

A narrow complex tachycardia

A 42-year-old woman presented to the accident and emergency department with a 3-month history of recurrent palpitations and breathlessness. She was otherwise fit and well and taking no regular medication. What are the abnormalities on the presenting electrocardiogram (ECG) in *Figure 1* and what is the diagnosis?

Discussion

The ECG demonstrates a regular narrow complex tachycardia at 215/min. The axis is normal. The differential diagnosis lies between atrioventricular node re-entrant tachycardia (AVNRT), orthodromic re-entrant tachycardia (ORT) and atrial tachycardia. In the adult population 60% of supraventricular tachycardias (SVTs) are the result of AVNRT. Accessory pathway-mediated atrioventricular re-entrant tachycardia (AVRT) represents 30% of SVT and 10% of SVTs are atrial tachycardias in this age group. Epidemiological studies have found an incidence of regular AVNRT or AVRT of 35/100 000 person-years and a prevalence of 2.29/100 000 persons.

The majority of AVRTs conduct antegradely through the atrioventricular node (AVN) and retrogradely through the accessory pathway (ORT) and are thus narrow complex tachycardias (*Figure 2a*). This contrasts with antidromic AVRT which conduct antegradely through the pathway and retrogradely through the AVN (*Figure 2b*). Antidromic AVRT is usually a broad complex arrhythmia with a delta wave visible on surface ECG during tachycardia, demonstrating that the antegrade limb is via an accessory pathway. In this case, the diagnosis of AVNRT was confirmed at electrophysiological study. There are a number of clues in the history and on the surface ECG which point to this diagnosis.

Presentation

AVNRT is more common in women than men and tends to present in young to middle-aged adults. The mean age of patients

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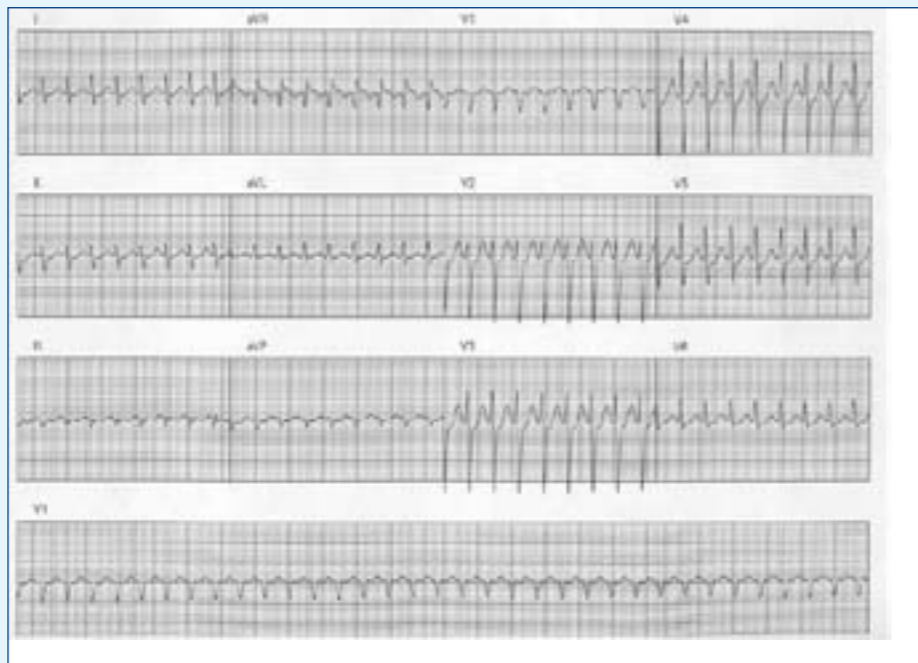


Figure 1. 12-lead electrocardiogram at presentation.

referred for catheter ablation of this arrhythmia is 45 years. The tachycardia rate ranges between 140 and 240 beats/min.

