

Diagnosing patients with su

AUTHORS
Dr Malini Govindan
MBBS, FRACP
research fellow

Professor A John Camm
BSc MD FRCP
Professor of Clinical
Cardiology, St George's
Hospital, London

What
investigations
should be
carried out?

What are
the treatment
options?

Which patients
should be
referred?



**THE MECHANISMS OF
CARDIAC ARRHYTHMIAS
ARE COMPLEX AND
RECOGNISING THESE**

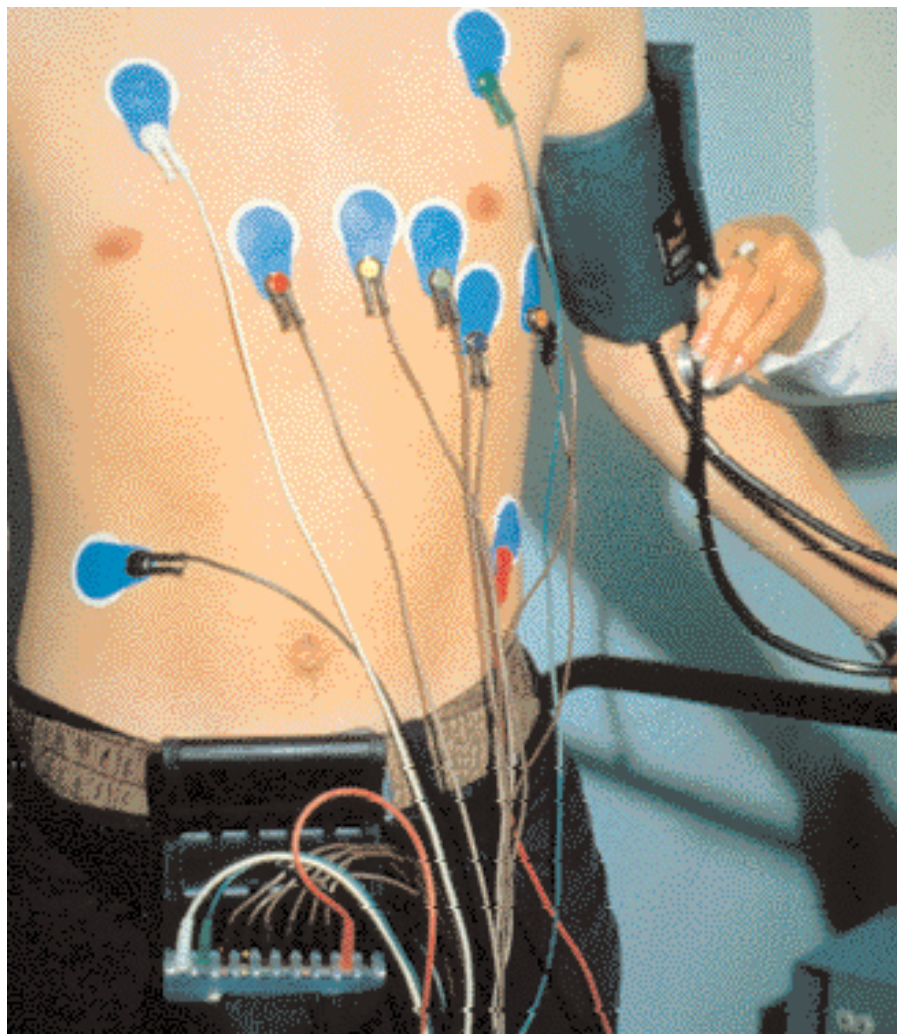
conditions can be difficult. When an arrhythmia is suspected, prompt recording of an ECG is vital, even if symptoms have subsided, as this may be useful for future reference, diagnosis and therapy.

Supraventricular tachycardias (SVTs) occur in 1% of the population.¹ They often develop in childhood but tend to have clinical presentations in early adulthood. Atrioventricular nodal re-entry tachycardia (AVNRT) is the most common type of SVT, followed by atrioventricular re-entrant tachycardia (AVRT).

Extensive research and improved understanding of the basic mechanisms of these conditions has led to improvements in diagnosis and management.

