BMJ

BMJ 2012;345:e7769 doi: 10.1136/bmj.e7769 (Published 11 December 2012)

CLINICAL REVIEW

Diagnosis and management of supraventricular tachycardia

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The term supraventricular tachycardia (SVT) encompasses many tachycardias in which atrial or atrioventricular nodal tissue are essential for sustaining the arrhythmia (box 1). In practice, however, the term SVT is generally used to refer to atrioventricular nodal re-entry tachycardia (AVNRT), atrioventricular re-entry tachycardia (AVRT), and atrial tachycardia[1]; in this review we follow that convention. We reviewed the literature to provide an up to date summary of our understanding of the mechanism for these arrhythmias, and we describe the approach to their diagnosis and management. Atrial fibrillation has been reviewed recently[2] so is not discussed in detail here. Much of the clinical evidence in this field is derived from observational and registry data, with a limited number of randomised control studies.

What is a supraventricular tachycardia?

SVTs are produced by disorders of impulse formation (causing generation of rapid electrical impulses from a small area) and/or disorders of impulse conduction, which result in re-entrant tachycardias, where tachycardia is produced because the electrical impulse repeatedly travels around a circuit. Re-entrant tachycardias typically require an extrasystole to initiate them. SVTs are among the few medical conditions that can be cured.

What are the different types of SVT?

AVNRT

AVNRT is caused by a re-entrant circuit involving the posterior and anterior inputs into the compact atrioventricular node.[3] A quarter of the population has two pathways that input into the compact atrioventricular node, one of which is a rapidly conducting pathway and the other a slower conducting pathway; these two pathways form the circuit for AVNRT (fig 1⇓).

The extrasystole that triggers the tachycardia is critically timed so that the faster pathway is still refractory from the last sinus beat (that is, it is not yet ready to conduct an impulse because it is still recovering from depolarisation). The electrical impulse therefore propagates exclusively down the slow pathway, which has a shorter refractory period (that is, it takes less time to recover from depolarisation) and then returns up the fast pathway, which has by then recovered. The circuit for typical AVNRT is thus formed.

Less commonly, activation travels anterogradely down the fast pathway and returns up the slow pathway, forming atypical AVNRT. These different mechanisms result in very different appearances on the surface electrocardiogram (ECG). In the typical form, atrial and ventricular activation occur virtually simultaneously (because the impulse returns up the fast pathway at the same time as travelling down the His-Purkinje system), so the P waves are invisible in the QRS complexes (a pseudo-RSR' pattern in lead V1 often provides a clue to the presence of the P wave), whereas the delayed atrial activation via the slow pathway in atypical AVNRT produces inverted P waves typically in the middle of or late in the RR interval.

Typical AVNRT presents as a short RP tachycardia (the ECG during tachycardia has a RP interval shorter than PR interval) while atypical AVNRT produces a long RP tachycardia (RP interval longer than PR interval) (fig 2⇓).

AVRT (including Wolff-Parkinson-White syndrome)

AVRT is also a re-entrant tachycardia; it requires the presence of an accessory pathway, a small strand of myocardium that bridges the normal insulation between atria and ventricles (fig 3⇓).

Some pathways can conduct impulses only from the ventricle to the atrium and are known as concealed accessory pathways. Others can conduct in both directions and usually produce pre-excitation of the ventricle because they conduct more rapidly than the atrioventricular node. This early ventricular activation shows on the 12 lead ECG as a delta wave at the start of the QRS (fig 3⇓). The terminal portion of the QRS complex is narrow, reflecting the rapid conduction via the His-Purkinje system once the atrioventricular node has been crossed. The degree of pre-excitation varies depending on the time required to cross the atrioventricular node and the location of the accessory pathway.

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Summary points

Supraventricular tachycardia comprises a group of conditions in which atrial or atrioventricular nodal tissues are essential for sustaining the arrhythmia

Common symptoms include palpitations, chest pain, anxiety, light headedness, pounding in the neck, shortness of breath, and uncommonly syncope

They are produced either by disorders of impulse formation and/or disorders of impulse conduction

For patients presenting with a regular narrow complex tachycardia, initial management is usually to slow atrioventricular node conduction, using either vagal manoeuvres or adenosine

Drug treatment may reduce the frequency of symptoms, but complete suppression is uncommon

Catheter ablation, a procedure done under local anaesthesia in the cardiac catheter laboratory, is usually curative

Sources and selection criteria

As well as using our personal reference collections, we searched PubMed to identify peer reviewed original articles, meta-analyses, observational studies, and reviews, as well as searching Clinical Evidence (http://clinicalevidence.bmj.com) and the Cochrane Collaboration databases. We used the search terms supraventricular tachycardia, atrioventricular nodal re-entry tachycardia, atrioventricular re-entry tachycardia, atrial flutter, atrial tachycardia, Wolff-Parkinson-White syndrome.

