Diagnosis of supraventricular tachycardias

Introduction

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

Classification of supraventricular tachycardias

In adults a heart rate above 100 beats per minute is classified as a tachycardia. The term supraventricular tachycardia describes a common group of tachycardias which arise from atrial or atrioventricular nodal tissue. The mechanism of tachycardia is either re-entrant, in which a single impulse repetitively travels around a re-entrant circuit, or focal in which a localized group of abnormal myocardial cells emits repeated depolarizations in rapid succession 'overdriving' the normal sinus rhythm. In practice, however, the term supraventricular tachycardia is generally restricted to five syndromes, classified according to their site of origin:

1. Sinus tachycardia (focal)
2. Atrioventricular nodal re-entrant tachycardia (re-entrant)
3. Atrioventricular re-entrant tachycardia (re-entrant)
4. Atrial flutter (re-entrant)
5. Atrial tachycardia (focal or re-entrant).

By convention, atrial fibrillation is considered a separate entity and was discussed in a separate article (Nijjer and Lefroy, 2012). Supraventricular tachycardia usually refers to a tachycardia in which the atrial rhythm is regular. The ventricular rhythm is usually also regular, although depending on the mechanism irregularities may occur as a result of variable conduction through the atrioventricular node.

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Clinical features

Patients typically recall a sudden onset of fast palpitations. They may have chest discomfort and breathlessness as a consequence of the fast heart rate. Typically the patient was previously well, but had sudden onset of palpitations. There may be transient pre-syncope, or even syncope, but consciousness usually returns rapidly. There may be a history of previous attacks. Patients feel anxious during the attack. Spontaneous termination is common. Sinus tachycardias by contrast subside gradually over minutes. Often there is prolonged tiredness after the episode (Wood et al, 1997).

The electrocardiogram usually shows a narrow QRS complex tachycardia which may require additional manoeuvres to resolve a diagnosis. A narrow QRS tachycardia occurs because during most supraventricular tachycardias the ventricles are activated via the atrioventricular node via the His–Purkinje system. Because activation occurs over the normal conduction system activation is rapid and therefore the QRS complex is narrow (less than 120 msec). Less commonly supraventricular tachycardias may result in a broad complex tachycardia. This may be because the patient has pre-existing bundle–branch block or develops rate-dependent bundle–branch block at higher heart rates. In both cases QRS morphology displays the typical features of either right or left bundle–branch block. Antidromic atrioventricular re-entrant tachycardia, where the ventricles are activated via an accessory pathway during the tachycardia, also produces a broad complex tachycardia. This is discussed in more detail later. While these exceptions exist, all broad complex tachycardias should be treated as ventricular tachycardia until proven otherwise.

Sinus tachycardia

Sinus tachycardia is discussed here for completeness and refers to a rapid narrow complex tachycardia that is driven by activity of the sino-atrial node in the atrium. It is usually appropriate, in response to physiological changes. The P waves preceding each QRS complex are normal in appearance and axis, and should have the same morphology as those seen in a resting electrocardiogram. At very fast rates the P wave may merge into the T wave making this difficult to discern. Although the heart rate is fast, it remains variable, changing with posture or activity.

Administration of adenosine results in transient atrioventricular block. As a result during sinus tachycardia ventricular rate will be slowed but the atrial rate continues to be rapid, P waves can be seen and the morphology can be examined. Adenosine will not terminate sinus tachycardia – the ventricular rate returns to the previous levels when the effect of adenosine has worn off after 15–30 seconds.

Common causes of sinus tachycardia are listed in *Table 1*. Treatment will depend on the cause and requires a careful diagnostic approach seeking the underly-

Table 1. Causes of a sinus tachycardia

Hypovolaemia
Shock
Pain
Sepsis
Fever
Anxiety
Heart failure
Pulmonary embolus
Hyperthyroidism
Exercise
Stimulants (salbutamol, cocaine, caffeine)

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ing problem. Thyrotoxicosis will require beta-blockers, occasionally at high doses.

Rarely, sinus tachycardia is inappropriate but this is a diagnosis of exclusion when the sinus rate is excessively high in the absence of any apparent trigger (Morillo et al, 1994). It may be caused by autonomic dysfunction and is difficult to treat effectively. Beta-blockers, verapamil and ivabradine are sometimes tried, with variable symptomatic response. Most cases remit spontaneously after a few years.

Atrioventricular nodal re-entrant tachycardia

Atrioventricular nodal re-entrant tachycardia is the most common form of supraventricular tachycardia presenting as an emergency, typically in early adulthood (Porter et al, 2004).

