JAMA | Review

Diagnosis and Management of Paroxysmal Supraventricular Tachycardia

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IMPORTANCE Paroxysmal supraventricular tachycardia (PSVT), defined as tachyarrhythmias that originate from or conduct through the atria or atrioventricular node with abrupt onset, affects 168 to 332 per 100 000 individuals. Untreated PSVT is associated with adverse outcomes including high symptom burden and tachycardia-mediated cardiomyopathy.

OBSERVATIONS Approximately 50% of patients with PSVT are aged 45 to 64 years and 67.5% are female. Most common symptoms include palpitations (86%), chest discomfort (47%), and dyspnea (38%). Patients may rarely develop tachycardia-mediated cardiomyopathy (1%) due to PSVT. Diagnosis is made on electrocardiogram during an arrhythmic event or using ambulatory monitoring. First-line acute therapy for hemodynamically stable patients includes vagal maneuvers such as the modified Valsalva maneuver (43% effective) and intravenous adenosine (91% effective). Emergent cardioversion is recommended for patients who are hemodynamically unstable. Catheter ablation is safe, highly effective, and recommended as first-line therapy to prevent recurrence of PSVT. Meta-analysis of observational studies shows single catheter ablation procedure success rates of 94.3% to 98.5%. Evidence is limited for the effectiveness of long-term pharmacotherapy to prevent PSVT. Nonetheless, guidelines recommend therapies including calcium channel blockers, β-blockers, and antiarrhythmic agents as management options.

CONCLUSION AND RELEVANCE Paroxysmal SVT affects both adult and pediatric populations and is generally a benign condition. Catheter ablation is the most effective therapy to prevent recurrent PSVT. Pharmacotherapy is an important component of acute and long-term management of PSVT.

JAMA. 2024;331(7):601-610. doi:10.1001/jama.2024.0076
Corrected on February 20, 2024.

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Section Editor: Kristin Walter, MD, Deputy Editor.

Supraventricular tachycardias (SVT) are tachyarrhythmias that originate from or conduct through the atria or atrioventricular (AV) node. Occurring at a heart rate of greater than 100/min, they typically conduct through the His-Purkinje system and appear as narrow QRS (≤120 ms) tachyarrhythmias on electrocardiogram (ECG). Paroxysmal SVT (PSVT) refers to a subgroup of SVT that begins episodically and terminates abruptly. The major types of PSVT are AV nodal reentrant tachycardia (AVNRT), AV reentrant tachycardia (AVRT), and focal atrial tachycardia (AT).[1,2] While atrial flutter and sometimes atrial fibrillation are considered forms of SVT and must be considered on the differential diagnosis, they are not covered in this Review because they tend to have different risk factors and management. Rarer forms of SVT including junctional ectopic tachycardia, nodoventricular tachycardia, and nodofascicular tachycardia are also not covered in this Review.

The prevalence of PSVT in the US is estimated to be 168 to 332 per 100 000 individuals based on retrospective analyses using large clinical databases.[3,4] Major associated comorbidities included chronic pulmonary disease (15.7%), diabetes (12.5%), heart failure (8.4%), cerebrovascular disease (7.8%), and peripheral vascular disease (7.6%). While pharmacologic therapies have largely remained unchanged over the past 2 decades, advances in monitoring techniques and ablation therapies have led to accurate diagnostic tools and curative treatment options for patients with PSVT. This Review summarizes current evidence regarding the diagnosis and treatment of PSVT (**Box**).

Methods

We performed a PubMed search for articles on SVT published between January 1, 1998, and July 1, 2023. We prioritized high-quality randomized clinical trials and observational prospective studies with large sample sizes. Of 6156 retrieved articles, 75 were included, including 10 randomized clinical trials, 43 observational studies, 5 meta-analyses, 1 systematic review, 2 consensus statements, 4 practice guidelines, 7 narrative reviews, and 3 case reports.

Epidemiology

Paroxysmal SVT occurs in both pediatric and adult populations. The precise prevalence of PSVT in the pediatric population is unknown, although it has been estimated to be 1 to 4 cases per 1000 individuals.[5] In a retrospective analysis of a national insurance dataset, the overall incidence of SVT among children up to 15 years of age regardless of symptom status was 1.03 per 1000 patient-years.[6] Among patients of all ages with PSVT, the majority are female (67.5%) and 50.3% are aged 45 to 64 years.[3] Older age is associated with

Box. Common Questions on the Diagnosis and Management of Paroxysmal Supraventricular Tachycardia (PSVT)

What Are the Most Common Subtypes of PSVT?
The most common subtypes of PSVT are atrioventricular (AV) nodal reentrant tachycardia (AVNRT), AV reentrant tachycardia (AVRT), and focal atrial tachycardia. AVNRT is mediated by reentry around the AV node when more than 1 AV nodal pathway exists. AVRT involves reentry using an accessory pathway. Focal atrial tachycardia originates from atrial tissue.

What Is the First-Line Acute Therapy for PSVT?
For hemodynamically stable PSVT, first-line therapy is vagal maneuver. One example is the modified Valsalva maneuver, which begins with a strain phase followed by performance of leg raise in the supine position. Carotid sinus massage is another vagal maneuver than can be effective. Intravenous adenosine can be used next if the arrhythmia persists. For hemodynamically unstable PSVT, emergent cardioversion is indicated.