We selected randomised controlled studies or meta-analyses when available; if none were available, we used observational studies and registry data.

Box 1 Differential diagnosis of a narrow complex tachycardia

Common causes

- Sinus tachycardia
- Atrioventricular nodal re-entry tachycardia (AVNRT)
- Atrioventricular re-entry tachycardia (AVRT)
- Atrial tachycardia
- Atrial flutter
- Atrial fibrillation

Rare causes

- Inappropriate sinus tachycardia
- Sinus node re-entry
- Permanent junctional reciprocating tachycardia
- Non-paroxysmal junctional tachycardia
- Focal junctional tachycardia

The diagnosis of Wolff-Parkinson-White syndrome describes patients with a delta wave on the surface ECG who also experience palpitations.

AVRT is usually triggered by a critically timed atrial extrasystole that finds the accessory pathway refractory, therefore antegrade conduction occurs exclusively down the atrioventricular node. The accessory pathway is no longer refractory by the time the wave front reaches its ventricular insertion and it therefore conducts retrogradely to the atrium—the re-entrant circuit is thus formed (fig 3⇓); this activation pattern is termed orthodromic AVRT, and produces a narrow complex tachycardia. Rarely, anterograde conduction travels down the accessory pathway returning to the atrium via the atrioventricular node, producing antidromic AVRT, which has broad QRS complexes that exaggerate the pattern of pre-excitation seen in sinus rhythm because ventricular activation occurs exclusively via the accessory pathway.

Atrial tachycardia

Atrial tachycardia may result from either abnormal impulse formation or a re-entrant mechanism. Atrial tachycardias are commonly classified according to whether they originate from a small localised area in the atrium (focal atrial tachycardia) or involve a larger re-entrant circuit (macro re-entry).

Focal atrial tachycardia

In focal atrial tachycardia there is generation of rapid electrical impulses from a small localised area in the atria (fig 4⇓).

Multifocal atrial tachycardia

Multifocal atrial tachycardia is less common than focal atrial tachycardia and occurs most often in acutely unwell patients and those with pulmonary disease and/or digoxin toxicity. At least three different morphologies of P wave are usually present.[4]

Macro re-entrant atrial tachycardia

The re-entrant circuit involves a large area of the atrium. The commonest is "typical" atrial flutter where the re-entrant loop circles the right atrium (fig 5⇓). 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).[5]

Who gets SVT?

People of all ages, either sex, and any ethnicity can develop SVT. In a large retrospective observational study in the United States the peak incidence of SVT presentation was in the middle decades of life (AVNRT at 48 (range 30-66) years; AVRT at 36 (18-64) years; and atrial fibrillation at 50 (31-69) years).[6]

A higher proportion of AVNRT cases occur in women, and AVRT is more likely to affect men.[7] No sex difference was observed for atrial fibrillation.

SVTs usually manifest themselves as recurrent palpitations, can seriously impair quality of life,[8] and often prompt visits to primary care doctors and acute medical units. The incidence in a large cohort in the United States was found to be 35/100 000 patient years, with a prevalence of 0.2%.[9] An estimated prevalence for atrial fibrillation is 0.4% to 1% in the general population.[10]

How does SVT present?

Common symptoms include palpitations, chest pain, anxiety, lightheadedness, pounding in the neck, shortness of breath, and uncommonly syncope.[11]

Sudden onset and offset of palpitations is typical for a re-entrant arrhythmia, while for sinus tachycardia onset and offset is usually gradual. Patients with AVNRT or AVRT may be able to terminate palpitations with vagal manoeuvres such as the Valsalva manoeuvre, breath holding, or coughing.

Some patients can identify triggers such as caffeine or alcohol intake, which can initiate re-entrant tachycardia by increasing the frequency of extrasystoles.