Mechanism

The re-entry circuit is entirely within the atrioventricular node, which has two separate conduction pathways (fast and slow) which are connected at the upper and lower ends, thus forming a potential re-entrant circuit.

Diagnosis

During tachycardia no distinct P waves are seen on the electrocardiogram as they occur simultaneously with the QRS complex (*Figure 1*). Careful comparison of the electrocardiogram in sinus rhythm with the supraventricular tachycardia electrocardiogram may show distortions of the QRS complex in tachycardia such as terminal pseudo r' waves in lead V1 or pseudo S or Q waves in II, III or aVF which are in fact P waves.

In patients with typical left or right bundle–branch block, the QRS will be broad. This can be distinguished from ventricular tachycardias by attempting vagotonic manoeuvres or intravenous adenosine; if the arrhythmia responds by slowing down or terminating, it is likely to be a supraventricular tachycardia although there are some special types of ventricular tachycardia that respond to adenosine. Adenosine should only be administered in a setting with full resuscitation facilities. Expert help should be sought and if there is concern about ventricular tachycardia, urgent senior support is mandatory.

Atrioventricular re-entrant tachycardia

The presence of an accessory pathway connecting the atrium and ventricle electrically allows for the occurrence of atrioventricular re-entrant tachycardia. This includes Wolff–Parkinson–White syndrome.

Mechanism

The orthodromic re-entry circuit travels from the atrium to the atrioventricular node to the ventricle, then back up the accessory pathway to the atrium again (*Figure 2*). This results in a narrow QRS complex tachycardia. The less common antidromic re-entry circuit travels from the atrium down the accessory pathway to ventricle, then back up the atrioventricular node to atrium. This results in a broad QRS complex tachycardia with bizarre QRS morphology similar to ventricular tachycardia. Patients with Wolff–Parkinson–White syndrome may be at risk of rapid ventricular rates (>300/min) if they develop atrial fibrillation with increased risk of sudden death (*Figure 3*). The rapid atrial activity is conducted to the ventricle via the accessory pathway without the safety net of the refractory period of the atrioventricular node.

Diagnosis

The resting electrocardiogram may be normal, or may have evidence of an accessory pathway, with a short PR interval, broad QRS complex and delta waves (e.g. Wolff–Parkinson–White syndrome). The delta wave is caused by the activation of a portion of the ventricular myocardium via the accessory pathway (*Figure 4*) (Wolff et al, 1930). During tachycardia, a rapid narrow complex tachycardia is seen in orthodro-

Figure 1. Narrow complex tachycardia. Retrograde P waves are seen within the QRS complexes, particularly in lead II. This likely represents an atrioventricular nodal re-entrant tachycardia.

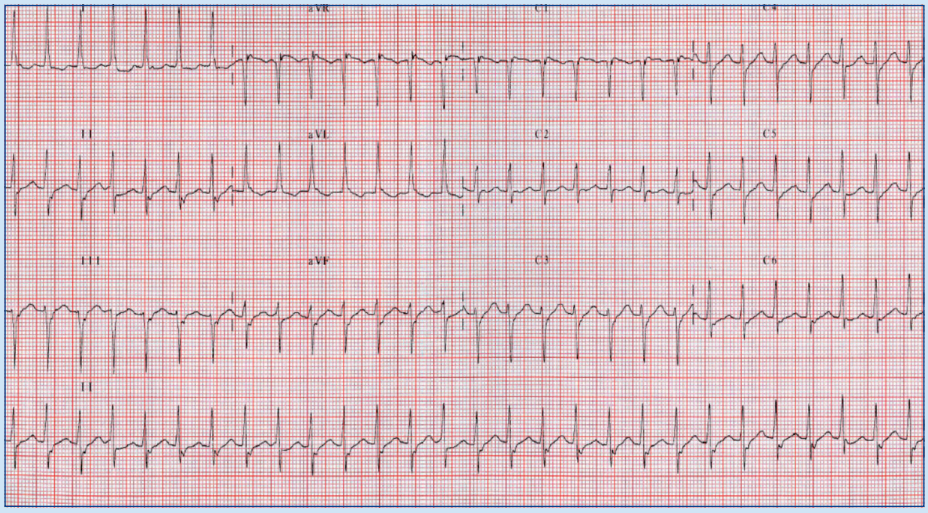
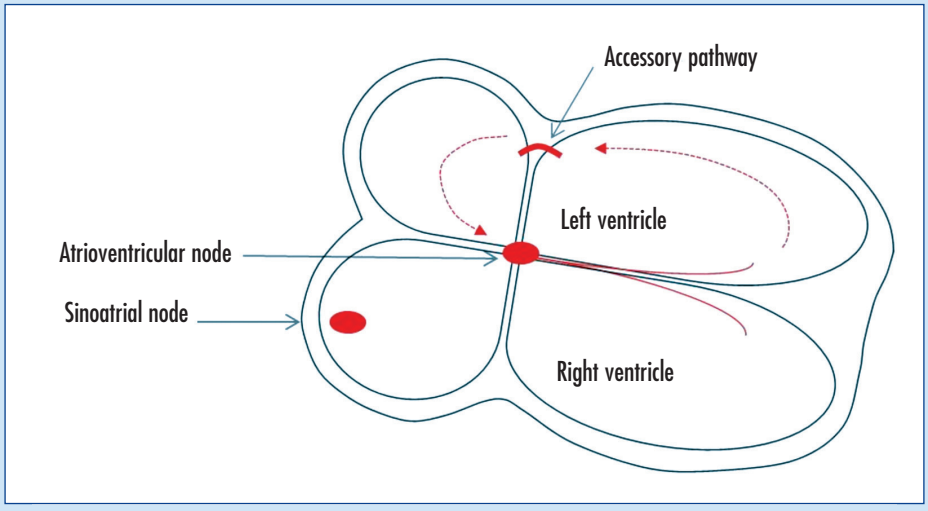


