Chapter 4
Supraventricular Tachycardia

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Abbreviations

AT	Atrial Tachycardia
AF	Atrial Fibrillation
AVN	Atrioventricular Node
AVNRT	Atrioventricular Nodal Reentrant Tachycardia
AVRT	Atrioventricular Reentrant Tachycardia
ECG	Electrocardiogram
JT	Junctional Tachycardia
MAT	Multifocal Atrial Tachycardia
PAC	Premature Atrial Contraction
PVC	Premature Ventricular Contraction
ST	Sinus Tachycardia
SVT	Supraventricular Tachycardia
TIA	Transient Ischemic Attack
VT	Ventricular Tachycardia
VF	Ventricular Fibrillation

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Supraventricular Tachycardia

Definition, Incidence and Prevalence

- Supraventricular tachycardia (SVT) is typically a regular, narrow complex tachycardia although underlying conduction disease (bundle branch blocks), aberrant conduction and, rarely, antegrade conduction (atrium to ventricle) through an accessory pathway will result in wide complex tachycardia.
- 90% of SVTs include a reentrant mechanism between the atria and the ventricle.
- Atrioventricular nodal reentrant tachycardia (AVNRT) comprises 60% of reentrant tachycardias, 30% are atrioventricular reentrant tachycardias (AVRT) and the remaining 10% are atrial tachycardias (AT).
- ATs increase in frequency in older patients (>50 years old) and AVRT becomes less common.
- SVT occurs in about 35 cases in 100,000 patient years with a prevalence of 2.25 per 1000 patients [2].
- For the purposes of this chapter we will exclude atrial fibrillation (AF) and atrial flutter as they are addressed in a separate chapter.

Interventions for SVT

Vagal Maneuvers

- During any intervention for SVT the patient should be connected to a continuous 12-lead ECG to aid in diagnosis of the arrhythmia.
- Vagal maneuvers, including carotid sinus massage (CSM) and the Valsalva maneuver (VM), increase the parasympathetic tone which slows conduction through the atrium and the atrioventricular node (AVN).

- Vagal maneuvers should be first line treatment for patients who are in SVT with stable hemodynamics.
- The VM involves inhaling followed by exhaling against a closed glottis for at least 10–15 seconds. The patient is generally in a supine or semi-recumbent position. The modified VM, which involves supine repositioning and passive leg raise at the end of strain, is significantly more effective at converting patients out of SVT [1].
- CSM, which is less efficacious than VM, may also be attempted. The patient should be in the supine position with the neck extended. The carotid sinus is located inferior to the angle of the mandible at the level of the thyroid cartilage. Constant pressure should be applied for 5–10 seconds. Contraindications to CSM include stroke or transient ischemic attack (TIA) within the last 3 months and presence of a carotid bruit [4].

Clinical Pearl
During any intervention for SVT the patient should be connected to a continuous 12-lead ECG to aid in diagnosis of the arrhythmia.

Adenosine

- Adenosine decreases the heart rate and slows conduction through the AVN, albeit briefly as the half-life is <10 seconds.
- IV Adenosine should be administered rapidly, over 1 to 2 seconds, followed by a rapid flush of normal saline. A 3-way stopcock may expedite the delivery of the flush. The patient should be in the supine position. The recommended initial dose through a peripheral IV is 6 mg followed by 12 mg.
- Adenosine should always cause transient atrioventricular (AV) block. If the bolus of adenosine fails to do so, then it

was not administered fast enough, or the patient requires a higher dose.
- Adenosine must be used with the utmost caution in cardiac transplant recipients as the denervated heart is incredibly sensitive to this medication and this may lead to profound bradycardia or asystole.

Electrical Cardioversion

- Patients with SVT who are hemodynamically unstable should undergo immediate synchronized electrical cardioversion.

Clinical Pearl
Patients with SVT who are hemodynamically unstable should undergo immediate synchronized electrical cardioversion.