In the absence of an acute episode the examination is usually normal. In a patient with tachycardia, prominent jugular venous A waves caused by atrial contraction against the closed tricuspid valve may be seen.[12]

What investigations are needed?

Electrocardiography

Attempts should always be made to capture the arrhythmia during an episode of palpitations. We recommend giving the patient a copy to ensure it is never lost and is easily available to health professionals. A rhythm strip should be recorded if 12 lead electrocardiography is not available, and every effort should be made to capture the termination of the tachycardia.

For patients with non-sustained episodes of palpitations, an ambulatory electrocardiograph or an event monitor can be useful in capturing an ECG during an episode.

Narrow complex tachycardia is most frequently seen (QRS <120 ms), but less commonly the QRS complexes are broad during tachycardia. Even though supraventricular tachycardias can present with broad QRS complexes, in the initial evaluation, a broad complex tachycardia should be treated as a ventricular tachycardia until proven otherwise.

A 12 lead ECG in sinus rhythm should also be recorded and carefully examined for the presence of a delta wave. In figure 2⇓ we suggest how to analyse the 12 lead ECG during tachycardia.

Echocardiography

Echocardiography is an important investigation for patients presenting with palpitations. The presence of structural heart disease such as left ventricular impairment should prompt urgent referral and investigation. Left ventricular impairment is associated with an increased risk of sudden cardiac death,[13] and patients are less likely to tolerate tachycardia. Such patients should not be prescribed class 1 antiarrhythmic drugs, such as flecainide.[1]

Most patients with SVT have a structurally normal heart. In a series of 145 patients referred for ablation, only 4% had heart failure.[14] However, certain types of structural heart disease are associated with particular SVTs. Patients with Ebstein's anomaly (congenital displacement of the septal tricuspid valve leaflet into the right ventricle) have an increased frequency of right sided accessory pathways.[15] Incisional atrial tachycardia occurs as a result of re-entry around surgical scars and is a major source of morbidity in the growing population of survivors of congenital heart disease.[16]

What are the acute management options in SVT?

Non-drug treatment

Patients presenting with sustained tachycardia should be assessed for haemodynamic stability; rarely, the arrhythmia is so poorly tolerated that immediate electrical cardioversion is needed. With the important exception of pre-excited atrial fibrillation (in which case the ECG shows an irregular, broad complex tachycardia, with variation of the QRS morphology (fig 6⇓)), the first line manoeuvre is usually to slow conduction through the atrioventricular node. This may either terminate the tachycardia if the mechanism is dependent on the atrioventricular node (AVNRT, AVRT) or provide diagnostic information in the case of atrial tachycardia (P wave morphology exposed and lack of dependence on the atrioventricular node to maintain the tachycardia). A continuous 12 lead ECG recording should be made during these manoeuvres (fig 7⇓).

Atrioventricular node conduction can be slowed by vagal stimulation with carotid sinus massage or the Valsalva manoeuvre.

Pre-excited atrial fibrillation should be managed with electrical cardioversion, or by flecainide infusion if the tachycardia is haemodynamically well tolerated and the heart is normal.

Drug treatment

If vagal manoeuvres are unsuccessful in slowing atrioventricular conduction then, unless the patient is asthmatic, the ACC/AHA/ESC guidelines[1] recommend intravenous adenosine as first line medication. Adenosine is preferred owing to its rapid onset of action and short half life. In a randomised double blinded placebo trial in participants with SVT, tachycardia was terminated in 91% of those who received adenosine (at a dose of either 6 mg or 12 mg) and in 16% of those receiving placebo.[17] When administering adenosine, ensure that resuscitation equipment is available in case the rare complications of bronchospasm or ventricular fibrillation occur.