AVNRT

AVNRT accounts for 60% of patients presenting with a narrow complex tachycardia.² It occurs mainly in young adults, particularly women. Most of these patients have structurally normal hearts. However, some patients with AVNRT may have associated mitral valve disease, pericarditis, myocardial



ischaemia or congenital heart anomalies.³

An appreciation of the electrophysiology of the AV nodal tissue is fundamental to understanding the mechanisms of AVNRT. See figure 1, right.

AVRT

AVRT is an accessory pathway-mediated tachycardia. The incidence of AVRT in the general population is 0.1–0.3% and it accounts for 30% of all SVTs.⁵ AVRT is twice as common in men than women and tends to present at a younger age than AVNRT.

Most patients with AVRT do not have structural heart disease. However, it is associated with Ebstein's anomaly and a higher prevalence of 0.55% has

been reported in first-degree relatives of patients with accessory pathways. See figure 2, p42.⁵

WOLFF-PARKINSON-WHITE SYNDROME

The most commonly described accessory pathway is that of Wolff-Parkinson-White syndrome (WPW). WPW should only be diagnosed in patients with evidence of pre-excitation and tachycardias. AVRT is the most common arrhythmia in patients with WPW and accounts for 95% of re-entry tachycardias.

Atrial fibrillation

About one-third of patients with bypass tracts such as in WPW have

paraventricular tachycardia

a propensity to develop dangerous atrial arrhythmias, including paroxysmal atrial flutter and fibrillation. Unlike the AV node, the accessory pathways have rapid nondecremental conduction and result in very rapid rates, which can degenerate into ventricular fibrillation and sudden cardiac death.⁴ As a result, patients with known pre-excitation syndromes who develop atrial fibrillation should not be administered AV nodal-blocking agents, as these may accelerate conduction down the accessory pathway and result in dangerous ventricular arrhythmias, see figure 3, p45.⁶

Patients with WPW have around a 0.1% risk of sudden arrhythmic death.⁷

DIAGNOSIS

The unpredictable nature of SVTs can present a diagnostic challenge. It is difficult to determine when an attack will occur, how long it will persist and when it will terminate. These problems can lead to some arrhythmias going undetected or undiagnosed, particularly if symptoms have abated before an examination is performed or an ECG can be recorded.

AVNRT and AVRT are characterised by abrupt onset and termination. In the majority of cases they are well tolerated in the absence of structural heart disease. However, the possibility of a bypass tract must be considered as a mechanism of SVT as, paradoxically, treatment with AV nodal-blocking agents or digoxin can cause haemodynamic instability and potentially fatal ventricular arrhythmias.

Symptoms of AVNRT and AVRT may be indistinguishable (see table 1, p45). They include palpitations, lightheadedness and shortness of breath. Syncope occurs more commonly in patients with accessory pathways as rapid ventricular rates may cause inadequate cerebral perfusion.

The diagnosis and management of these arrhythmias is by review of 12-lead ECG and ambulatory recordings with correlation to the clinical setting. Continuous ECG recordings with Holter monitors, event recorders or implantable loop recorders can be carried out in the outpatient setting.

It often takes several episodes before the arrhythmia can be documented and, rarely, the arrhythmia may be elusive enough to escape recording.

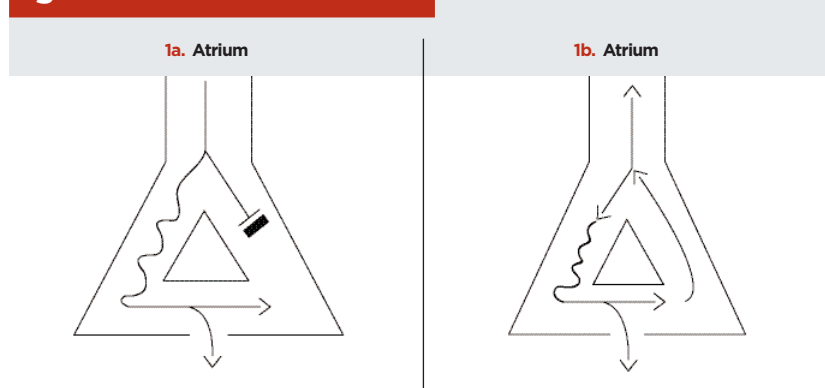
Usually, SVTs are documented by an ECG showing the presence of a regular narrow complex tachycardia, with a heart rate of about 150-250bpm (see figure 4, p46).