Approach to the Patient with SVT

12-Lead Electrocardiogram (ECG)

- Obtaining a 12-lead ECG is paramount for the diagnosis of SVT as P waves may not be evident on telemetry.
- The RP relationship refers to the interval between the R wave and the ensuing P wave and can aid in the diagnosis of SVT. In a short RP tachycardia, the P wave will closely follow the R wave (RP < PR), whereas the P wave will be closer to the ensuing R wave in long RP tachycardia (RP > PR).

Response to Adenosine

- SVTs that are adenosine responsive include AVNRT, AVRT, junctional tachycardia (JT), and some focal ATs.
- Any AVN dependent SVT will terminate with an adequate bolus of IV adenosine.

- Adenosine may be diagnostic as the delay in conduction through the AVN may reveal discrete atrial activity.

AV Nodal Reentrant Tachycardia

- AVNRT is more common in women than men and the ventricular rate is generally between 180 beats per minute (BPM) and 200 BPM, although this may range from 110 BPM to >250 BPM.
- AVNRT requires dual AV nodal physiology, in which patients have a fast and slow pathway within the AVN. The fast pathway allows for rapid conduction but has a long recovery period. The slow pathway results in slower conduction but a shorter recovery time.

Typical AVNRT

- In typical AVNRT, or "slow-fast" conduction, a premature atrial contraction (PAC) will be conducted antegrade through the slow pathway as the fast pathway is refractory from the previous sinus beat. During this period of slow conduction, the fast pathway will repolarize and allow for retrograde conduction (ventricle to atrium) of the impulse. Thus, the retrograde P will be very close in proximity to the QRS complex (short RP interval). Conversely, a premature ventricular contraction (PVC) may conduct up the fast pathway and start the tachycardia.
- P waves may appear as a pseudo-r' pattern in the anterior precordial leads. Retrograde P waves may also appear as pseudo-S waves in the inferior leads (Fig. 4.1). An inferiorly directed retrograde P wave axis (upright in leads II, III and aVF) excludes AVNRT.
- Vagal maneuvers should be first line for the treatment of acute AVNRT, followed by adenosine. Adenosine should successfully terminate AVNRT with appropriate dosing