The effectiveness of intravenous verapamil seems similar to that of adenosine in terminating SVT. In a randomised controlled trial, intravenous verapamil (2.5-7.5 mg) resulted in termination of tachycardia in 91% of patients, compared with termination in 93% of those receiving adenosine (6 mg dose followed by a 12 mg dose if required).[17] A meta-analysis of eight trials with a total of 605 patients, found similarly high rates of termination with both adenosine and verapamil (91% *v* 90%), with slightly higher rates of minor adverse effects and lower rates of hypotension described with adenosine.[18] β blockers are a further option and are effective in lowering heart rate, but in a small randomised study intravenous esmolol converted only 6% of SVTs to sinus rhythm.[19] Amiodarone[20] and flecainide are further options. Acute administration of flecainide successfully restored sinus rhythm in 72% of patients with AVRT and 83% with AVNRT.[21]

Macro re-entrant atrial tachycardias are not usually responsive to antiarrhythmic drugs and usually require electrical

cardioversion (or overdrive atrial pacing, where tachycardia is terminated by rapid pacing of the atrium using either an existing pacemaker or temporary pacing lead in the right atrium) to restore sinus rhythm. The ACC/AHA/ESC guidelines recommend that before cardioversion the same guidelines for thromboembolic prophylaxis are followed for atrial flutter as for atrial fibrillation (international normalised ratio 2-3, or arrhythmia duration <48 hours, or no evidence of atrial thrombus on transoesophageal echocardiography).[1]

What are the long term management options?

In general, improvement of quality of life is the major therapeutic goal for SVTs, and treatment strategies should be guided according to symptoms and patient preference. Patients troubled by recurrent symptomatic episodes should be offered treatment; options include medication or catheter ablation. Patients can be taught the Valsalva manoeuvre and some find this helpful in controlling their symptoms. This can be described to the patient as simulating straining on the toilet, or trying to breathe out forcefully while keeping the mouth closed and nose pinched.

There have been no large scale randomised studies comparing these treatments. However, data from prospective non-randomised studies suggest that catheter ablation results in a greater reduction in symptoms and higher quality of life scores compared with medical treatment.[22 23]

Patients with pre-excitation (delta wave on ECG) warrant special consideration. Such patients are at risk of sudden cardiac death from ventricular fibrillation induced by rapidly conducted atrial fibrillation. The findings from a recent meta-analysis suggest that in patients who do not have palpitations, this risk seems to be relatively low (1.25 per 1000 person years, 95% confidence interval 0.57 to 2.19 per 1000 person years).[24] Symptoms of palpitations appear to be associated with an increased risk of ventricular fibrillation.[25]

We advise referring patients with pre-excitation to an electrophysiologist for further assessment. Patients with documented broad complex tachycardia (QRS duration >120 ms and heart rate >100 beats/min), even if they have only had a single episode, are also best referred early. Box 2 outlines which patients to refer to an electrophysiologist.

Drug treatment

In general drug treatment is reserved for minimising symptoms while awaiting catheter ablation or for long term management of patients who decline catheter ablation or in whom the procedure carries an unacceptably high risk. Drug treatment may be effective in reducing the frequency of symptoms but complete suppression is uncommon.[26]

A major limitation in evaluating antiarrhythmic agents for treatment of SVTs is the absence of large, multicentre, randomised, placebo controlled studies.

For AVNRT and AVRT without pre-excitation, agents to block atrioventricular node conduction are commonly used as first line medication, although only limited data are available on the long term efficacy of these medications. Data from a small, randomised, double blind trial, showed that verapamil, propranolol, and digoxin all reduced the frequency of symptoms, with all equally effective.[26]

For patients who do not respond to these drugs, alternatives include flecainide and sotalol.[27] Although only relatively small numbers of patients have been included in randomised studies of flecainide and sotalol, the available data suggest they can be effective in reducing symptoms. In one small placebo controlled study 79% of patients in the flecainide group and 15% in the placebo group did not experience symptoms, though the follow-up period was only eight weeks.[28] Proarrhythmia, including ventricular tachycardia, has been reported in 6% of patients.[29] Agents to slow atrioventricular node conduction are often co-administered, as flecainide may predispose to atrial flutter with 1:1 atrioventricular conduction.[29]

Flecainide is not recommended in patients with structural or ischaemic heart disease, in whom it is associated with an increased risk of sudden cardiac death.[30] Amiodarone is generally not recommended owing to the potential for serious adverse effects in patients with a benign condition. However, it may be used to treat atrial tachycardias resistant to other treatments, particularly in patients with structural heart disease and in elderly people.

Catheter ablation

Catheter ablation provides a definitive management option for SVT and is usually done under local anaesthesia as a day case. Catheters capable of recording electrical activation in the heart are inserted via the femoral vessels and manipulated under x ray guidance. Radiofrequency energy delivered via a catheter is used to create small localised areas of scar.