Distinguishing AVNRT from AVRT is not straightforward, although the surface ECG may provide some useful clues as to the diagnosis. Antegrade conduction in typical AVNRT is relatively slow and retrograde

conduction is faster. Therefore the atria are often depolarised at the same time as the ventricles, causing the P-wave to be partially or completely hidden in the QRS complex (see figure 4, p46). This is a reliable sign of typical AVNRT.⁸

Furthermore, the arrhythmia may terminate with a retrograde P-wave, which suggests the site of termination is within the AV node. Following termination, a very brief period of asystole may occur, as the sinus node has been suppressed for the duration of the tachycardia and may take a few seconds to recover as the dominant pacemaker. »

Figure 1. Mechanism of AVNRT



In the normal heart, the AV node has a *single* conducting pathway. This transmits impulses from the atria and conducts them in an antegrade manner to the ventricle via the His bundle, with the inherent physiological delay over the AV node.

In AVNRT, the substrate for re-entry depends on the presence of *two* distinct AV nodal pathways. The normal conducting pathway is often thought of as the fast pathway; it conducts the electrical impulse rapidly but has a long refractory period. The slow pathway conducts the impulse more slowly but has a short refractory period.³

Many patients may have dual pathway physiology on electrophysiological testing. However, not all will develop AVNRT. For AVNRT to develop, one pathway must have sufficiently slower conduction than the other, in order

to allow a re-entry circuit to be formed. Only 20% of patients with dual pathways will actually develop clinical arrhythmias.

AVNRT is triggered by a premature impulse. If a premature atrial impulse is discharged, it will be unable to conduct via the fast pathway, which will still be refractory from the previous impulse, and hence will be directed down the slow pathway in an antegrade manner (figure 1a). Depending on the timing of the impulse, this may then be conducted retrogradely back to the atria if the fast pathway has recovered, thereby initiating the re-entry circuit for AVNRT (figure 1b). Therefore the common AVNRT circuit comprises an antegrade slow pathway and a retrograde fast pathway: the 'slow-fast' pathway.^{3,4}

Importantly, in AVNRT the ventricles are not involved in the re-entry circuit.

In the atypical form of AVNRT, the P-wave may be clearly visible *before* the QRS complex. In such cases, the usual functional circuit is reversed, with antegrade conduction down the fast pathway and retrograde conduction over the slow pathway, resulting in delayed atrial activation in relation to the QRS complex. These are often referred to as 'fast-slow' AVNRTs (see figure 5, p46).⁹

In cases of AVRT with accessory pathways, the ECG may reveal delta waves of pre-excitation or a short PR interval. However, many bypass tracts are concealed and only conduct from ventricle to atrium and evidence of pre-excitation on the surface ECG will be absent. The relationship of the P-wave to the QRS complex may aid diagnosis.

In more resistant cases, stress testing may be used as an ancillary test to reproduce the suspected transient arrhythmia. Alternatively,

electrophysiology studies can be used if patients have recurrent symptoms but ECG recordings have failed to document the nature of the arrhythmia. They may also be helpful in identifying the nature of the underlying pathway and its exact anatomical location to determine the best method of treatment, map the re-entrant circuit and plan for ablation.

MANAGEMENT

Initial therapy for an acute episode of a narrow complex tachycardia includes vagal manoeuvres, such as carotid sinus massage and the Valsalva manoeuvre. These can be safely performed in general practice and may increase vagal tone to terminate the tachycardia. Vagal manoeuvres should not be attempted in patients with hypotension.

