Management of Supraventricular Tachyarrhythmias*

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Supraventricular tachyarrhythmias are commonly encountered in clinical practice. Clinical consequences of these tachyarrhythmias range from insignificant to life-threatening. Management consists of acute termination of an episode of tachyarrhythmia and, in certain patients, is followed by chronic prophylaxis against future recurrence. Recent advances in the understanding of clinical cardiac electrophysiology has allowed the development of new pharmacologic and nonpharmacologic therapeutic options in both acute and chronic management of these tachyarrhythmias.

Supraventricular tachyarrhythmias are characterized by a rapid heart rhythm of greater than 100 bpm and a site of origin at or above the level of the His bundle of the atrioventricular (AV) conduction system. Morphology of the QRS complex during a supraventricular tachyarrhythmia is identical to that seen during sinus rhythm, unless there is development of functional bundle branch block or anterograde conduction over an anomalous AV connection.

Acute Management

The immediate approach to a patient with any supraventricular tachyarrhythmia is dictated by the hemodynamic status related to the rhythm disturbance. The presence of chest pain, change in mental status or symptomatic hypotension mandates immediate QRS-synchronized electrical cardioversion. Recommended energy levels for initial cardioversion are 100 joules or less and can be as low as 25 joules for atrial flutter. Cardioversion of atrial fibrillation usually requires 150 joules or more. Electrical cardioversion is not advisable in the presence of digitalis intoxication due to the potential development of intractable, postconversion ventricular tachyarrhythmias.

In hemodynamically stable patients, maneuvers that transiently increase vagal tone can be diagnostically and therapeutically useful by suppressing conduction and prolonging refractoriness in the AV node. In atrial flutter, careful unilateral carotid sinus massage or the Valsalva maneuver may transiently increase the degree of AV block and allow easier identification of atrial activity. Vagal maneuvers may also terminate paroxysmal reentrant supraventricular tachycardia which involves the sinus node or AV node as a part of the reentrant circuit ( sinoatrial reentry, AV nodal reentry and AV reciprocation).1 2 Breath-holding coupled with facial immersion in ice water stimulates the diving reflex, increases vagal tone and may thereby slow or terminate a reentrant supraventricular tachyarrhythmia.3 This reflex is more prominent and clinically effective in young patients than in the elderly.

When the use of pharmacologic agents is necessary for acute termination of a reentrant supraventricular tachycardia, administration of IV verapamil (5 to 10 mg or 0.075 to 0.15 mg/kg) over two minutes is quite efficacious. It has been reported that intravenous verapamil therapy can successfully convert 80 percent of episodes of paroxysmal supraventricular tachycardia to sinus rhythm (Fig 1).5 6 The effectiveness of verapamil is mediated by its calcium-channel blocking properties which slow conduction and prolong refractoriness of the sinus and AV nodes. However, verapamil should be avoided in patients with severe left ventricular dysfunction or overt congestive heart failure as its negative inotropic effects may result in clinical deterioration.7

Intravenous preparations of edrophonium, digitalis and propranolol also slow conduction and prolong refractoriness of the sinus and AV nodes, and may thus be used to terminate an acute episode of paroxysmal supraventricular tachycardia. In patients with significant left ventricular dysfunction, digitalis is the preferred initial pharmacologic agent.

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In hypotensive patients, pressor agents (including phenylephrine and metaraminol) have been used to terminate paroxysmal supraventricular tachycardia. Pressor agents increase blood pressure, thereby stimulating carotid baroreceptors and causing a reflex increase in vagal tone. However, an exaggerated hypertensive response to pressor agents may cause clinical deterioration in patients with ventricular dysfunction. Electrical cardioversion should be considered in this situation prior to the use of pressor agents. Temporary transvenous right atrial or transesophageal left atrial rapid pacing may also be used for terminating an acute episode of reentrant supraventricular tachycardia. Rapid atrial pacing may penetrate and alter refractoriness of reentrant pathways, thereby terminating a reentrant arrhythmia. The procedure is confined to the atrium; it can thus be safely applied even in patients with digitalis intoxication.

The acute treatment of hemodynamically stable patients with atrial fibrillation or atrial flutter is initially directed toward control of the ventricular response. In patients without ventricular preexcitation, therapy with intravenous preparations of digitalis, propranolol or verapamil may be used to slow the ventricular rate.

