate.

Antidromic tachycardia occurs when antegrade conduction proceeds via a free-wall accessory pathway and retrograde conduction occurs over the normal His-Purkinje-AV nodal route. The ventricles are activated eccentrically beginning at the annular insertion of the accessory pathway. The resulting maximally preexcited wide QRS rhythm may be difficult to distinguish from ventricular tachycardia. Patients with multiple accessory pathways may have preexcited tachycardias using separate accessory pathways for antegrade and retrograde conduction. Bystander antegrade accessory pathway conduction may occur during AVNRT or atrial tachycardia. Atrial flutter may be conducted regularly or rapidly and irregularly like atrial fibrillation.

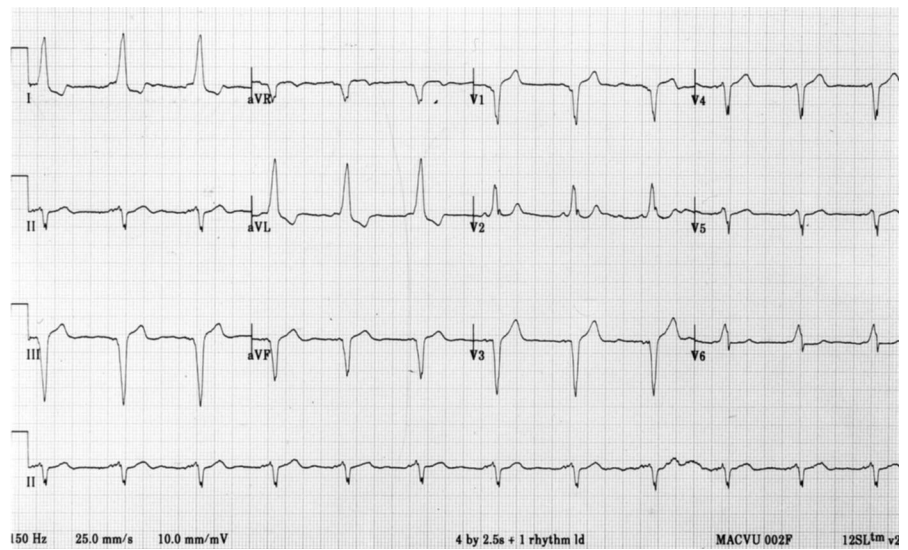


Figure 2. The short PR intervals and wide QRS complexes with slurred upstrokes (delta waves) are characteristic of manifest preexcitation in the Wolff-Parkinson-White Syndrome. This right posteroseptal accessory pathway was successfully ablated. I, II, III, aVR, aVL, aVF, V<sub>1</sub>-V<sub>6</sub>, standard ECG leads.

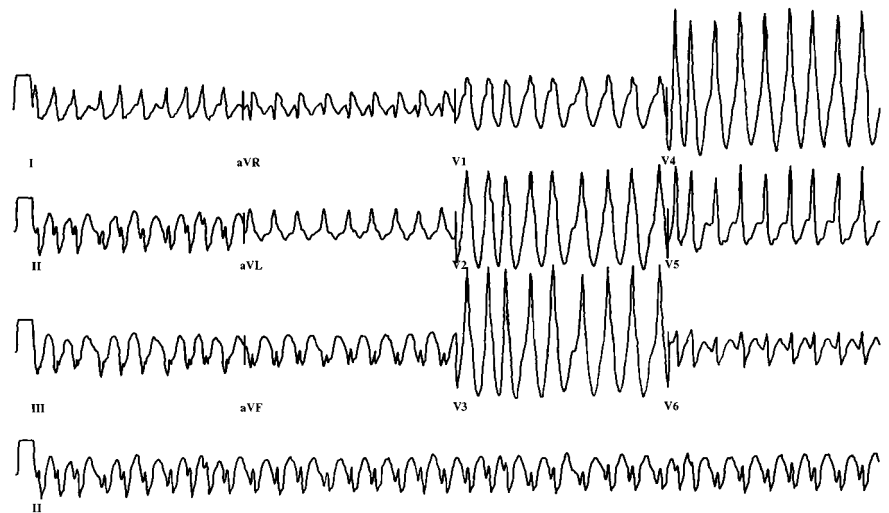


Figure 3. A rapid irregularly, irregular wide QRS tachycardia is present. Preexcited atrial fibrillation, as seen here, may degenerate to ventricular fibrillation. This ECG pattern should be promptly recognized by all practicing clinicians. I, II, III, aVR, aVL, aVF, V<sub>1</sub>-V<sub>6</sub>, standard ECG leads.

