Supraventricular tachyarrhythmias and their management in the perioperative period

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1A01, 1A02, 1B04,
2A03 and 2A06

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Key points

- Cardiac arrhythmias are a significant cause of morbidity and mortality in the perioperative period.
- Potential triggers for perioperative supraventricular tachyarrhythmias should be identified and treated.
- Haemodynamically unstable supraventricular tachyarrhythmias should be treated with immediate synchronized electrical cardioversion.
- Resuscitation equipment should be immediately available when attempting electrical cardioversion of supraventricular tachyarrhythmias.
- Classification of supraventricular tachycardia (SVT) can guide appropriate pharmacological treatment in the perioperative period.
- For haemodynamically stable SVT where there is doubt regarding the diagnosis, seek advice.

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This review will provide an overview of supraventricular tachycardias (SVTs), their classification, diagnostic features, and management in the perioperative period of adult non-cardiac surgery.

The term 'SVT' refers to paroxysmal tachyarrhythmias that require atrial or atrioventricular (AV) nodal tissue, or both, for their initiation and maintenance.[1]

The incidence of persistent SVT is 2% before operation and 6% in the postoperative period.[2] In non-cardiac surgery, perioperative arrhythmias are more likely to be supraventricular than ventricular in origin.[3] Atrial arrhythmias occur most frequently 2–3 days post-surgery similar to perioperative acute coronary syndromes and are likely related to sympathetic stimulation associated with an inflammatory response.[4] Other precipitants of SVT often associated with high sympathetic tone are summarized in Table 1. Such arrhythmias are important for the anaesthetist; in particular, atrial fibrillation (AF) is associated with haemodynamic derangement, postoperative stroke, perioperative myocardial infarction, ventricular arrhythmia, heart failure, and longer hospital stay.[5]

Classification and electrocardiogram features[6,7]

The diagnostic features of SVTs are summarized in Table 2 and diagrammatically represented in Figure 1.

Regular SVTs

Sinus tachycardia

Sinus tachycardia (ST) is an appropriate autonomic response to a physiological stress. The normal maximum ventricular rate in a physiological ST can be estimated by subtracting the patient's age from 220 bpm. Inappropriate ST is a rare exception to this rule and should be considered a diagnosis of exclusion.

Atrial flutter

Atrial flutter (AFL) is the second most common pathological SVT and usually originates from a re-entrant circuit at the tricuspid valve. This forms an organized regular rhythm characterized by atrial depolarization at ~300 bpm and with 2:1 conduction in the AV node results in ventricular depolarization at ~150 bpm. Differentiating this arrhythmia from other SVTs can be difficult as flutter waves are often obscured by T waves at this rate. Furthermore, variable conduction from a ratio of 2:1 to 3:1 can give the appearance of an irregular SVT.

Atrioventricular nodal re-entrant tachycardia

Both AV nodal re-entrant tachycardia (AVNRT) and AV re-entrant tachycardia (AVRT) can present at any age but more commonly in young adults. Severe haemodynamic decompensation is rare in the absence of concurrent structural cardiac disease.

AVNRT is caused by a re-entrant circuit involving posterior and anterior inputs to the AV node, one of which is rapidly conducting and the other slower conducting (Fig. 1). An atrial ectopic during the refractory period of the fast pathway propagates down the slow pathway to the ventricles and then conducts retrogradely up the repolarized fast pathway to the atria. A re-entrant circuit is formed with almost simultaneous activation of atria and ventricles, thus obscuring the P wave within the QRS complex (Table 2).

