# Management of supraventricular tachycardias

## Introduction

This pair of articles addresses the common arrhythmias seen in hospital, covering diagnosis, electrocardiogram findings and management strategies. The first article discussed the individual tachycardias in more detail, and this article looks at the best approach to management of the patient with supraventricular tachycardia.

## Management of supraventricular tachycardias

A common approach can be applied to the acute management of all supraventricular tachycardias and these are described first. Specific approaches to the management of the different types can be applied when the mechanism is clearer and these are described subsequently. *Figure 1* summarizes the approach to managing supraventricular tachycardias.

The choice of agents will be determined by the arrhythmia, cardiac and other system comorbidities, and patient preference. Beta-blockers can be used in the majority of patients but consider calcium-channel antagonists if severe asthma or peripheral vascular disease is present.

While amiodarone can maintain sinus rhythm, long-term use is complicated by many potential permanent side effects and as such may be used mainly when significant left ventricular impairment is present.

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Sotalol is a beta-blocker with additional unique class III actions. While a safe medication in many cases it can cause QTc prolongation and electrocardiograms should be repeated after initiation and uptitration, which reduces its routine use.

Flecainide can be used safely in the absence of structural heart disease and has efficacy in treating paroxysmal atrial fibrillation and Wolff–Parkinson–White syndrome.

Both flecainide and beta-blockers may be used for a 'pill in the pocket' approach in patients keen to avoid daily medications, but patient education will be required. Recurrent symptomatic arrhythmias should be referred for a specialist opinion and if suitable considered for radiofrequency ablation.

## Acute management

Supraventricular tachycardias should be managed in an appropriate environment with access to full resuscitation facilities. Electrocardiogram and blood pressure monitoring should be in place before undertaking any therapeutic manoeuvres.

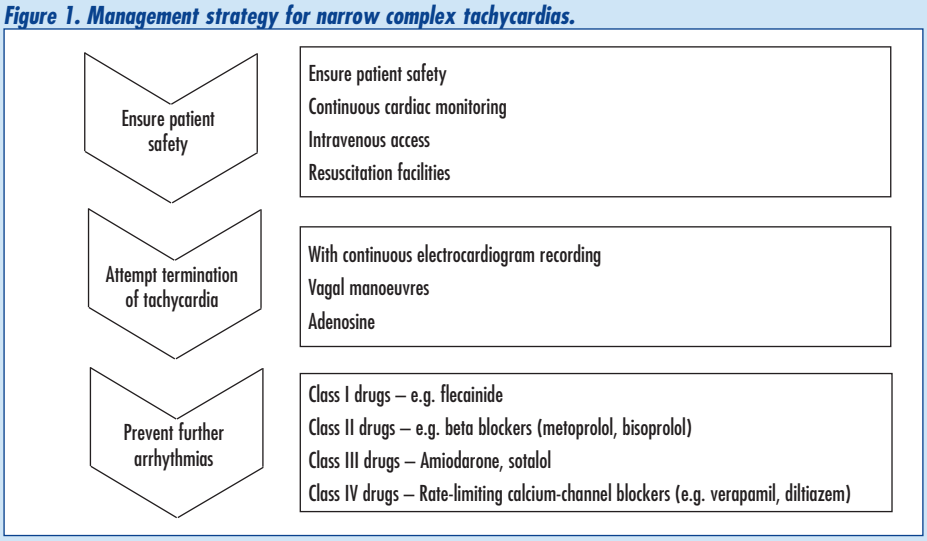
A useful first-line manoeuvre for both diagnostic and treatment purposes is vagal stimulation. This can include asking the patient to blow into a 20 ml syringe, carotid sinus massage, or asking the patient to perform a Valsalva manoeuvre. Should this not work escalating boluses of adenosine (6 mg, 12 mg) should be administered (Belhassen and Viskin, 1993). During either of these procedures it is essential to ensure that either an electrocardiogram rhythm strip or ideally a 12-lead electrocardiogram is being printed as this will aid further diagnosis and management.

