

# Cardiac Arrhythmias – Part III

## Narrow Complex Tachycardia

MD O'Neill & DW Davies

### Abstract

*Narrow complex tachycardia usually refers to an abnormality of cardiac rhythm involving the tissues of the sinus node, atrial tissue, the atrioventricular node or an accessory atrioventricular communication. Although atrial fibrillation is the most common supraventricular arrhythmia, the term "supraventricular tachycardia" conventionally refers to the group of rhythm disturbances encompassing sinus tachycardia (appropriate and inappropriate), atrial tachycardia, atrial flutter, atrioventricular nodal reciprocating tachycardia (AVNRT) and atrioventricular reciprocating tachycardia (AVRT) including the Wolff Parkinson White syndrome (WPW). Atrial fibrillation is beyond the scope of this article which focuses on the diagnosis and acute management of the patient presenting with one of these common causes of a regular, narrow complex tachycardia.*

### Keywords

*Narrow complex tachycardia, supraventricular tachycardia, reentry, accessory pathway, ablation*

| Key Points                                                                                                                                                                                                                                                                 |
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| <ul style="list-style-type: none"> <li>Narrow complex tachycardia is almost always of supraventricular origin (SVT) but may also include rare ventricular tachycardias originating in the interventricular septum or fascicles of the cardiac conduction system</li> </ul> |
| <ul style="list-style-type: none"> <li>Atrial tachycardia (focal and reentrant), AVNRT and AVRT account for the majority of cases of narrow complex tachycardia</li> </ul>                                                                                                 |
| <ul style="list-style-type: none"> <li>Adenosine is both a diagnostic and a therapeutic tool but not a long term treatment for narrow complex tachycardia.</li> </ul>                                                                                                      |
| <ul style="list-style-type: none"> <li>All patients with narrow complex tachycardia should be considered for curative ablation therapy for their arrhythmia.</li> </ul>                                                                                                    |

### Introduction

There are three electrophysiological mechanisms by which most cardiac arrhythmias, including narrow complex tachycardia, may be explained: enhanced automaticity, triggered activity and re-entry. Cardiac cells which are capable of spontaneous diastolic depolarization may, under certain circumstances, depolarize at rates in excess of

the sinus node rate, thereby giving rise to a tachycardia. Triggered activity refers to abnormal cell membrane depolarisation during the repolarisation phase of the action potential. If the depolarization repetitively reaches the electrical threshold to "trigger" a further action potential, a focal source of ectopic depolarisations may perpetuate a SVT. By far the most common arrhythmia mechanism however, for initiation and maintenance of SVT is re-entry. This phenomenon refers to the conduction of an electrical wavefront around an obstacle to conduction (which may be anatomical eg scar tissue or a function of the electrical properties of the tissue comprising the circuit) and within a defined circuit. Atrial flutter, AVRT and AVNRT can all be explained by a re-entrant mechanism. That a SVT is manifest on the 12 lead electrocardiogram (ECG) as narrow complex is dependent on normal conduction of the impulse in an antegrade direction through the AV node and normal function of the left and right bundle branches. The differential diagnosis of a broad complex tachycardia has been discussed in the second paper in this series of reviews, and includes all the of the SVTs mentioned above in the presence of abnormal left or right bundle branch function or antegrade atrioventricular conduction via an accessory pathway.<sup>1</sup>