Atrioventricular re-entrant tachycardia

AVRT uses an accessory pathway bypassing the AV node, which may conduct in an anterograde direction (from the atria to ventricle via the accessory pathway) or in a retrograde direction (from the ventricle to atria via the accessory pathway). In contrast to AVNRT, an AVRT re-entrant circuit depends upon the presence of congenitally abnormal myocardial fibres (accessory pathways) connecting the atria and ventricles that bypass the AV node (Fig. 1). Three

doi:10.1093/bjaceaccp/mku018 Advance Access publication 7 June, 2014

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Table 1 Risk factors for SVT in the perioperative period

Acute anaesthetic factors	Acute surgical factors
Anaesthesia-induced cardiac depression	Pain
Inotropes	Trauma
Hypervolaemia → acute atrial stretch	Anaemia
Auto-PEEP	Local and systemic inflammation (elevated IL-6 and CRP)
Shock	Mediastinal manipulation
Pulmonary artery catheter/misplaced central line	
Local anaesthetic toxicity	
Acute medical factors	**Chronic medical factors**
Hypoxia	Ageing (fibrosis and inflammation)
Hypovolaemia	Atrial distension (heart failure, valvular disease)
Electrolyte disturbances	Ischaemic heart disease
Acute myocardial infarction	Congestive cardiac failure
Metabolic/respiratory acidosis	Chronic hypoxia, e.g. COPD, OSA
Pneumonia	Hypertension
Sepsis	Cardiomyopathy
Pulmonary embolism	Persistent tachycardia-induced atrial remodelling
Hypoglycaemia/hyperglycaemia	Accessory pathways
Hypothermia/hyperthermia	Congenital heart disease
Myocarditis/pericarditis	Scarring post-cardiac surgery
Pneumothorax	Pulmonary hypertension
Excessive alcohol and caffeine intake	Hyper/hypothyroidism
Recreational drugs	Malignancy

arrhythmias are seen with these accessory pathways (Table 2): (i) a regular narrow complex tachycardia (orthodromic; anterograde conduction via the AV node and retrograde conduction via the accessory pathway); (ii) a regular broad complex tachycardia (antidromic; anterograde conduction via the accessory pathway and retrograde conduction via the AV node); (iii) an irregular broad complex tachycardia (AF with rapid anterograde conduction via the accessory pathway).

Wolff–Parkinson–White syndrome

The term Wolff–Parkinson–White (WPW) syndrome is a specific form of AVRT and is applied to the patient with both preexcitation manifest on an ECG and symptomatic arrhythmias (orthodromic or antidromic AVRT) involving the accessory pathway. The term *WPW pattern* is applied to the patient with preexcitation manifest on an ECG in the absence of symptomatic arrhythmias. The classic ECG pattern of preexcitation in sinus rhythm has two major features:

- A short PR interval (<120 ms) due to rapid AV conduction through the accessory pathway and bypass of the AV node.
- The QRS complex consists of fusion between the early depolarization via the accessory pathway and later depolarization via the AV node producing a *delta wave* and a broad QRS complex.

The AV node acts as a rate-limiting filter between the atria and ventricles. The accessory pathway bypasses this filter and can conduct rapid supraventricular arrhythmias directly to the ventricles. In the context of WPW syndrome with AF, conduction of rapid AFs to the ventricle via the accessory pathway can precipitate ventricular fibrillation and sudden death.

Focal atrial tachycardia

Focal atrial tachycardia (FAT) is a regular atrial rhythm at a constant rate of >100 beats min^{-1} originating within the left or right atrium outside of the sinus node. Usually, there is a subtle difference in P wave morphology from sinus rhythm, so comparison with a sinus ECG is valuable in diagnosis. Abrupt onset over 3–4 beats favours FAT, as ST requires 30 s to minutes for onset and offset.

Irregular SVTs

Atrial fibrillation

AF is the most common pathological SVT and is caused by multiple electrical wavelets appearing in the atria simultaneously with an irregular ventricular response. AF is classified acute when occurring suddenly in a patient previously in sinus rhythm. Conversely, in chronic AF, the onset of tachycardia is gradual and is precipitated by physiological stressors similar to an ST.

Characteristic ECG findings for AF are the absence of P waves, with disorganized electrical activity in their place, and irregular R–R intervals due to irregular conduction of impulses to the ventricles.