Figure 2. Orthodromic atrioventricular re-entrant tachycardia.



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mic atrioventricular re-entrant tachycardia. P waves may or may not be visible and the electrocardiogram can be similar in appearance to atrioventricular nodal re-entrant tachycardia.

Atrial tachycardia

Mechanism

Atrial tachycardias are commonly classified according to whether they originate from a small localized area in the atrium (focal atrial tachycardia) or involve a larger re-entrant circuit (macro re-entry).

In focal atrial tachycardia, rapid electrical impulses are generated from a small localized area in the atria at a rate faster than the sinoatrial node, typically 110–240 beats per minute. Rarely, multi-focal atrial tachycardias may occur, mostly in the elderly or those with theophylline toxicity. Multifocal atrial tachycardias have a propensity to develop atrial flutter or atrial fibrillation (Chen et al, 1998).

Macro re-entry atrial tachycardias involve a re-entrant circuit which includes a large area of the atrium. Atrial flutter where the re-entrant loop circles the right atrium is an example. Because it is so common and has characteristic features it is often discussed separately from other atrial tachycardias. Other re-entrant atrial tachycardias are seen in patients who have structural heart disease and in those who have had previous surgery (increasingly in those who have had catheter ablation procedures to treat atrial fibrillation).

Figure 3. Atrial fibrillation occurring in Wolff–Parkinson–White syndrome. The accessory pathway allows rapid conduction of the irregular atrial rhythm, leading to haemodynamic instability and high risk of ventricular fibrillation.

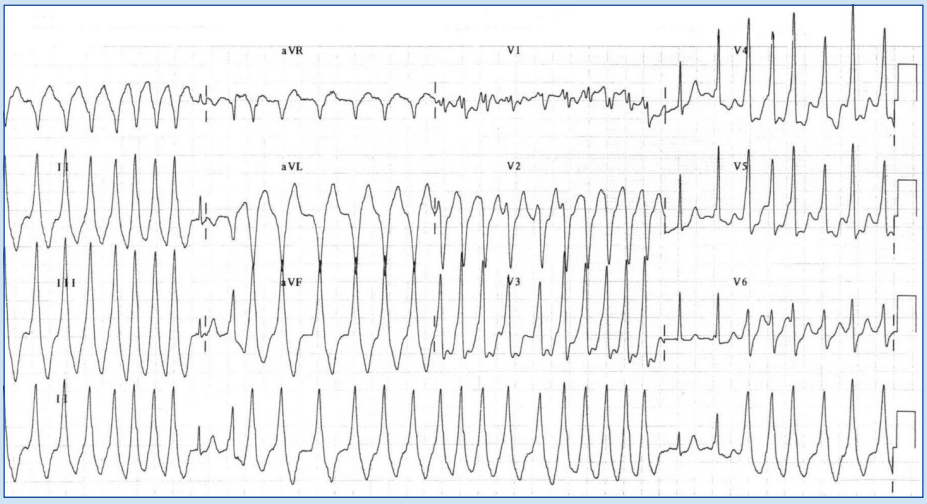


Figure 4. Wolff–Parkinson–White syndrome. In resting sinus rhythm, a shortened PR interval and slurred upstroke of the QRS (delta wave) can be seen.