How Effective Is Ablation for PSVT?
Ablation for PSVT is highly effective and safe. It is recommended as the first-line therapy for prevention of recurrent symptomatic PSVT. Acute success rates for AVNRT and AVRT ablation are upward of 94%. The acute success rate for atrial tachycardia is also high (77%).

increased incidence of PSVT. A subset of patients presenting without baseline cardiovascular disease (including hypertension, coronary artery disease, heart failure, cardiomyopathy, valvular disease, atrial fibrillation, and sick sinus syndrome) is often termed to have *lone PSVT*. One observational study including 1763 patients showed that lone PSVT accounts for 39% of incident cases of PSVT.[7] Patients with lone PSVT were on average younger (mean age, 37 vs 69 years) and had faster mean heart rate on index presentation (186/min vs 155/min) compared with those with PSVT and cardiovascular disease. Risk factors for lone SVT are not well established.

Pathophysiology

Electrical activity in the heart originates in the sinoatrial node, propagates through the atria to reach the AV node, and then travels through the bundle of His, the bundle branches, and the Purkinje fibers to depolarize the rest of the ventricles (**Figure 1**A). Three key mechanisms may disrupt this process and cause arrhythmia: enhanced automaticity, triggered activity, and reentry. Enhanced automaticity occurs when either pacemaker cells increase their firing rate or nonpacemaker cells acquire the ability to spontaneously depolarize. Triggered activity occurs when a myocardial action potential leads to abnormal oscillations in the transmembrane potential, known as afterdepolarizations, and causes extra heartbeats. Reentry typically describes the circuitous movement of electrical impulse along pathways that differ in properties including refractoriness and conduction velocity (**Figure 2**). Among these mechanisms, reentry plays the most dominant role in the pathogenesis of the common forms of PSVT. Understanding the pathophysiology of different PSVT subtypes helps clinicians make correct diagnoses and provide appropriate management.

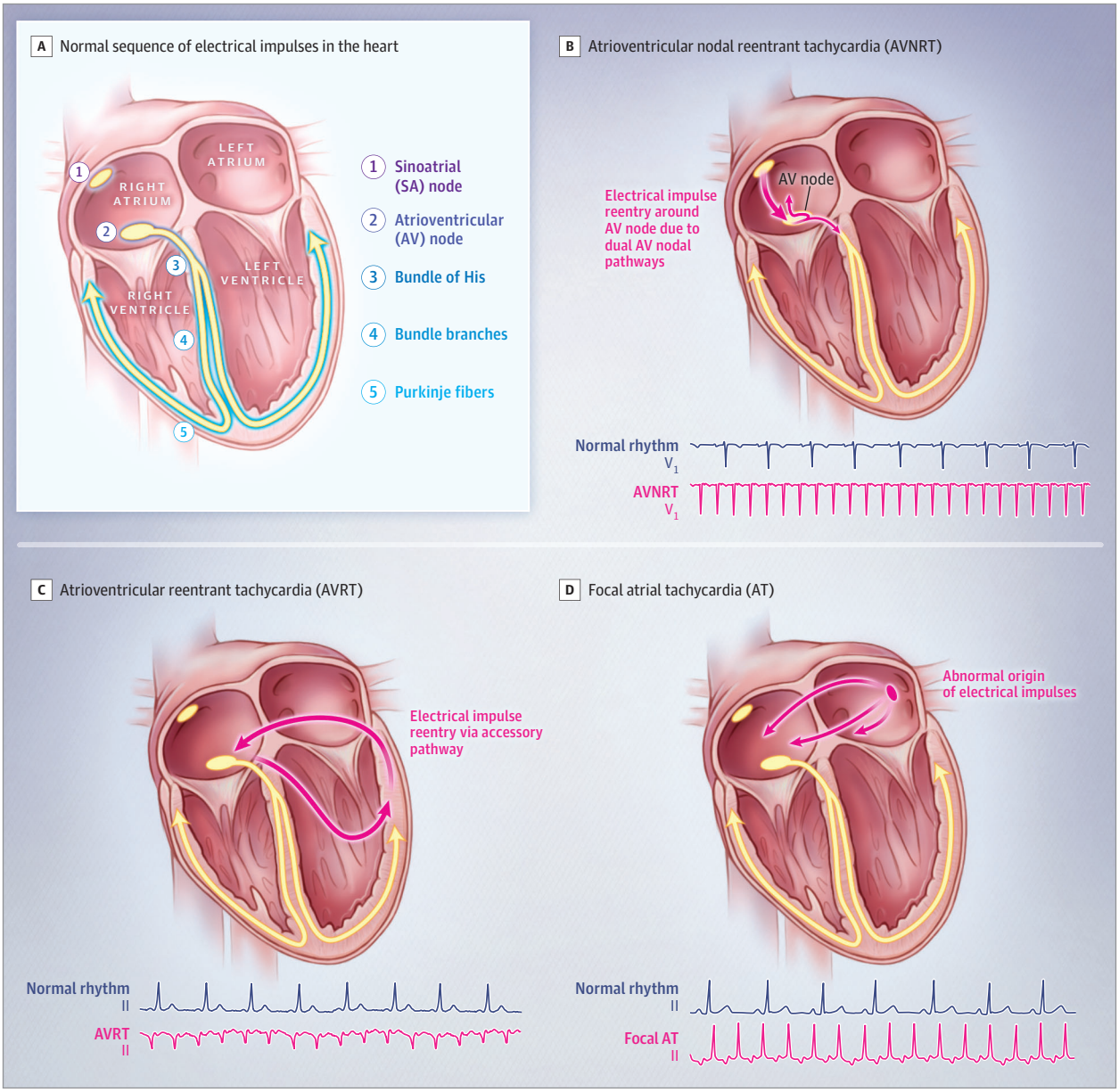
Atrioventricular nodal reentrant tachycardia is the most common subtype of PSVT and represents approximately 56% of cases referred for catheter ablation. In typical AVNRT, anterograde conduction occurs over the slow pathway, which usually extends anatomically from the compact AV node inferiorly along the tricuspid valve annulus to the floor of the coronary sinus (Figure 1B). Electrical activity propagates up this pathway to the compact AV node and then exits the fast pathway at the top of the AV node. The retrograde P wave is typically inscribed close to the QRS and can be difficult to discern on ECG, leading to the so-called no-RP tachycardia.