Although accessory pathways may be located anywhere along the atrioventricular groove except where the mitral and aortic annuli connect, two variants with characteristic locations deserve mention. Atriofascicular pathways (Mahaim fibers) connect the right atrium and right bundle branch. During sinus rhythm there is minimal or no preexcitation. Typical Mahaim reentry goes antegrade down the bypass tract and retrograde via the normal conduction system, usually beginning with the right bundle branch. A regular wide QRS rhythm with a typical left bundle branch block pattern is seen. These pathways conduct antegrade in a

decremental fashion (retrograde conduction is absent) and occur much less frequently than typical atrioventricular accessory pathways. Patients with atriofascicular fibers frequently have multiple accessory pathways and/or AVNRT (12).

Paroxysmal junctional reciprocating tachycardia is a form of AV reentry that may be nearly incessant. The antegrade limb of the circuit is the normal AV conduction system. The retrograde limb is a concealed, decrementally conducting accessory pathway usually located posteroseptally. Incessant tachycardia may result in a tachycardia-mediated cardiomyopathy.

*Acute Management of Tachycardia Associated with Wolff-Parkinson-White Syndrome.* Acute tachycardia treatment depends on characteristics of its QRS complexes. A 12-lead ECG should be obtained whenever possible.

Orthodromic AVRT usually presents with a narrow QRS complex; functional or fixed bundle branch block can widen the QRS. Treatment should begin with vagal maneuvers. If these do not terminate the arrhythmia, intravenous adenosine is the initial drug of choice. Intravenous verapamil should be administered when adenosine is ineffective. These therapeutic interventions interrupt tachycardia by creating transient AV nodal conduction block.

Orthodromic AVRT may degenerate to preexcited atrial fibrillation. If verapamil has already been given, hypotension may ensue. Adenosine has been used as a provocative measure to induce atrial fibrillation in preparation for focal ablation and could, in theory, induce the rapid preexcited version of this tachyarrhythmia. Once this has occurred, treatment should proceed as outlined below for wide QRS tachycardias.

Treatment of wide QRS tachycardia should be directed at blocking conduction via the accessory pathway. Although adenosine may terminate antidromic tachycardia (at the AV node), it will not affect atrial tachyarrhythmias conducting rapidly across the accessory pathway. Because of its short half-life, adenosine administration poses little risk, unless it precipitates atrial fibrillation.

Verapamil is contraindicated in the presence of wide QRS tachycardia. Its hypotensive effects may make patients hemodynamically unstable or even contribute to the onset of ventricular fibrillation. Intravenous digoxin is not given to patients with WPW and atrial fibrillation because it may (in one third of patients) enhance antegrade accessory pathway conduction and likewise result in degeneration to ventricular fibrillation.

Patients having supine systolic blood pressure  $\geq 90$  mm Hg can be given intravenous procainamide. It is administered in a loading dose of 10 mg/kg infused at a rate  $\leq 50$  mg/min. Blood pressure should be monitored every minute and the infusion rate should be decreased if hypotension develops. Procainamide will depress conduction across the accessory pathway, decrease ventricular rate, and stabilize

the patient. It may also terminate the wide QRS tachycardia.

Direct current cardioversion should be available, preferably at the bedside, whenever treatment of a wide QRS tachycardia is undertaken. If, at baseline, there are signs of hemodynamic compromise (angina, heart failure, or hypotension), drug therapy should be eschewed and direct current cardioversion used to promptly restore sinus rhythm. If hemodynamic instability develops during drug therapy, DC cardioversion should be performed immediately. DC cardioversion should be the next elective therapy when pharmacotherapy is unsuccessful.

#### **AUTOMATIC ATRIOVENTRICULAR JUNCTIONAL AND NONPAROXYSMAL ATRIOVENTRICULAR JUNCTIONAL TACHYCARDIAS; PAT WITH BLOCK**

Automatic AV junctional tachycardia, also known (particularly in pediatrics) as junctional ectopic tachycardia, primarily affects children and infants. It is often incessant. In patients without congenital heart disease, it may present as a tachycardia-mediated cardiomyopathy. This tachyarrhythmia results in marked hemodynamic deterioration after corrective surgery for congenital heart disease. It generally appears within 12 hrs postoperatively and terminates within a few days if the patient survives.

Digitalis, beta-blockers, and class IA antiarrhythmics are ineffective in children. Amiodarone (which may suppress tachycardia or control its rate) should be administered when rates  $< 150$  beats/min cannot be achieved by other means (13). In adults, beta blockade may successfully control the rate. Adult automatic AV junctional tachycardia may be difficult to manage medically. Recent reports suggest that catheter ablation can eliminate

tachycardia while preserving AV conduction (14, 15).

