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AHC Media

Supraventricular Tachycardia: A Review for the Practicing Emergency Physician

I have been practicing emergency medicine long enough to have experience treating paroxysmal supraventricular tachycardia (PSVT) without adenosine or even verapamil. I just looked at my first edition of "Tintinalli," the one that came in two three-ring binders. How many of you still have it? If you are willing to part with it, let me know. But I digress. The treatment sequence recommended for PSVT in the Cardiac Arrhythmias chapter from that first version was: 1) facial immersion in ice water; 2) sedation with diazepam; 3) edrophonium IV; 4) vasopressor infusion with metaraminol or levarterenol; 5) digoxin IV; 6) propranolol IV; and 7) synchronized cardioversion. We have come a long way since that chapter was written. We now have better understanding of these dysrhythmias and better drugs to manage them. As they say, the good old days were not always so good. Enjoy this issue.

—J. Stephan Stapczynski, MD, FACEP, Editor

Introduction

Supraventricular tachycardias (SVT) are a common category of dysrhythmias seen in the emergency setting. The term SVT refers to a variety of rhythm disturbances that are characterized by a fast or tachycardiac rhythm (ventricular rate > 100 beats per minute [bpm] in an adult) that originates above the atrioventricular (AV) node. SVT types vary from the generally benign sinus tachycardia and paroxysmal supraventricular tachycardia (PSVT), to unifocal and multifocal atrial tachycardia (MAT), to the more serious conditions of atrial flutter and atrial fibrillation. Confusion can arise as practitioners may also use the general term interchangeably with a specific type of SVT, like PSVT. Further confusion arises because there are two types of PSVT based on the specific conduction pathway abnormality: atrioventricular reentrant tachycardia (AVRT) and atrioventricular nodal reentrant tachycardia (AVNRT). In this review each rhythm is referred to by its specific name.

Recognition, diagnosis, and treatment are important skills for emergency

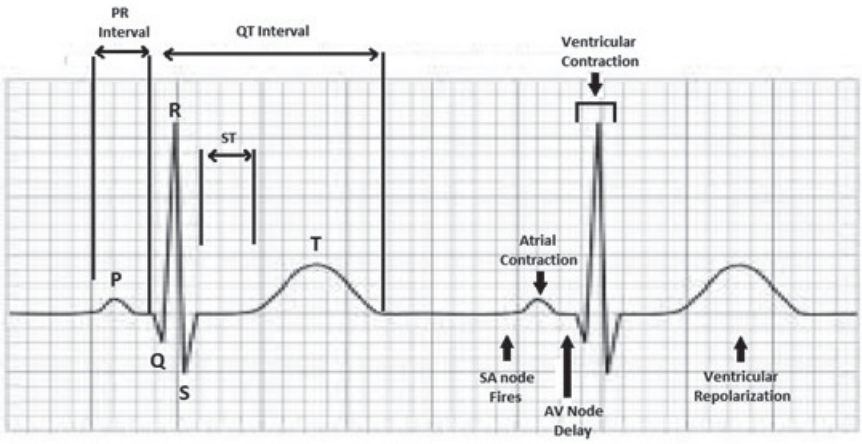
EXECUTIVE SUMMARY

- Current guidelines recommend DC cardioversion when patients become hemodynamically unstable due to supraventricular tachycardia (SVT).
- Adenosine is generally safe to terminate SVTs (with the exception of wide complex rhythms caused by Wolff-Parkinson-White syndrome) and assist in the diagnosis of atrial fibrillation or flutter.
- AV node blocking agents (beta-blockers, calcium channel blockers, adenosine, and digoxin) are CONTRAINDICATED in patients with wide-complex rhythms associated with WPW.
- If adenosine fails to terminate the SVT, calcium channel blockers (verapamil) or beta-blockers (esmolol or metoprolol) can be used for conversion or ventricular rate control.
- Anticoagulation (heparin) is recommended before cardioversion of atrial fibrillation of greater than 48 hours duration.

physicians because of the distinct differences in diagnosis and treatments for each type of SVT. SVTs may be physiological (i.e., an appropriate sinus tachycardia during an asthma exacerbation) or pathological (i.e., atrial fibrillation associated with a dilated cardiomyopathy). SVTs may arise from either an ectopic pacemaker or, more commonly, impulse reentry. SVTs are classified based on physiology and typically diagnosed from the surface electrocardiogram (ECG). Diagnosis is sometimes facilitated through a careful history regarding the abruptness of onset of palpitations and ECG tracings at the onset and/or termination of the dysrhythmia. Antiarrhythmic treatments are aimed at terminating the impulse pathology responsible for maintaining the dysrhythmia. Reentry may be terminated using vagal maneuvers, medications, cardioversion, or surgical ablation, depending on the clinical stability of the patient and origin and location of the abnormal conduction.

This review focuses on the two most common forms of pathological SVT: atrioventricular nodal reentrant tachycardia (AVNRT) and atrioventricular reentrant tachycardia (AVRT), which is commonly associated with the Wolff-Parkinson-White syndrome (WPW). Sinus tachycardia, atrial fibrillation, atrial flutter, and multifocal atrial tachycardia are briefly discussed for purposes of recognition.[1-4]

Figure 1. A Normal PQRST Complex with the Physiologic Actions Responsible for Each Event Labeled



AV, atrioventricular; SA, sinoatrial

Table 1. SVT Classification Based on Electrical Origin and Regularity on ECG

Origin	Regular	Irregular
Atrial	Sinus tachycardia Atrial tachycardia (AT) Atrial flutter (A flutter) Sinoatrial node reentrant tachycardia	Atrial fibrillation (A fib) Atrial flutter with variable block Multifocal atrial tachycardia (MAT)
Atrioventricular	Atrioventricular reentrant tachycardia (AVRT) AV nodal reentrant tachycardia (AVNRT) Automatic junctional tachycardia	

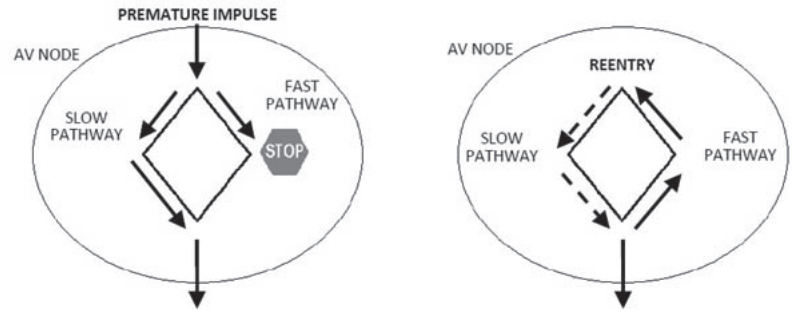
Epidemiology

SVTs are estimated to have an incidence of 35 cases per 100,000 persons-years, with a two-fold risk in females compared to males.[3] SVT is seen in all age groups, and has even been observed in utero.[9] SVTs are also the most common dysrhythmia found in pediatric populations, affecting 1:250 to 1:1000 infants and children.[5,6] Children with congenital heart disease, accessory conduction pathways, and inherited ion channelopathies are the most common pediatric groups afflicted with SVTs.[7] When examined by subtype, AVNRT accounts for approximately 50-60% of adult SVT, and AVRT accounts for 30% of pediatric SVT.[8]

SVT Classification and ECG Recognition

In order to discuss the electrical abnormalities seen in SVTs, the normal

Figure 2. AV Node Reentry Circuit



Two separate conduction pathways exist within (or in close proximity) to the atrioventricular (AV) node that have differing conduction velocities (indicated by "slow" and "fast" pathways) and refractory periods. Reentry is initiated when a premature impulse reaches the node and finds the fast pathway refractory, and travels down the slow pathway. Upon reaching lower end of the circuit, the fast pathway has now recovered and the depolarization is able to travel back up that recovered limb. The reentrant loop is maintained by the impulse traveling retrograde up the fast pathway and anterograde back down the slow pathway again, resulting in rapid ventricular response.

Figure adapted from: Ahluwalia M, Quest T. Supraventricular tachycardia (SVT): Strategies for diagnosis, risk stratification, and management in the emergency department setting. *Emerg Med Reports* 2002; 23 (4):41–52.

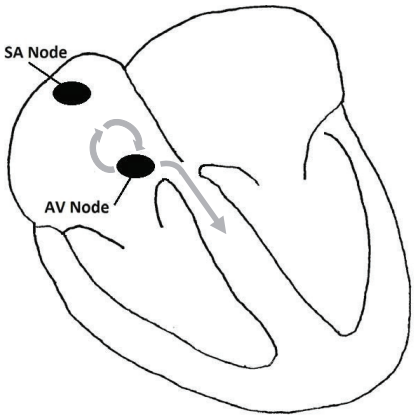
conduction pathways will be briefly reviewed. (*See Figure 1.*) The sinoatrial (SA) node is normally the dominant pacemaker of the heart and SA depolarization initiates cardiac activity during sinus rhythm. SA depolarization is electrically silent on the ECG. The inherent rate of SA node depolarization is between 60 and 100 bpm, compared to the inherent rate of pacemaker tissue found in nodal and ventricular tissue of 40 to 60 bpm. The faster firing rate of the SA causes it to be the dominant pacemaker of the heart. However, other areas in the heart can become the dominant pacemaker if its spontaneous depolarization rate is faster than that of the SA node.

The SA discharge depolarizes the adjacent atrial tissue, then subsequently spreads through the atria and toward the AV node. The P wave on the ECG is produced by atrial depolarization and is typically upright (a positive deflection) in leads I and AVF during sinus rhythm, reflecting that atrial depolarization is right to left, and superior to inferior.