In those with asthma or chronic obstructive pulmonary disease, adenosine can cause severe bronchospasm and should be used with caution. Instead, verapamil (5–10 mg intravenous) is effective – although it should be avoided if the patient is in heart failure or already on beta-blockers.

If there is haemodynamic instability with the tachycardia, then emergency direct current cardioversion should be considered early. Drugs blocking the atrioventricular node (adenosine, digoxin, verapamil) are contraindicated in atrial fibrillation with pre-excitation suggestive of an accessory pathway – by blocking the atrioventricular node they may precipitate arrest by allowing all the rapid atrial activity to be conducted 1:1 via the accessory pathway. Emergency treatment of atrial fibrillation in these patients is with intravenous flecainide or urgent direct current cardioversion.

When electrocardiograms from sinus rhythm, tachycardia, and during the administration of adenosine or vagal stimulation are available the supraventricular tachycardia mechanism may become clear. Adenosine will not terminate atrial flutter or atrial tachycardia, only delay conduc-

***Figure 1. Management strategy for narrow complex tachycardias.***



tion through the atrioventricular node to demonstrate the atrial rhythm disturbance like the classic flutter waves of atrial flutter. Alternatively, if the supraventricular tachycardia circuit is dependent on the atrioventricular node, as in atrioventricular nodal re-entrant tachycardia or atrioventricular re-entrant tachycardia, then adenosine-induced blockade will terminate the rhythm. If early re-initiation occurs, then intravenous verapamil can be used to terminate the arrhythmia and it has a longer duration of action.

## Management of atrioventricular re-entrant tachycardia and atrioventricular nodal re-entrant tachycardia

A similar approach is also used for the longer term management of atrioventricular re-entrant tachycardia and atrioventricular nodal re-entrant tachycardia. Pharmacological therapy can be used to prevent supraventricular tachycardia recurrence – a beta-blocker or verapamil can sometimes be used in combination with flecainide. Treatment can be taken by the patients as required to treat further attacks – a 'pill in the pocket' strategy. The patient can also be instructed to perform vagal manoeuvres should he/she feel the symptoms start.

The prospect of a cure achieved by radiofrequency catheter ablation can be offered to nearly all patients with supraventricular tachycardia, and in patients with recurrent symptoms, this is the optimal treatment and avoids long-term pharmacological therapy. In atrioventricular nodal re-entrant tachycardia, radiofrequency catheter ablation of the slow atrioventricular nodal pathway has a success rate around 98%, with a risk of inadvertent atrioventricular block requiring pacemaker implantation of 0.5% (Feldman et al, 2011).

Ablation of accessory pathways in atrioventricular re-entrant tachycardia can be similarly undertaken. By eliminating the accessory pathway the patient is protected from potential risk of rapidly conducted atrial fibrillation which may degenerate to ventricular fibrillation.

## Atrial flutter

The substrates for atrial flutter and atrial fibrillation often co-exist in the same patient. Acute management of both is also similar. However, it is a myth that atrial flutter is less harmful. The stroke risk is similar to atrial fibrillation and anticoagulation is an urgent consideration (Sparks and Kalman, 2001). Low molecular weight heparin is commonly used in the acute setting before switching the patient on to oral anticoagulants, of which warfarin continues to be most commonly used. Atrial flutter can be resistant to rate control with even multiple drugs. Patients should be treated with a view to either rate control or rhythm control depending on the time of onset of the arrhythmia and the clinical state of the patient.

Those presenting in flutter within 48 hours of onset can have cardioversion to sinus rhythm by medical therapy, external direct current cardioversion or radiofrequency catheter ablation without needing formal anticoagulation. Those presenting later than 48 hours should be anticoagulated, with an international normalized ratio between 2 and 3 for ≥3 weeks before cardioversion. Alternatively, transoesophageal echocardiography to exclude the presence of left atrial appendage thrombus may allow earlier cardioversion.