If these measures are unsuccessful and the patient is symptomatic but

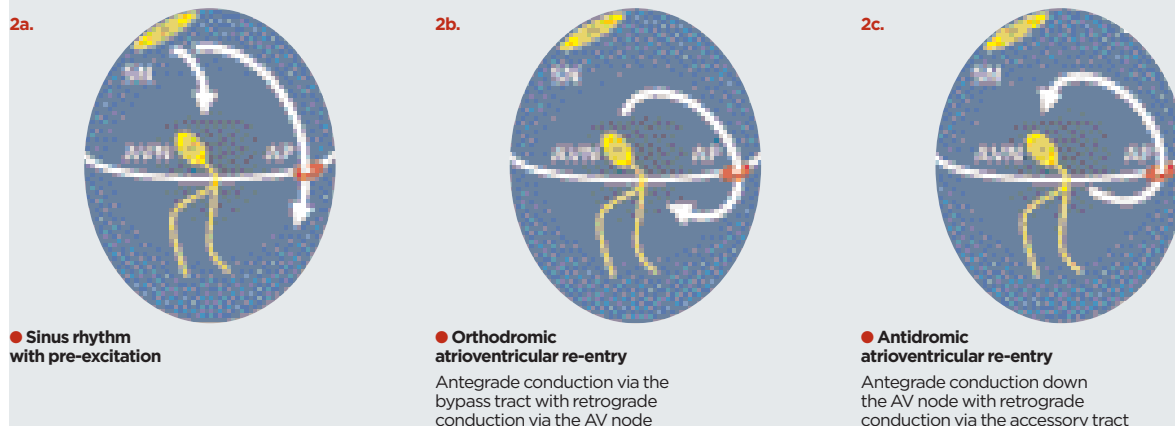
stable, drugs can be used to terminate the tachycardia. These include calcium-channel blockers (verapamil or diltiazem), beta-blockers (atenolol, metoprolol or bisoprolol) or digoxin, providing that there is no evidence of pre-excitation on the surface ECG. Indications for referral to a cardiologist are listed in table 1, p45.

Patients with any evidence of haemodynamic instability or severe prolonged symptoms should be referred to the emergency department for monitoring and administration of iv bolus doses of adenosine. Short-acting beta-blockers or calcium-channel blockers may also be given. Immediate termination can also be achieved with direct current cardioversion, although this is rarely required.

AVNRT

Preventative therapy for AVNRT may be required in patients with frequent, prolonged or highly symptomatic >>

Figure 2. Mechanism of AVRT



AVRT is the result of two distinct pathways: one involving the AV node, the other an accessory pathway or bypass tract. These are anomalous pathways connecting the atrial and ventricular myocardium and are capable of conducting in an antegrade or retrograde direction, or both. AVRT is distinct from AVNRT as these pathways do not share the same decremental physiology of the AV node and hence have the propensity to conduct at very rapid rates.

These accessory pathways cause an activation wavefront that is separate from that resulting from conduction over the normal AV nodal-His bundle pathway (see figure 2a). When an

impulse is generated, it travels antegradely down two pathways: the accessory pathway and the normal pathway. The fusion of the two independent wavefronts causes an abnormal QRS complex, with slurring of the upstroke known as the delta wave. These delta waves, along with a short PR interval, are signs of pre-excitation shown on the surface ECG and are often associated with symptomatic tachycardias.

It is important to note that not all bypass tracts conduct in an antegrade manner during sinus rhythm; these tracts are referred to as concealed pathways and are only capable of retrograde conduction. Patients with concealed pathways will have a normal

ECG during sinus rhythm without the appearance of delta waves.

In patients with concealed tracts, the impulse conducts antegradely through the AV node and retrogradely via the accessory pathway, producing an orthodromic AVRT (see figure 2b), while antidromic AVRT involves antegrade conduction through the bypass tract and retrograde conduction through the AV node (see figure 2c).

Orthodromic AVRT is associated with a narrow complex tachycardia, while antidromic AVRT can produce a broad complex tachycardia but is only seen in 5-10% of patients with WPW.⁴

episodes that do not resolve spontaneously or are difficult to terminate. Similar drugs are used for prevention of recurrence as for acute episodes.

Radiofrequency catheter ablation of the slow pathway may be considered for some patients. Indications for ablation depend on clinical judgement and patient preference. Factors that may contribute to referral for ablation include:

- The duration and frequency of tachycardia
- Tolerance of symptoms
- Efficacy and tolerance of antiarrhythmic drugs
- The need for lifelong drug therapy
- The presence of concomitant structural heart disease.

Success rates for ablation procedures approach 95%, while drug efficacy is in the range of 30–50%.⁶ Catheter ablation is preferred to long-term drug therapy and may be offered as an initial therapy in cases such as athletes, pilots, drivers, people who operate dangerous machinery or women planning pregnancy. However, patients undergoing radiofrequency ablation must be advised that there is a 1% risk of AV block, requiring permanent pacemaker implantation.^{5,10}

AVRT or pre-excitation

Patients with AVRT or pre-excitation may be more difficult to treat. Accessory pathway conduction is not affected by calcium-channel blockers or beta-blockers and digoxin is contraindicated in patients with WPW.

