



## Contemporary Management of Paroxysmal Supraventricular Tachycardia

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**C**ase History: A 15-year-old female high school student developed rapid palpitations with dizziness while playing soccer. A rescue squad obtained a rhythm strip showing a regular narrow complex tachycardia at 220 bpm. The tachycardia stops abruptly during an attempt to start an intravenous line. Her past medical history is unremarkable. Her physical exam and ECG in sinus rhythm are now normal. She is referred to an electrophysiologist to discuss therapy.

### Initial Evaluation

Paroxysmal supraventricular tachycardia (PSVT) is a common arrhythmia occurring with an incidence of 2.5 per 1000 adults.<sup>1</sup> PSVT in the absence of structural heart disease can present at any age but most commonly first presents between ages 12 and 30. Most patients with PSVT due to atrioventricular nodal reentrant tachycardia (AVNRT) or atrioventricular reentrant tachycardia (AVRT) do not have associated structural heart disease, although exceptions (e.g., Epstein's anomaly, familial preexcitation) do exist. Atrial tachycardias are more commonly associated with structural heart disease. In patients without structural heart disease, the physical exam during

PSVT is significant mainly for rapid heart rate. Prominent jugular venous pulsations due to atrial contraction against closed AV valves may be a clue to AVNRT. History, physical exam, and an ECG constitute an appropriate initial evaluation. Further diagnostic studies are indicated only if there are signs or symptoms that suggest structural heart disease.

A 12-lead ECG during tachycardia is helpful for defining the mechanism of PSVT. In patients with brief, self-terminating episodes, an event recorder is the most effective way to obtain ECG documentation.<sup>2</sup> Patients with panic disorder report symptoms similar to those of PSVT, and an ECG during palpitations aids in diagnosis.

### Mechanisms of PSVT

Figure 1 illustrates schematically the common forms of PSVT. The AV node sits in the triangle of Koch in the floor of the right atrium. Separate pathways, characterized by their conduction velocities as fast or slow, provide input into the AV node.<sup>3</sup> If these pathways have different refractory periods, reentry using one pathway for antegrade conduction and one for retrograde conduction may occur. The P-wave position during AVNRT de-

pends on the types of pathways used. In the most common form, slow pathway–antegrade, fast pathway–retrograde, the P wave is either not seen or is visible in the terminal portion of the QRS. If 2 slow pathways or a fast antegrade pathway and a slow retrograde pathway are used, the RP' interval will be either short or long, respectively. Although uncommon, AV block is possible during tachycardia if block occurs in either the distal AV node or the Bundle of His.

In AVRT, an extranodal accessory pathway connects the atrium and ventricle. Accessory pathways may exhibit both antegrade and retrograde conduction, or either only antegrade (rare) or retrograde (concealed pathways) conduction. When the pathway manifests antegrade conduction, a delta wave will be present on the surface ECG, and a diagnosis of Wolff-Parkinson-White syndrome is made if the patient has PSVT. Accessory pathways usually exhibit rapid, non-decremental conduction, but a minority of them may manifest slow, decremental conduction. The most common form, orthodromic AVRT, uses the accessory pathway as the retrograde limb and the AV node–His as the antegrade limb, which results in a narrow QRS. Functional or fixed bundle branch