P wave relationships and morphology

The identification of the P wave is often the key to determining the tachycardia diagnosis. Close inspection of lead V1 demonstrates a retrograde P wave lying 70 msec after the onset of the QRS and negative P waves in the inferior leads. In typical AVNRT the retrograde P wave

is either hidden in the QRS complex or appears immediately after it as a pseudo R wave (*Figure 3a* and *b*). Therefore, the more common form of AVNRT (which uses a slowly conducting antegrade limb in the AVN and rapidly conducting retrograde limb – known as the ‘fast’ AVN pathway) is a short RP tachycardia (*Figure 4*). The RP interval is longer than the subsequent PR interval. The other differential of a short RP tachycardia in this age group is ORT with a postero-septal accessory pathway but a further clue to

Figure 2. Schematic representation of (a) orthodromic and (b) antidromic re-entrant accessory pathway (AP) mediated tachycardia. The terms orthodromic and antidromic refer to the direction of atrioventricular node activation, orthodromic being the normal direction of activation in sinus rhythm.

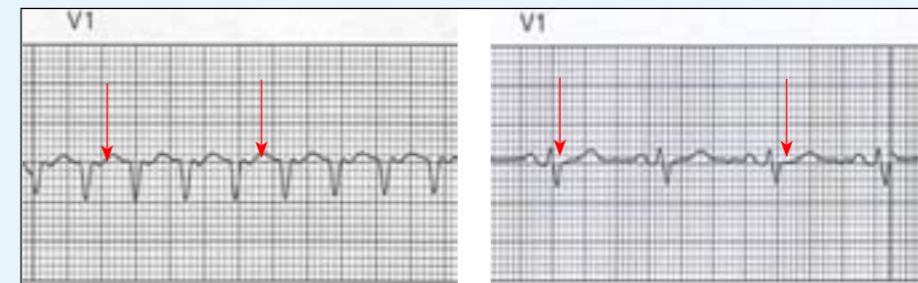
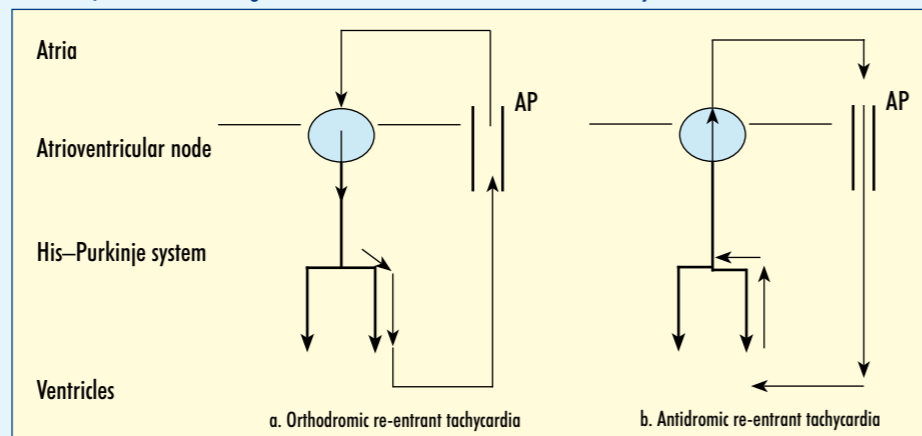


Figure 3a. Lead V1 during tachycardia illustrating retrograde P waves (RP < PR interval), *b.* Lead V1 in sinus rhythm. Note the absence of the retrograde P wave indicating that the deflection in the ST segment during tachycardia was not simply a repolarization abnormality.

diagnosis may be obtained if one looks carefully for bundle-branch block (aberrancy) during tachycardia.