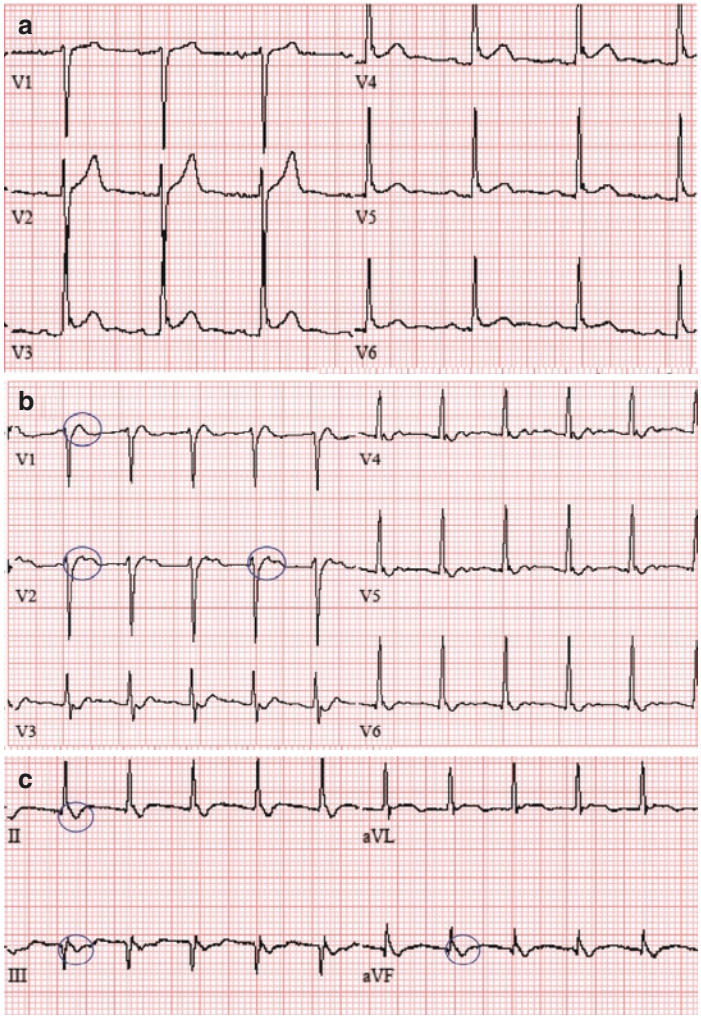

Figure 4.1 (**a**) This image demonstrates the precordial leads from the baseline ECG of a 32 year-old male who was later found to have typical AVNRT. (**b**) During tachycardia in the same patient a pseudo-r' pattern (retrograde P wave) develops conferring a short RP interval. (**c**) Note the superior axis of the retrograde P waves in the inferior leads during tachycardia, causing pseudo-S waves

and administration. Synchronized cardioversion is highly recommended for both stable and unstable patients who have failed to convert with vagal maneuvers and adenosine. However, IV nodal blockade is a reasonable option in hemodynamically stable patients.

Atypical AVNRT

- Atypical AVNRT, or "fast-slow" conduction, involves antegrade conduction down the fast pathway with retrograde conduction up the slow pathway. The retrograde P wave will be delayed due to the longer conduction time through the slow pathway resulting in a long RP tachycardia (Fig. 4.2).
- The treatment algorithm for atypical AVNRT is identical to that of typical AVNRT.

AV Reentrant Tachycardia

- AVRT requires an accessory pathway outside of the AVN to complete the reentrant circuit. Accessory pathways are termed manifest if they conduct antegrade, which will demonstrate ventricular pre-excitation in sinus rhythm with a delta wave on the ECG (Fig. 4.3). Manifest pathways may conduct impulses antegrade, retrograde or both. Those pathways that only conduct retrograde, termed concealed pathways, will not produce a delta wave.

Pre-Excited Atrial Fibrillation

- AF is particularly dangerous in patients with manifest pathways as this can lead to rapid conduction to the ventricle and degenerate to ventricular fibrillation (VF). For those with pre-excited AF (Fig. 4.4), procainamide or ibutilide are first line treatment for stable patients. *IV digoxin,*