Conduction through the AV node, corresponding to the PR interval on the ECG, is slow and serves as protection that prevents excessive ventricular stimulation from accelerated atrial rhythms. As it leaves the AV node, the impulse is conducted to the ventricles via the His-Purkinje fibers, which are specialized fibers that distribute the impulse very rapidly and efficiently to the ventricles. The right bundle branch distributes the signal to the right ventricle, and the left bundle to the left ventricle. The normal, narrow QRS wave on ECG results from ventricular depolarization rapidly spread via the His-Purkinje system. Blockage of impulse conduction in either of the bundles produces the characteristic ECG pattern of a bundle branch block. Blockage in the AV node, Bundle of His, or both bundles results in complete AV dissociation or third-degree heart block.

Wide-QRS complex rhythms (abbreviated WCT) are usually produced by tachycardias that arise below the AV node, as they do not utilize the His-Purkinje fiber system. Tachycardias from supraventricular origin normally produce narrow QRS complexes as they travel on the normal conduction pathways. However, important exceptions exist, including tachycardias that produce rate-related bundle branch blocks, patients with underlying bundle branch disease or ventricular conduction abnormalities, or bypass tracts that circumvent the AV node altogether. As a result, one cannot always identify SVTs simply based on the absence of wide-QRS complexes.

Figure 3. AV Nodal Reentrant Tachycardia (AVNRT)

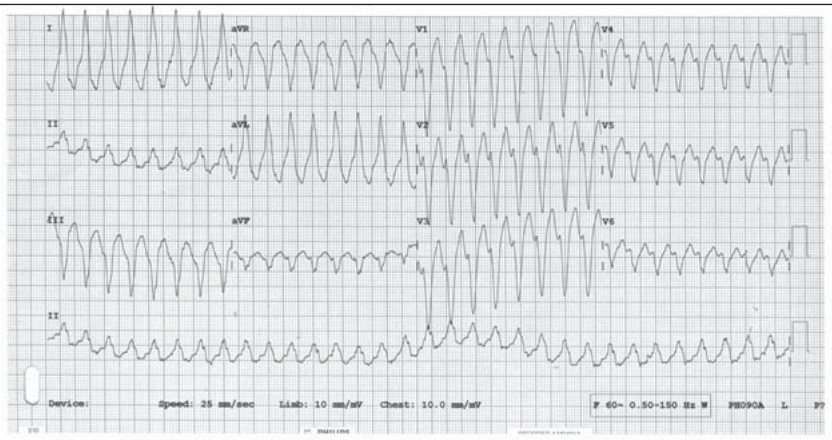


Re-entry is sustained in a small circuit within or close by the AV node.

Sinus tachycardia is the most common SVT, followed by atrial fibrillation, atrial flutter, and finally AVNRT, AVRT, and atrial tachycardia.[10] (*See Table 1.*) Pathological SVTs due to enhanced "automaticity" result from an abnormal site of impulse formation. As mentioned above, tachycardia can be caused by any pacemaker focus in the heart whose firing rate exceeds that of the SA node.[3] Such ectopic pacemaker tissue has been demonstrated in the atria, AV junction, or neighboring tissues in the vena cava and pulmonary veins.[3]

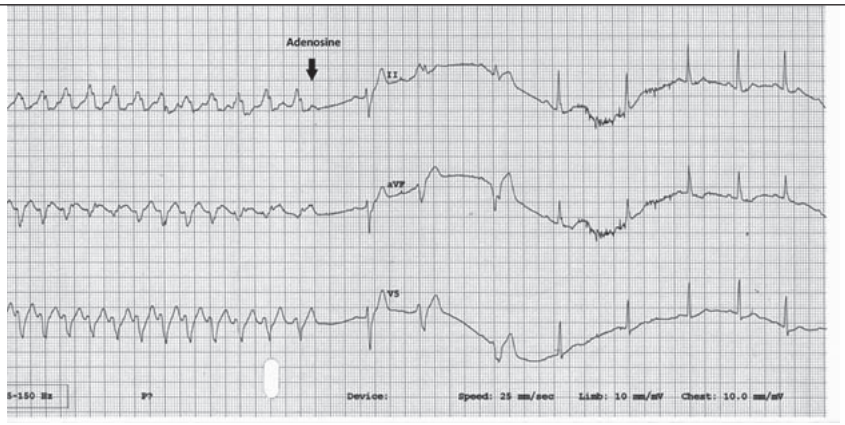
SVTs resulting from "reentry" are those with an impulse sustained via conduction around a loop of conductive tissue. In sustained reentrant tachydysrhythmias, two separate conduction pathways (either inside or outside the AV node) are connected at either end, forming a loop of conductive tissues. In order for reentry to be initiated and sustained, one of the pathways must usually conduct more slowly than the other and also have different refractory periods. (*See Figure 2.*)

Figure 4A. 12-lead ECG of AVNRT and Aberrant Conduction (Rate-dependent LBBB)



Differentiation between AVNRT with aberrant conduction and ventricular tachycardia is not always possible.

Figure 4B. Conversion of AVNRT with Aberrancy to Sinus Rhythm with Adenosine 6 mg IV



Reentry is usually initiated by an ectopic beat (premature atrial or ventricular contraction, PAC or PVC, respectively) that encounters one tract refractory and the other capable of conduction. During conduction on the open limb, the refractory limb recovers and is capable of conducting retrograde back toward where the impulse originated. As the impulse travels back toward the origin, the original tract is ready to conduct again resulting in a "closed loop" impulse conduction that sustains itself and produces a rapid ventricular response. Reentrant SVT can be induced by processes that increase ectopy, such as alcohol, recreational stimulant drugs, or hyperthyroidism, and possibly nicotine and caffeine.[11]

Sinus Tachycardia. Sinus tachycardia is a normal physiologic response to an intrinsic or environmental stressor. Common stressors include prescription medications (albuterol, aminophylline), stimulants (caffeine, nicotine, etc.), hypovolemia, anemia, as well as pain, fever, and recreational stimulants (amphetamines, cocaine, ecstasy, or MDMA).[13] The rhythm is distinguished by gradual onset and resolution, with P waves preceding each QRS complex, and a ventricular rate that usually does not exceed 220 bpm minus the patient's age.[10]

Atrioventricular Nodal Reentrant Tachycardia (AVNRT). By definition, the AVNRT reentrant circuit is in or closely related to the AV node and is dependent on AV nodal conduction to maintain the electrical circuit.[12] (*See Figure 3.*) This is the most common type of reentrant SVT in adult patients with structurally normal hearts, accounting for 50-60% of cases.[14] It is typically paroxysmal, and is more common in women at 75% of all cases.[12] The tachycardia is typically regular, usually around 180 bpm, but with a range of 140-280 bpm, and the QRS complex is typically narrow unless a bundle branch block or accessory pathway is present.[4]

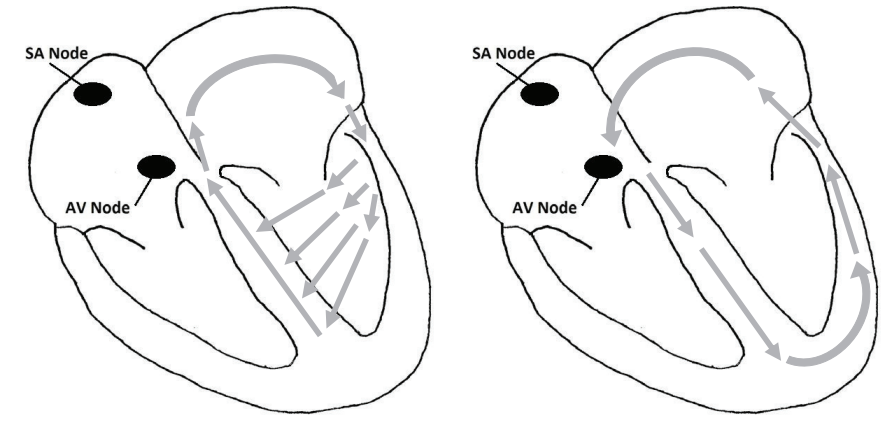
There are two types of AVNRT, typical and atypical.[4] In sinus rhythm the impulse normally travels through the fast pathway to depolarize the ventricles. In typical AVNRT, if a PAC occurs while the fast pathway is still refractory, the impulse instead travels down the slow pathway, resulting in ventricular depolarization with prolonged PR interval. In typical AVNRT, this impulse then continues in a retrograde direction up the fast pathway that is now recovered from its refractory period, establishing the reentry circuit.[3,15] (*See Figure 2.*) Typical AVNRT is about nine times more common than typical AVNRT.

In atypical AVNRT, the reentry circuit travels in the opposite direction, anterograde down the fast pathway and retrograde through the slow pathway.[4,13] Propagation around this circuit can produce different ECG findings. In typical AVNRT, the retrograde P waves are seen within or in close proximity to the terminal portion of the QRS complex. In atypical AVNRT, the retrograde P waves are within or after the T wave.