The effect of bundle-branch block

If the patient develops functional bundle-branch block during the tachycardia one should be careful to measure the RR intervals. An increased RR interval during bundle-branch block *vs* normal conduction indicates an accessory pathway-mediated tachycardia involving the His-Purkinje system as opposed to AVNRT or atrial tachycardia (AT) if the

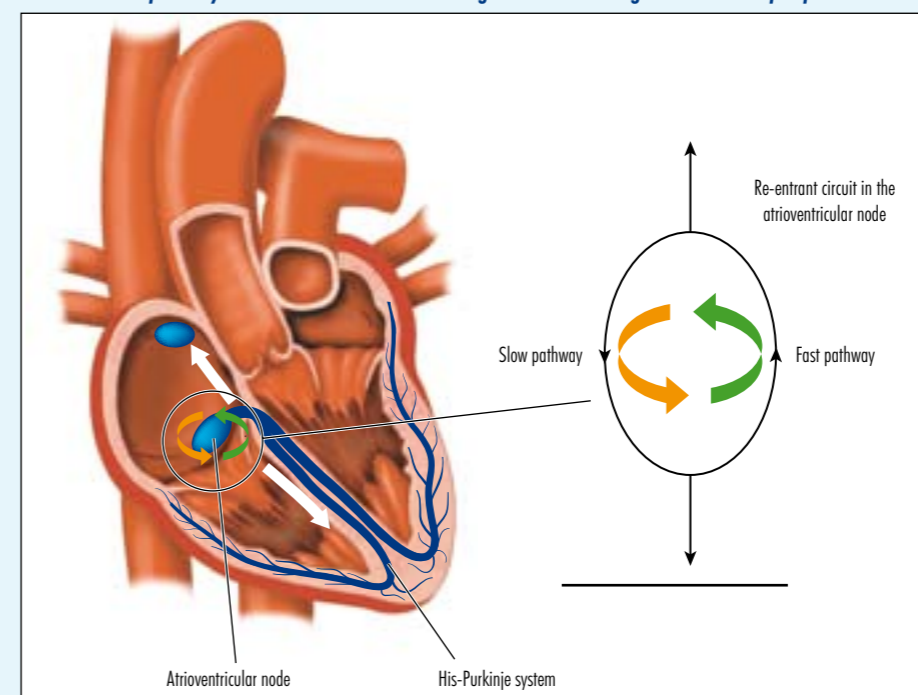
bundle-branch block is on the same side of the heart as the accessory pathway.

Onset and termination of tachycardia

AVNRT is an AVN-dependent tachycardia and therefore the diagnosis may become evident at onset or termination. The onset of tachycardia is often preceded by a premature atrial beat that results in sudden prolongation of the PR interval as conduction blocks in the fast pathway and proceeds down the slow pathway (*Figure 4*).

When spontaneous termination occurs and the P waves are easily identified, the

Figure 4. The circuit in typical atrioventricular node re-entrant tachycardia (AVNRT) illustrating almost simultaneous activation of the atria and ventricles as a result of re-entry in the atrioventricular node (AVN). In typical AVNRT the antegrade limb of the circuit consists of a slow pathway. Initially an atrial premature beat causes block to occur in the fast pathway and activation continues down the slow pathway. This delay allows recovery of the fast pathway and so the activation wavefront can conduct retrogradely around the AVN. The slow pathway recovers in time to allow antegrade activation again and the loop repeats itself.



last QRS of the tachycardia is frequently associated with a single P wave since AVNRT usually stops by blocking in the antegrade limb of the circuit. Termination of a tachycardia with a P wave usually indicates that the AVN is an important part of the circuit, i.e. the tachycardia is most probably AVNRT or an accessory pathway-mediated tachycardia as opposed to an atrial tachycardia. In atrial tachycardia, block in the AVN reveals a sequence of non-conducted P waves. Therefore, close inspection of the rhythm strip after injection of an AVN blocking drug such as adenosine, verapamil or a beta-blocker will enable confirmation of AVN dependence in the mechanism of a tachycardia.

Treatment

AVNRT can be treated pharmacologically with a number of agents. The principal agents used include verapamil, beta-blockers, digoxin, sotalol, flecainide or disopyramide. Their efficacy is by no means optimal and, although the frequency of symptoms may be reduced, patients are rarely ‘cured’ with medication alone. The recurrence rate on antiarrhythmic drugs is about 20% for AVRT and AVNRT.