Atrioventricular reentrant tachycardia is the second most common form of PSVT referred for ablation (27% of cases) and involves reentry via an accessory pathway (Figure 1C).[8,9] Also known as a bypass tract, the accessory pathway is an abnormal electrical and anatomical connection across the AV ring between atria and ventricles. Accessory pathways occur in approximately 1 in 1500 individuals and are present from birth.[10] Unlike AVNRT, AVRT is slightly more common in men (54.6%) compared with women.[9] Orthodromic AVRT occurs when conduction through the AV node is antegrade, leading to activation of the His-Purkinje system in the same fashion as sinus rhythm, but it is then followed by retrograde conduction through the accessory pathway back to the atria to complete the circuit. Therefore, the QRS complex in orthodromic AVRT is typically narrow (≤120 ms) unless conduction delay or block distal to the AV node is present. On the other hand, antidromic AVRT involves antegrade conduction through the accessory pathway, leading to cell-to-cell ventricular depolarization and contraction followed by retrograde conduction up the His-Purkinje system and the AV node. Therefore, the QRS complex in antidromic AVRT is typically wide (≥120 ms) and can appear like basal ventricular tachycardia arising from the location of the accessory pathway.

Antegrade conduction through the accessory pathway during sinus rhythm can inscribe a slurred QRS upstroke associated with a short PR segment known as the Wolff-Parkinson-White (WPW) pattern. The slurred QRS upstroke (also known as a delta wave) occurs because ventricular depolarization through the accessory pathway occurs earlier than through the native conduction system. This phenomenon is also known as ventricular preexcitation. Patients are diagnosed with WPW syndrome if their ECG demonstrates a WPW pattern and they experience symptomatic SVT. However, patients with orthodromic AVRT may not manifest a WPW pattern on ECG in sinus rhythm if they have a concealed conduction pathway, which conducts only retrogradely. Patients with a WPW pattern are at risk of sudden cardiac death, with an estimated incidence rate of 2.4 (95% CI, 1.3-3.9) per 1000 person-years.[11] These patients should be referred for cardiology for further risk stratification and management.

Focal AT is the third most common type of PSVT, accounting for 17% of cases referred for ablation (Figure 1D).[8,9] Among patients with PSVT, the prevalence of focal AT increases with age[9] and can be caused by enhanced automaticity, triggered activity, or reentry in diseased atria. On ECG, focal AT tends to present with monomorphic P waves that may appear different from P waves in sinus rhythm. However, the P waves appear similar when the arrhythmia originates from the superior aspect of the crista terminalis near the sinoatrial node. Distinct P waves may be difficult to identify at higher ventricular rates seen in 1:1 conduction when they coincide with QRS or T waves. The presence of discrete P waves with intervening

Figure 1. Three Major Subtypes of Paroxysmal Supraventricular Tachycardia



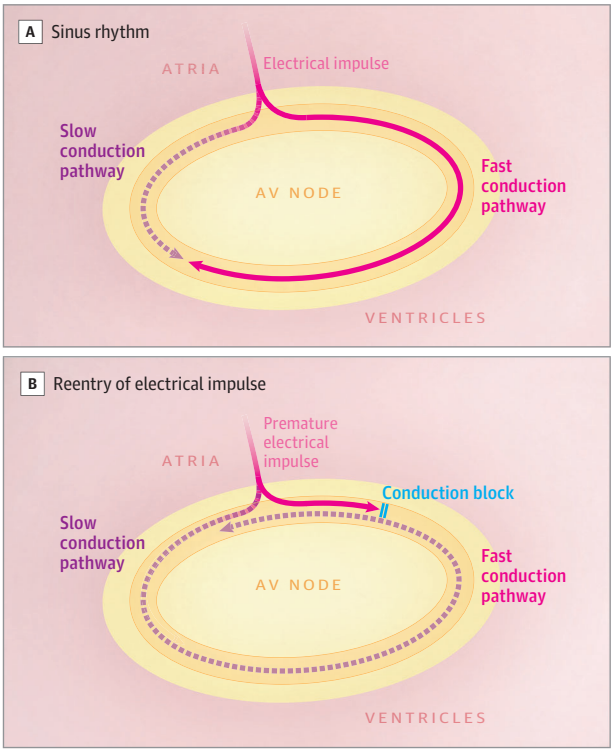
A, In normal physiology, electrical impulse originates in the sinoatrial node, travels across the atria to the atrioventricular (AV) node, then down the bundle branches and Purkinje fibers, leading to ventricular depolarization. B, In atrioventricular nodal reentrant tachycardia (AVNRT), reentry occurs around the AV node due to the presence of dual AV nodal pathways. On electrocardiogram (ECG), a pseudo R′ in lead V_1 or a pseudo S wave in inferior leads are often observed due to the presence of a buried retrograde P wave. However, the P wave may be difficult to identify because the timing of atrial depolarization nearly overlaps with ventricular depolarization. C, In atrioventricular reentrant tachycardia (AVRT), an accessory pathway participates in the reentrant circuit. On ECG, orthodromic AVRT is a narrow complex tachyarrhythmia (in the absence of conduction block). Patients with an accessory pathway may demonstrate as preexcitation in sinus rhythm, manifesting as a slurred QRS upstroke and short PR segment. Note that an ECG of unspecified supraventricular tachycardia subtype is provided. D, In focal atrial tachycardia (AT), electrical activity originates from an abnormal focus within the atria. On ECG, focal AT may feature P waves with a distinct morphology from those observed in sinus rhythm.

isoelectric segments can help distinguish focal AT from atrial flutter, which often has an undulating baseline. Multifocal AT is an uncommon arrhythmia with a predilection for patients with pulmonary disease. It can be distinguished from focal AT by the presence of 3 or more P-wave morphologies and an irregularly irregular rhythm.