In some patients aberrant infranodal conduction may be present, such as pre-existing or rate-dependent bundle branch blocks. (*See Figure 4A.*) Conversion to sinus rhythm can occur with adenosine, a drug with specific effects on the AV node. (*See Figure 4B.*)

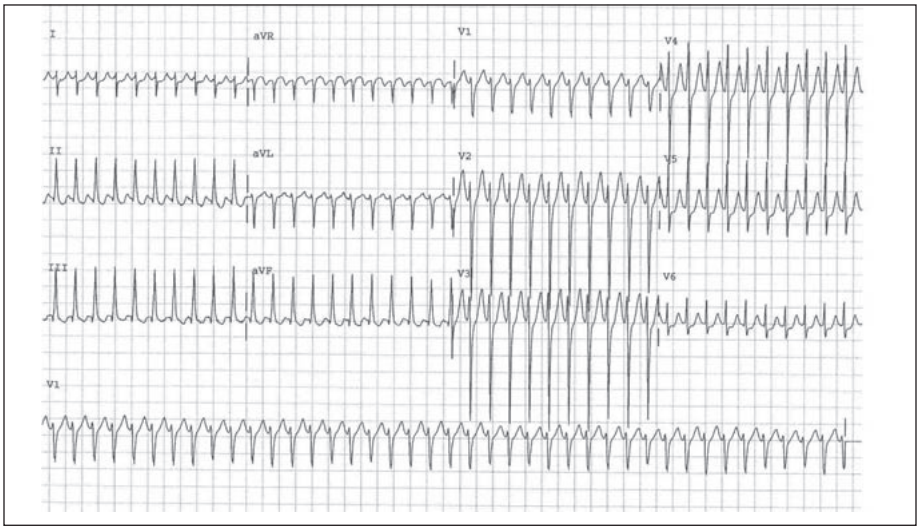
AVRT/Wolff-Parkinson-White Syndrome (WPW). AVRT is the next most common reentrant SVT in patients with otherwise structurally normal hearts, accounting for 25-30% of cases, most commonly seen in children and teenagers.[14] In AVRT there is conduction through both an electrically

Figure 5. Antidromic (left) and Orthodromic (right) AV Reentrant Tachycardia



During Antidromic conduction, the impulse is propagated down the bypass tract, slowly through the ventricular myocardium, retrograde up the His-Purkinje system where it is conducted through the atria back to the bypass tract, thus sustaining re-entry. During Orthodromic conduction, the impulse is conducted down the His-Purkinje system where it spreads rapidly through the ventricular myocardium, and is conducted retrograde up the bypass tract and through the atria back to the AV node, thus completing the re-entry circuit.

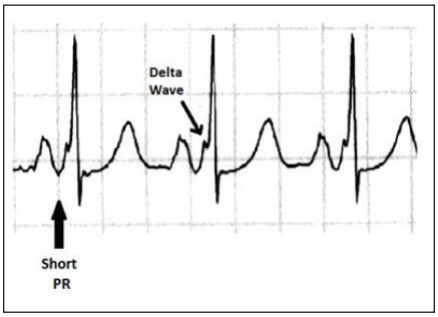
Figure 7. Orthodromic SVT in a Patient with WPW Syndrome



normal AV node and a physically separate, accessory extra-nodal pathway. Reentry most commonly occurs with orthodromic conduction where the impulse travels down the AV node (antegrade) and back up (retrograde) the accessory tract producing a narrow QRS complex. (*See Figure 5.*) Antidromic conduction occurs in the reverse direction where the impulse travels down the accessory tract (antegrade) and back up (retrograde) through the AV node.

During sinus rhythm, antidromic conduction of the atrial depolarization stimulates a portion of the ventricle before the impulse can reach the ventricles via the AV node-His bundle-Purkinje pathway. This "pre-excitation" of a portion of the ventricles produces an initial slurring of the QRS complex on

Figure 6. Delta Wave



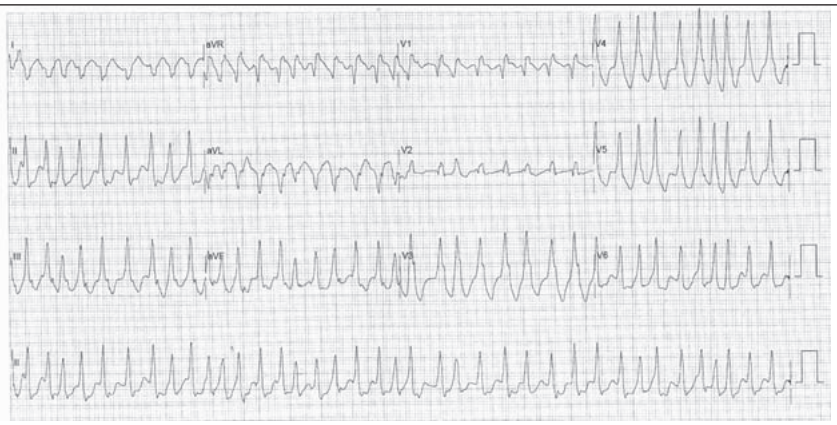
ECG. (*See Figure 6.*) The initial slurring of the QRS seen during sinus rhythm is called the delta wave.

Wolff-Parkinson-White (WPW) syndrome is the most common condition associated with AVRT. WPW syndrome is diagnosed when a patient is in sinus rhythm by identification of ventricular pre-excitation (delta wave), shortened PR interval, and wide QRS complex.[10,17] (*See Figure 6.*) In some cases, conduction via the accessory pathway is variable, and delta waves and shortened PR intervals are present only intermittently, with periods of normal-appearing QRS complexes. Thus, the baseline ECG may not show evidence of accessory pathway during sinus rhythm. WPW is uncommon, seen in 0.2% of the population.[19] While WPW can be associated with structural abnormalities, like Ebstein anomaly (tricuspid valve placed abnormally low in the right ventricle), most patients have otherwise structurally normal hearts.

AVRT in patients with WPW is usually orthodromic with a narrow QRS complex. (*See Figure 7.*) Narrow complex tachycardia in these patients does not require deviation from the standard approach, but wide complex tachycardias are a different situation.

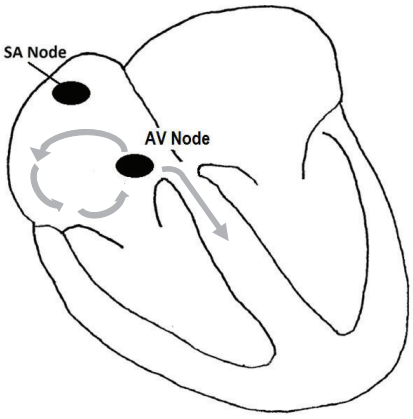
The occurrence of atrial fibrillation or flutter in patients with accessory tracts can result in excessive ventricular stimulations because the rapid atrial impulses can reach the ventricles, bypassing the protective function of the AV node. Atrial fibrillation or atrial flutter in WPW patients generally presents with an irregular wide complex tachycardia, distinguishing it from ventricular tachycardia, which is a regular wide complex

Figure 8. 12-lead ECG Wide Complex, Irregular Tachycardia Produced by Atrial Fibrillation in a Patient with Underlying WPW Syndrome



The irregular rate distinguishes this rhythm from ventricular tachycardia, which usually produces a regular, wide complex rhythm.

Figure 9. Atrial Flutter



Atrial flutter is most commonly sustained by a re-entry circuit involving the inferior portion of the right atrium.

Figure 10. 12-lead ECG of Atrial Flutter with a 2:1 Block

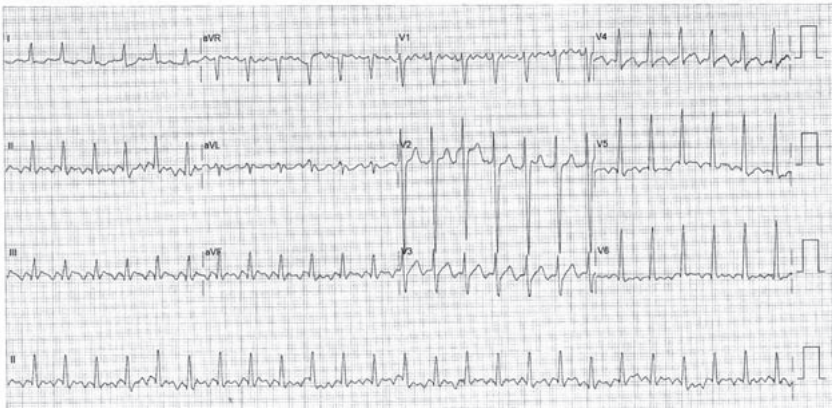
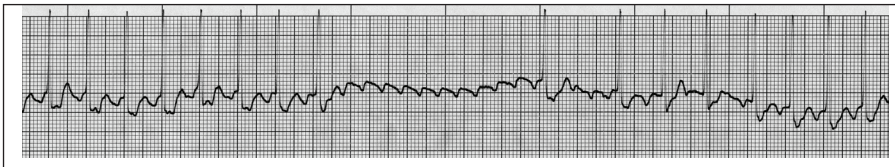


Figure 11. Transient Induction of AV Nodal Block by Carotid Sinus Massage in Atrial Flutter Making the Flutter Waves More Visible



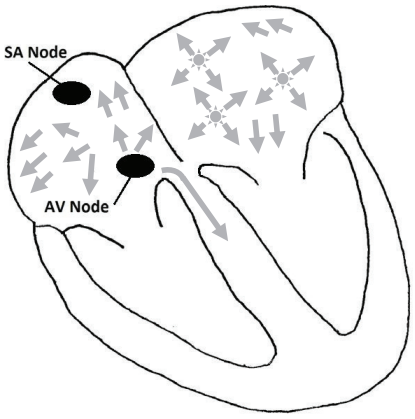
tachycardia. (*See Figure 8.*)

Atrial fibrillation in WPW is a very characteristic rhythm presentation, not only demonstrating the widened QRS complex but also beat-to-beat variation in the QRS complex morphology and extremely rapid rate.