Digoxin is administered in a dose of 0.5 mg intravenously and is followed by 0.25 mg every six hours until the ventricular response is controlled or a maximum dosage over 24 hours is given. Verapamil and propranolol may be substituted in patients without left ventricular dysfunction. Intravenous verapamil (5 to 10 mg) is given over two minutes and repeated after 15 min if adequate ventricular slowing is not achieved. This loading dose may be followed by a loading infusion of 0.375 mg/min for 30 min, then a constant infusion of 0.25 mg/min if prolonged ventricular rate control is required. Control of the ventricular response may be achieved with intravenous propranolol in 1 mg boluses over one minute repeated every five minutes to a total dose of 0.15 mg/kg. Temporary rapid atrial pacing (transvenous or transesophageal) may be effective in converting some forms of atrial flutter to sinus rhythm. However, atrial pacing is not effective for converting atrial fibrillation to sinus rhythm.

In patients with the Wolff-Parkinson-White syndrome, atrial flutter and atrial fibrillation may result in a ventricular response in excess of 250 bpm by anterograde conduction over a rapidly conducting anomalous AV connection necessitating emergent electrical...

**Figure 1.** Rhythm strip showing paroxysmal supraventricular tachycardia terminated by intravenous verapamil treatment. Retrograde atrial activation is present with an inverted P-wave trailing the QRS complex during tachycardia. Tachycardia termination occurs following anterograde block in the atrioventricular node. Blood pressure = 100/60 mm Hg at 1:30 PM after administration of 5 mg verapamil IV. Paper speed = 25 mm/s in this and the following figures.

**Figure 2.** Standard 12-lead electrocardiogram showing atrial fibrillation and the Wolff-Parkinson-White syndrome. Markedly irregular R-R intervals are present with both near normal and maximally preexcited QRS complexes.
cardioversion (Fig 2). Pharmacologic control of the ventricular response during atrial flutter and atrial fibrillation in hemodynamically stable patients with the Wolff-Parkinson-White syndrome requires depression of conduction and prolongation of refractoriness in the anomalous AV connection. Intravenous procainamide, 50 mg/min to a total dose of 10 to 20 mg/kg, followed by a 2 to 6 mg/min constant infusion, is the therapy of choice. Intravenous lidocaine treatment may also be effective in certain patients. Medications that exert depressive action on the AV node but not the anomalous AV connection may cause deleterious effects when ventricular preexcitation is present. Intravenous digitalis therapy has a variable effect on the refractory period of the anomalous AV connection and, in some cases, may actually shorten it and accelerate the ventricular response. Beta-adrenergic blocking agents have little effect on conduction in the anomalous AV connection, while intravenous verapamil therapy may indirectly shorten the refractory period of the anomalous AV connection by a reflex sympathetic discharge in response to verapamil-induced hypotension. Intravenous verapamil has been reported to have provoked degeneration of atrial flutter and atrial fibrillation in the setting of ventricular preexcitation into ventricular fibrillation (Fig 3). Intravenous digitalis and verapamil should be considered contraindicated in the acute treatment of atrial flutter and atrial fibrillation associated with ventricular preexcitation.

Atrial tachycardias commonly accompany chronic pulmonary or cardiac disease. P-wave morphology of atrial tachycardias is often different from that of sinus rhythm. Vagal maneuvers may transiently increase AV block but will usually not terminate atrial arrhythmias. Intravenous verapamil and propranolol are effective in slowing the ventricular response if they are not clinically contraindicated, and digitalis preparations may also be useful if digitalis intoxication is not the underlying cause of arrhythmias. Type 1 antiarrhythmic agents such as procainamide, quinidine and disopyramide slow conduction and prolong refractoriness of the atrium, and thus offer the best chance of converting atrial tachycardias to sinus rhythm.

Digitalis not only depresses AV nodal conduction but also enhances automaticity and promotes triggered activity. Clinically, digitalis intoxication may induce atrial tachycardia with AV block and nonparoxysmal AV junctional tachycardia. For the treatment of these
supraventricular arrhythmias, digitalis should be withheld and associated hypokalemia or hyperkalemia corrected. Intravenous preparations of an antiarhythmic agent such as diphenylhydantoin or procainamide may be needed for suppressing symptomatic atrial arrhythmias caused by digitalis intoxication. If complete AV block or severe bradycardia is present, insertion of a temporary pacemaker is necessary. Administration of digitalis antibody is indicated in patients with metabolic derangements associated with severe digitalis overdose.17

Multifocal atrial tachycardia is an irregular atrial tachyarrhythmia in which at least three different P-wave morphologies with variable PR intervals are seen.1 This tachycardia is not uncommonly seen in critically ill patients with severe underlying cardiopulmonary disease. Antiarrhythmic treatment has been disappointing and resolution of the tachyarrhythmia usually requires effective treatment and improvement of the underlying condition. Both intravenous verapamil and metoprolol slow the ventricular rate,18,19 but intravenous verapamil is the preferred therapeutic regimen as it may terminate multifocal atrial tachycardia in certain patients.