In AVNRT, the slow pathway is targeted with the aim of modifying conduction so that re-entrant tachycardia can no longer be sustained. Both acute and long term success rates for this procedure are high. In a large observational study acute success was achieved in 98% of cases.[31] A meta-analysis of 10 observational studies comprising 1204 patients reported a 4.3% recurrence rate.[32] Serious complications are uncommon, the most serious being atrioventricular block requiring pacemaker therapy (affecting 1% of patients in early series).[31 33 34]

AVRT is also amenable to catheter ablation. No randomised prospective studies have been conducted, but observational studies and registries have observed acute success rates of more than 95% and recurrence rates less than 5%.[33 35] Atrioventricular block is a risk in cases where the accessory pathway is close to the atrioventricular node and the His bundle; the use of cryothermal energy may reduce this risk.[36] Other complications are reported to occur in less than 2-3% of patients and include vascular injury, bleeding, venous thrombosis, pulmonary embolism, myocardial perforation, systemic embolism (in the case of a left sided accessory pathway), and rarely, death (0-0.2%).[33-35]

Focal atrial tachycardia can also be successfully treated with catheter ablation, although randomised control trials are lacking, and evidence is limited to small observational studies. Acute success rates of 85%[37 38] with recurrence rates of 8% have been reported.[39]

For re-entrant atrial tachycardias, radiofrequency ablation has high success rates and is often used as first line treatment.[5 40] Radiofrequency ablation is the treatment of choice for typical atrial flutter. This practice is supported by the findings from a medium sized randomised study which found that, compared with medical treatment, as first line treatment ablation produced higher rates of sinus rhythm (80% *v* 36%), fewer hospital admissions, and a lower occurrence of atrial fibrillation.[40]

After the procedure, most patients can return to their normal activities very quickly. We recommend they avoid heavy lifting for two weeks after the procedure. In the UK, the Driver and Vehicle Licensing Agency states that car drivers (group 1

Box 2 Which patients should you refer to an electrophysiologist?

Urgent referral

Patients with:

- Syncope with palpitations on exertion
- Broad complex tachycardia
- Pre-excitation (delta wave) on a 12 lead ECG
- Structural heart disease
- Severe symptoms

Routine referral

Those with:

- Drug resistance or intolerance
- Preference not to take medication
- Diagnostic uncertainty

entitlement) may not drive for two days after successful catheter ablation (vocational drivers (group 2) for two weeks afterwards) if the arrhythmia was not or did not have the potential to be incapacitating. If the arrhythmia was or had the potential to be incapacitating, the restriction is six weeks after successful ablation.[41]

Contributors: All authors contributed fully to all stages of the planning, researching, and preparation of the manuscript. DWD and ZIW are the guarantors.

Competing interests: All authors have completed the ICMJE uniform disclosure form at www.icmje.org/coi_disclosure.pdf (available on request from the corresponding author) and declare: SMAS is funded by the British Heart Foundation; no financial relationships with any organisations that might have an interest in the submitted work in the previous three years; no other relationships or activities that could appear to have influenced the submitted work.

Provenance and peer review: Commissioned; externally peer reviewed.

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Tips for non-specialists

- If a patient presents with tachycardia always record an ECG and give the patient a copy; this can be helpful for future management
- Broad complex tachycardia is ventricular tachycardia until proved otherwise
- For narrow complex tachycardia, the first step is usually to slow AV node conduction with either a Valsalva manoeuvre or adenosine. This provides diagnostic information and will usually terminate AVNRT and AVRT
- For patients with recurring symptoms, catheter ablation has high acute and long term success rates

Additional educational resources

For healthcare professionals

- The Resuscitation Council provides free algorithms for the acute management of tachycardia: www.resus.org.uk/pages/glalgos.htm
- ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias. 2003. www.escardio.org/guidelines-surveys/esc-guidelines/Pages/supraventricular-arrhythmias.aspx
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For patients (free web based resources)

- Arrhythmia Alliance (www.heartrhythmcharity.org.uk)—The patient pages give information on the full range of rhythm disturbances and treatments available, and downloadable single sheet summaries for different arrhythmias
- British Heart Foundation (www.bhf.org.uk)—The "Heart Health" pages give information on different cardiac conditions and investigations including electrophysiology studies; telephone advice line (Heart Helpline) is also available.
- NHS Choices (www.nhs.uk/conditions/supraventricular-tachycardia)—Simple overview of supraventricular tachycardia
- Patient.co.uk website (www.patient.co.uk/health/Supraventricular-Tachycardia-(SVT).htm)—Brief overview of SVT in lay terms