Multifocal atrial tachycardia

Multifocal atrial tachycardia (MAT) is less common than FAT and occurs most often in acutely unwell patients. The typical patient is elderly with acute pulmonary infection and may have associated ischaemic heart disease, electrolyte derangement, or theophylline toxicity. This arrhythmia usually progresses into other atrial tachyarrhythmia typically AF.

MAT is characterized by irregular atrial activity with at least three morphologically distinct P waves and an isoelectric baseline between the P waves.

Preoperative diagnosis and investigations[7,8]

History

A classical history for a regular SVT describes an abrupt onset of rapid regular palpitations. Irregular palpitations suggest AF and gradual onset regular palpitations infer an ST. Associated symptoms offer a guide to severity and commonly include pounding in the neck or head, anxiety, shortness of breath, or light-headedness. Less commonly SVT is associated with chest pain or syncope.

Some patients can identify triggers such as caffeine or alcohol intake and develop techniques to terminate palpitations such as vagal manoeuvres, which support the diagnosis of AVNRT or AVRT.

Patients for elective surgery with these symptoms should be postponed pending appropriate investigations and treatment from a cardiologist.

Electrocardiogram

Patients with paroxysmal SVT typically have a normal resting ECG, but evidence of preexcitation and the appearance of WPW pattern should be sought.

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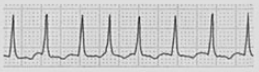
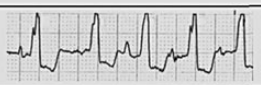
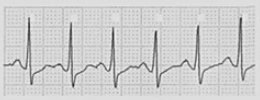
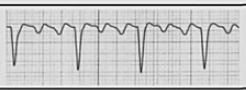
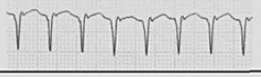
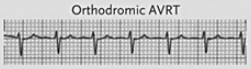
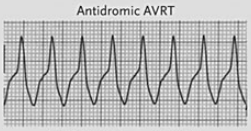
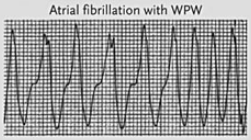
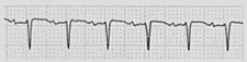
Table 2 Differential diagnosis of the SVTs. AV, atrioventricular; AVRT, atrioventricular re-entrant tachycardia; WPW, Wolff–Parkinson–White syndrome. From Link,[6] 1440. Copyright © (2013) Massachusetts Medical Society. Reprinted with permission from Massachusetts Medical Society

SVT	Underlying Causes	Regularity	Rate (beats/min)	Onset	Atrial Activity and P:QRS Relationship	Response to Adenosine	Electrocardiogram
Atrial fibrillation	Cardiac disease, pulmonary disease, pulmonary embolism, hyperthyroidism, postoperative	Irregular	100–220	Sudden or gradual (if in chronic atrial fibrillation)	Fibrillatory waves, no relationship to QRS	Transient slowing of ventricular rate	
Multifocal atrial tachycardia	Pulmonary disease, theophylline therapy	Irregular	100–150	Gradual	Changing P morphologic features before QRS	None	
Sinus tachycardia	Sepsis, hypovolaemia, anaemia, pulmonary embolism, pain, fear, fright, exertion, myocardial ischaemia, hyperthyroidism, heart failure	Regular	220 minus the patient's age	Gradual	P before QRS	Transient slowing	
Atrial flutter	Cardiac disease	Regular (occasionally irregular if variable AV conduction)	150	Sudden	Flutter waves, usually 2:1	Transient slowing of ventricular rate	
AV nodal reentrant tachycardia	None	Regular	150–250	Sudden	No apparent atrial activity or R' at termination of QRS	Termination of tachycardia	
AV nodal tachycardia	Rarely, Ebstein's anomaly	Regular	150–250	Sudden	In narrow complex, P after QRS; in wide complex, P rarely observed; in irregular rhythm (atrial fibrillation), no apparent P wave	Termination of tachycardia	Orthodromic AVRT  Antidromic AVRT  Atrial fibrillation with WPW 
Focal atrial tachycardia	Cardiac disease, pulmonary disease	Regular	150–250	Sudden	P before QRS	Termination of 60–80%	

Echocardiography

Patients with structural heart disease are at greatest risk for developing SVT under anaesthesia. A preoperative echocardiogram may elicit potential precipitants of SVT such as valvular heart disease, atrial dimensions, and left ventricular size and function. These abnormalities may also help guide therapy upon onset of acute SVT.

