

SVT

**Causes & Triggers**  
Usually none found but consider:

- Acute illness: infection, hyperthyroidism, PE, hypovolaemia
- Cardiac: abnormalities of conduction system, ischaemic heart disease, pericarditis / myocarditis, alcohol, mitral valve disease, post cardiac surgery
- Drugs: B2 agonists, antiarrhythmics, digoxin toxicity, tricyclics, lithium, cocaine, MDMA, amphetamines – see [toxbase](#)
- Metabolic: high or low K or Ca, hypoxia, high CO<sub>2</sub>, acidosis, high or low temp

**Anti-arrhythmics – cautions & contra-indications:**  
B-blockers and Ca channel blockers should not be used together  
**Adenosine:** acute bronchospasm, dipyridamole, carbamazepine  
**Amiodarone:** Sino-atrial block and conduction disturbances, severe hypotension, thyroid disease, CCF, pregnancy & breast-feeding.  
**B-blockers:** acute bronchospasm, uncontrolled heart failure, sick sinus syndrome, AV block,  
**Ca channel blockers:** heart failure, hypotension, sick sinus syndrome, heart block, AF with WPW, VT, pregnancy & breast-feeding

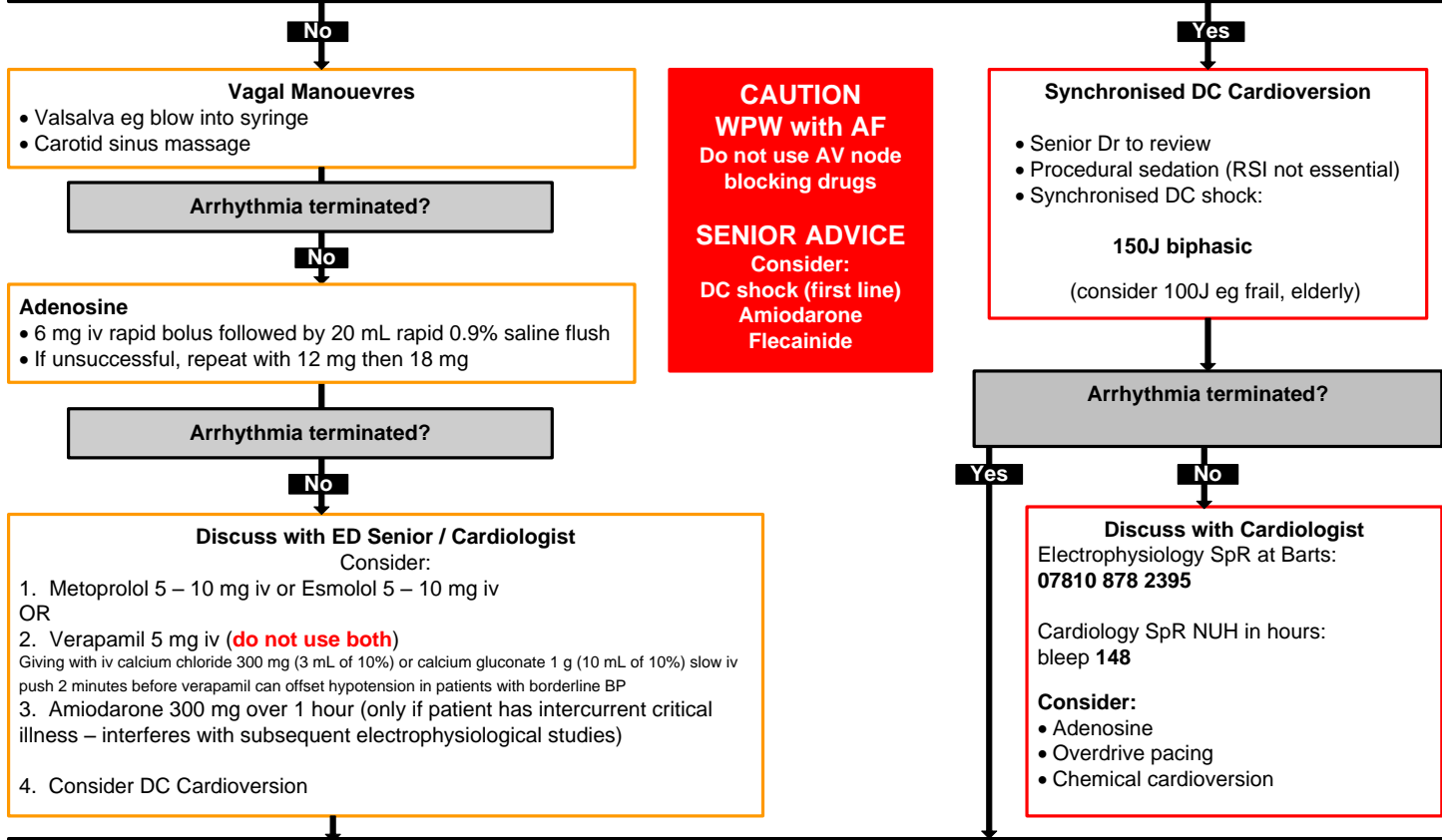
**Classification of SVT**  
SVT usually produces a regular narrow complex tachycardia. P waves may not be visible, but if seen may give a clue as to the origin of the SVT:  
AVNRT (AV nodal reentry tachycardia): Accessory pathway within AV node. May see pseudo R in V1, or pseudo S in II, III, aVF. RP shorter than PR  
AVRT (AV reentry tachycardia): Accessory pathway outside AV node (WPW). May see delta wave on resting ECG. Retrograde P waves may be seen in the ST segment. RP shorter than PR.  
Junctional tachycardia: Retrograde P waves before, during or after QRS. P usually inverted in II, III, aVF and upright in aVR, V1  
Atrial tachycardia: Trigger within the atria but outside SA node, AV node or accessory pathway. P waves abnormal morphology. Isoelectric baseline seen between P waves (cf atrial flutter). P waves often inverted in II, III, aVF. RP longer than PR (cf AVRT, AVNRT)