Questions for future research

- Uncertainty remains about the optimal management of patients with asymptomatic pre-excitation (delta wave on the ECG)—in particular, whether sufficiently sensitive risk stratification is possible or whether most patients should be managed with catheter ablation of their accessory pathway
- There is ongoing development of invasive and non-invasive methods for mapping complex arrhythmias, such as atrial tachycardias occurring after ablation for atrial fibrillation. These have the potential to help with diagnosis and to guide the catheter ablation procedure

40 Natale A, Newby KH, Pisanó E, Leonelli F, Fanelli R, Potenza D, et al. Prospective randomized comparison of antiarrhythmic therapy versus first-line radiofrequency ablation in patients with atrial flutter. *J Am Coll Cardiol* 2000;35:1898-904.

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Accepted: 12 November 2012

Cite this as: *BMJ* 2012;345:e7769

Figures

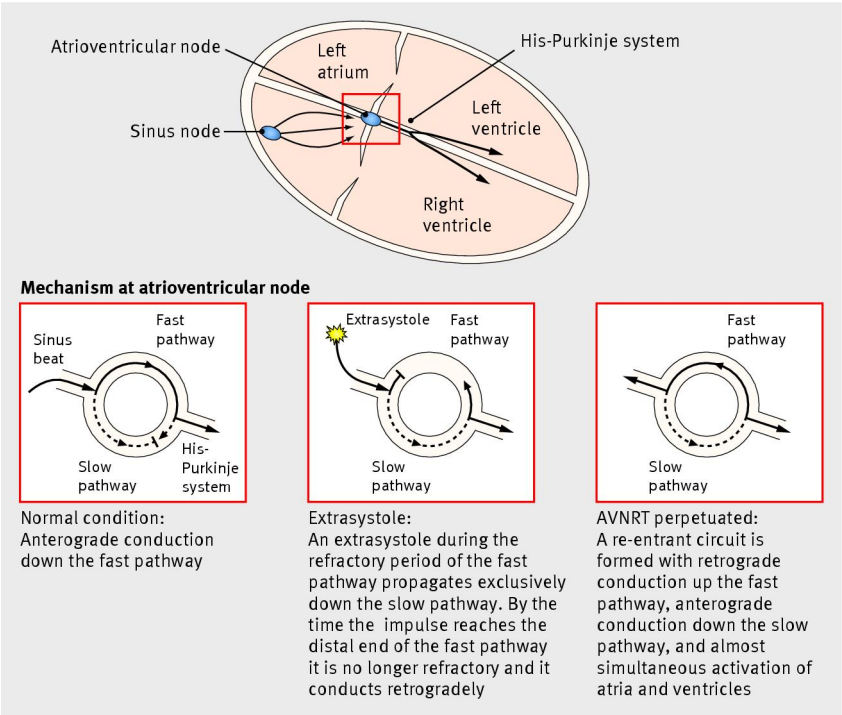

Fig 1 Mechanism for atrioventricular nodal re-entry tachycardia

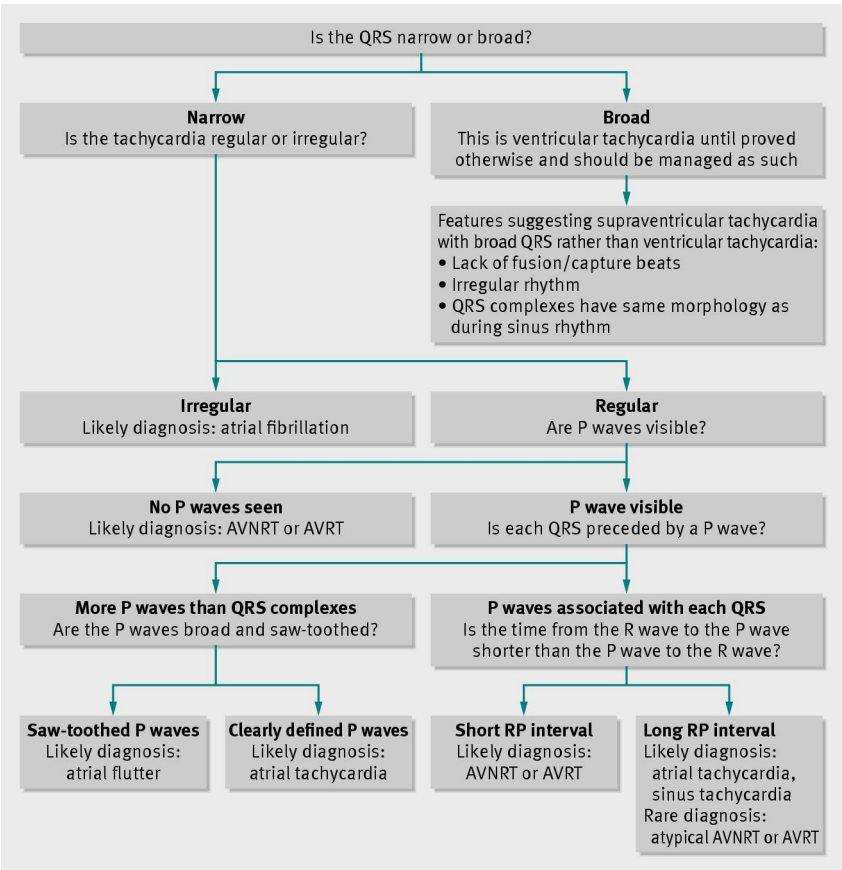

Fig 2 Interpretation of ECG showing tachycardia

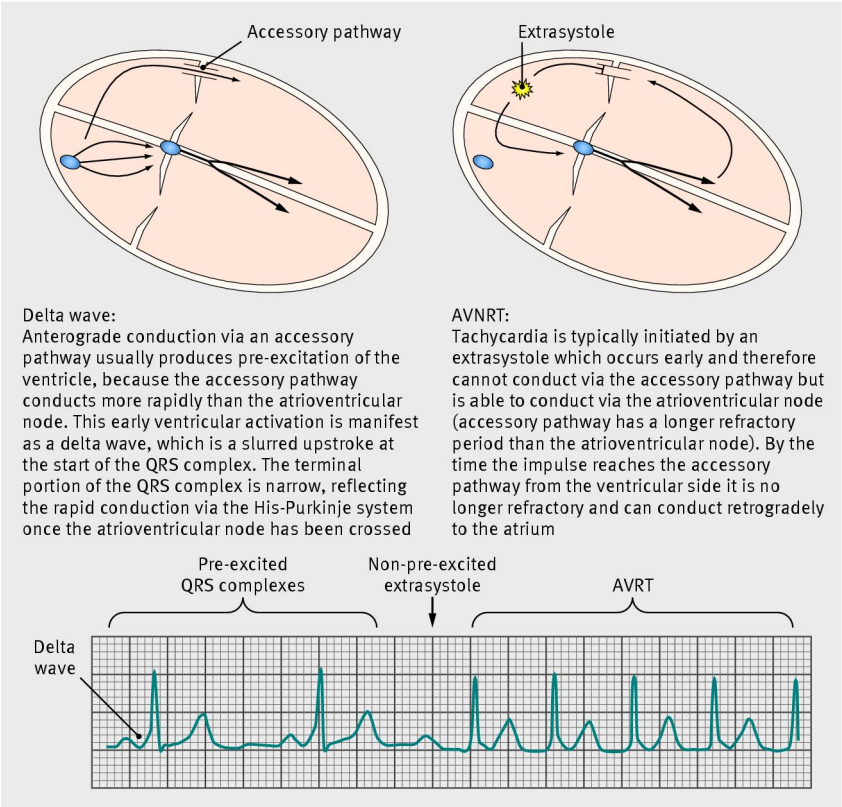

Fig 3 Mechanism for atrioventricular re-entry tachycardia

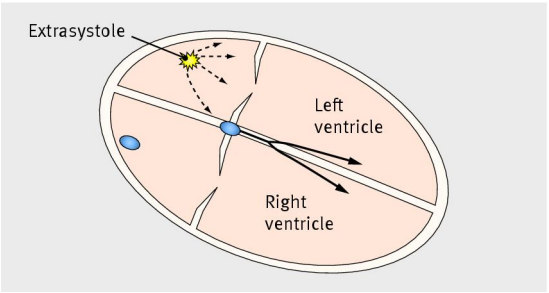

Fig 4 Mechanism for focal atrial tachycardia. Generation of rapid electrical impulses from a small localised area in the atria (in this example, the left atrium)