Clinical Presentation, Diagnosis, and Evaluation

Patients may experience abrupt onset of symptoms associated with PSVT ranging from palpitations (86%) to chest discomfort (47%),

Figure 2. Schematic of Reentry



A, When 2 conduction pathways exist, one may conduct electrical impulse faster than the other. In sinus rhythm, conduction occurs in the same direction (from top to bottom) along both pathways. The 2 impulses collide and reentry does not occur. B, A premature impulse arrives within a time window where the fast pathway remains refractory from the previous depolarization while the slow pathway has recovered from depolarization. Conduction block occurs in the fast pathway and the electrical impulse travels down the slow pathway, then back up along the recovered fast pathway, establishing a circuit.

shortness of breath (38%), and lightheadedness (19%).[12] Symptoms are typically more pronounced at more elevated heart rates or in the background of coronary artery disease. However, the presence of ST depressions during SVT or mild troponin elevations in young patients with low clinical risk of coronary artery disease generally does not indicate coronary artery disease. Patients with heart failure can experience acute exacerbation and pulmonary edema in association with PSVT. Syncope is rarely caused directly by SVT and is more likely a vagally mediated response. Symptomatic PSVT may terminate spontaneously or persist until medical intervention.

Typical evaluation for PSVT begins with a comprehensive history that includes onset, timing, and duration of symptoms as well as cardiovascular history. In the absence of structural heart disease, physical examination findings may be normal outside of arrhythmic episodes. A 12-lead ECG should be obtained whenever possible, both during an arrhythmic episode and following cardioversion. Additional diagnostic tests that detect outpatient arrhythmia episodes, in order of increasing monitoring duration, include Holter monitors, "patch" monitors, event monitors, and implantable loop recorders. Despite interest in use of consumer wearable devices for arrhythmia detection, evidence for their use is currently limited. One study involving 52 patients investigated the diagnostic accuracy of consumer wearable devices for detecting SVT induced in the electrophysiology laboratory and showed that sensitivity was poor (1.5%-37.7%) for short episodes of SVT (<15 seconds) but improved (36%-100%) for longer episodes (>60 seconds) compared with the reference standard of continuous 12-lead ECG.[13] Additional data are needed for the diagnostic accuracy of PSVT for lead-based direct-to-consumer recording devices. Artificial intelligence may be used to identify patients at risk of PSVT using machine learning and integrating large volumes of ECG data. A deep learning model developed from sinus rhythm ECGs of 9069 patients and externally validated in 3886 patients was found to be accurate in identifying patients who later developed PSVT (sensitivity, 86.8%; specificity, 97.2%).[14]

A complete blood cell count, thyroid function tests, and a basal metabolic panel are typically obtained for patients with PSVT. However, the association between electrolyte abnormalities and PSVT is not well established. Transthoracic echocardiogram is useful for evaluating structural heart disease that potentially accompanies PSVT. Patients with history of angina and other clinical risk factors may benefit from screening for coronary artery disease using coronary computed tomography angiography or stress testing.

Treatment

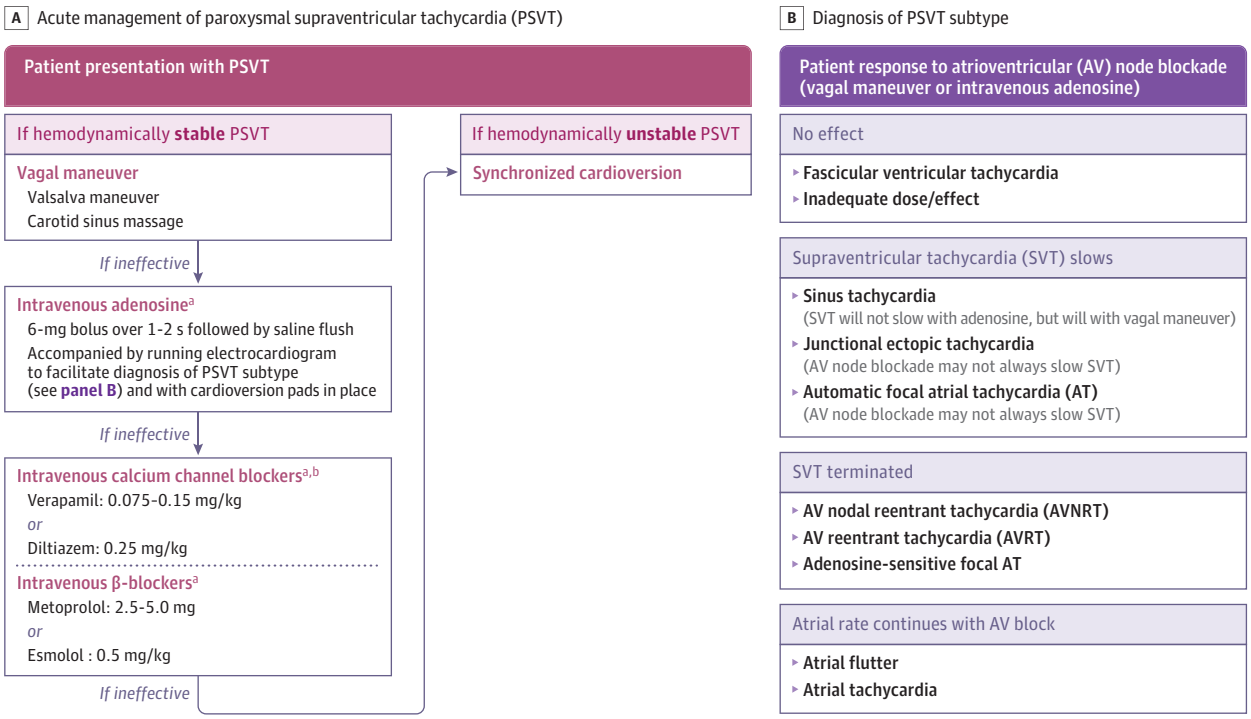
Both acute and long-term therapy are important components of PSVT management. Acute therapy is intended to terminate an arrhythmia episode and resolve symptoms. Greater symptom severity may warrant earlier institution of acute therapy. Long-term therapy serves to prevent recurrence and reduce arrhythmia burden. Advances in catheter ablation have led to safe, effective, and often curative interventions for PSVT.