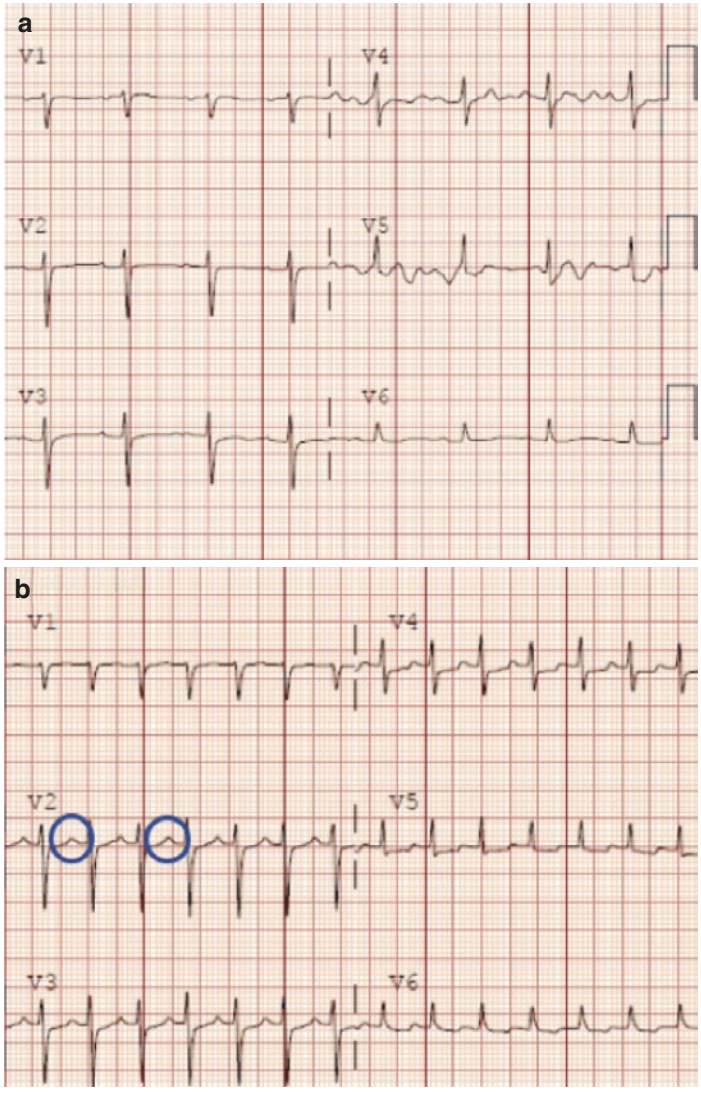

FIGURE 4.2 (**a**) The ECG on the left represents the precordial leads from a patient with atypical or "fast-slow" AVNRT which was captured in the precordial leads on the right. (**b**) The RP interval, which is evident with the retrograde p waves in the anterior leads, is longer than the PR interval

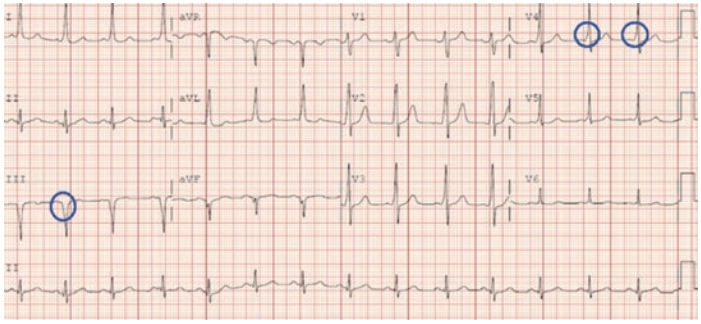

FIGURE 4.3 A 34 year-old male presented with syncope and was noted to have ventricular pre-excitation on the above 12-lead ECG. Note the short PR interval in sinus rhythm and the delta waves in the precordial and inferior limb leads

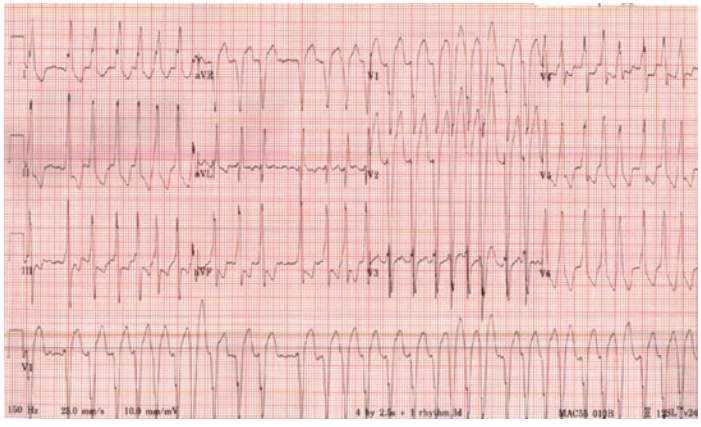

FIGURE 4.4 12-lead ECG from a 32 year-old male with pre-excited atrial fibrillation

IV amiodarone, beta blockers, diltiazem and verapamil are harmful in the acute treatment of pre-excited AF [3].

- For patients who have pre-excited AF and are hemodynamically unstable immediate synchronized cardioversion should be performed.

Orthodromic AVRT

- Orthodromic AVRT involves antegrade conduction through the AVN with retrograde conduction through the accessory pathway. The resultant ECG demonstrates a narrow complex tachycardia (the delta wave will resolve as the accessory pathway will be refractory following retrograde conduction), barring aberrant conduction, with a short RP interval. The RP interval will typically be longer than AVNRT as the accessory pathway is extranodal.
- Vagal maneuvers and adenosine, which should terminate the tachycardia, are first line therapies for AVRT. If the tachycardia persists cardioversion can be considered. If the patient is stable and does not have evidence of pre-excitation on the baseline ECG then IV beta blockers, diltiazem or verapamil may terminate the tachycardia. However, these drugs, including adenosine, should be avoided if there is evidence of pre-excitation given their potential to precipitate AF with rapid conduction to the ventricle. Therefore, cardioversion should be available.