Excessive stimulation of the ventricles can deteriorate into ventricular fibrillation. Fortunately, the incidence of spontaneous sudden death from ventricular fibrillation secondary to rapid conduction of atrial fibrillation in patients with pre-excitation syndromes is relatively rare, about 0.15-0.45% deaths per patient-year.[11] Risk of decompensation is dramatically increased when conduction down the AV node is iatrogenically blocked, leaving only the accessory pathway conducting rapid atrial impulses to the ventricles. Thus, any AV node-blocking agent is contraindicated in patients with a wide complex tachycardia from known or suspected WPW. Specifically, adenosine is not an appropriate treatment in this case, as it inhibits conduction down the AV node, allowing uninhibited conduction down the accessory pathway.[3] As accessory pathways may be concealed in narrow-complex orthodromic SVT, a defibrillator should always be available when administering adenosine in the event of decompensation into VF. If WPW is suspected or known from a baseline ECG, procainamide or ibutilide are the drugs of choice to slow conduction down the accessory pathway.[3]

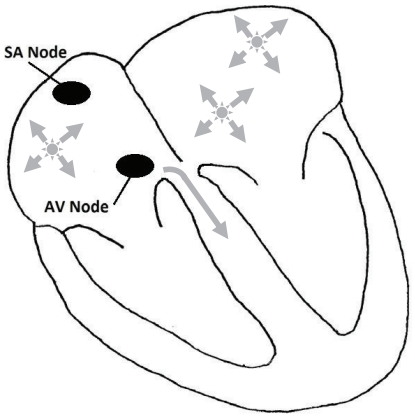
Atrial Flutter. Typical atrial flutter refers to a reentry circuit that develops in the right atrium around the tricuspid annulus, where the cavotricuspid isthmus is the path of slow conduction.[4] (*See Figure 9.*) Nearly all typical atrial flutters revolve counterclockwise around the tricuspid annulus, which produces negative flutter waves in inferior leads and positive waves in the anterior precordial leads on ECG.[20] (*See Figure 10.*) This rhythm typically produces an atrial

Figure 12. Atrial Fibrillation



Atrial fibrillation is due to multiple small re-entry circuits within the atria that produce a chaotic pattern of atrial depolarizations. The AV node blocks most of these impulses and those that are conducted produce an irregular ventricular rhythm.

Figure 14. Multifocal Atrial Tachycardia (MAT)



At least two areas within the atria develop spontaneous depolarization, thus competing with the SA node for cardiac pacing. MAT has at least three different P wave morphologies and an irregular ventricular rhythm.

rate of 250-300 bpm, which is blocked at the AV node, most often in a 2:1 fashion, yielding a very steady ventricular rate of around 150 bpm. (*See Figure 10.*) Unfortunately, with 2:1 block, the non-conducting P waves (flutter waves) are often obscured by the T waves, making the diagnosis on ECG less obvious. Even so, the persistent ventricular rate of 150 bmp is highly suggestive of atrial flutter. If unsure of the underlying rhythm, inducing a transient AV block by carotid sinus massage or adenosine can often expose the underlying "saw-tooth" pattern of atrial flutter. (*See Figure 11.*)

While 2:1 AV block is most common in atrial flutter, variable or higher blockade ratios can occur, producing slower ventricular rates. In addition, patients taking AV-nodal blocking agents (beta-blockers, calcium channel blockers), or those with underlying AV nodal disease, may have rates less than 150 bpm.

Atrial Fibrillation. Atrial fibrillation is a common form of SVT, with a prevalence of 1-2% in the general population.[21] This number rises to nearly 15% in those older than 80 years of age,[22] and the average 40-year-old has a lifetime risk of 25% for developing atrial fibrillation.[23] Atrial fibrillation is associated with a wide variety of medical disorders: hypertension,[24] congestive heart failure,[25] valvular heart disease,[26] chronic kidney disease,[27] obesity,[28] alcohol intake,[29] chronic obstructive pulmonary disease,[30] and pulmonary embolism.[31] Atrial fibrillation typically begins as an episodic arrhythmia, but as time passes the heart structurally remodels to support a constant abnormal rhythm. Thus, the more time spent in atrial fibrillation, the higher the likelihood that one will remain in atrial fibrillation. The consequences of atrial fibrillation are significant, as the risk of embolic stroke and dilated cardiomyopathy are increased.[32]

ECG features of atrial fibrillation will help the practitioner differentiate it from other types of SVTs. In this rhythm, multiple foci spread impulses through the atria, disrupting the normally organized atrial contraction. (*See Figures 12 and 13.*) Small reentry-type circuits produce extremely rapid impulses with local rates from 300 to 500 bpm.[33] These impulses reach the AV node in a haphazard fashion, and the refractory period of the AV node results in chaotic conduction of impulses to the ventricles. Thus, the hallmark of atrial fibrillation on ECG is an irregularly irregular rhythm with absent P waves. QRS complexes are narrow unless there is underlying bundle branch disease. Often, patients present with a rapid ventricular rate, which, unfortunately, makes the absence of P waves more difficult to detect. As in atrial flutter, a trial of adenosine will slow the ventricular rate and may reveal the underlying lack of P waves.

Atrial Tachycardia (AT). ATs are relatively rare and only comprise about 10% of all SVT cases. ATs may originate from an isolated atrial focus in the atrium (focal atrial tachycardia), from a large re-entrant circuit (macro re-entry, such as atrial flutter), or simultaneously from multiple atrial foci (multifocal atrial tachycardias or MAT).[11,34] (*See Figure 14.*) (*See Figure 15.*) In contrast

Figure 13. Atrial Fibrillation with Chaotic Atrial Depolarization and Irregular Ventricular Rhythm

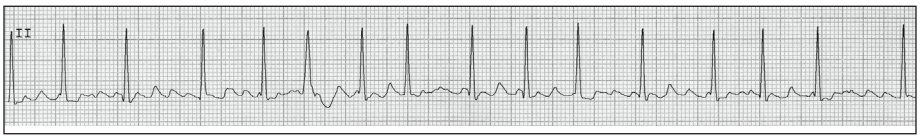
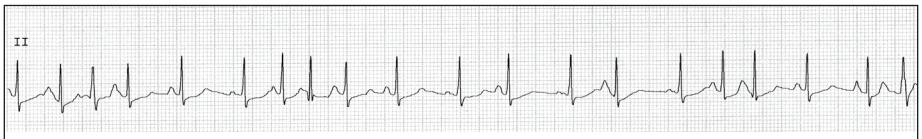
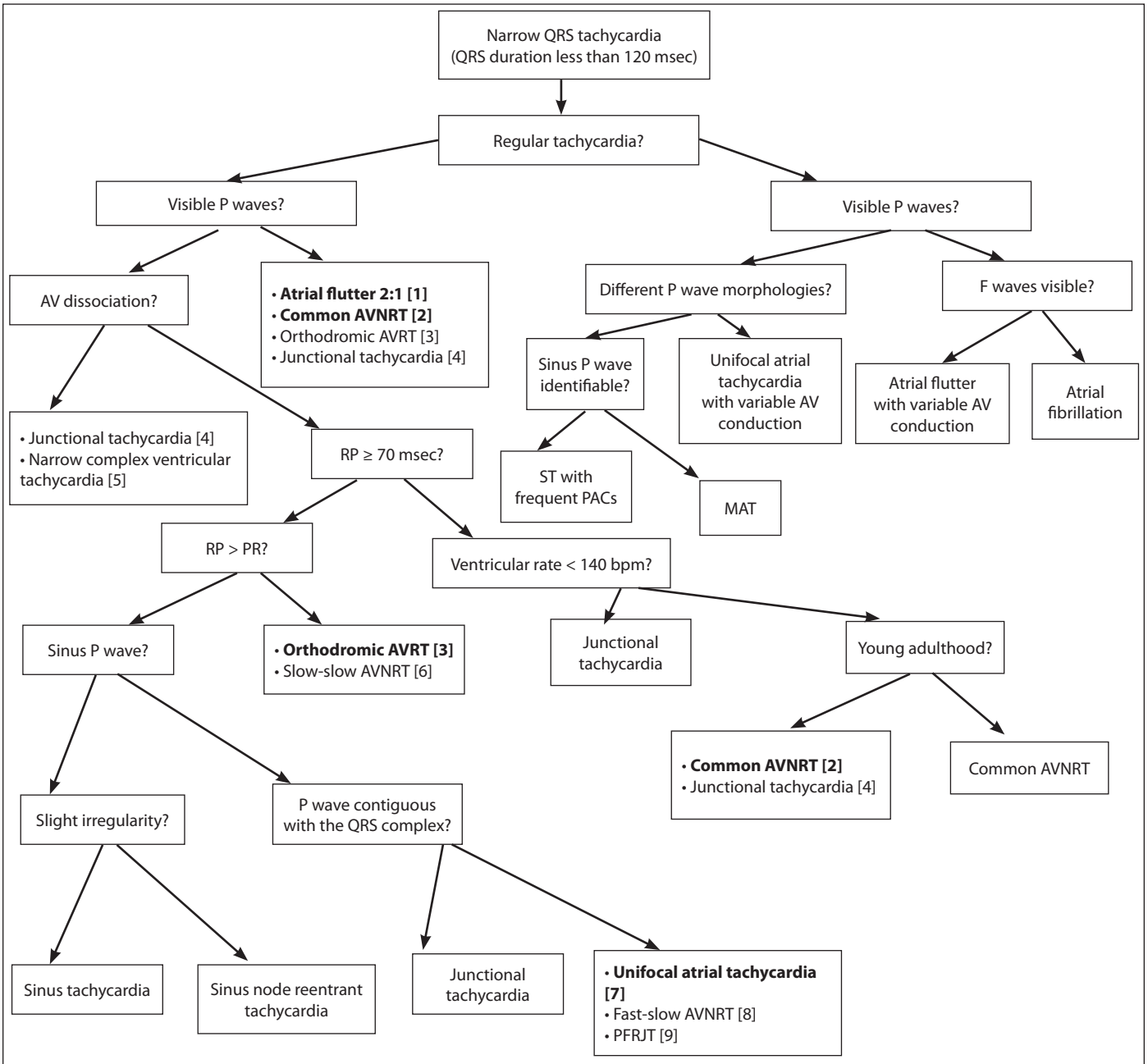