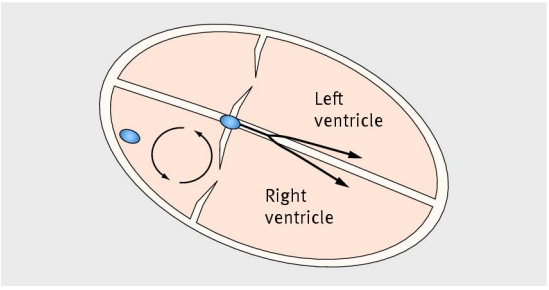

Fig 5 Mechanism for atrial flutter, an example of a macro re-entrant circuit. The electrical activation circles the right atrium

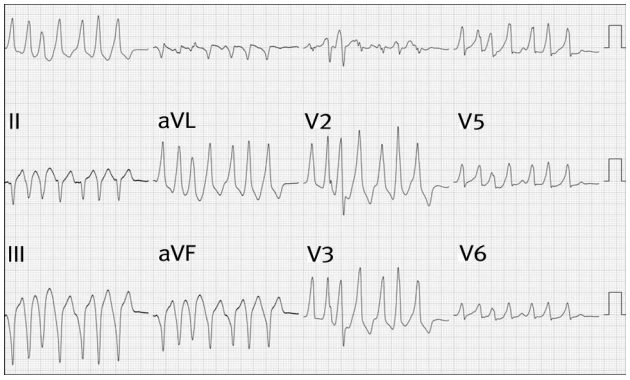

Fig 6 Pre-excited atrial fibrillation

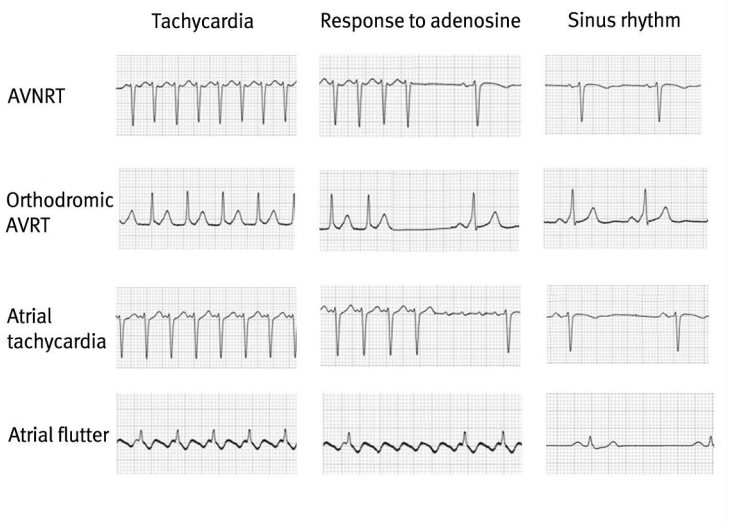

Fig 7 Typical electrocardiographic recordings for four common SVTs during tachycardia, administration of adenosine, and sinus rhythm. AVNRT: As atrial and ventricular activation occurs virtually simultaneously in typical AVNRT, the retrograde P waves are either hidden in the QRS complex or produce a pseudo r' in V1. Tachycardia is usually terminated with adenosine. Orthodromic AVRT: During tachycardia ventricular activation occurs exclusively via the atrioventricular node and His-Purkinje system; tachycardia is therefore narrow complex. Typically, the RP interval is short; less commonly, if the accessory pathway conducts slowly, a long RP interval is observed. Adenosine usually terminates the tachycardia and in sinus rhythm a delta wave is commonly seen. Atrial tachycardia: Often the RP interval is long. Tachycardia is not dependent on the atrioventricular node so it usually continues even after the administration of adenosine (though adenosine may terminate focal atrial tachycardia). When atrioventricular conduction is blocked the underlying P waves can be clearly seen. Atrial flutter: The characteristic flutter waves are seen clearly after administration of adenosine, and tachycardia continues because it does not depend on the atrioventricular node.