**CHRONIC PROPHYLAXIS**

With successful control of an acute episode of supraventricular tachyarrhythmia, the decision to proceed with detailed evaluation of the rhythm disturbance and to institute chronic antiarrhythmic therapy must be considered. In frequent, short-lived, self-limited supraventricular tachycardia that is hemodynamically inconsequential may require neither extensive evaluation nor chronic therapy. Further, most patients with supraventricular tachyarrhythmias who require chronic therapy can be managed empirically without invasive electrophysiologic studies.

Detailed, invasive electrophysiologic studies are indicated:1) to rapidly achieve an effective therapeutic regimen in patients with hemodynamically serious consequences such as syncope, cardiac arrest or congestive heart failure due to supraventricular tachyarrhythmias; 2) to precisely characterize the underlying mechanism of supraventricular tachyarrhythmias in symptomatic patients resistant to empiric therapy in order to allow more rational drug selection; 3) to precisely characterize the supraventricular tachyarrhythmias resistant to pharmacologic therapy prior to proceeding to nonpharmacologic options including antitachycardia permanent pacemakers, electrode catheter ablation or surgical intervention; 4) to identify symptomatic patients with the Wolf-Parkinson-White syndrome who are at risk for development of a rapid ventricular rate during atrial fibrillation; or 5) to differentiate between supraventricular tachyarrhythmias with aberrant conduction and ventricular tachyarrhythmias when clinically important.

Multiple oral medications have been found to reduce the frequency of recurrence of supraventricular tachyarrhythmias. Verapamil (80 to 120 mg every eight hours) is effective in preventing paroxysmal supraventricular tachycardia,20 but successful tachycardia termination with intravenous verapamil does not predict longterm efficacy with oral verapamil therapy.8 Oral diltiazem (60 to 90 mg every eight or six hours) is also efficacious treatment.8 Oral beta-adrenergic blocking agents may also chronically suppress paroxysmal supraventricular tachycardia. A regimen of nadolol, 80 to 160 mg daily, has been shown to be useful in chronically suppressing reentrant supraventricular tachycardia.21 Digitalis therapy also suppresses paroxysmal supraventricular tachycardia in some patients.

Premature atrial and ventricular beats often initiate paroxysmal supraventricular tachycardia. Therefore, therapy with quinidine gluconate (324 to 648 mg every eight hours), procainamide (500 to 1,000 mg every six hours), disopyramide (100 to 200 mg every eight hours), flecainide (100 to 200 mg every 12 hours) and encainide (25 to 50 mg every eight hours) may also be clinically efficacious by reducing or eliminating these initiating events. These antiarrhythmic medications may also work by prolonging conduction and refractoriness of the atrium, His-Purkinje system, retrograde fast AV nodal pathway, ventricular myocardium and anomalous AV connection.7

For patients with chronic atrial fibrillation, control of the ventricular response may be achieved with oral preparations of digitalis, diltiazem, verapamil or beta-adrenergic blocking agents used either alone or in various combinations.22,23 In patients with atrial fibrillation who are candidates for cardioversion, quinidine, procainamide and disopyramide have been used to achieve sinus rhythm. Flecainide has been shown to be as efficacious as quinidine for medical cardioversion of patients with atrial fibrillation.20 However, flecainide should not be administered to patients with compromised left ventricular function (ejection fraction <40 percent) because of its negative inotropic effect. In patients resistant to therapy with conventional medications, amiodarone (200 to 400 mg daily) is effective in restoring sinus rhythm.24 Unfortunately, side effects of amiodarone therapy, including tremor, corneal deposits, ataxia, thyroid dysfunction, skin rash, hepatotoxicity and pulmonary fibrosis preclude long-term use in all but the most difficult circumstances.25

Chronic prophylaxis of episodic atrial flutter and atrial fibrillation in the presence of ventricular preexcitation may be attained with medications that prolong atrial and anomalous AV connection refractoriness. Quinidine, procainamide, disopyramide, flecainide and amiodarone are effective in slowing the ventricular response during atrial fibrillation associ-
ated with ventricular preexcitation, and may even suppress occurrence of atrial flutter and atrial fibrillation. Propafenone, currently under clinical investigation, slows conduction and prolongs refractoriness of the atrium, AV node, His-Purkinje system, ventricular myocardium and anomalous AV connection, and thus appears to be a promising antiarrhythmic agent for both ventricular and supraventricular tachyarrhythmias including atrial flutter-fibrillation associated with ventricular preexcitation.\textsuperscript{20,31}

Recently, nonpharmacologic methods (including implantation of antitachycardia pacemakers, percutaneous catheter ablation and surgical interruption of cardiac tissues) have become available to treat recalcitrant supraventricular tachyarrhythmias. Extensive electrophysiologic study is necessary prior to implementing any of these therapeutic options.