Figure 15. Multifocal Atrial Tachycardia



Multiple P wave morphologies in a single lead and irregular ventricular rhythm

Figure 16. ECG-based Algorithm for Diagnosis of Regular, Narrow QRS Tachycardias



Bolded text indicates the most likely diagnosis in each box. AV, atrioventricular; VT, ventricular tachycardia; AVRT, atrioventricular reentry tachycardia; AVNRT, atrioventricular nodal reentrant tachycardia; MAT, multifocal atrial tachycardia. Adapted from Butta C, Tuttolomondo A, Di Raimondo D, et al. Supraventricular tachycardias: Proposal of a diagnostic algorithm for the narrow complex tachycardias. *J Cardiol* 2013;61(4):247-255.

to the other SVT types, ATs may occur in repetitive short bursts.[10] ATs may be caused by digoxin toxicity, structural abnormalities such as atriotomy scars, catecholamines, heart failure, or chronic obstructive pulmonary disease.[8,12]

ECG findings in AT vary somewhat depending on the variety. Focal atrial tachycardias involve rapid electrical impulse generation from a distinct atrial location, producing simple normal-appearing P waves that precede each QRS.[34] However, the P-R interval is typically longer than it should be. As the name suggests, multifocal ATs are characterized by multiple premature beats that originate in several distinct areas in the atria, producing at least three different P wave morphologies. Because MAT has multiple foci of electrical activity, it can be difficult to

distinguish from atrial fibrillation.[34] MAT is seen less often than in the past, as theophylline use was historically one of the most common causes.[10] Other causes include atrial hypoxia or increased atrial pressure, and thus MAT is classically found in patients who are critically ill with pulmonary disease, sepsis, or digitalis toxicity.[3]

Clinical Presentation

Symptoms of SVT typically include acute onset and termination of palpitations, lightheadedness, chest pain, abnormal pulsations in the neck, and dyspnea that last between seconds to several hours.[11] This is in contrast to sinus tachycardia, which is non-paroxysmal and has gradual onset and termination. Less commonly, symptoms of SVT may include syncope secondary to vasovagal response or compromised cardiac output (occurring in 15% of presentations), and polyuria secondary to the diuretic effects of atrial natriuretic factor.[3,11,12] Metabolic derangements may induce tachydysrhythmias, so consider checking serum potassium, magnesium, and TSH, although these tests are usually low-yield.[11]

SVT is more challenging to diagnose in pediatric populations. Symptoms range from irritability, tachypnea, poor feeding, and pallor in infants, to complaints of chest pain, palpitations, shortness of breath, and fatigue in older children of verbal age.[35] SVT is typically well-tolerated in infants, but may progress to heart failure if SVT persists for longer than 12-48 hours.[35]

Troponins in SVT. Up to 33% of patients with SVTs experience chest pain.[36] As such, troponin levels may be ordered on these patients in many emergency departments through pre-established protocols — often before the physician has a chance to fully recognize and treat the SVT. Troponin elevation occurs after episodes of prolonged tachycardia in up to 12-48% of patients.[37] It is thought that mild, diffuse subendocardial ischemia is responsible for the enzyme elevations. The subendocardial tissue experiences mild ischemia after prolonged exposure to ventricular rates that are too fast to allow for adequate coronary flow during diastole. Thus, the physician may encounter the dilemma of dispositioning patients who have had their SVT successfully treated in the emergency department, but have documentation of an elevated troponin.

Specific guidelines for this situation have yet to be established, but several general recommendations can be given. Ideally, this dilemma can first be avoided by not obtaining troponin levels in younger/healthy patients with SVTs terminated in the emergency department. Secondly, the type of SVT can be helpful to consider when selecting which patients should have a troponin level tested. A literature review of patients with troponin elevations resulting from more benign rhythms, such as AVRT and AVNRT, found that no patients were ultimately diagnosed with coronary disease.[36] Lastly, elevations resulting from rhythms known to be more likely associated with underlying pathology — such as atrial flutter and fibrillation — should prompt cardiac consultation and/or further evaluation (i.e., trending of troponin levels). Therefore, in the vast majority of cases, further evaluation of troponin elevations in patients with successfully treated SVTs should be reserved for those with significant risk for coronary disease, older age, or those with persistent symptoms in the emergency department.

Diagnosis Using ECG

There are many algorithms for diagnosis of SVT based on ECG.[4,8] (*See Figure 16.*) The main ECG components used for making a diagnosis include: 1) QRS duration (narrow vs wide), 2) characterization of onset and termination, 3) heart rate, and 4) relative position of the P wave within the R-R interval.[10,12]

The first characteristic that should be noted on ECG is whether the QRS is narrow or wide (QRS > 120 msec). ECG documentation of arrhythmia onset and termination may not be possible when patients present after onset of the tachyarrhythmia; however, knowing this information may also be helpful in classifying the type of SVT. For example, AVNRT will usually start with a premature atrial beat and end with a P wave.[12]

Heart rate may also be helpful in diagnosing certain subtypes of SVTs. A rate of 150 bpm may indicate atrial flutter with 2:1 AV block, since the atrial rate during flutter is 300 bpm. Likewise, atrial flutter rates less than 160 bpm without clearly identifiable P waves may be seen in dysrhythmias in which a slow AV nodal pathway is present.[12]

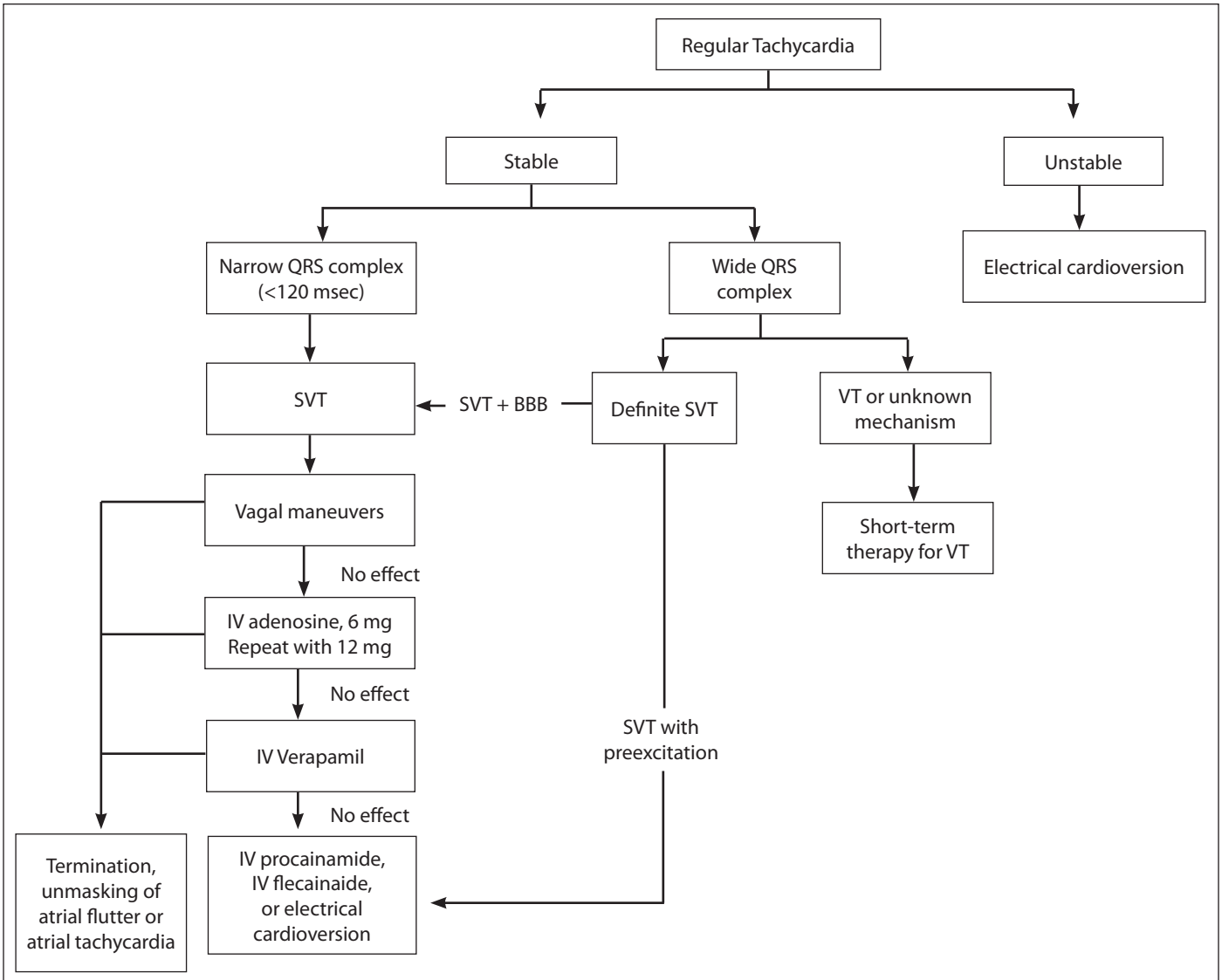
Finally, when P waves are present, identifying P wave position can be helpful in visualizing the pathological conduction. Leads II, III, aVF, and V1 are most useful for P wave examination, and P waves are normally upright in these leads. In AVNRT, AVRT, and atrial tachycardias, P waves are retrograde, and negative in the inferior leads because the atrium is activated in the opposite direction.[12] When the impulse is generated from a right atrial foci, the P wave is positive or biphasic in lead aVL and negative or biphasic in V1. In contrast, when the impulse is generated from the left atrium, the P wave is negative or isoelectric in lead aVL and positive in lead V1.[12]

In some cases the SVT will have resolved by the time the patient is evaluated in the emergency department, either spontaneously or from pre-hospital treatment. A baseline 12-lead ECG should also be performed to screen for pre-excitation if no previous ones are available.