Antidromic AVRT

- Antidromic, AVRT only occurs in 5-10% of patients with accessory pathways. Antidromic AVRT involves antegrade conduction down the accessory pathway resulting in a wide complex tachycardia as the antegrade depolarization does not propagate through the His-Purkinje system. It may be difficult to discern from ventricular tachycardia (VT).
- When suspected the treatment for atypical AVRT is identical to that of typical AVRT.

Atrial Tachycardia

Focal Atrial Tachycardia

- Atrial tachycardias are regular atrial rhythms with a single focus or small (<2 cm) microreentrant circuits that produce atrial rates generally between 100 and 250 BPM. AT can arise from specific sites in the atria and commonly occur in structurally normal hearts.
- Like other automatic mechanisms, ATs may display a gradual onset and offset or "warm up" and "warm down" over 3–4 beats if the patient is on telemetry [5]. On the other hand, ATs may start abruptly as in Fig. 4.5.
- ATs may conduct 1:1 to the ventricle, although this relationship varies, and generally appear as long RP tachycardias on the 12 lead ECG. However, the RP interval will be short in patients with first degree AV block. Further, due to the decremental property of the AVN (more frequent stimulation causes slower conduction through the node), the delay through the node may not be apparent in sinus rhythm.
- The P wave axis should at least demonstrate subtle differences when compared to sinus rhythm (Fig. 4.6). A consistent PP interval, the duration between consecutive P

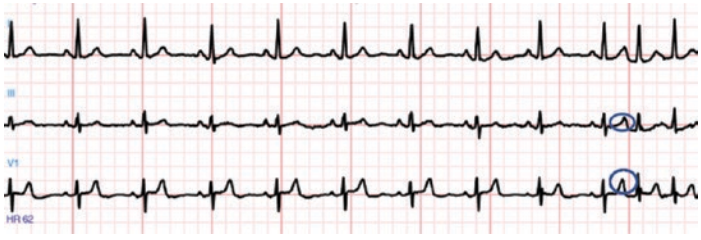

FIGURE 4.5 This 3-lead telemetry strip captured the onset of atrial tachycardia from a patient admitted for palpitations. Note the change in the T wave with the superimposed P wave, conferring a long RP interval, as well as the abrupt increase in the rate

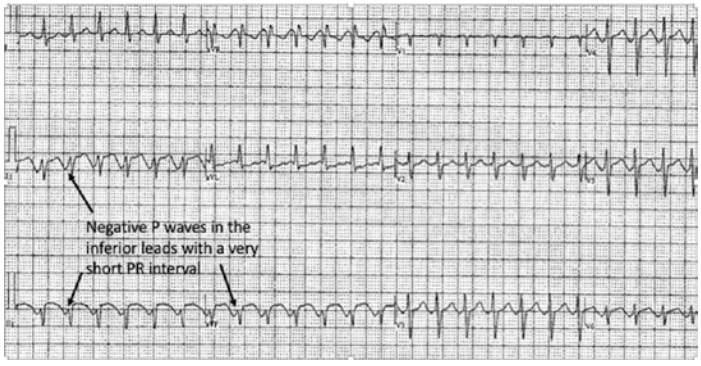

FIGURE 4.6 This 68 year-old male experienced recurrent tachycardia which was captured on this 12-lead ECG. The P wave axis is superior, opposite of that in sinus rhythm, in this long RP tachycardia. The patient was taken to the electrophysiology lab where he was found to have a focal AT emanating from the ostium of the coronary sinus

waves, despite variable PR and RP intervals on the ECG during tachycardia suggests AT.