Most patients with a history of AVNRT or AVRT have a structurally normal heart. However, certain types of structural heart disease are associated with particular SVTs. Re-entrant atrial tachycardias around surgical scars are a major source of morbidity in the growing population of survivors of congenital heart disease.

Other investigations

Abnormalities that may precipitate SVT under anaesthesia may be detected with the following tests:

- full blood count,
- urea and electrolytes,
- liver function tests,
- thyroid function tests,
- drug screening,
- arterial blood gas,
- blood glucose,
- chest radiograph.

Management of perioperative SVT[3,8–10]

Initial assessment

Whenever possible, cardiac arrhythmias should be controlled before operation as surgery and anaesthesia can cause marked deterioration. Management of medical comorbidities and correction of underlying electrolyte imbalance are part of the strategy for treatment and prevention of perioperative SVT (see Table 1 for perioperative risk factors for SVT).

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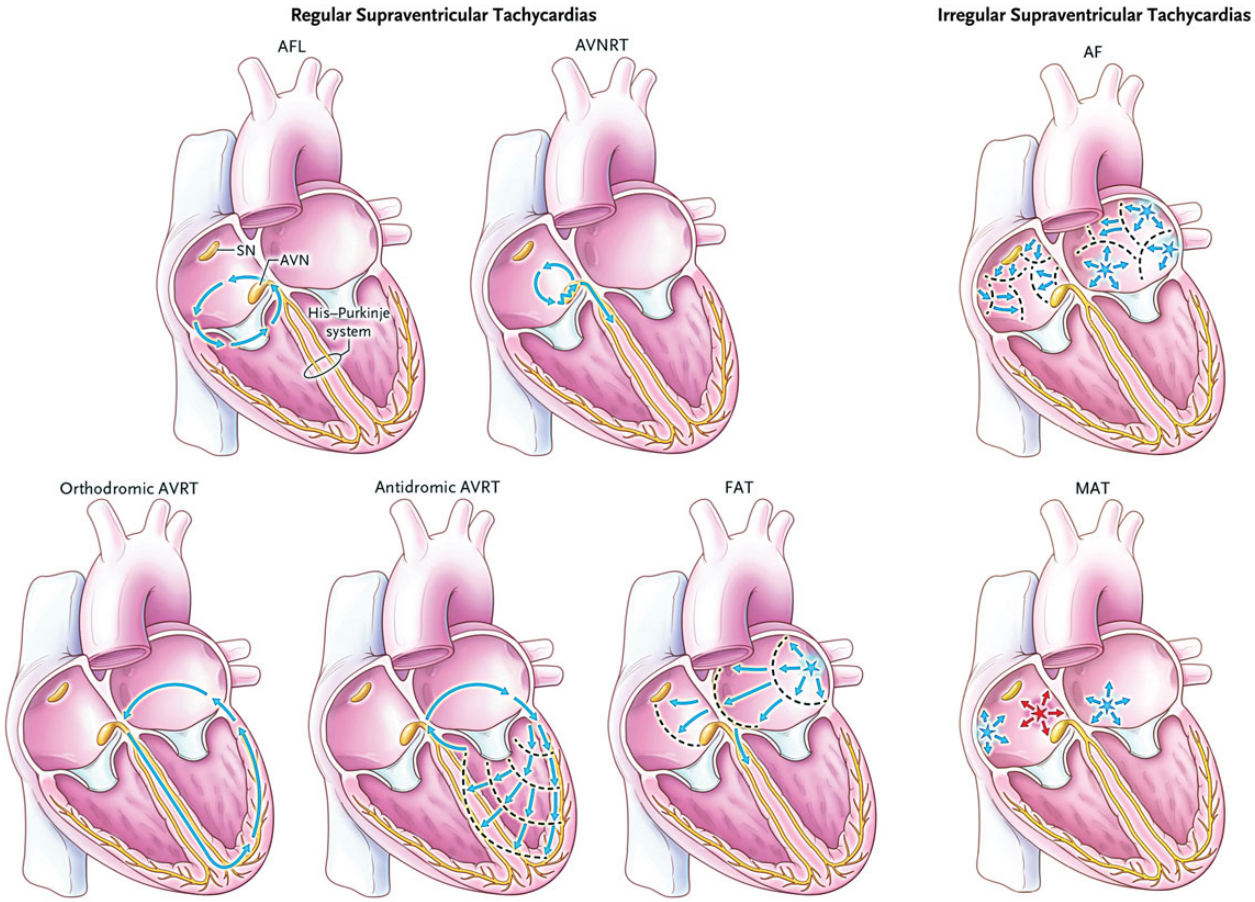