Treatment: Hemodynamically Unstable Patient

The first and most critical feature of a patient with SVT is hemodynamic stability. Determining stability may sound straightforward, but in practice, identifying subtle hemodynamic instability can be more challenging. Systolic blood pressures must be interpreted according to the expected baseline of the patient. While systolic pressures below 90 mmHg are rarely normal, a pressure of 90mm Hg can be close to baseline in a pregnant patient or in patients of smaller stature. Likewise, this may be significantly low in a patient with history of hypertension whose "normal" systolic is closer to 200 mmHg. When available, old records that provide evidence of the patient's normal blood pressure range may be very useful.

Figure 17. Treatment-based Algorithm for Management of SVT in the ED



VT, ventricular tachycardia; SVT, supraventricular tachycardia; BBB, bundle branch block; msec, milliseconds.
Reprinted with permission from Delacretaz E. Clinical practice. Supraventricular tachycardia. *N Engl J Med* 2006;354 (10):1039-1051.

When prior blood pressure information is not present, the best approach is to assess for physiologic signs of instability. These include altered mental status, chest pain, diaphoresis, and delayed capillary refill. If all of these parameters are normal, one usually has some time to further assess the patient and give intravenous fluids to help support blood pressure before pharmacologic treatment.

When it is determined that an SVT rhythm has caused the patient to become hemodynamically unstable, then one should proceed immediately with direct current (DC) synchronized cardioversion. Begin with lower energy (50 J), and escalate as needed up to 200 J. Most cases of SVT will resolve after only 1–2 shocks.

The question of increased risk of embolic stroke arises when discussing cardioversion of atrial tachydysrhythmias. This has been well studied in patients with atrial fibrillation.[38] Hemodynamically unstable patients with acute onset atrial fibrillation (< 48 hours) are treated the same as unstable patients with any other SVT, as the risk of embolic stroke in these patients is less than 1%.[39] For unstable patients with more than 48 hours of atrial fibrillation, initiation of heparin therapy followed by outpatient oral anticoagulation for 4 weeks is recommended after electrical cardioconversion.[38]

Treatment: Hemodynamically Stable Patient

Guidelines for treatment of SVTs have been developed.[11,40] (*See Figure 17.*) After assessing for hemodynamic stability, the first step is to distinguish narrow vs. wide QRS complex. Narrow QRS complex is defined as < 120 ms in width.[40]

Vagal maneuvers are recommended

Table 2. Drug Treatment for SVT in Adults

Drug	Dosage	Comments
Drugs to Block AV Nodal Conduction		
Adenosine	6 mg rapid IV push, if after 2 minutes the dysrhythmia persists, repeat rapid IV push with 12 mg, may repeat once more of dysrhythmia persists	Effective in terminating narrow QRS complex reentrant tachydysrhythmias involving the AV node
Verapamil	2.5-5 mg IV bolus over 2-3 minutes, if after 15 minutes the dysrhythmia persists, may repeat doses 5-10 mg after 15-30 min	Effective in terminating narrow QRS complex reentrant tachydysrhythmias involving the AV node and reducing ventricular rate in atrial fibrillation or flutter
Diltiazem	15-20 mg IV bolus over 2 minutes, followed by IV infusion at 5-20 mg/h	
Esmolol	500 mcg/kg IV bolus over 60 seconds, followed by IV infusion starting at 50 mcg/kg per minute, titrate infusion to desired heart rate	
Metoprolol	5 mg IV bolus, may repeat 5 mg IV every 5 minutes up to total dose of 15 milligrams	
Propranolol	30 mcg/kg IV over 1 minute, may repeat same dose every 2 minutes, up to total dose of 100 mcg/kg	
Drugs to Terminate Tachydysrhythmias		
Procainamide	15-17 mg/kg IV over 30 minutes, followed by IV infusion at 1-4 mg (20-80 mcg/kg) per min	Used in wide-complex tachydysrhythmias and new onset atrial fibrillation Median time to conversion of new-onset atrial fibrillation about an hour Caution in patients with AMI and LV dysfunction Infuse at rate of 20 milligrams/min to reduce adverse effects
Amiodarone	Stable patient: 150 mg IV over 10 minutes, may repeat same dose every 10 minutes up to total dose of 2 grams OR use IV infusion 0.5 mg/min Ventricular fibrillation or pulseless ventricular tachycardia: 300 mg IV bolus, may repeat with additional dose of 150 mg IV bolus	Used in wide-complex tachydysrhythmias and new-onset atrial fibrillation Preferred in setting of AMI or LV dysfunction Contraindicated in pregnancy
Ibutilide	Weight < 60 kg: 10 mcg/kg IV over 10 min Weight > 60 kg: 1 mg IV over 10 min	Used for conversion of new-onset atrial fibrillation or flutter. Median time to conversion 20-30 minutes
Flecainide	2 mg/kg IV over 10 minutes	Used for conversion of new-onset atrial fibrillation or flutter. Median time to conversion up to 4 hours Avoid in patients with ACS or cardiomyopathy
Propafenone	2 mg IV over 10 minutes	Used for conversion of new-onset atrial fibrillation or flutter. Median time to conversion 2 hours Avoid in patients with ACS, cardiomyopathy, or severe COPD
Vernakalant*	3 mg/kg IV infusion over 10 min, if dysrhythmia persists after 15 min, a second infusion of 2 mg/kg IV over 10 min can be given	Used for conversion of new-onset atrial fibrillation or flutter Median time to conversion 8-11 min Avoid in patients with hypotension, ACS within 30 days, severe aortic stenosis, and prolonged QT interval

Abbreviations: ACS = acute coronary syndrome; AMI = acute myocardial infarction; COPD = chronic obstructive pulmonary disease; LV = left ventricle; mg = milligrams; mcg = micrograms
* not available in the U.S.

as a first-line treatment to convert SVT; however, these techniques tend to be more successful early in the SVT episode and less successful once the patient is in the emergency department. Vagal maneuvers include carotid sinus massage, Valsalva maneuver, or ice pack to the face (most commonly used in children). Vagal maneuvers trigger an increase in activity of the vagal nerve and result in slowed conduction through the AV node, which may be effective at terminating AV nodal-dependent tachyarrhythmias.[11] The Valsalva maneuver is the most effective technique in adults, as is facial application of ice in infants. The Valsalva maneuver is performed by placing the patient supine, having the patient inhale a large breath and hold exhalation while at the same time tensing up the abdominal

wall muscles, ideally for 15–20 seconds until exhalation and resumption of normal breathing.[41] The increased vagal tone and potential termination or slowing of the SVT occurs during the release phase. Having the patient blow through a straw is another way to perform a Valsalva maneuver. If there is no history of carotid artery disease and no bruits on exam, carotid sinus massage may be performed by applying steady circular pressure at the level of the cricoid cartilage for no more than 5 seconds at a time. Massage only one carotid artery at a time.

All vagal maneuvers should be performed under continuous ECG-monitoring in order to better deduce the mechanism and SVT type in the event of successful termination.[11] If vagal maneuvers are unsuccessful, pharmacological treatments may be attempted to slow the conduction through the AV node or accessory pathway.[11]

Pharmacological treatments typically begin with a trial of adenosine (see below), except in obvious cases of atrial fibrillation or flutter, and where one can proceed directly to ventricular rate control. Often the SVT can be either terminated (as in AVNRT and AVRT) or the underlying rhythm exposed (as with atrial flutter or atrial fibrillation) after use of adenosine. If repeated doses of adenosine are ineffective, conversion can be attempted with intravenous verapamil. Remember that this agent is contraindicated in patients with wide complex tachycardias from known or suspected WPW, where procainamide is the drug of choice. Rate control for atrial flutter or fibrillation is typically achieved with calcium channel blockers or beta-blockers (see below).

Adenosine. Adenosine slows impulse formation in the SA node and slows conduction time through the AV node. As a result, adenosine is successful at terminating SVT in 60-80% of those patients treated with 6 mg, and 90-95% of those treated with 12 mg.[11] Dose range for pediatric populations is 100-250 micrograms/kg IV bolus.[7] Adenosine has an extremely short therapeutic duration (about 10 seconds), so it is administered via a rapid IV push followed by a saline flush when given via a peripheral IV line. The dose should be decreased by half the dose if given through a central access line. In rhythms that do not terminate with adenosine administration, adenosine has the advantage of slowing the heart rate to better determine the rhythm focus.[8] Adenosine is typically successful at terminating narrow complex AVRT and AVNRT, and will gradually slow and then reaccelerate sinus tachycardia. If no effect is seen after 2-3 doses of adenosine, a narrow-complex ventricular tachycardia may exist.[13] Atrial tachycardias (including atrial flutter and atrial fibrillation) are generally not converted to sinus rhythm by adenosine, but the transient AV block produced by adenosine often allows the underlying atrial rhythm to be revealed.