Patients with ECG evidence of pre-excitation or a family history of bypass tracts or sudden death should be referred to a cardiologist or

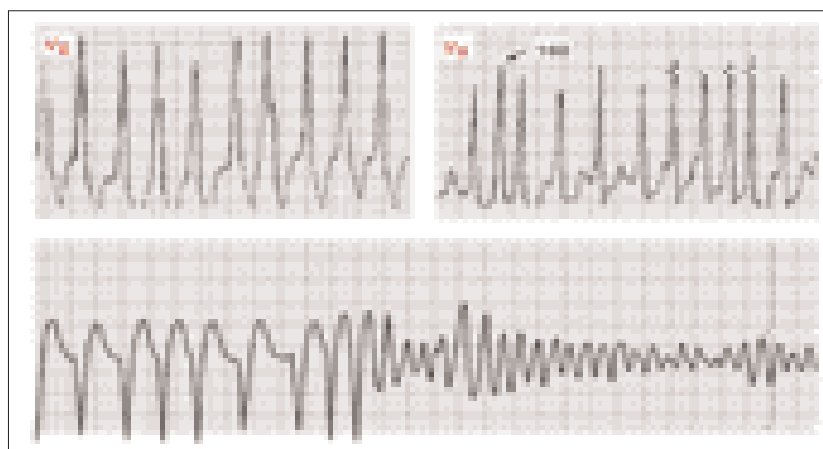


FIGURE 3

Atrial fibrillation in Wolff-Parkinson-White syndrome. Broad complex atrial fibrillation with 1:1 conduction via the bypass tract and a rapid ventricular rate which subsequently degenerates to ventricular fibrillation

electrophysiologist for further evaluation. Drugs blocking conduction in accessory pathways include class Ia (procainamide) and class Ic drugs (flecainide, propafenone). These are indicated if drug therapy becomes necessary for bypass-mediated tachycardias. In patients with WPW who develop atrial fibrillation and ventricular tachycardia cannot be excluded, the drugs of choice include amiodarone or class Ia drugs such as procainamide.

Long-term medical therapy for the prevention of recurrence usually involves combination therapy with an AV nodal-blocking agent and a class Ic (flecainide) or class III (amiodarone) drug. Class Ic drugs should be avoided if structural heart disease is present.

Unfortunately, drug therapy is poorly tolerated and can be unpredictable and variable in its response. Therefore patients with bypass tracts and symptomatic

tachycardias (WPW) should undergo catheter ablation as first-line therapy, which has a success rate of 85–90%. The NASPE prospective catheter ablation registry reported an overall incidence of 1% for AV block.¹⁰ This tends to occur with attempted ablation of septal pathways as these are located close to the AV junction. These data have been confirmed by others.^{11,12} »

Table 2

Indications for referral to a cardiologist

- Recurrent regular symptomatic SVT that has been unsuccessfully treated with one type of medication
- Patients with recurrent symptomatic SVT who are reluctant to take drug therapy
- Referral for ablation of SVT
- Recurrent palpitations and unclear diagnosis
- Recurrent syncope associated with palpitations
- Syncope and pre-excitation
- WPW syndrome or asymptomatic pre-excitation
- Pre-excited atrial fibrillation

Table 1

Summary: diagnosis and investigation of SVTs

Symptoms	Physical findings	ECG findings	Echo	Electrophysiology study
● Abrupt onset and termination ● Episodic, lasting seconds to days ● Palpitations ● Lightheadedness ● Shortness of breath ● Angina (especially if underlying coronary artery disease is present) ● Syncope (if ventricular rate is rapid and associated with hypotension)	● Rapid heart rate (range from 150–250bpm) ● Blood pressure usually normal but hypotension can occur with very rapid rates ● Signs of cardiac failure in patients with left ventricular dysfunction ● Diaphoresis	● Narrow complex regular tachycardia (unless conduction abnormality coexists) ● QRS rate 150–250bpm ● P-waves not usually visible in AVNRT (buried in the QRS) ● Abrupt termination may occur with a retrograde P-wave or bradycardia ● In typical AVNRT the PR>RP interval ● Evidence of pre-excitation on the ECG (delta wave)	● Structural heart disease ● Congenital anomalies (Ebstein's anomaly) ● Left ventricular function ● Associated conditions such as hypertrophic cardiomyopathy	● Induction of the tachycardia for diagnosis ● Demonstration of dual AV nodal pathways ● Determine the presence of an accessory pathway ● If an accessory pathway is present to determine the risk of threatening arrhythmias ● Mapping of the arrhythmia circuit ● Ablation of the circuit if required