**Summary of Treatment Response**  
Cardioversion achieved by vagal manoeuvres, adenosine or DC shock. Rate control achieved with B blockers or verapamil  
AVNRT and AVRT may cardiovert with vagal manoeuvres, adenosine, verapamil or DC shock. B blockers will slow rate  
Junctional and atrial tachycardia will not cardiovert with vagal or adenosine, but usually respond to DC shock. Atrial tachycardias respond better to b blockers than Ca blockers

**If broad complex – exit pathway, follow ALS protocol**

**Investigations:** FBC & VBG (all), HCG, TFT (if first presentation) & CXR (only if clinically indicated); additional tests if condition requires

**Are there signs of shock or acute pulmonary oedema?**  
**Caution:** compromise due to SVT is rare if structurally normal heart. Consider underlying cause and treat as appropriate eg sepsis



**Discharge Criteria:**  
No signs ACS or LVF, full recovery post sedation  
Follow up: Refer to Rapid Access Heart Rhythm Clinic at Barts Hospital:  
fax referral form and a copy of the ECGs and discharge summary to 0203 465 5769  
Barts Hospital is located in West Smithfield, EC1A 7BE. Nearest tube is St Pauls or Barbican. Enquiries 0203 465 6767

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Reference Documents

Use of calcium with verapamil in the management of supraventricular tachyarrhythmias; *Weiss et al; International Journal of Cardiology; October 1983*

Treatment of atrial arrhythmias: effectiveness of verapamil when preceded by calcium infusion; *Jacob et al; Archives Internal Medicine; 1986*

Administration of intravenous calcium before verapamil to prevent hypotension in elderly patients with paroxysmal supraventricular tachycardia; *Miyagawi et al; Journal of Cardiovascular Pharmacology; August 1993*

ALS 2010 Resuscitation Guidelines