Uncommon but worrisome side effects of adenosine include bronchospasm and, in very rare cases, ventricular fibrillation, so adenosine should be administered under continuous ECG monitoring, and resuscitation equipment should be available.[11] To date, only a single case report each of cardiac arrest and respiratory arrest resulting from use of adenosine to terminate PSVT has been published.[42]

Common side effects of adenosine include chest tightness, flushing, and sense of dread.[10] Adenosine is contraindicated in patients with severe bronchospastic lung disease or wide complex QRS due to the high risk of increasing conduction down a bypass tract leading to atrial fibrillation and subsequent ventricular fibrillation.[7,8,10] Adenosine should be used with caution in heart-transplant recipients and patients who are on certain medications, such as theophylline, carbamazepine, or dipyridamole, although dose adjustments may be made in order to attempt administration to these patients.[11]

Calcium Channel Blockers. If adenosine is ineffective at terminating the narrow complex tachydysrhythmia, additional agents such as intravenous calcium channel blockers or beta-blockers may be tried.[11] Verapamil is a calcium channel blocker that induces a conduction block in the AV node and is useful for terminating AVNRT and orthodromic AVRT, although its success rate is less than adenosine.[8] The initial verapamil dose used to terminate SVT is 2.5-5.0 mg IV (0.1 mg/kg in children with maximum dose of 5 mg). Onset of action is slower than adenosine, so an observation period of 15 to 30 minutes is required to judge effectiveness and decide if an additional dose is required. The repeat dose is 5 to 10 mg (0.2 to 0.3 mg/kg in children with a maximum dose of 10 mg). Verapamil in not recommended in children younger than 2 years old and should not be administered as an IV preparation in patients with prior beta-blockade due to risk of asystole.[7]

In cases of atrial fibrillation or flutter, diltiazem is generally the first-line choice, followed by verapamil. Instead of terminating the abnormal conduction like adenosine, calcium channel blockers act to slow the heart rate.[8] The typical diltiazem dose is 0.25 mg/kg IV bolus followed by an infusion of 5 to 15 mg/hr. Adverse effects may include dizziness and exacerbation of heart failure or bradyarrhythmias.[7,8] Again, calcium channel blockers should be avoided in patients with WPW or wide complex tachycardias as they may cause increased conduction down the accessory pathway and result in induction of ventricular fibrillation.[8]

Beta Blockers. Beta blockers are used as additional agents in refractory SVT mediated by an accessory pathway,[7] and first-line agents in atrial fibrillation/flutter. Beta blockers are negatively ionotropic and are effective at decreasing heart rate. They are contraindicated in asthma, bradycardia, and congestive heart failure.[7] They are also relatively contraindicated in diabetes due to the blockage of hepatic gluconeogenesis and risk of hypoglycemia.[7] Side effects of beta blockers include bronchospasm, bradycardia, fatigue, insomnia, and depression.

Other Pharmacological Agents. If adenosine, calcium channel blockers, and/or beta blockers are ineffective (or contraindicated), third-line agents may be considered, including procainamide, ibutilide, propafenone, or flecainide.[11] These agents are useful for lengthening the antegrade refractoriness of accessory pathways and are usually effective in rapidly terminating atrial fibrillation.[11]

Again, if wide complex tachycardia is present, it is best to avoid adenosine and calcium channel blockers unless the physician is certain the dysrhythmia is an SVT with aberrant infranodal conduction. In almost all other circumstances, it is recommended to treat wide-complex tachycardias as ventricular tachycardia with procainamide, ibutilide, lidocaine, amiodarone, or sotalol.[10] Because of its long history of use, procainamide is recommended, and, if procainamide is unsuccessful at arrhythmia termination, it will decrease the risk of ventricular fibrillation in pre-excitation syndromes by impairing anterograde conduction down the accessory pathway.[16] Procainamide is contraindicated in second- or third-degree heart block and polymorphic ventricular tachycardia.

Referral

After successful treatment of SVT in the ED, referral to a cardiologist or an electrophysiologist is appropriate, especially if the patient had syncope with the dysrhythmia, if the patient works in a profession where a subsequent recurrence may create a high-risk situation (such as airline pilot, truck driver, or operator of large machinery), or if the patient engages in high-risk activities such as scuba diving or rock climbing. Phone consultation or consultation in the emergency department with a cardiologist is recommended for patients with exertional syncope or palpitations, wide-complex tachycardia, history of structural heart disease, severe symptoms, or evidence of pre-excitation (delta wave) on 12-lead ECG (regardless of whether SVT is present). Routine referral may be considered in situations in which there is diagnostic uncertainty.[8,11,34]

Long-term Therapy

If lifestyle adjustments such as decreasing environmental and drug triggers are ineffective, and SVT frequently recurs, patients may be considered candidates for prophylactic or curative therapy options.[7] Some patients may be managed on verapamil or beta blockers taken on an intermittent basis, but this may not be a feasible option in SVT cases that interfere with daily function.[8] Catheter ablation is growing in its use as a curative therapy for selectively destroying abnormal conduction pathways using radiofrequency ablation or cryoablation.[12] Catheter ablation may be used in nearly any type of SVT, but traditionally targets the accessory pathway in WPW syndrome, the slow pathway in AVNRT or AVRT (success rate of 95-97 %), or the abnormal impulse foci in atrial tachycardias, flutter, and fibrillation (success rate of 80%).[7,12] The main risk of catheter ablation is inadvertent production of AV block.[12]

Conclusion

SVTs encompass a spectrum of tachydysrhythmias each defined by unique electrical circuitry and ECG presentations. Proper treatment selection depends on the clinician's ability to understand the different types of SVT and rapidly recognize the specific dysrhythmia from the ECG. Consider comorbid conditions and available drug therapies for dysrhythmia management. Proper referral for long-term treatment after successful return of sinus rhythm is important.

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CME Questions

1. Which statement about AVNRT is true?
   A. AVNRT involves an accessory extra-nodal pathway.
   B. AVNRT is typical in the Wolff-Parkinson-White syndrome.
   C. AVNRT is more common than AVRT in pediatric and young adult populations.
   D. Typical AVNRT involves anterograde conduction down the fast pathway and retrograde conduction up the slow pathway.
2. In which scenario would adenosine be the most appropriate first-line therapy for SVT?
   A. 32-year-old male with sudden-onset palpitations, HR 160, BP 83/60, RR 28
   B. 18-year-old female with wide-complex irregular tachycardia, HR 180, BP 110/75, RR 24
   C. 45-year-old male with history of severe asthma and paroxysmal SVT, HR 160, BP 130/90, RR 20
   D. 23-year-old female with history of ADHD and sudden-onset palpitations, HR 170, BP 124/86, RR 25

EMERGENCY MEDICINE REPORTS

CME Objectives

Upon completion of this educational activity, participants should be able to:

- recognize specific conditions in patients presenting to the emergency department;
- apply state-of-the-art diagnostic and therapeutic techniques to patients with the particular medical problems discussed in the publication;
- discuss the differential diagnosis of the particular medical problems discussed in the publication;
- explain both the likely and rare complications that may be associated with the particular medical problems discussed in the publication.

3. Which patient should be urgently referred to an electrophysiologist?
   A. a 45-year-old male with paroxysmal SVT who is compliant with his prophylactic medications
   B. a 39-year-old female with paroxysmal SVT who presents with palpitations after a syncopal episode
   C. a 29-year-old female with paroxysmal SVT who is noncompliant with prophylactic medications
   D. a 35-year-old man with recurrent episodes of paroxysmal SVT
4. Which of the following statements is true?
   A. Catheter ablation may be an alternative curative therapy in MAT where pharmacologic therapies have been unsuccessful.
   B. Antidromic conduction is the most common form of AVRT conduction.
   C. SVT is rarely seen in children.
   D. AVNRT is the most common reentrant SVT seen in adults with structurally normal hearts.
5. When treating stable narrow complex SVT, in which order should treatments be attempted?
   A. vagal maneuvers → adenosine → verapamil → procainamide
   B. vagal maneuvers → verapamil → adenosine → flecainide
   C. vagal maneuvers → adenosine → ibutilide → esmolol
   D. vagal maneuvers → esmolol → verapamil → procainamide
6. Which of the following is *not* considered a common environmental trigger for sinus tachycardia?
   A. caffeine
   B. marijuana
   C. ecstasy (MDMA or 3,4-methylenedioxy-methamphetamine)
   D. nicotine
7. What is the most effective therapy for long-term prevention of atrial flutter?
   A. radioablation
   B. propranolol
   C. atropine
   D. verapamil
8. How is WPW syndrome diagnosed on ECG?
   A. ventricular pre-excitation (delta wave) + shortened PR interval + wide QRS complex
   B. ventricular pre-excitation (delta wave) + shortened PR interval + narrow QRS complex
   C. ventricular pre-excitation (delta wave) + lengthened PR interval + wide QRS complex
   D. ventricular pre-excitation (delta wave) + shortened PR interval + narrow QRS complex
9. Which is *not* a common presenting symptom of SVT?
   A. lightheadedness
   B. dyspnea (or shortness of breath)
   C. palpitations
   D. cyanosis
10. Which of the following statements is true?
    A. "Automaticity" refers to abnormal impulse formation.
    B. "Automaticity" refers to abnormal impulse conduction.
    C. "Reentry" refers to multiple abnormal impulses.
    D. "Reentry" refers to abnormal impulse formation.