Fig 1 Mechanisms of SVTs. AFL, atrial flutter; AVNRT, atrioventricular nodal re-entrant tachycardia; AF, atrial fibrillation; AVRT, atrioventricular re-entrant tachycardia; FAT, focal atrial tachycardia; MAT, multifocal atrial tachycardia. From Link,[6] 1442. Copyright© (2013) Massachusetts Medical Society. Reprinted with permission from Massachusetts Medical Society.

β-Blockers and non-dihydropyridine calcium channel blockers (e.g. verapamil, diltiazem) should, where possible, be continued in the perioperative period as they reduce the incidence of perioperative SVT.

For patients with AF at high risk of thromboembolism, therapeutic anticoagulation should be stopped and therapeutic low molecular weight heparin bridging therapy considered in the pre- and postoperative period.

Upon acute onset of intraoperative SVT, aetiology should be considered before therapy is instituted except in extreme haemodynamic instability where immediate synchronized direct current (DC) cardioversion is required (refer to Fig. 2 for the SVT management algorithm and Table 3 for the therapeutic options for acute SVT management).

Regular narrow complex supraventricular tachyarrhythmia

In the haemodynamically stable patient, slowing conduction through the AV node may either terminate the tachycardia if the mechanism is dependent on the AV node (AVNRT or AVRT) or provide diagnostic information in the case of AFL (the P wave morphology is exposed and this arrhythmia is not dependent on the AV node for maintenance).

I.V. adenosine is recommended as a first-line medication due to a rapid onset and short half-life. The use of adenosine necessitates full ECG monitoring and resuscitation equipment in theatre should the rare complications of bronchospasm or asystole occur. Adenosine should be administered with extreme caution in asthmatics and patients taking digoxin and/or verapamil.

Second-line agents such as the longer-acting AV nodal blocking agent diltiazem can assist in the diagnosis and treatment of a narrow complex tachycardia but carry a higher risk of prolonged hypotension and should be used with caution in the perioperative setting.

Irregular narrow complex SVT

Atrial fibrillation

Management of acute-onset intraoperative AF should begin with assessment of haemodynamic status and correction of precipitants. For