Nonparoxysmal AV junctional tachycardia occurs primarily in the setting of digitalis toxicity. It is also associated with cardiac surgery, myocardial infarction, and rheumatic fever. Hypokalemia may cause or exacerbate this arrhythmia. Sympathetic stimulation increases the tachycardia rate.

Digitalis toxicity may also precipitate atrial tachycardia (so-called PAT with block). This tachycardia is usually managed by withholding digoxin and administering potassium. Lidocaine, phenytoin, and digoxin antibody fragments may also be used. Direct current cardioversion is not effective treatment for automatic arrhythmias. Shocks delivered in the setting of digitalis toxicity may result in refractory ventricular fibrillation.

#### **MULTIFOCAL ATRIAL TACHYCARDIA**

Multifocal atrial tachycardia (MAT) is generally regarded to be an automatic arrhythmia. It is characterized by multiple (three or more) morphologically distinct (nonsinus) P waves, atrial rates of 100–130 beats/min, and variable AV block (Fig. 4).

MAT is commonly associated with respiratory disease and congestive heart failure. Hypoxemia is a frequent finding. MAT may be exacerbated by digitalis or theophylline toxicity, hypokalemia, hypomagnesemia, and hyponatremia. These precipitants usually do not result in MAT if respiratory decompensation is absent. Although MAT is an uncommon arrhythmia in general, it is relatively common in the critical care setting. It has been reported in patients with cancer, lactic acidosis, pulmonary emboli, renal disease, and infection.

MAT may superficially resemble atrial fibrillation. Careful examination of a 12-lead ECG may be required to distinguish these entities. Differentiation is impor-

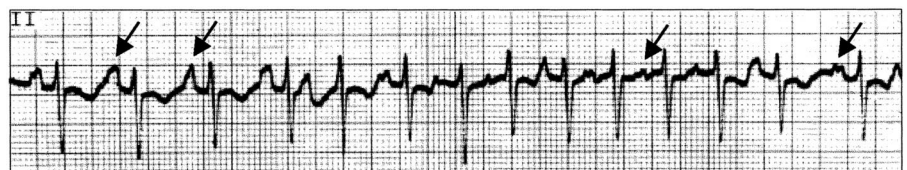


Figure 4. This rhythm strip demonstrates multifocal atrial tachycardia with at least four (arrows) distinct P wave morphologies. II, ECG lead. Adapted with permission from Chou T, Knilans TK (Eds): *Electrocardiography in clinical practice: Adult and pediatric*. Philadelphia, WB Saunders, 1996, p 353.

tant for proper patient management. MAT does not respond to DC cardioversion. Treatment of MAT is usually directed at elimination of the underlying precipitants. Metoprolol (used cautiously when bronchospasm is present) or verapamil may provide atrial and ventricular rate control and occasionally restore sinus rhythm (16, 17). Potassium and magnesium supplements may help suppress MAT. Amiodarone has also been useful in restoring sinus rhythm (5, 11).

## SINUS TACHYCARDIA

Sinus tachycardia is usually a normal reflex response to changes in physiologic, pharmacologic, or pathophysiologic stimuli such as exercise, emotional upset, fever, hemodynamic or respiratory compromise, anemia, thyrotoxicosis, poor physical conditioning, sympathomimetic or vagolytic agents, and abnormal hemoglobins (18). The resulting increase in cardiac output is usual beneficial. Heart rate is generally  $\leq 180$  beats/min, except in young patients who may achieve rates  $\geq 200$  beats/min during vigorous exercise (19). Tachycardia resolves when conditions return to baseline.

## ATRIAL FLUTTER

Although the precise reentrant circuit is unknown, typical atrial flutter traverses (with either counterclockwise or clockwise rotation) through an isthmus formed by the inferior vena cava, tricuspid valve, eustachian ridge, and coronary sinus ostium. Counterclockwise rotation is more common and results in negative "flutter" waves in ECG leads II, III, aVF, and  $V_6$ . Atrial activity in lead  $V_1$  is positively directed. Clockwise atrial flutter produces oppositely directed P waves in these leads. Atrial rates generally range between 250 beats/min and 350 beats/min; however, slower rates may be seen in the presence of specific pharmacotherapy (which slows conduction within the circuit) or marked right atrial enlargement, presumably because of a larger circuit. Atrial flutter usually presents with 2:1 atrioventricular block and ventricular rates  $\sim 150$  beats/min. Pharmacotherapy for typical and atypical atrial flutter is similar to that outlined below for atrial fibrillation. Special care must be taken to avoid inadvertent precipitation of 1:1 AV conduction and subsequent hemodynamic deterioration.