Acute Therapy

Vagal Maneuvers

Vagal maneuvers comprise noninvasive techniques that stimulate carotid baroreceptors, generate reflex parasympathetic outflow, and slow conduction through the AV node. Currently, they constitute first-line therapy for patients with hemodynamically stable PSVT (**Figure 3**A). The REVERT trial, a pragmatic, randomized clinical trial involving 428 patients with PSVT, evaluated the effectiveness in the emergency department of the modified Valsalva maneuver vs the standard Valsalva maneuver. In this study, the maneuver involved (1) maintaining 15 seconds of strain at a pressure of 40 mm Hg by blowing into an aneroid manometer while positioned in a semirecumbent position, then (2) lying supine with legs raised to 45° by medical staff for 15 seconds, and (3) returning to the semirecumbent position. This modification to the standard Valsalva maneuver (**Video 1**) was designed to promote venous return during the recovery phase of the maneuver and subsequently stimulate the baroreceptor reflex. Compared with the standard maneuver, the modified maneuver was associated with an increased rate of successful conversion to sinus rhythm (43% vs 17%).[15] Similar findings were observed in another randomized clinical trial in which patients blew into a 10-mL syringe instead of a manometer.[16] The procedure is safe and can be taught to patients for treatment at home. Carotid sinus massage (**Video 2**) is a potentially useful vagal maneuver that directly stimulates carotid baroreceptor by the application of gentle pressure over the bifurcation of the common carotid artery

Figure 3. Diagnosis and Acute Management of PSVT



A, Acute management of paroxysmal supraventricular tachycardia (PSVT). PSVT typically manifests as a regular narrow QRS complex tachyarrhythmia. Patients who are hemodynamically unstable because of their arrhythmia should undergo synchronized cardioversion. Otherwise, first-line therapy involves use of vagal maneuvers such as the modified Valsalva maneuver or carotid sinus massage. If this is ineffective, intravenous adenosine is recommended. Other agents that can help terminate PSVT include intravenous calcium channel blockers and β-blockers. B, Diagnosis of PSVT subtype based on response to atrioventricular node blockade. Potential responses to vagal maneuver or intravenous adenosine include arrhythmia slowing, termination, continuation with atrioventricular block, and no effect. Slowing of atrial or junctional rate may occur for arrhythmias due to enhanced automaticity because of the baroreceptor reflex or drug effect of adenosine. Termination of arrhythmia may occur for reentrant arrhythmias whose circuits involve the atrioventricular node. Certain focal atrial tachycardia due to triggered activity may also terminate with adenosine. Atrial flutter and certain atrial tachycardia persist despite the effects of adenosine. Atrioventricular nodal blockade helps reveal underlying atrial activity in these arrhythmias and assists with diagnosis. Adenosine may have no apparent effect in cases in which dosing is inadequate or fascicular ventricular tachycardia is present and manifesting as a narrow-complex tachycardia like PSVT.

[a] Doses are for adults; see Page et al.[1]

[b] In clinical practice, the average intravenous bolus doses for calcium channel blockers are verapamil, 5 mg, and diltiazem, 20 mg.

for 5 seconds. One randomized clinical trial showed that the efficacy of carotid sinus massage in terminating PSVT is similar to the standard Valsalva maneuver (10.5% vs 19.4%).[17] Carotid sinus massage should be avoided in patients with carotid bruit or known carotid artery stenosis.

Adenosine

Adenosine is a fast-acting adenosine receptor agonist that has a short half-life (approximately 10 seconds) and may be used to treat PSVT. Its effects include AV nodal blockade through agonism of the A1 receptor, which may interrupt reentry-based SVTs that are dependent on AV nodal conduction. It is typically administered as a 6-mg intravenous bolus administered over 1 to 2 seconds followed by saline flush.[18] A recent meta-analysis of 3 randomized trials and 1 nonrandomized trial (178 patients) showed similar efficacy in terminating PSVT whether adenosine was combined with normal saline and administered through a single syringe (85% terminated PSVT) vs sequential administration in which adenosine was administered first, followed by a saline flush (77% terminated PSVT; odds ratio, 2.08; 95% CI, 0.65-6.64), although the analysis was limited by small sample size.[19] Adenosine administration should be accompanied by a running ECG to facilitate diagnosis of PSVT subtype (Figure 3B)[20] and with electrical cardioversion pads in place. Adverse effects include flushing (62%), chest tightness (12%), and dyspnea (7%).[21] The success rate of PSVT termination by adenosine is 89.7% based on a meta-analysis of 7 randomized trials comparing adenosine (622 patients) with calcium channel blockers (verapamil or diltiazem).[21] Adenosine should generally be avoided in heart transplant recipients because the denervated heart is more sensitive to this drug. However, 1 prospective study involving 80 pediatric and young adult cardiac transplant recipients suggested that a low initial dose of adenosine (25 µg/kg or 1.5 mg if weight exceeded 60 kg) was safe and did not lead to need for rescue pacing.[22] Adenosine is contraindicated in patients with atrial fibrillation with preexcitation (ie, WPW) because AV blockade can induce rapid ventricular conduction over the accessory pathway and result in ventricular fibrillation. Adenosine administration may also induce atrial fibrillation by shortening the atrial cardiomyocyte refractory period. Because there are some case reports of bronchoconstriction with adenosine used to treat PSVT, guidelines recommend caution with use in patients with asthma.