### Mechanism of arrhythmia in narrow complex tachycardia

#### Atrioventricular Nodal Reciprocating Tachycardia

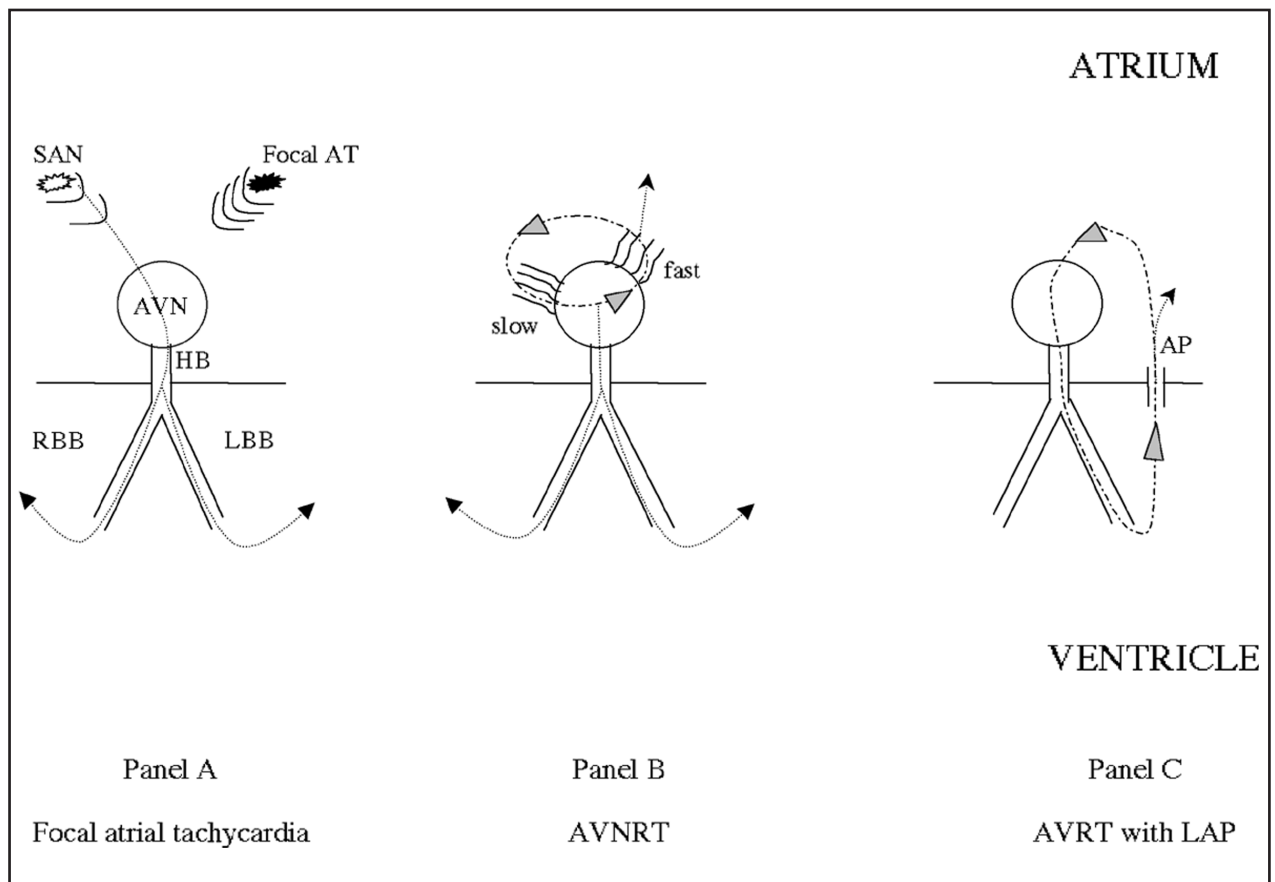
The AV node is the only normal electrical connection between the atria and the ventricles. It is best conceptualised as a complex of structures incorporating electrically distinguishable atrial inputs ("fast" and "slow" pathways), the compact AV node itself and the AV bundle. The fast pathway conducts impulses rapidly but repolarises slowly (i.e. long refractory period) while the slow pathway conducts slowly but repolarises rapidly (i.e. short refractory period). During sinus rhythm, the atrial impulse is normally conducted to the ventricles via the fast pathway. If an atrial ectopic reaches the AV

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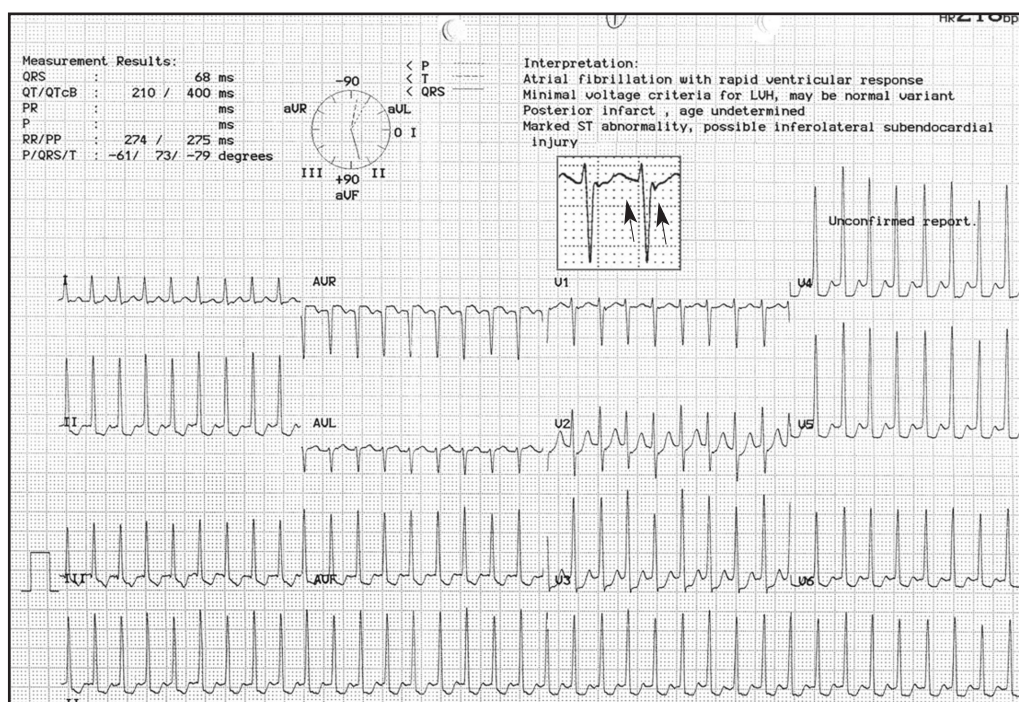
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## Cardiac Arrhythmias – Part III: Narrow Complex Tachycardia