## ATRIAL FIBRILLATION

Atrial fibrillation is the most important sustained supraventricular arrhythmia both in frequency and in potential for long-term sequelae. More than two million people in the United States suffer from atrial fibrillation. The frequency of this arrhythmia increases dramatically after the age of 60.

Atrial fibrillation is most often associated with structural cardiac (diffuse atrial) disease. Unlike typical atrial flutter, left atrial enlargement is more important than right atrial enlargement in its pathogenesis (20). The chaotic appearance of this arrhythmia is usually the result of shifting reentrant circuits (multiple wavelet hypothesis). Paroxysmal atrial fibrillation may have focal origins (usually in pulmonary veins) (4).

Atrial fibrillation and flutter frequently coexist. Dr. Chung has outlined the principals of managing atrial fibrillation in a separate article in this supplement to *Critical Care Medicine* (21). Therefore, only a limited review is presented below.

*Acute Management of Atrial Fibrillation.* Treatment of atrial fibrillation has three important components: control of the ventricular rate, restoration (and maintenance) of sinus rhythm, and prevention of embolic phenomena.

Control of the ventricular rate (Table 1) is most frequently achieved using digoxin, beta-blockers, calcium channel blockers (verapamil or diltiazem), or combinations of these agents. Verapamil should be administered cautiously to patients with significant left ventricular dysfunction. The time-honored use of digitalis preparations has recently come under considerable scrutiny. Although digoxin is effective in controlling rates at rest, exercise rate control is not often achieved. Digoxin remains appropriate for patients with concomitant left ventricular dysfunction and congestive heart failure. Intravenous diltiazem is effective and well tolerated. Diltiazem may be administered by continuous intravenous infusion. This combination of efficacy, ease of parenteral delivery, and tolerance makes this agent an attractive option in the critical care setting.

A variety of agents may be used to restore sinus rhythm. It has become customary to manage acute episodes with intravenous procainamide. This is appropriate when relatively rapid conversion is desired or if the patient is unable to take

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medications orally. Dosing and management of associated hypotension has already been discussed. Intravenous procainamide has been reported to restore sinus rhythm in  $\sim 43\%$  to  $65\%$  of patients with atrial fibrillation/flutter (22).

Ibutilide, a unique class III agent, prolongs action potential duration by increasing inward sodium current flow. This results in QT interval prolongation. Patients receiving intravenous ibutilide should be carefully monitored for development of torsade de pointes. Ibutilide is suitable for acute cardioversion; however, prolonged intravenous (or oral) dosing is not available to prevent arrhythmia recurrence. Ibutilide restores sinus rhythm in  $\sim 30\%$  to  $50\%$  of patients with atrial fibrillation and up to  $76\%$  of patients with atrial flutter (22). Preliminary data suggests that ibutilide may be administered safely to patients on concomitant antiarrhythmic agents (23). At first glance, the efficacy of intravenous procainamide and ibutilide appears to be similar. However, most patients in early procainamide trials had atrial fibrillation/flutter of recent onset (the most important predictor of pharmacologic conversion is pretreatment arrhythmia duration). Newer, direct comparisons of these agents have demonstrated the clear superiority of ibutilide in conversion of atrial fibrillation and flutter. Restoration of sinus

Table 1. Intravenous drug therapy for atrial fibrillation

Drug	Acute Dose	Maintenance Dose	Comments
Intravenous drugs for atrial fibrillation: rate control			
Digoxin	1 mg over 24-hr in increments of 0.25 to 0.5 mg	0.125 to 0.25 mg	Not very effective in high catecholamine states. Caution with renal disease.
Esmolol <sup>c</sup>	0.5 mg/kg/min for one min	0.05–0.2 mg/kg/min	Short half-life. Hypotension common.
Verapamil	5–20 mg in 5 mg increments	5–10 mg boluses every 30 mins or 0.005 mg/kg/min	Caution with LV dysfunction.
Diltiazem	20–25 mg or 0.25 to 0.35 mg/kg	10–15 mg/hr	Well-tolerated. May cause hypotension.
Intravenous drugs for cardioversion of atrial fibrillation			
Procainamide	10–15 mg/kg at ≤50 mg/min	2–6 mg/min	May cause hypotension.
Amiodarone	150 mg over 10 mins, then 1 mg/min for 6-hr	0.5 mg/min	May cause hypotension. Many long-term side effects.
Ibutilide	1 mg over 10 mins. A second dose may be given 10 mins after first.	None	Prolongs QT, may cause torsade de pointes. May lower energy requirement for direct current cardioversion.