Antitachycardia pacemakers capable of delivering single or multiple atrial or ventricular extrastimuli during supraventricular tachycardia can render a critical portion of the re-entrant circuit refractory to oncoming excitation and terminate a reentrant arrhythmia (Fig 4).\textsuperscript{24,32} Pacemaker generators may be completely automatic or patient-activated. In patients with the Wolff-Parkinson-White syndrome at risk for rapid anterograde conduction over the anomalous AV connection, implantation of antitachycardia pacemakers is less desirable for fear of potential induction of atrial flutter or atrial fibrillation during antitachycardia pacing. Prior to permanent implantation, it is essential to confirm that the proposed antitachycardia device will reproducibly and reliably terminate the arrhythmia.

A percutaneous catheter technique has recently been developed to control supraventricular tachyarrhythmias by ablation of AV nodal or anomalous AV connection tissue. For catheter ablation of the AV node, several direct current shocks of between 35 and 500 (usually 200) joules are administered via a defibrillator to a catheter electrode that is recording the His bundle potential (cathode) and to a patch electrode near the left scapula (anode).\textsuperscript{24} Successful ablation produces complete AV block, effectively controlling refractory supraventricular tachyarrhythmias but mandating permanent pacemaker placement. Immediate complications of the catheter ablation technique include ventricular fibrillation, cardiac tamponade, transient hypotension and myocardial damage as reflected by small post-ablation rises in creatine phosphokinase MB. Further, over a followup period of 9.9 ± 8.2 months, three of 127 patients died suddenly, four died of progressive heart failure and four died of noncardiac causes.\textsuperscript{34} Further work is needed to refine the technique so that supraventricular tachyarrhythmias are controlled, AV conduction is modified rather than eliminated, and the need for post-ablation permanent pacemaker implantation avoided.

Direct current catheter ablation of anomalous AV connections has met with limited success. Ablation of posteroseptal accessory connections has been successful,\textsuperscript{35} but electrical shocks delivered in the region of the coronary sinus can cause perforation followed by development of cardiac tamponade.\textsuperscript{36 More extensive work is necessary to improve the efficacy and safety of electrical catheter ablation of anomalous AV connections.}

The most common supraventricular tachyarrhythmias that are made amenable to surgical correction are those associated with the Wolff-Parkinson-White syndrome.\textsuperscript{37} Surgical ablation of anomalous AV connections in symptomatic patients with this syndrome has yielded excellent results at low risk in experienced hands. Current clinical indications for surgical transection or cryoablation of an anomalous AV connection include: 1) tachyarrhythmias due to the anomalous AV connection refractory to medical therapy, 2) intolerance of medical therapy necessary to control tachyarrhythmias, and 3) atrial fibrillation with very rapid anterograde conduction over the anomalous connection.

The currently favored surgical technique for control of medically refractory AV nodal re-entry is cryoabla-

![Figure 4. Simultaneous standard leads V_{4} (top) and III showing termination of paroxysmal supraventricular tachycardia (PSVT) by paired ventricular extrastimuli. PSVT is initiated by a premature atrial beat (fourth complex) and the first pair of ventricular extrastimuli capture the ventricle (ninth and tenth complexes) but fail to terminate tachycardia. The second pair of ventricular extrastimuli (16th and 17th complexes) with a decreased premature coupling interval capture the ventricle and terminate the tachycardia. (The antitachycardia pacemaker is the Programmable Automatic Scanning Arrhythmia Reversion, model 4171, Telectronics.){/figure}
The treatment of the AV node. 

Discrete cryoablation of perinodal tissue has allowed modification of one limb of the re-entrant circuit without the production of complete AV block in a small number of patients. Postoperative permanent pacemaker therapy may thus be avoided.

SUMMARY

Supraventricular tachyarrhythmias are common and treatment is based on the frequency and hemodynamic severity caused by these arrhythmias. Empiric therapy with currently available medications often satisfactorily controls symptomatic arrhythmias. Nonpharmacologic therapy with permanent antitachycardia pacemakers, percutaneous catheter ablation or surgery can be effective for selected patients with medically refractory supraventricular tachyarrhythmias after thorough electrophysiologic evaluation. In selected patients with life-threatening supraventricular tachyarrhythmias due to the WPW syndrome, surgical ablation is the therapy of choice.

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