Direct current cardioversion can be very successful (~97%) and requires very low levels of defibrillation energy (50–100 J) to be effective. The previous article provided a detailed approach to cardioversion (Nijjer and Lefroy, 2012). The authors recommend this is done in a controlled environment with full resuscitation facilities and with sedation given by an anaesthetist. Paddles can be placed antero-posterior or in the usual antero-lateral position. Defibrillation shocks should be synchronized with the electrocardiogram to reduce the risk of 'R on T', which triggers ventricular fibrillation and cardiac arrest.

Medical rhythm control should be achieved using class I or III anti-arrhythmics; the most commonly used agents are flecainide or amiodarone. *Table 1* shows a simplified approach to the Vaughan-Williams classification of antiarrhythmics. Class I agents (flecainide or propafenone) should not be used alone in atrial flutter because changes to the refractory period of the atrioventricular node could allow flutter waves to be conducted one-to-one to the ventricle. This would lead to a ventricular rate of 300 beats per minute which may rapidly degenerate into ventricular fibrillation. Therefore, a class II or IV anti-arrhythmic (a beta-blocker or calcium channel blocker respectively) should be used in combination. These agents can also be used alone if a rate control strategy is being pursued, although high doses and multiple agents may be required to achieve ventricular rates of 60–80 beats per minute at rest.

Radiofrequency catheter ablation can be used to eliminate the right atrial circuit causing atrial flutter with a high success rate (>90%) and low risk of complications (Lesh et al, 1994). This is performed by introducing a catheter through the right femoral vein into the right atrium. The ablation procedure involves creating a line of scar from the most caudal point of the tricuspid annulus across an isthmus of

**Table 1. Simplified Vaughan-Williams classification of anti-arrhythmics**

| Class | Action | Main target tissue | Examples |
|---|---|---|---|
| I | Sodium channel blockade | Atrial and ventricular myocardium | Flecainide<br>Lidocaine (ventricular only)<br>Propafenone |
| II | Beta adrenoreceptor blockade | Sinus and atrioventricular node | Bisoprolol<br>Metoprolol<br>Esmolol (intravenous only, short acting)<br>Sotalol (also class III action) |
| III | Potassium channel blockade | Atrial and ventricular myocardium | Amiodarone<br>Dronedarone<br>Sotalol (also class II action) |
| IV | Calcium channel blockade | Sinus and atrioventricular node | Verapamil<br>Diltiazem |

Sinus tachycardia is helped by class II and IV drugs; atrial fibrillation and flutter can be treated using class I, II, III and IV drugs, as well as digoxin; supraventricular tachycardias are treated with class I, II and III drugs as well as adenosine. From Williams (1984)

atrial myocardium to the junction between the right atrium and the inferior vena cava. This creates electrical block in the typical flutter circuit, preventing recurrence.

## Atrial tachycardia

Management options for atrial tachycardia include medication to slow ventricular rate such beta blockers or calcium-channel antagonists. Sinus rhythm may be restored medically using antiarrhythmic medication such as class I (flecainide) or III agents (amiodarone, sotalol), or patients can be direct current cardioverted to sinus rhythm.

Radiofrequency catheter ablation can eliminate tachycardia in >85–95% of atrial tachycardia cases (Lesh et al, 1994).

## Conclusions

A simple and systematic approach should be used for the diagnosis and management of supraventricular tachycardia. Where possible documentation of the arrhythmia with a 12-lead electrocardiogram is essential and can guide further management with pharmacological therapy and in many cases radiofrequency ablation. As a cure from the disabling symptoms can be achieved with ablation in >90% of cases, referral to a specialist should be considered at an early stage. BJHM

### KEY POINTS

- The management of supraventricular tachycardias requires assessment of patient safety, termination of the arrhythmia and then prevention of recurrence; correct identification of the arrhythmia is essential.
- Termination of arrhythmia may occur with conservative vagal manoeuvres, medical anti-arrhythmics including adenosine or direct current cardioversion.
- Longer term management requires anti-arrhythmics, of which there are many; compliance is important.
- Radiofrequency ablation can be curative in some supraventricular tachycardias with a low rate of complications.

*Conflict of interest: none.*

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