key points

SELECTED BY

Dr Peter Saul

GP, Wrexham and associate GP Dean for North Wales

AVNRT accounts for 60% of patients presenting with a narrow complex tachycardia. It occurs mainly in young adults, particularly women. In AVNRT, the substrate for re-entry depends on the presence of two distinct AV nodal pathways. The 'normal' pathway is fast but recovers slowly, the aberrant pathway is slow but recovers quickly. If a premature impulse is discharged, it will be unable to conduct via the fast pathway and hence will be directed down the slow pathway. Depending on the timing of the impulse this may then be conducted retrogradely back to the atria if the fast pathway has recovered.

AVRT is an accessory pathway-mediated tachycardia and accounts for 30% of all SVTs. AVRT is twice as common in men than women and tends to present at a younger age than AVNRT. In AVRT there are anomalous pathways connecting atrial to ventricular myocardium, which bypass the AV node and conduct at very rapid rates. The most commonly described accessory pathway is that of Wolff-Parkinson-White syndrome (WPW). About one-third of patients with bypass tracts such as in WPW have a propensity to develop dangerous atrial arrhythmias, including paroxysmal atrial flutter and fibrillation.

The unpredictable nature of SVTs can present a diagnostic challenge. It is difficult to determine when an attack will occur, how long it will persist and when it will terminate.

The diagnosis and management of these arrhythmias is by review of 12-lead ECG and ambulatory recordings with correlation to the clinical setting. Continuous ECG recordings with Holter monitors, event recorders or implantable loop recorders can be carried out in the outpatient setting. Usually, SVTs are documented by an ECG showing the presence of a regular narrow complex tachycardia, with a heart rate of about 150–250bpm.

Initial therapy for an acute episode of a narrow complex tachycardia includes vagal manoeuvres, such as carotid sinus massage and the Valsalva manoeuvre. If these measures are unsuccessful and the patient is symptomatic but stable, drugs can be used to terminate the tachycardia. These include calcium-channel blockers (verapamil or diltiazem), beta-blockers (atenolol, metoprolol or bisoprolol) or digoxin, providing that there is no evidence of pre-excitation on the surface ECG.

Treatment for AVRT is more difficult. Drugs blocking conduction in accessory pathways include class Ia (procainamide) and class Ic drugs (flecainide, propafenone) but radiofrequency ablation is now the first-line treatment.

Patients with any evidence of haemodynamic instability or severe prolonged symptoms should be referred acutely for monitoring and treatment.

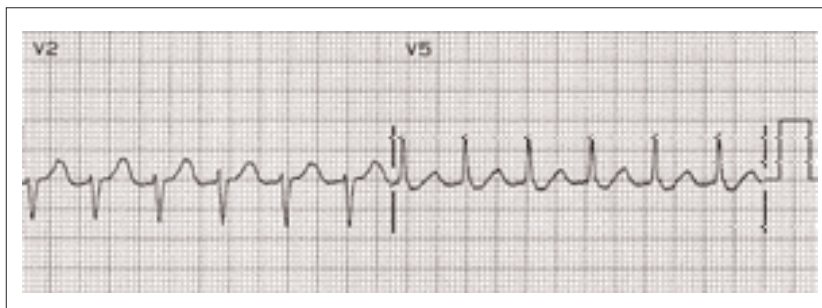


FIGURE 4 (ABOVE)
AVNRT. Narrow complex regular tachycardia. Q waves are buried in the end of the QRS complexes, causing a short RP interval