Calcium Channel Blockers

Nondihydropyridine calcium channel blockers may be used in acute treatment of PSVT. Intravenous verapamil, 5 mg, or diltiazem, 20 mg, can be administered over 2 minutes as a bolus. A meta-analysis including 7 randomized clinical trials (622 patients) showed that the rate of cardioversion did not significantly differ between intravenous calcium channel blockers (verapamil and diltiazem) and adenosine (93% vs 90%).[21] Patient should be monitored for hypotension following administration, although the risk of hypotension with calcium channel blockers was only 0.7% (622 patients).[21] Calcium channel blockers should be avoided in patients with heart failure with reduced ejection fraction. Etripamil is a short-acting, intranasal calcium channel blocker that may be efficacious in terminating PSVT.[23-25] In a phase 3 randomized clinical study involving 692 patients, etripamil significantly increased probability of cardioversion 30 minutes after administration compared with placebo (64% vs 31%).[25]

β-Blockers

There is limited evidence for use of β-blockers in termination of PSVT. A prospective randomized crossover study involving 44 patients showed that intravenous diltiazem at 0.25 mg/kg was more effective at terminating PSVT than intravenous esmolol at 0.5 mg/kg (100% vs 25%).[26] However, β-blockers are useful as rate control agents and are guideline recommended for acute therapy as an alternative to calcium channel blockers if adenosine is ineffective. In a study of 42 patients, diltiazem, 120 mg, plus propranolol, 80 mg, led to a higher cardioversion rate at 2 hours compared with placebo (94% vs 52%).[27] β-Blockers should be avoided in patients with acute decompensated heart failure.

Antiarrhythmics

Despite limited evidence, antiarrhythmics may be useful in acute management of PSVT. Class Ic agents including propafenone and flecainide can restore sinus rhythm. In a single-group prospective study involving 70 patients, intravenous propafenone, 2 mg/kg, terminated AVNRT in 76% of patients and AVRT in 88% of patients with these types of PSVT induced in the electrophysiology laboratory.[28] In another study, oral flecainide, 3mg/kg, led to successful cardioversion in 61% of patients.[27] While there is ongoing investigation on the effectiveness of inhaled flecainide in treating atrial fibrillation, there is currently no evidence favoring its use in treating PSVT.[29] Ibutilide and amiodarone can be used in treatment of AVRT or focal AT. One observational study involving 38 patients with focal AT showed that ibutilide was effective at cardioverting 39% of arrhythmic episodes.[30] Procainamide may also be used in refractory cases of PSVT. Antiarrhythmic agents are preferred over AV nodal agents in patients with antidromic AVRT. All antiarrhythmic agents have potential proarrhythmic effects, and therefore outpatient follow-up with ECG monitoring is advised.

Direct-Current Cardioversion

Emergent synchronized cardioversion is the therapy of choice for patients with PSVT and hemodynamic instability. A prospective study of 84 patients with unstable SVT showed that cardioversion was 100% successful at terminating the arrhythmia within 1 to 3 attempts when performed out of hospital.[31] Synchronized cardioversion in a hemodynamically stable and conscious patient requires sedation. Although the optimal energy setting is not well studied, typically 50 to 100 J is used for initial attempts at electrical cardioversion for PSVT.

Prevention of Recurrent PSVT

Catheter Ablation

Existing data demonstrate that catheter ablation is safe,[32] cost-effective,[33,34] and often curative for PSVT; thus, its use is favored as first-line therapy for symptomatic patients with recurrent PSVT. Ablation is performed percutaneously using a catheter that delivers energy that ablates critical sites within reentrant circuits or at or near automatic foci. An electrophysiology study at the time of ablation is typically performed, allowing confirmation of the arrhythmia subtype and thus the appropriate ablation target. Patients should therefore be referred to an electrophysiologist for consideration of ablation following diagnosis and initial acute management of PSVT.[1]

Multiple lines of evidence exist for the efficacy and safety[35] of catheter ablation with radiofrequency or cryoablation energy in treating symptomatic AVNRT. A meta-analysis from 2009 of 23 different treatment groups from both randomized trials and observational studies (4249 patients) showed that the single-procedure success rate of AVNRT ablation was 94.3% (95% CI, 91.2%-97.4%).[36] In a randomized trial of 61 patients (aged 18-65 years) comparing ablation with medical therapy (bisoprolol, 5 mg/d, and/or diltiazem, 120-300 mg/d), patients in the ablation group experienced lower risk of arrhythmia recurrence (0 vs 68%) over a 5-year period.[37] AVNRT ablation is also effective and safe in patients older than 75 years.[38]

For patients with symptomatic AVRT, ablation of the accessory pathway constitutes first-line preventive therapy. In 1 longitudinal cohort study of 2169 patients, the acute success rate of accessory pathway ablation was 98.5%.[11] The risk of complications for AVRT ablation includes a 0.3% risk of AV block requiring a pacemaker.[36] In a meta-analysis of 54 studies that included both observational studies and randomized trials (4244 patients) assessing long-term outcomes for septal accessory pathway ablation (a more challenging location due to proximity to the conduction system), radiofrequency ablation was associated with 11.6% risk of arrhythmia recurrence over 37.5 months of follow-up, while cryoablation was associated with 24.1% risk over 13.5 months.[39] Patients with asymptomatic preexcitation likely also benefit from prophylactic accessory pathway ablation. A study of 72 patients with WPW syndrome who were identified on ECG as being at an elevated risk of arrhythmia reported a significantly lower primary end point of arrhythmic events over a 5-year follow-up period among patients who were randomized to radiofrequency catheter ablation of accessory pathways vs controls (7% vs 77%). The risk reduction with ablation was 92% (relative risk, 0.08; 95% CI, 0.02-0.33; $P<.001$).[40]