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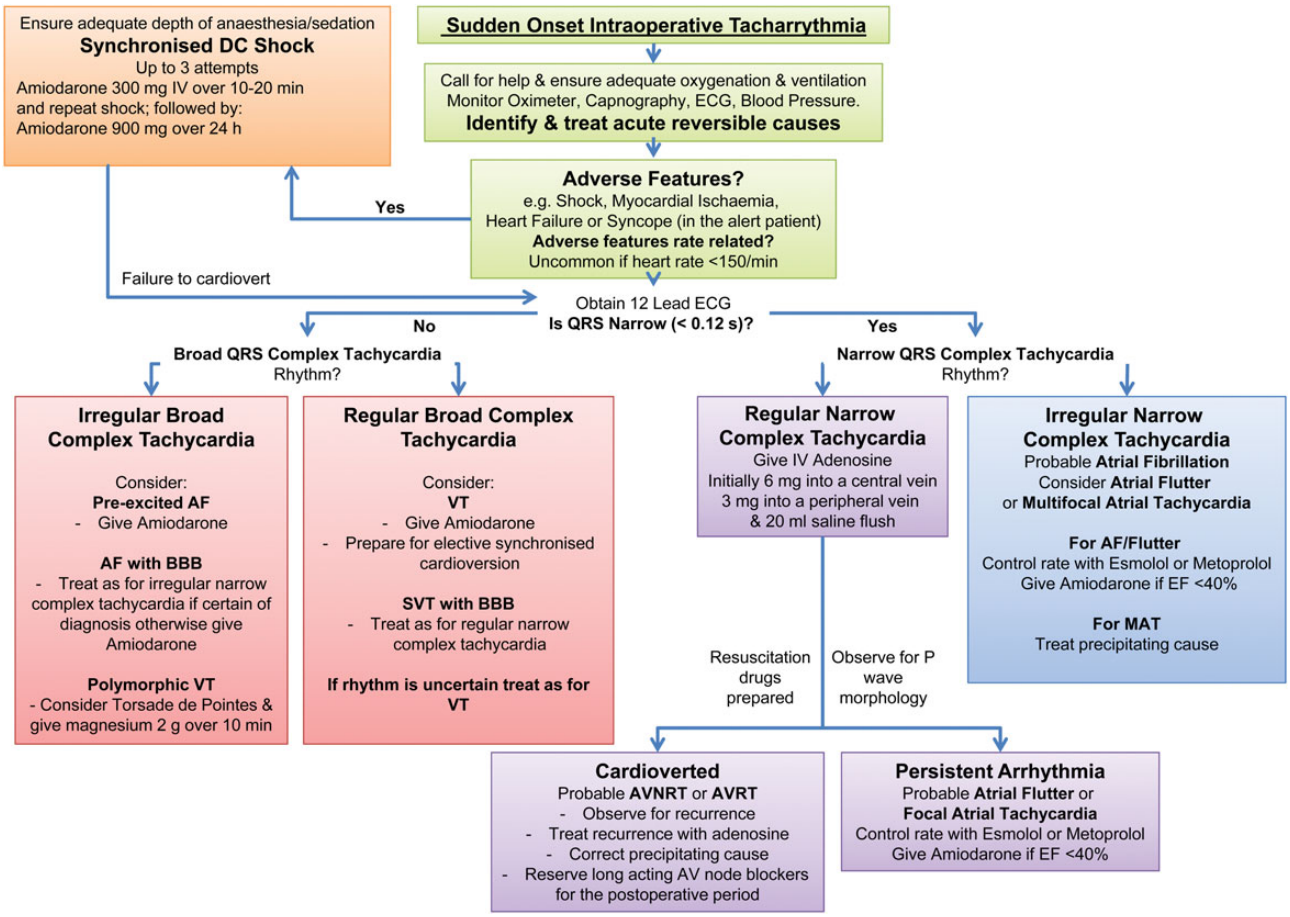

Fig 2 Management algorithm for acute onset intraoperative SVT.

perioperative AF, the goal of management is rate control. Slowing the ventricular rate lengthens diastole, enhancing stroke volume and reduces myocardial oxygen consumption. The β-blocker esmolol is rapidly hydrolysed by plasma esterases and therefore is easily titratable. While this drug is β_1-receptor selective and useful in patients with obstructive airways disease, it is negatively inotropic precluding use in patients with severe left ventricular impairment. Metoprolol is less titratable than esmolol and has similar β_1 receptor selectivity and negative inotropic effects. The non-dihydropyridine calcium channel blocker diltiazem has less negative inotropic action than esmolol but is less easily titrated. Both diltiazem and amiodarone are recommended for heart rate control in patients with left ventricular impairment. Diltiazem is more efficacious at rate control than amiodarone but is more likely to cause hypotension. I.V. digoxin slows the ventricular rate during SVT through vagotonic effects but is not recommended in the perioperative period due to slow onset (~6 h) and low efficacy in high adrenergic states such as surgery.