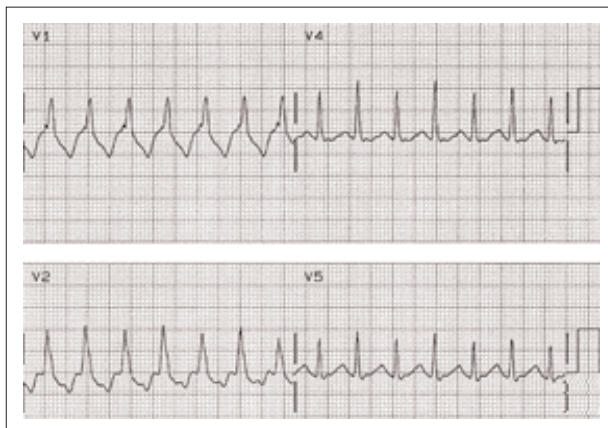


FIGURE 5 (LEFT)
Atypical AVNRT. Here the P waves actually occur at the onset of the T wave

Patients with SVT and infrequent symptoms who do not have evidence of pre-excitation can be treated with a variety of approaches based on patient preference. These arrhythmias are rarely dangerous. Catheter ablation has sufficient efficacy and low risk to be used for symptomatic patients, either as initial therapy or for patients experiencing side-effects or arrhythmia recurrence during drug therapy.

CONCLUSIONS

AVNRT and AVRT are common, mostly stable cardiac arrhythmias that can be clinically difficult to distinguish. Routine ECG assessment will provide clues to the diagnosis. Simple manoeuvres can terminate these arrhythmias and patients with recurrent symptoms or more complex presentations can be referred to a specialist to help elucidate the underlying mechanisms and determine appropriate treatment.

Systems are being developed for more urgent referral of patients with sustained or compromising arrhythmias. The development of rapid access multidisciplinary arrhythmia clinics will allow access to expert opinion for initial assessment and formulation of a care plan. This should improve long-term management in primary care and reduce hospital admissions. ●

REFERENCES

- 1 Monahan KM, Josephson ME. Recent advances in AVNRT: mechanisms and therapy. *Card Electrophysiol Rev* 1997;1:434–8
- 2 Department of Health. *National Service Framework for Coronary Heart Disease – arrhythmias and sudden cardiac death*. London:DH 2007
- 3 Fogoros RN. *Electrophysiologic Testing (Practical Cardiac Diagnosis)*. 3rd ed. London:Blackwell Science 1999
- 4 Ward DE, Camm AJ. *Clinical Electrophysiology of the Heart*. 1st ed. London:Edward Arnold 1987
- 5 Blomström-Lundqvist C, Scheinman MM, Aliot EM et al. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias – executive summary: a report of the American college of cardiology/American heart association task force on practice guidelines and the European society of cardiology committee for practice guidelines (writing committee to develop guidelines for the management of patients with supraventricular arrhythmias). Developed in collaboration with NASPE-Heart Rhythm Society. *J Am Coll Cardiol* 2003;42:1493–531
- 6 Calkins H, Sousa J, el-Atassi R, et al. Diagnosis and cure of the Wolff-Parkinson-White syndrome or paroxysmal supraventricular tachycardias during a single electrophysiologic test. *N Engl J Med* 1991;324:1612–8
- 7 Munger TM, Packer DL, Hammill SC et al. A population study of the natural history of Wolff-Parkinson-White syndrome in Olmsted County, Minnesota, 1953–1989. *Circulation* 1993;87:866–73
- 8 Kumar UN, Rao RK, Scheinman MM. The 12-lead ECG in supraventricular tachycardia. *Cardiol Clin* 2006;24:427–37
- 9 Zipes DP. Clinical application of the electrocardiogram. *J Am Coll Cardiol* 2000;36:1746–8
- 10 Scheinman MM, Huang S. The 1998 NASPE prospective catheter ablation registry. *Pacing Clin Electrophysiol* 2000;23:1020–8
- 11 Clague JR, Dagues N, Kotthamp H et al. Targeting the slow pathway for atrioventricular nodal reentrant tachycardia: initial results and long-term follow-up in 379 consecutive patients. *Eur Heart J* 2001;22:82–8
- 12 Calkins H, Yong P, Miller JM et al. Catheter ablation of accessory pathways, atrioventricular nodal reentrant tachycardia, and the atrioventricular junction: final results of a prospective, multicenter clinical trial. The Atakr Multicenter Investigators Group. *Circulation* 1999;99:262–70