Catheter ablation offers a 98% cure rate but does carry a 1% chance of atrioventricular block and lifelong dependence upon permanent pacing with all its attendant problems. Ablation requires delivery of radiofrequency energy to the slow pathway which can lie anywhere from the low right atrium, 3 cm from the compact AVN to just a few millimetres away higher up on the septum. Therefore, the risks of catheter ablation should be discussed carefully with the patient and weighed against the frequency and severity of the patient’s symptoms. Clearly, permanent pacing in a 25-year-old patient who faces 50 years of pacemaker battery changes, lead replacements and veno-occlusive problems has significant implications compared to a 60-year-old where such issues are less of a concern. However, given the high success rates of ablation for SVT, which is now a day-case procedure, the following categories of patient with SVT should be referred to an electrophysiologist to consider ablation:

1. All patients with regular SVT on anti-dysrhythmic drugs
 2. All patients with recurrent regular SVT.
- Advances in techniques for catheter ablation

mean that indications for this procedure now include atrial flutter and fibrillation. Success rates for atrial flutter are quoted at 95% if the flutter circuit involves the low right atrium isthmus region. Strategies and indications to perform atrial fibrillation (AF) ablation are in a state of rapid evolution. At the time of writing, indications for AF ablation include:

1. Symptomatic paroxysmal AF in patients who have had a trial of at least one anti-dysrhythmic drug and have a structurally normal heart
2. New onset persistent AF in an atrium <5 cm diameter.

Success rates for paroxysmal AF vary from 70 to 98% depending upon the centre's experience and techniques used, often requiring at least one ablation procedure. The next decade will certainly see significant advances in catheter ablation strategies for AF and will also allow us to assess whether ablation offers long-term cure for this arrhythmia. **BJHM**

Conflict of interest: none.

Further reading

Supraventricular tachycardia mechanisms

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interventions, and specific reference to anatomic boundary of the reentrant circuit. *Cardiol Clin* **11**(1): 151–81

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KEY POINTS

- Supraventricular arrhythmias are either atrioventricular node dependent, i.e. atrioventricular node re-entrant tachycardia and atrioventricular re-entrant tachycardias, or atrioventricular node independent, e.g. atrial tachycardia, atrial flutter and atrial fibrillation.
- The electrocardiogram diagnosis requires close inspection for P wave morphology and its relationship to the QRS complexes.
- Termination of a supraventricular tachycardia with a P wave indicates atrioventricular node dependence of the tachycardia and with a QRS complex an atrial tachycardia.

RSM YOUNG FELLOWS' AUDIT PROJECT PRIZE

Colorectal cancer imaging delays: speeding up the diagnostic pathway by cutting out the middle man

The British Journal of Hospital Medicine is pleased to be publishing some abstracts from the Royal Society of Medicine's Young Fellows' Audit Project Prize for 2004–5. This is the winning abstract by Dr David Burling – the runners up will be published in future issues. For information about entering this year's prize, please contact young.fellows@rsm.ac.uk

Abstract Objective

To audit radiological colorectal cancer staging time delays in order to meet the NHS cancer plan waiting time target.

Method

Time intervals between barium enema diagnosis of cancer and staging computed tomography (CT) scans were calculated for a 6-

month period, establishing that there were significant delays. A new strategy was implemented by which the radiologist rather than the clinician organized the CT appointment when they found a cancer at barium enema. Time intervals were reaudited for 6 months following implementation.

Results

A subsequent staging CT was performed on 49 patients with cancer found at barium enema. Average waiting times between barium enema and CT fell from 42 days (range 13–101, median =15, $n=18$) to 16 days (range 5–40, median =35, $n=31$)

in the 6 months before and after implementing the new strategy.

Discussion

The target of 1 month between diagnosis and treatment was unfeasible with existing practice. This audit identified unnecessary procedural delay and led to successful streamlining of the CT referral process while saving a second outpatient clinic visit.

Conclusions

Reduced waiting times between barium enema and staging CT have made the NHS cancer plan's target achievable. **BJHM**

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