Postoperative AF predisposes patients to a considerably increased risk of stroke and thromboembolism. Patients with AF persisting for >48 h should be risk stratified against the CHADS2 score and if appropriate anticoagulated as soon as it is safe to do so from a surgical wound perspective.[11] The CHADS2 score stratifies patients for risk of stroke associated with AF according to the presence of congestive heart failure, hypertension, age ≥75, diabetes mellitus, and prior stroke or transient ischaemic attack.[12]

AF with preexcitation

In patients with an accessory pathway capable of anterograde conduction who develop AF, conduction to the ventricle often occurs through a combination of the AV node and the accessory pathway. However, because most accessory pathways have a shorter refractory period than the AV node, the ventricular rate can be more rapid if AV conduction occurs preferentially via the accessory pathway. For this reason, AV nodal blocking drugs (adenosine, diltiazem, verapamil, β-blockers, and digoxin) are contraindicated since blocking the AV node will promote conduction down the accessory pathway and potentiate ventricular fibrillation.

Haemodynamically unstable preexcited AF should be treated with synchronized DC cardioversion. Amiodarone, which is not selective for the AV node in prolongation of the action potential and

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Table 3 Therapeutic options for the acute treatment of SVTs

Treatment	Usual dose	Major acute side-effects	Cautions/contraindications
Haemodynamically unstable tachyarrhythmia			
Synchronized direct current electrical cardioversion	Broad complex tachycardia or atrial fibrillation, start with a 120–150 J biphasic shock (200 J monophasic) and increase increments if this fails up to 360J. Atrial flutter and regular narrow-complex tachycardia use lower energies: start with 70–120 J biphasic (100 J monophasic).	• Bradycardia, heart block, VT, VF, and asystole • Stroke • Burns • Complications of sedation	Contraindications • Digitalis toxicity • Haemodynamically stable AF of duration $>$ 48 h without anticoagulation/TOE Conditions unlikely to cardiovert • FAT, MAT, and Permanent AF
SVT with narrow QRS complex			
Adenosine	6 mg rapid i.v. bolus followed by flush into a proximal vein. If no response within 1–2 min; administer 12 mg	• Facial flushing, chest pain • Bronchospasm • Hypotension, rarely asystole	Contraindications • WPW syndrome and AF • Cardiac transplant recipients Cautions • Obstructive airways disease, digoxin, and verapamil
Esmolol	0.5 mg kg^{-1} over 1 min, then 50–200 μg kg^{-1} min^{-1} infusion titrated to response	• Hypotension, heart block, bradycardia, negative inotropy • Bronchospasm	Cautions • Asthma • Heart failure, AV block, Ca-channel blocker treatment
Metoprolol	1–5 mg over 10 min		
Diltiazem	0.25 mg kg^{-1} over 2 min, then 5–15 mg h^{-1} infusion titrated to response	• Hypotension, heart block, bradycardia, negative inotropy	Cautions • Heart failure, AV block, β-blocker treatment
SVT with preexcitation and refractory SVT			
Amiodarone	300 mg i.v. loading dose over 10 min Repeat 150 mg i.v. over 10 min as needed. Follow loading dose with 900 mg over 23 h. Maximum dose of 2.2 g in 24 h	• Hypotension, bradycardia, AV block, QTc prolongation • Phlebitis • Ocular, pulmonary, hepatic, haematological, neurological complications with chronic use	Contraindications • Pregnancy • Porphyria • AV block, marked sinus bradycardia Cautions • Administer via central vein and dilute in 5% dextrose
SVT with hypokalaemia (target serum K: $\geq$ 4.5 and < 5.5 mmol $litre^{-1}$)			
Potassium chloride	Infusion via peripheral access: Maximum infusion rate of 20 mmol KCl h^{-1} Maximum concentration of 40 mmol KCl $litre^{-1}$	Infusion via central access: Maximum infusion rate of 40 mmol KCl h^{-1} Maximum concentration of 1 mmol KCl ml^{-1}	• Hyperkalaemia • Arrhythmias, cardiac arrest • Paraesthesia, confusion, weakness • Phlebitis	• Continuous ECG monitoring is required for infusions $>$20 mmol h^{-1} • Cautiously replace potassium in renal impairment • Monitor blood glucose levels
SVT with hypomagnesaemia (target serum MG: $\geq$ 1 and < 2 mmol $litre^{-1}$, i.e. supranormal levels)			
Magnesium sulphate	Infusion via peripheral access: 2.5 g (10 mmol) Mg in 100 ml (2.5%) solution at 3 ml min^{-1}	Infusion via central access: 5 g (20 mmol) Mg in 50 ml (10%) solution at 1.5 ml min^{-1}	• Flushing, drowsiness, hypotension, bradycardia, arrhythmias • Respiratory depression and coma	• Continuous ECG monitoring is required for infusions • Cautiously replace magnesium in renal impairment • Monitor blood calcium levels