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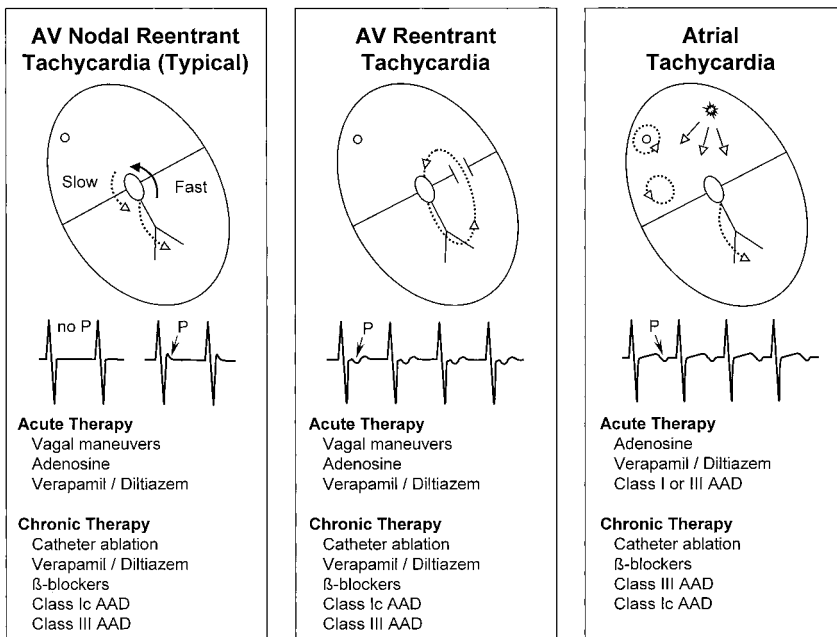
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**Figure 1.** PSVT: Mechanisms, typical ECG findings, and recommended therapies. In typical AV nodal reentrant tachycardia (antegrade conduction down the slow AV nodal pathway and retrograde conduction up the fast pathway), the retrograde P wave may not be seen or may be visible early after the QRS complex. When visible, it often appears as a pseudo-R' in lead V<sub>1</sub>. In AV reentrant tachycardia, there is typically a short RP interval with the timing and morphology of the P-wave dependent on the site and conduction velocity of the accessory pathway. Atrial tachycardia typically produces variable RP and PR intervals as AV conduction depends on AV nodal properties and the tachycardia rate. In atrial tachycardia, the morphology and axis of the P wave are influenced by atrial site of origin and tachycardia mechanism. Acute and chronic therapies are discussed in the text. P indicates P wave; AAD, antiarrhythmic drug.

block, a reversal of the circuit (antidromic AVRT), or the presence of 2 accessory pathways can lead to a wide QRS complex during PSVT. Accessory pathways can also conduct as passive bystanders during AVNRT or atrial tachycardias. In AVRT, the ventricle is an obligate part of the circuit, and thus AV block cannot occur.

Atrial tachycardia is the least common form of PSVT in normal individuals but predominates in patients with significant atrial scarring, especially from prior atrial surgery. Atrial tachycardias may be caused by enhanced or triggered automaticity or by reentry. Because the AV node and ventricle are not required participants in the arrhythmia, AV block commonly occurs. The PR or apparent RP' intervals depend on AV conduction properties. P-wave morphology depends on the site of origin in the atrium. If the site of origin is within or involves the sinus node region, sinus node reentrant

or inappropriate sinus tachycardia is identified.

### Management of Acute Episodes

Because PSVT will rarely be so poorly tolerated that it requires immediate termination with electrical cardioversion, most patients can be managed with physiological maneuvers or drugs.

Most episodes of PSVT require intact 1:1 AV nodal conduction for continuation and are therefore classified as AV nodal-dependent. AV nodal conduction and refractoriness may be modified by vagal maneuvers and by many pharmacological agents and thus are the 'weak links' targeted by most acute therapies.


Many patients learn to terminate acute episodes of PSVT by using vagal maneuvers early during an episode of PSVT. Valsalva is the most effective technique in adults, but carotid massage may also be effective.<sup>4</sup> Facial immersion

is the most reliable method in infants. Vagal maneuvers are less effective once a sympathetic response to PSVT has become established, so patients should be advised to try them soon after onset.

Oral antiarrhythmic drug tablets are not reliably absorbed during rapid PSVT,<sup>5</sup> but some patients may respond to self-administration of crushed medications. In one small study,<sup>6</sup> a combination of diltiazem (120 mg) plus propranolol was shown to be superior to placebo and flecainide. Hypotension and bradycardia after termination are rare complications. Many patients with prolonged episodes require parenteral therapy.

Adenosine and the non-dihydropyridine calcium antagonists verapamil and diltiazem are the intravenous (IV) drugs of choice for termination of PSVT.<sup>4</sup> Adenosine is an endogenous purine nucleoside that slows AV nodal conduction and results in transient AV nodal block. Conduction in rapidly conducting accessory pathways is not affected, but decremental pathways may exhibit block. Exogenous adenosine is cleared extremely rapidly from the circulation by cellular uptake and metabolism, with an estimated half-life of <5 seconds.<sup>7</sup> Adenosine effect is typically seen 15 to 30 seconds after rapid peripheral infusion as a first-pass effect. Administration via a central line requires dose reduction; 1 mg would be the appropriate initial dose. The effective dose range for peripheral administration in adults is 2.5 to 25 mg. If no upper dosage limit is imposed, at least transient termination of AV node-dependent PSVT can be produced in all patients. The recommended adult dosage for peripheral infusion is 6 mg, followed by a 12 mg dose if needed. In pediatric patients, the dose range is 50 to 250 μg/kg administered via an upward dose titration. Because of the ultrashort duration of action, cumulative effects of sequential doses are not seen.