LV, left ventricular.

<sup>c</sup>Metoprolol and propranolol may also be used. “Rate control” assumes no pre-excitation. Amiodarone is also effective for rate control.

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rhythm with ibutilide occurred in 32% to 51% of patients with atrial fibrillation and 64% to 76% of patients with atrial flutter vs. conversion rates of 0% to 5% (atrial fibrillation) and 0% to 14% (atrial flutter) after intravenous procainamide (22, 24, 25).

Initially, intravenous amiodarone is primarily a calcium channel- and beta-blocker. It may be effective for rate control when other agents fail. The temptation to use intravenous amiodarone to restore sinus rhythm should be tempered by knowledge of its acute electrophysiologic effects. Its class I and, particularly, class III effects require time to occur, making this a poor choice for rapid conversion. Bolus treatment with intravenous amiodarone has been very disappointing (4% conversion) for acute conversion of atrial fibrillation (22). In contrast, ~20% to 50% of patients with persistent AF (>24–48 hrs in duration) revert to sinus rhythm with sustained administration (loading periods of up to 4 wks) of oral amiodarone (26). As noted, intravenous amiodarone may result in hypotension and negative inotropic effects particularly in the setting of pre-existing left ventricular dysfunction. It is also quite costly and should not be used in a cavalier fashion.

Electrical cardioversion remains the most effective way of restoring sinus rhythm in patients with atrial fibrillation (70% to 85% effective). It is often required even in the presence of antiarrhythmic drugs, which are conversely often required to prevent arrhythmia recurrence. Internal cardioversion or

higher energy transthoracically may be effective in up to 84% of patients refractory to 360 joules (27, 28). Newer external defibrillators delivering energy via biphasic waveforms also increase DC cardioversion efficacy (29).

Episodes of atrial fibrillation may be precipitated and perpetuated by metabolic disturbances (such as hypoxemia) and the presence of increased circulating catecholamines (exogenous and endogenous). Restoration of sinus rhythm may be difficult and impractical when *severe* metabolic derangements or multi-system organ failure are present. Serial attempts to restore sinus rhythm via DC cardioversion in this setting is futile and may be hemodynamically deleterious.

Anticoagulation is pivotal in preventing embolic phenomena during DC cardioversion of atrial fibrillation that has persisted ≥48 hrs (30). The risk/benefit ratio of anticoagulation for episodes of shorter duration is less clear. Intravenous heparin may be used before DC cardioversion if sinus rhythm must be restored quickly. Therapeutic international normalized ratios should be achieved (with warfarin) and maintained for ≥4 wks after DC cardioversion. TEE provides a good look at the left atrial appendage and is helpful to avoid a high risk of embolization when anticoagulation is short-term or contraindicated. A negative TEE should not, however, be viewed as a substitute for adequate anticoagulation. The role of TEE in providing anticoagulant “shortcuts” is being studied in a large multicenter trial (31).

Initiation of specific oral antiarrhythmic

drugs is not really in the realm of acute care. Nevertheless, the intensivist must have some familiarity with these agents as well as current philosophies regarding their use. The current trend for patients with structural heart disease is to avoid class I agents. Class IA agents, for many years the drugs of choice, pose an acute risk of torsade de pointes and have poor side effect profiles. Concerns about increases in long-term mortality (32) have diminished their use.

Flecainide (class IC) is a good choice for patients without coronary artery disease or significant left ventricular dysfunction (ejection fraction should be ≥40%). It reduces the number of episodes by 80%. Flecainide is convenient (twice a day dosing) and has very few noncardiac side effects. Propafenone is also helpful, but requires more dose titration and individualization to achieve similar success.

Class III agents are generally chosen for patients with structural heart disease. Sotalol (with class III and beta-blocking effects) may be used with efficacy similar to quinidine. A 3% to 7% risk of torsade de pointes exists with sotalol therapy, usually during the first week of therapy. QT intervals should be monitored rigorously during sotalol therapy. Amiodarone is the most effective long-term therapy for maintaining sinus rhythm in patients with atrial fibrillation. Amiodarone does not increase mortality in patients with structural heart disease. This agent may actually contribute to improved survival in patients with atrial fibrillation and advanced heart failure (33). The benefits of

amiodarone therapy must always be balanced against its considerable side effect profile. Dofetilide, a pure class III antiarrhythmic agent (recently market released in the United States) that blocks the rapid component of the delayed-rectifier potassium current, is also effective without increasing mortality in patients with congestive heart failure (34).

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