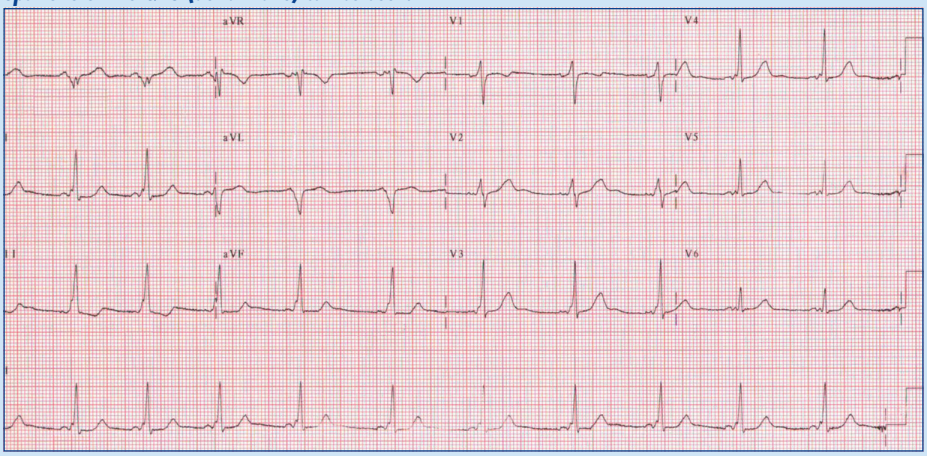
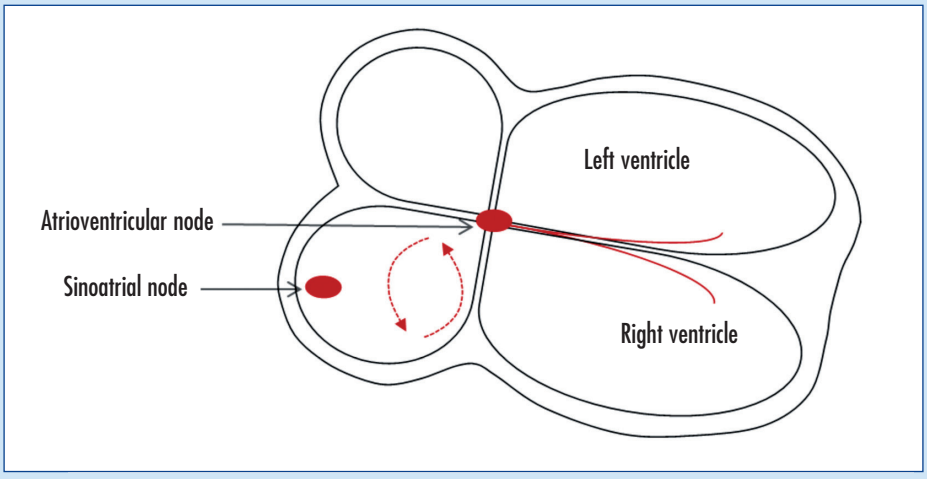


Figure 5. Typical atrial flutter.



Diagnosis

Examining the electrocardiogram it may be apparent that the P wave morphology is different to that seen in sinus rhythm. Ventricular rate tends to remain constant and it may be possible to see more P waves than QRS complexes, for example they may conduct in a 2:1 or 3:1 pattern.

Atrial flutter

Atrial flutter is a form of macro-reentrant atrial tachycardia. It is common in patients with hypertension, hyperthyroidism, lung disease or structural heart problems, such as atrial septal defects or mitral valve disease. Atrial flutter may be paroxysmal or persistent, and may be symptomatic or found incidentally.

Mechanism

Atrial flutter arises from the right atrium in most cases. In structurally normal hearts, there is a potential re-entrant circuit with an electrically protected pathway occurring between anatomical barriers that allows the electrical impulse from one beat to be passed on to the next beat. The pathway exists between the tricuspid annulus anteriorly and the crista terminalis posteriorly (*Figure 5*) (Lee et al, 2005). The anatomical length of this circuit around the tricus-

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pid annulus and the conduction velocity determines the cycle length, typically 200 ms, meaning that the atria usually flutter at a rate of around 300 per minute. This gives rise to the very characteristic electrocardiogram pattern with saw-tooth shaped flutter (F) waves which replace normal P wave activity.

Commonly, the impulses are conducted from the atria to the ventricles with a ratio of 2:1 or 4:1, giving rise to ventricular rates which are regular, at around 150/min or 75/min. Variable conduction ratios are common, giving rise to irregular ventricular rates. 1:1 conduction is possible, particularly on physical exertion, giving rise to ventricular rates of 300/min leading to collapse.