- Transient AV block following the administration of adenosine may demonstrate persistent atrial activity without ventricular conduction, allowing differentiation of AT from AVNRT and AVRT. On the other hand, depending upon the mechanism of the AT, adenosine may terminate the tachycardia, although AV block usually occurs before termination of the arrhythmia.
- If AT is diagnosed, IV beta blockers, diltiazem or verapamil are first line therapies for the acute treatment of hemodynamically stable patients.

Multifocal Atrial Tachycardia

- Multifocal atrial tachycardia (MAT) involves multiple atrial foci and manifests as 3 distinct P wave morphologies with a ventricular rate of >100 BPM on the ECG. This

arrhythmia usually occurs in older, critically ill patients with severe pulmonary or cardiac disease.
- IV metoprolol may be used in the acute setting to slow the ventricular rate, although it should be avoided in patients with severe pulmonary disease. IV verapamil is relatively successful at converting patients with MAT to sinus rhythm.

Junctional Tachycardia

- JT arises from the atrioventricular junction, specifically the perinodal tissue and His bundle. P waves may appear before, after or even within the QRS complex with a ventricular rate > 100 BPM. Thus, JT may appear on the ECG as a short or long RP tachycardia.
- JT is more common in infants and children and rarely occurs in adults. This arrhythmia may be seen in adults after valve surgery and tends to be transient [5].
- IV beta blockers, diltiazem, verapamil or procainamide are reasonable options for the treatment of acute JT.

Clinical Pearls
- During any intervention for SVT the patient should be connected to a continuous 12-lead ECG to aid in diagnosis of the arrhythmia.
- Patients with SVT who are hemodynamically unstable should undergo immediate synchronized electrical cardioversion.
- Adenosine should always cause transient atrioventricular (AV) block. If the bolus of adenosine fails to do so, then it was not administered fast enough or the patient requires a higher dose.
- IV digoxin, IV amiodarone, beta blockers, diltiazem and verapamil are potentially harmful in the acute treatment of pre-excited AF.
- Adenosine should be avoided in patients with pre-excited AF and heart transplant recipients.

Key Learning Points

1. Supraventricular tachycardia (SVT) is typically a regular, narrow complex tachycardia although underlying conduction disease, aberrant conduction and, rarely, antegrade conduction (atrium to ventricle) through an accessory pathway will result in wide complex tachycardia.
2. Any SVT that is dependent on the AV node, such as AVNRT and AVRT, should terminate with an adequate bolus of adenosine.
3. Adenosine can be a diagnostic and therapeutic intervention for SVT as it may terminate the arrhythmia or block AV conduction, allowing for visualization of discrete atrial activity. However, this medication should be used with extreme caution or avoided in patients who have received an orthotopic heart transplant those patients with evidence of ventricular pre-excitation.

References

1. Appelboam A, Reuben A, Mann C, Gagg J, Ewings P, Barton A, et al. Postural modification to the standard Valsalva manoeuvre for emergency treatment of supraventricular tachycardias (REVERT): a randomised controlled trial. Lancet. 2015;386(10005):1747–53. https://doi.org/10.1016/s0140-6736(15)61485-4.
2. Garcia MA, Macle L, Khairy P. Electrophysiology for clinicians. Minneapolis: Cardiotext Publishing; 2012.
3. Page RL, Joglar JA, Caldwell MA, Calkins H, Conti JB, Deal BJ, et al. 2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia. J Am Coll Cardiol. 2016;67(13):e27–e115. https://doi.org/10.1016/j.jacc.2015.08.856.
4. Shen W, Sheldon RS, Benditt DG, Cohen MI, Forman DE, Goldberger ZD, et al. 2017 ACC/AHA/HRS guideline for the evaluation and management of patients with syncope: a

report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines and the Heart Rhythm Society. Circulation. 2017;136(5):e60–e122. https://doi.org/10.1161/cir.0000000000000499.

5. Zipes DP, Jalife J, Stevenson WG. Cardiac electrophysiology: from cell to bedside. Philadelphia, PA: Elsevier; 2018.