Catheter ablation is recommended for the prevention of recurrent symptomatic focal AT. One study examining 105 patients undergoing radiofrequency ablation for focal AT demonstrated an immediate postprocedural success rate of 77%.[41] Over a mean follow-up of 33 months, 90% of patients who had early procedural success did not experience AT recurrence. In another study of 28 patients with focal AT arising from the pulmonary veins, freedom from AT after undergoing focal ablation was 96% over a mean follow-up of 25 months.[42] In a retrospective analysis examining patients with tachycardia-mediated cardiomyopathy (left ventricular ejection fraction <50%) secondary to focal AT, 97% of patients with

Table. Oral Preventive Therapies for Paroxysmal Supraventricular Tachycardia (PSVT)[a]

	Class	Commonly treated arrhythmia subtype	Avoid in	Common/notable adverse events
First-line therapy	Calcium channel blockers (diltiazem, verapamil)	AVNRT, AVRT, focal atrial tachycardia	Heart failure with reduced ejection fraction, Wolff-Parkinson-White syndrome	Hypotension, bradycardia, headache
	β-Blockers (eg, metoprolol, atenolol)	AVNRT, AVRT, focal atrial tachycardia	Decompensated heart failure	Bradycardia
Second-line therapy	Class Ic agents (flecainide, propafenone)	AVRT, focal atrial tachycardia, AVNRT	Structural or ischemic heart disease	Arrhythmia, vision changes (flecainide), unusual taste (propafenone)
	Dofetilide	AVNRT, AVRT, focal atrial tachycardia	Prolonged QT, severe kidney impairment	QT prolongation, headache
Refractory cases	Amiodarone	Focal atrial tachycardia, AVNRT, AVRT		Pulmonary toxicity, thyroid toxicity, liver toxicity, corneal deposition
Adjunctive therapy	Ivabradine	Focal atrial tachycardia	Decompensated heart failure, severe hepatic impairment, atrioventricular block	Bradycardia

Abbreviations: AVNRT, atrioventricular nodal reentrant tachycardia; AVRT, atrioventricular reentrant tachycardia.

[a] The initial pharmacologic management strategy to prevent PSVT involves use of calcium channel and β-blockers. However, long-term use of AV nodal agents, especially calcium channel blockers, are avoided in patients with Wolff-Parkinson-White syndrome due to potential for atrial arrhythmia with rapid conduction over accessory pathways. Calcium channel blockers are also avoided in patients with heart failure with reduced ejection fraction given their varying degrees of negative inotropic effect. Class Ic agents including flecainide and propafenone are helpful in the management of PSVT but should be avoided in patients with structural heart disease. Dofetilide is a class III agent that helps suppress arrhythmia recurrence but requires inpatient monitoring for QT prolongation during initiation. Given its renal clearance, dofetilide should be avoided in patients with severe kidney impairment. Amiodarone is a useful agent in preventing PSVT but is reserved for refractory cases given potential for pulmonary, thyroid, and liver toxicities. Ivabradine may serve as a useful adjunct to β-blockers in controlling atrial tachycardia.

successful ablation experienced normalization of ejection fraction,[43] demonstrating that ablation can reverse myocardial dysfunction secondary to PSVT.

Pharmacologic Therapy

There is limited evidence for the effectiveness of pharmacotherapy to prevent recurrent PSVT. Current guidelines recommend β-blockers or nondihydropyridine calcium channel blockers as first-line agents (**Table**). Class Ic antiarrhythmics including flecainide and propafenone are considered second-line agents. An as-needed, "pill-in-the-pocket" approach using flecainide (300 mg if weight ≥70 kg; 200 mg if weight <70 kg), diltiazem, verapamil, or β-blockers may be preferred over maintenance therapy in patients with infrequent arrhythmia recurrence or adverse effects of maintenance therapy. Dofetilide is an antiarrhythmic agent that may also be considered for long-term therapy. One randomized clinical trial including 122 adults showed that the risk of PSVT recurrence while taking dofetilide was similar to propafenone (50% vs 46%) and significantly lower than placebo (94%) over a 6-month period.[44] Initiation of dofetilide requires 72-hour hospitalization for monitoring. Amiodarone can be considered for refractory cases of PSVT but may cause pulmonary, hepatic, and/or thyroid toxicity. Ivabradine may serve as an adjunct to β-blocker therapy in preventing AT recurrence.

Prognosis

The overall prognosis of patients with PSVT in the absence of structural heart disease is good, especially when treated with catheter ablation. However, with pharmacotherapy alone, 50% of patients continue to have at least monthly symptoms.[45] One retrospective study investigated the risk of SVT-related adverse events in 1770 patients with AVNRT or AVRT followed up over 2.8 years and demonstrated the risk of heart failure/cardiomyopathy to be 1.2%.[46] Independent predictors of adverse events were older age and preexisting heart disease. Catheter ablation for patients with tachycardia-mediated cardiomyopathy effectively restored left ventricular systolic function in 97% of 30 patients in a retrospective study.[43] In another retrospective cohort study of 4 806 830 patients using a claims database, PSVT was independently associated with increased risk of stroke,[47] with an absolute risk of 0.94%, though the mechanism of this effect requires further elucidation. Paroxysmal SVT may lead to unnecessary shocks in patients with implantable cardioverter-defibrillators due to misclassification of arrhythmia as ventricular tachyarrhythmia. This risk may be mitigated by dual-chamber arrhythmia detection.[48]