**Figure 1.** Mechanisms of narrow complex tachycardia. The figure shows conduction of the cardiac impulse from the atrium to the ventricle across the atrioventricular node (AVN), Bundle of His (HB), left (LBB) and right bundle branches (RBB). Panel A depicts sinus rhythm and a focal atrial tachycardia where the ectopic focus overdrives the normal sinus rate. Panel B shows route of impulse propagation in “typical” slow-fast AVNRT, where the antegrade limb of the re-entry circuit is down the slow atrial input to the AV node and the retrograde limb is up the fast atrial input to the node. Panel C depicts orthodromic AVRT where the antegrade limb of the tachycardia circuit is via the AV node and the retrograde limb via a left-sided accessory pathway (LAP). In the presence of disease of the infranodal cardiac conduction system, all of these may manifest as broad complex tachycardia.



**Figure 2.** Twelve lead electrocardiogram of a 24 year old female patient presenting with palpitations. This shows a regular narrow complex tachycardia, with widespread ST segment change and a pseudo-r' in V1 (inset – marked with arrows). AVNRT is the most likely diagnosis.

## Cardiac Arrhythmias – Part III: Narrow Complex Tachycardia

node while the fast pathway is refractory, it may penetrate the slow pathway. By the time the impulse has completed conduction through the slow pathway, the fast pathway has recovered i.e. the refractory period is complete, and may accept retrograde invasion by the impulse which has just exited the slow pathway, thereby initiating “slow-fast” AV node re-entry. Atrioventricular nodal reentrant tachycardia can then be described as a re-entrant circuit in which the tachycardia is perpetuated by antegrade conduction via the “slow” pathway and retrograde conduction from node to atrium via the “fast” pathway (so called typical AVNRT – see Figure 1 panel b). Less commonly, antegrade conduction occurs via the “fast” pathway and retrograde conduction via the “slow” pathway (so called atypical AVNRT).

The 12 lead electrocardiogram may offer a clue to the diagnosis in typical AVNRT. As retrograde conduction is via the fast pathway during tachycardia, the resultant atrial depolarization occurs during or very shortly after the QRS complex (<70ms, so called short RP tachycardia).

This may be manifest as either no visible p waves on the 12 lead electrocardiogram, or a pseudo-r’ in lead V1 (Figure 2)

### ***Atrioventricular Reciprocating Tachycardia (Extra Nodal Accessory Pathways)***

Accessory pathways are congenital bridges of myocardium across the atrioventricular groove, providing an intact electrical connection between the atrial and ventricular myocardium. These pathways may conduct in both directions i.e. atrioventricular and ventriculoatrial, or in a purely retrograde direction. In the former, antegrade conduction is manifest on the 12 lead ECG as a delta wave and short PR interval. This is the WPW pattern. (Figure 3) In retrograde conduction, the accessory pathway is said to be “concealed” and the surface ECG shows a normal PR interval and QRS duration. The incidence of WPW pattern varies from 0.1 to 3 per 1000 ECGs, in which the incidence of paroxysmal tachycardias is between 10% and 30% of patients.<sup>2</sup>

**Figure 3.** Twelve lead electrocardiogram of a patient with Wolff Parkinson White syndrome, showing a short PR interval and a slurred QRS upstroke (delta wave), best seen in leads V4 to V6.



**Figure 4.** 12 lead electrocardiogram of typical counterclockwise right atrial flutter. Note the negative sawtooth flutter waves in the inferior leads (II, III and aVF) and the positive flutter wave in V1 with transition to negative in V6. There is 2:1 atrioventricular conduction with a flutter rate of 250bpm and a ventricular response rate of 125bpm.