Minor side effects, including transient dyspnea or chest pain, are common with adenosine. Sinus arrest or bradycardia may occur but resolve quickly if appropriate upward dosing is used. With PSVT termination, atrial and ventricular premature beats are frequently seen, and



a few patients with adenosine-induced polymorphic ventricular tachycardia have been reported.<sup>4</sup> These patients had long baseline QT intervals and long pauses during adenosine-induced AV block. Adenosine shortens the atrial refractory period, and atrial ectopy may induce atrial fibrillation. This may be dangerous if the patient has an accessory pathway capable of rapid antegrade conduction. Because adenosine is cleared so rapidly, re-initiation of PSVT after initial termination may occur. Either repeat administration of the same dose of adenosine or substitution of a calcium channel blocker will be effective.

Adenosine mediates its effects via a specific receptor cell surface receptor, the A<sub>1</sub> receptor. Theophylline and other methylxanthines block the A<sub>1</sub> receptor. Caffeine levels achieved after beverage ingestion may be overcome by the doses of adenosine used to treat PSVT. Dipyridamole blocks adenosine elimination, thereby potentiating and prolonging its effects. Cardiac transplant recipients are also unusually sensitive to adenosine. If adenosine is chosen in these latter situations, much lower starting doses (ie, 1 mg) should be selected.

The AV node action potential is calcium channel-dependent, and the non-dihydropyridine calcium channel blockers verapamil and diltiazem are very effective for terminating AV node-dependent PSVT.<sup>8,9</sup> The recommended dosage of verapamil is 5 mg IV over 2 minutes, followed in 5 to 10 minutes by a second 5 to 7.5 mg dose. The recommended dosage of diltiazem is 20 mg followed, if necessary, by a second dose of 25 to 35 mg. PSVT termination should occur within 5 minutes of the end of the infusion, and over 90% of patients with AV node-dependent PSVT respond.

As with adenosine, transient arrhythmias, including atrial and ventricular ectopy, atrial fibrillation, and bradycardia, may be seen after PSVT termination with calcium channel blockers. Hypotension may occur with calcium channel blockers, particularly if the PSVT does not terminate. Calcium channel blockers are not recommended in infants and

neonates with PSVT because of reports of cardiovascular collapse.<sup>10</sup>

Adenosine and verapamil have been shown to have equivalent efficacy in several randomized clinical trials.<sup>4,7,11</sup> Most PSVT patients can be acutely managed with either agent. To minimize the potential for adverse effects, adenosine should be selected in patients with severe hypotension or heart failure, in infants and neonates, and in those at risk for severe bradycardia. Verapamil and diltiazem should be chosen for patients with poor venous access, patients with bronchospasm, and those taking agents that interfere with adenosine action or metabolism.

AV node-dependent PSVT can present with a wide QRS complex in patients with fixed or functional aberration, or if an accessory pathway is used for antegrade conduction. Most wide complex tachycardias, however, are due to mechanisms that may worsen after IV administration of adenosine and calcium channel blockers. Unless there is strong evidence that a wide QRS tachycardia is AV node-dependent, test doses of adenosine, verapamil, or diltiazem should not be used.<sup>4</sup>

Limited data are available on acute pharmacological therapy of atrial tachycardias. Automatic or triggered tachycardias and sinus node reentry should respond to adenosine, verapamil, diltiazem, or  $\beta$ -adrenergic blockers. Other atrial tachycardias may respond to class I or class III antiarrhythmic drugs given orally or parenterally.

### Chronic Therapy

Patients with well-tolerated episodes of PSVT that always either terminate spontaneously or can be broken easily by the patient do not require chronic prophylactic therapy. Selected patients may be treated only for acute episodes.<sup>6</sup> For patients whose PSVT is not well-tolerated or not easily broken, either catheter ablation or chronic drug therapy may be appropriate.

### Pharmacological Therapy

For AV node-dependent PSVT, calcium channel blockers and  $\beta$ -adrenergic

blockers will improve symptoms in 60% to 80% of patients.<sup>8,9</sup> Flecainide and propafenone affect both the AV node and accessory pathways and will also reduce episode frequency.<sup>12,13</sup> Sotalol, dofetilide, and amiodarone are second-line agents. Because sympathetic stimulation can antagonize the effects of many antiarrhythmic agents, concomitant therapy with a  $\beta$ -adrenergic blocker may improve efficacy.<sup>14</sup>

Pharmacological management of atrial tachycardias has not been well evaluated in controlled clinical trials. Depending on the mechanism responsible for the arrhythmia,  $\beta$ -adrenergic blockers, calcium channel blockers, and class I or class III antiarrhythmic drugs may reduce or eliminate symptoms.