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refractory period, may be considered in haemodynamically stable preexcited AF.

Multifocal atrial tachycardia

Treatment of the arrhythmia's precipitant takes therapeutic precedent, as the condition is usually transient resolving when the underlying disease process improves.

Cardioversion is rarely successful in MAT. Owing to the multiple atrial foci, DC cardioversion is not effective in restoring sinus rhythm and may precipitate ventricular arrhythmias.

Broad complex SVT

SVT may occasionally present as a broad complex tachycardia due to a bundle branch block or conduction via an accessory pathway. Broad complex regular tachycardia should be treated as ventricular in origin, unless a diagnosis of SVT with aberrant conduction or of antidromic AVRT is certain. Attempts to treat VT with AV nodal blocking agents such as adenosine will prove ineffective and potentially deleterious.

Irregular broad complex tachycardias should be treated according to the established advanced life support guidelines.[9]

Future treatment options for SVT

Landiolol is an i.v. administered, ultra-short-acting β_1-blocker with an elimination half-life of 3–4 min and approximately eightfold greater cardioselectivity than esmolol. The rapid onset, readily titratable, and rapidly reversible effects make landiolol a potentially important agent for the management of perioperative tachyarrhythmias.[13]

Tecadenoson is an adenosine A_1 receptor-selective agonist undergoing clinical trials for termination of SVT and rate control of AF. Animal studies demonstrated significant prolongation of AV nodal conduction and refractoriness without negative inotropic effects. The A_1 receptor-selectivity of Tecadenoson avoids the adverse effect profile of adenosine, which also stimulates the A_{2A}, A_{2B}, and A_3 receptor subtypes.[14]

Pregnancy

Synchronized DC cardioversion is recommended for the haemodynamically unstable gravid patient with SVT. Electrical cardioversion does not result in compromise of blood flow to the fetus and the risk of inducing fetal arrhythmia is very small. Almost all antiarrhythmic drugs cross the placenta, although there is no evidence for teratogenicity or increased risk of adverse fetal/neonatal effects with adenosine, digoxin, diltiazem, or verapamil. Similarly, there is no evidence for teratogenicity with β-blockers, although there is risk of impaired fetal growth with atenolol and new-borns are at risk of β-blockade with prolonged treatment. Amiodarone is associated with congenital thyroid disease and prolonged QT interval and should be considered a drug of last resort.[15]

Areas of controversy

Published guidelines on the acute management of AF emphasize the role of rhythm control with electrical cardioversion or antiarrhythmic drugs such as flecainide.[12] In the perioperative setting, additional emphasis should be placed upon the trigger of arrhythmia. Treatment of the underlying disease, analgesia, oxygenation, and correction of haemodynamic or electrolyte derangement may restore sinus rhythm in the majority of cases without the need for antiarrhythmic drugs. Furthermore, the efficacy of electrical cardioversion is considered limited if underlying triggers are not eliminated and the adverse effects of antiarrhythmic drugs such as flecainide or procainamide may be exaggerated in the critically unwell, patients under general anaesthesia, or both.[8,16]

Declaration of interest

None declared.

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