Practical Considerations and Application of Evidence in Special Populations

Pediatric Populations

In the pediatric population, PSVT most commonly presents in newborns and young infants.[49] AVRT accounts for most PSVT cases in this age group due to the presence of accessory pathways associated with immaturity of the cardiac conduction system. Permanent junctional reciprocating tachycardia is a rare form of orthodromic AVRT in this age group that is characterized by slow conduction through the accessory pathway and can be challenging to control. Paroxysmal SVT diagnosed in infancy resolves spontaneously in 90% cases, but a minority (up to one-third) of patients develop recurrence later in childhood.[5] In contrast to AVRT, AVNRT is typically diagnosed in older children. Symptoms and signs of PSVT range from irritability to failure to thrive, gastrointestinal symptoms, and palpitation. Acute treatment includes vagal maneuvers, adenosine, class Ic agents (flecainide, propafenone), and

emergent cardioversion for hemodynamically unstable patients. Suppressive pharmacologic therapies include propranolol, digoxin, class Ic agents, amiodarone, and sotalol.[50-54]

Ablation therapy can be an important component of long-term management of PSVT in children.[55,56] A prospective cohort study including 481 pediatric patients showed that the overall acute success rate of radiofrequency ablation for PSVT was 97.8%.[57] Pharmacotherapy may be preferred over ablation in children younger than 5 years because of technical limitations due to small anatomical size. Cryoablation is more commonly used in pediatric patients than adult patients because of their smaller-sized hearts and its lower long-term risk of heart block compared with radiofrequency ablation.[58-60] A retrospective analysis of 49 adolescent patients with AVNRT at a single center showed that cryoablation had an immediate postprocedural success rate of 100%.[61] Although recurrence occurred in 22% patients over a follow-up of 30 months, repeat catheter ablation resulted in a 100% long-term success rate over a mean follow-up of 30 months after the second procedure.

Pregnancy

The risk of SVT increases during pregnancy and is estimated to be 22 per 100 000 pregnancies.[62-64] Paroxysmal SVT is associated with risk of composite severe maternal morbidity which includes heart failure and shock among other comorbidities (odds ratio, 3.52; 95% CI, 2.65-4.67) and increased risk of cesarean delivery (odds ratio, 1.61; 95% CI, 1.44-1.81).[65] However, there is a paucity of data regarding the efficacy and safety of antiarrhythmic drugs for SVT during pregnancy.[66] Antiarrhythmic agents are generally avoided in the first trimester given concerns for teratogenic effects. First-line long-term preventive therapy includes β-blockers such as propranolol and metoprolol. However, atenolol is generally avoided given greater association with fetal growth restriction.[67] Second-line therapies include digoxin and verapamil. Flecainide and propafenone can be useful in patients with WPW syndrome. Flecainide may also be useful in patients with incessant AT, which has limited response to β-blockers and calcium channel blockers. Sotalol can also be useful in the management of SVT in this population. Amiodarone can be used in refractory cases of PSVT but is typically avoided given risk of fetal hypothyroidism. Dronedarone is contraindicated in pregnancy. Catheter ablation in pregnant patients is generally avoided given fetal exposure to ionizing radiation, but it can be used to treat drug-refractory SVT when performed at experienced centers, particularly where ablation without fluoroscopic guidance is possible.[68-71] Preconception catheter ablation in eligible patients with known PSVT should be recommended.

Exercise-Associated PSVT

Paroxysmal SVT can be provoked by intense physical activity. Individuals with exercise-associated PSVT should undergo evaluation for structural heart disease.[72] Catheter ablation is recommended for patients with WPW syndrome, which can be a cause of cardiac arrest in young athletes.[73] On the other hand, those with asymptomatic preexcitation may be risk-stratified by a stress ECG, in which abrupt disappearance of ventricular preexcitation during exercise suggests low risk of malignant arrhythmia. For professional/competitive athletes with asymptomatic preexcitation, invasive electrophysiology study allows for additional risk stratification.

Catheter ablation can also be considered for other forms of PSVT including AVNRT, AVRT with concealed accessory pathway, and focal AT. According to the 2020 European Society of Cardiology guidelines on sports cardiology, patients with a history of PSVT without ventricular preexcitation are allowed to participate in sports activities (class I). Pharmacotherapy such as β-blockers and sodium channel blockers are generally avoided given their potential to affect an athlete's performance. In addition, β-blockers are prohibited by the World Anti-Doping Agency in certain sports. Ablation may be mandated as "definitive" therapy in occupations such as airline pilots, commercial drivers, and military personnel.

Limitations

This Review has several limitations. First, the search may have missed relevant publications. Second, a formal quality assessment of included articles was not performed. Third, this was not a systematic review. Fourth, SVTs commonly occur in the growing population of patients with congenital heart disease,[74,75] but we did not include a systematic review of this special population of patients.

Conclusions

Paroxysmal SVT affects both adult and pediatric populations and is generally a benign condition. Catheter ablation is the most effective therapy to prevent recurrent PSVT. Pharmacotherapy is an important component of acute and long-term management of PSVT.

ARTICLE INFORMATION

Accepted for Publication: January 3, 2024.

Correction: This article was corrected on February 20, 2024, to fix data in the abstract regarding prevalence of PSVT.

Conflict of Interest Disclosures: Dr Zei reported receipt of grants from Biosense Webster and personal fees from Abbott and Medtronic. No other disclosures were reported.

Submissions: We encourage authors to submit papers for consideration as a Review. Please contact Kristin Walter, MD, at kristin.walter@jamanetwork.org.

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