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EMERGENCY MEDICINE REPORTS

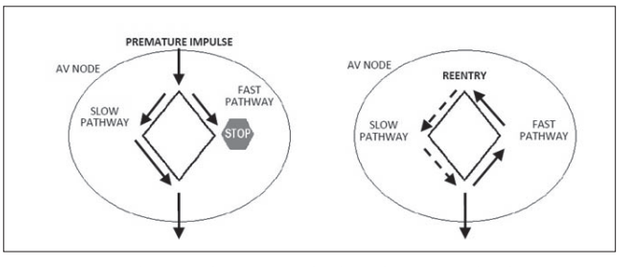
Supraventricular Tachycardia: A Review for the Practicing Emergency Physician

Drug Treatment for SVT in Adults

Drug	Dosage	Comments
Drugs to Block AV Nodal Conduction		
Adenosine	6 mg rapid IV push, if after 2 minutes the dysrhythmia persists, repeat rapid IV push with 12 mg, may repeat once more of dysrhythmia persists	Effective in terminating narrow QRS complex reentrant tachydysrhythmias involving the AV node
Verapamil	2.5-5 mg IV bolus over 2-3 minutes, if after 15 minutes the dysrhythmia persists, may repeat doses 5-10 mg after 15-30 min	Effective in terminating narrow QRS complex reentrant tachydysrhythmias involving the AV node and reducing ventricular rate in atrial fibrillation or flutter
Diltiazem	15-20 mg IV bolus over 2 minutes, followed by IV infusion at 5-20 mg/h	
Esmolol	500 mcg/kg IV bolus over 60 seconds, followed by IV infusion starting at 50 mcg/kg per minute, titrate infusion to desired heart rate	
Metoprolol	5 mg IV bolus, may repeat 5 mg IV every 5 minutes up to total dose of 15 milligrams	
Propranolol	30 mcg/kg IV over 1 minute, may repeat same dose every 2 minutes, up to total dose of 100 mcg/kg	
Drugs to Terminate Tachydysrhythmias		
Procainamide	15-17 mg/kg IV over 30 minutes, followed by IV infusion at 1-4 mg (20-80 mcg/kg) per min	Used in wide-complex tachydysrhythmias and new onset atrial fibrillation Median time to conversion of new-onset atrial fibrillation about an hour Caution in patients with AMI and LV dysfunction Infuse at rate of 20 milligrams/min to reduce adverse effects
Amiodarone	Stable patient: 150 mg IV over 10 minutes, may repeat same dose every 10 minutes up to total dose of 2 grams OR use IV infusion 0.5 mg/min Ventricular fibrillation or pulseless ventricular tachycardia: 300 mg IV bolus, may repeat with additional dose of 150 mg IV bolus	Used in wide-complex tachydysrhythmias and new-onset atrial fibrillation Preferred in setting of AMI or LV dysfunction Contraindicated in pregnancy
Ibutilide	Weight < 60 kg: 10 mcg/kg IV over 10 min Weight > 60 kg: 1 mg IV over 10 min	Used for conversion of new-onset atrial fibrillation or flutter. Median time to conversion 20-30 minutes
Flecainide	2 mg/kg IV over 10 minutes	Used for conversion of new-onset atrial fibrillation or flutter. Median time to conversion up to 4 hours Avoid in patients with ACS or cardiomyopathy
Propafenone	2 mg IV over 10 minutes	Used for conversion of new-onset atrial fibrillation or flutter. Median time to conversion 2 hours Avoid in patients with ACS, cardiomyopathy, or severe COPD
Vernakalant*	3 mg/kg IV infusion over 10 min, if dysrhythmia persists after 15 min, a second infusion of 2 mg/kg IV over 10 min can be given	Used for conversion of new-onset atrial fibrillation or flutter Median time to conversion 8-11 min Avoid in patients with hypotension, ACS within 30 days, severe aortic stenosis, and prolonged QT interval

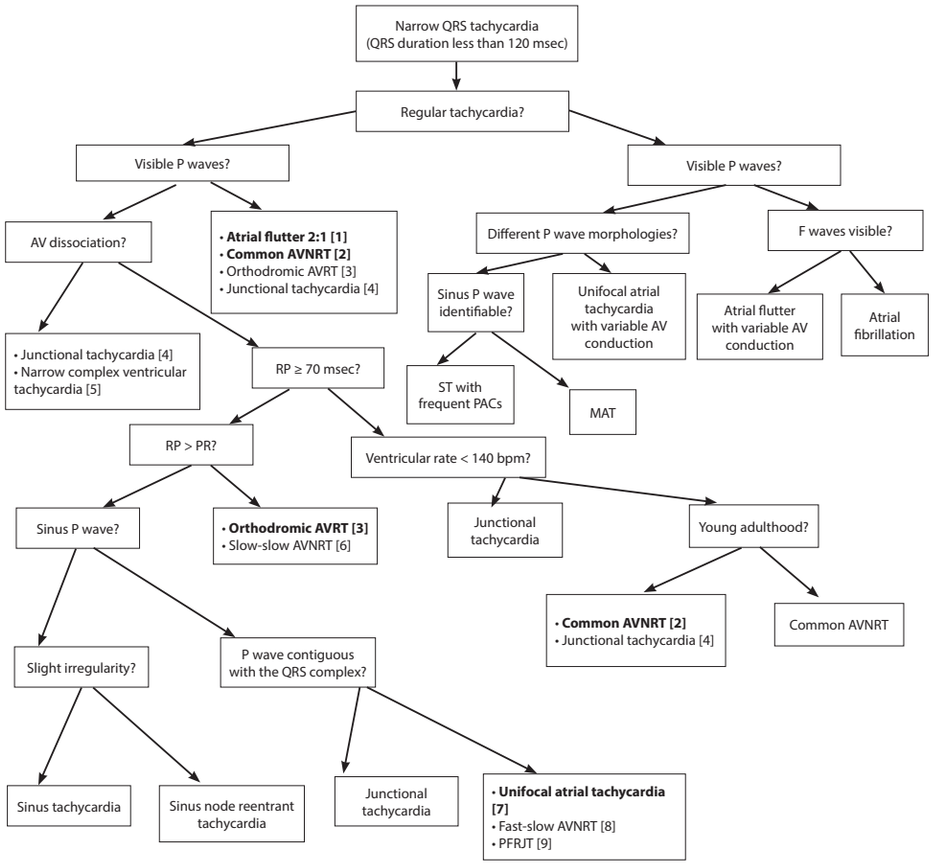
Abbreviations: ACS = acute coronary syndrome; AMI = acute myocardial infarction; COPD = chronic obstructive pulmonary disease; LV = left ventricle; mg = milligrams; mcg = micrograms
* not available in the U.S.

AV Node Reentry Circuit



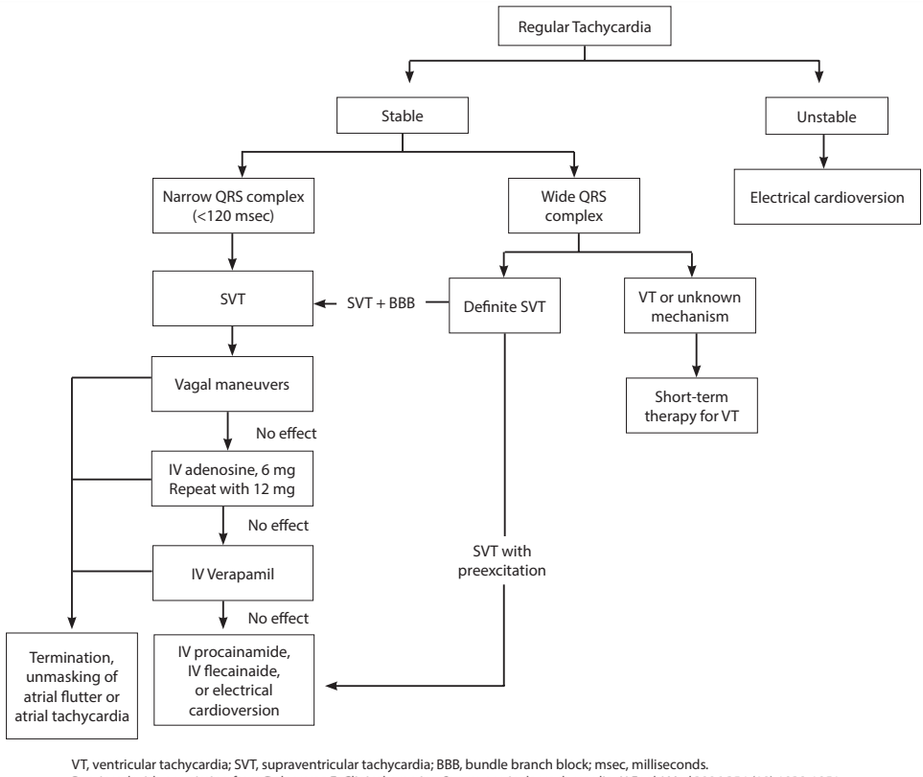
Two separate conduction pathways exist within (or in close proximity) to the atrioventricular (AV) node that have differing conduction velocities (indicated by "slow" and "fast" pathways) and refractory periods. Reentry is initiated when a premature impulse reaches the node and finds the fast pathway refractory, and travels down the slow pathway. Upon reaching lower end of the circuit, the fast pathway has now recovered and the depolarization is able to travel back up that recovered limb. The reentrant loop is maintained by the impulse traveling retrograde up the fast pathway and anterograde back down the slow pathway again, resulting in rapid ventricular response.
Figure adapted from: Ahluwalia M, Quest T. Supraventricular tachycardia (SVT): Strategies for diagnosis, risk stratification, and management in the emergency department setting. *Emerg Med Reports* 2002; 23 (4):41–52.

ECG-Based Algorithm for Diagnosis of Regular, Narrow QRS Tachycardias



Bolded text indicates the most likely diagnosis in each box. AV, atrioventricular; VT, ventricular tachycardia; AVRT, atrioventricular reentry tachycardia; AVNRT, atrioventricular nodal reentrant tachycardia; MAT, multifocal atrial tachycardia. Adapted from Butta C, Tuttolomondo A, Di Raimondo D, et al. Supraventricular tachycardias: Proposal of a diagnostic algorithm for the narrow complex tachycardias. *J Cardiol* 2013;61(4):247-255.

Treatment-Based Algorithm for Management of SVT in the ED



VT, ventricular tachycardia; SVT, supraventricular tachycardia; BBB, bundle branch block; msec, milliseconds.

Supplement to *Emergency Medicine Reports*, June 14, 2015: "Supraventricular Tachycardia: A Review for the Practicing Emergency Physician." Authors: Gary Hals, MD, PhD, Attending Physician, Richland Memorial Hospital, Columbia, SC. Chloe McCoy, MD, PhD, Richland Memorial Hospital, Columbia, SC.