Figure 6. Atrial flutter. F waves are best seen in lead II: they are 'negative' (point downwards) suggesting a 'typical' counter-clockwise atrial flutter. The ventricular rate is regular here, with 2:1 conduction.

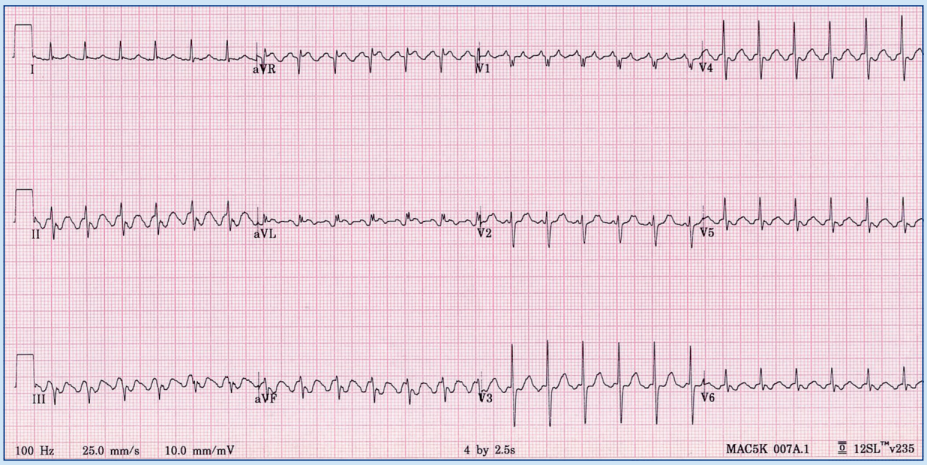
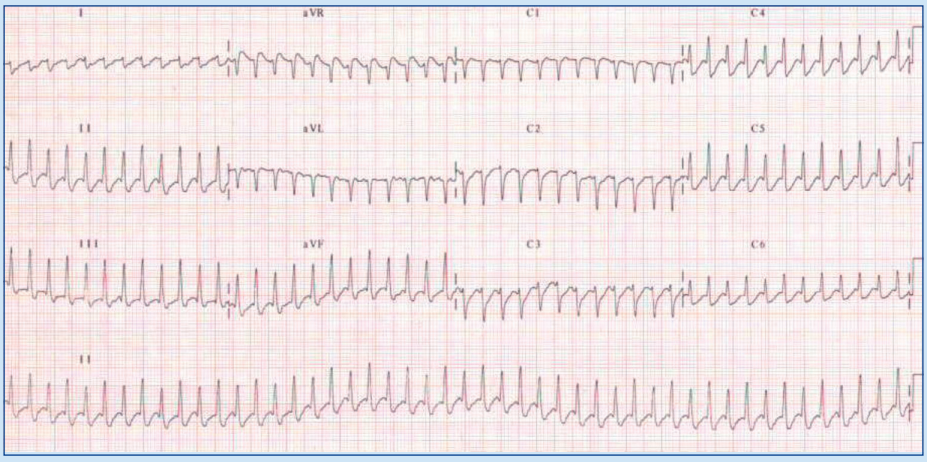


Figure 7. Atrial flutter with 1:1 conduction, seen as narrow complex tachycardia without obvious P waves. The QRS complexes are regular and approximately 300bpm – giving a clue to flutter. Adenosine would reveal flutter 'F' waves.



KEY POINTS

- Supraventricular tachycardias are common and are likely to be one of five subtypes.
- Recognition of key electrocardiographic features can help identify which specific type of arrhythmia is present.
- Adenosine can help to terminate most supraventricular tachycardias but must be used with full resuscitation facilities close at hand and with senior and expert support.
- Recognition of the appropriate arrhythmia will determine the appropriate management, which will be discussed in the next article.

Diagnosis

The electrocardiogram appearance shows characteristic 'saw-tooth' flutter waves which replace normal P wave activity and are best seen in leads II and V1 (*Figure 6*).

At fast ventricular rates, flutter waves are difficult to discern (*Figure 7*), more so if bundle–branch block is present. Atrial flutter should be suspected with rapid regular heart rates of 150 bpm. Intravenous adenosine will induce temporary block at the atrioventricular node, thereby revealing flutter waves. Adenosine will aid diagnosis but will not terminate atrial flutter: a common error is to continue giving adenosine in rising doses in the forlorn hope that it will.

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**

Conflict of interest: none.

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