### Catheter Ablation

The introduction of invasive electrophysiological studies provided a much better understanding of the mechanisms and anatomical structures responsible for PSVT. Catheter ablation is an attractive alternative for patients who either desire to avoid or are unresponsive or intolerant to drug therapy.

The initial approach for ablation of AVNRT targeted the anterior or fast AV nodal pathway. Although this technique was effective, it resulted in a prolonged PR interval and was associated with a significant risk of AV block due to the para-Hisian location of the fast pathway. Therefore, techniques were developed to target the slow pathway in the posteroseptal tricuspid annular region near the coronary sinus os. Ablation is guided either by mapping a multi-component slow pathway potential or by anatomical landmarks. Slow pathway ablation does not prolong the PR interval in sinus rhythm and has a lower risk of producing AV block.<sup>15,16</sup> Slow pathway ablation is also the procedure of choice in patients with the fast-slow and slow-slow variants of AVNRT. In the North American Society for Pacing and Electrophysiology (NASPE) Prospective Voluntary Registry of 1197 patients, the immediate success rate was 96.1%, with a 1% risk of AV block.<sup>15,17</sup> Late recurrence is seen in 3% to 7% of patients.

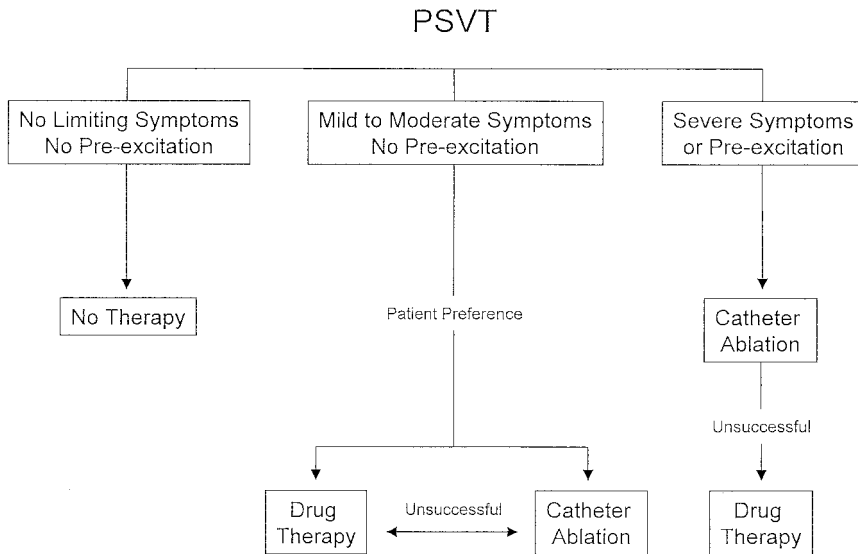


Figure 2. Algorithm for the management of PSVT.

In patients with AVRT, the target for catheter ablation is the accessory pathway. Mapping is performed along the mitral or tricuspid annulus to identify the shortest AV or VA interval. Accessory pathway potentials can sometimes be seen. Success rates for ablation of a single accessory pathway are approximately 95%, with a recurrence rate of 5%.<sup>15,17</sup> Right-sided and septal pathways have a lower initial success rate, and conduction in the former recurs more frequently. Complications of accessory pathway ablation include those from obtaining vascular access, myocardial perforation, valvular damage, coronary artery injury, AV block, and embolism. In experienced centers, major complications requiring treatment should occur in less than 3% of cases.

Catheter ablation of atrial tachycardia is also feasible, but the success rate is somewhat lower.<sup>15</sup> Activation mapping, entrainment mapping, electroanatomical mapping, and non-contact mapping are used to identify the ablation target. Mapping may be complex and difficult in patients with post-surgical atrial anatomy and in those with multiple foci.<sup>18</sup>

### Summary

PSVT is the best-understood clinical arrhythmia. Symptoms in almost all patients can be controlled or eliminated with appropriate therapy (Figure 2). In patients without structural heart disease, catheter ablation offers the potential for a complete long-term cure.

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