## Cardiac Arrhythmias – Part III: Narrow Complex Tachycardia

Atrioventricular re-entry tachycardia may be either orthodromic or antidromic. During orthodromic tachycardia (Figure 1, panel c), the antegrade limb of the circuit is the AV node and specialized conduction tissues while the accessory pathway is the route of conduction from ventricle to atrium. In the presence of normally functioning conduction tissues, this is manifest as a narrow complex tachycardia on 12 lead ECG. If the accessory pathway is the antegrade limb, and the AV node the retrograde limb (antidromic tachycardia), the QRS complex will be broad during tachycardia.<sup>1</sup>

Robust accessory pathways with short refractory periods may allow extremely rapid antegrade conduction during atrial fibrillation, resulting in an irregular, broad complex tachycardia with rapid ventricular response rates and subsequent degeneration to ventricular fibrillation. The prophylactic ablation of accessory pathways in asymptomatic children with WPW remains a controversial area.<sup>3</sup>

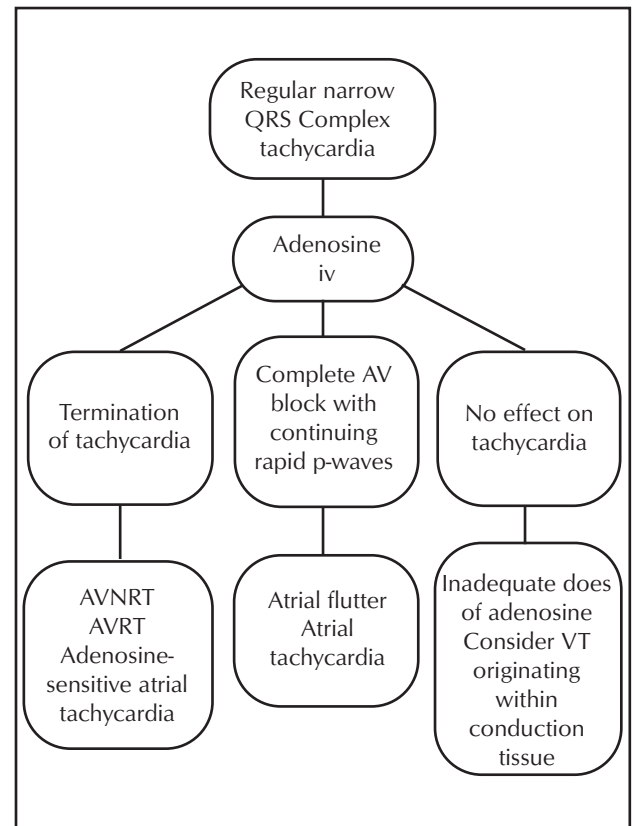
### Atrial Flutter

Atrial flutter is characterised by an organized atrial rhythm with a rate of between 250 and 350 bpm. The ventricular response rate is determined by the conduction properties of the AV node. The most common re-entrant circuit for atrial flutter involves counter-clockwise propagation of the impulse wavefront around the annulus of the tricuspid valve and is characterised electrocardiographically by negative flutter waves in the inferior leads (II, III and aVF) and a positive flutter wave in V1 (Figure 4). Flutter with a variable degree of AV block can result in a grossly irregular heart rhythm.

### Assessment of a patient with narrow complex tachycardia

#### History

Although this article deals with patients presenting acutely with a documented narrow complex tachycardia, the majority of patients with palpitations are asymptomatic by the time they are referred for clinical evaluation by a cardiologist. The age and sex of the patient can offer useful clues to the diagnosis of a narrow complex tachycardia prior to electrocardiographic examination. As accessory AV nodal conduction pathways are congenital abnormalities, it is not surprising that patients with accessory pathway-mediated tachycardia have a mean age of symptom onset that is almost a decade less than patients who have AVNRT.<sup>4</sup> Atrioventricular nodal reentrant tachycardia is more common in women than in men while the reverse is true for both atrial flutter<sup>5</sup> and accessory pathway mediated tachycardia.<sup>6</sup> Of patients who present to a hospital with a SVT without evidence of WPW morphology on their sinus rhythm ECG, approximately 30% have AVRT (via a concealed pathway), 60% have AV nodal reentrant tachycardia and the remainder have sinoatrial or intra-atrial tachycardia as the tachycardia mechanism.<sup>7</sup>



**Figure 5.** Flow chart depicting the response of a narrow complex tachycardia to an intravenous bolus of adenosine. AVNRT indicates atrioventricular nodal reciprocating tachycardia; AVRT, atrioventricular reciprocating tachycardia; VT, ventricular tachycardia (adapted from ref 11, by kind permission of the publishers)

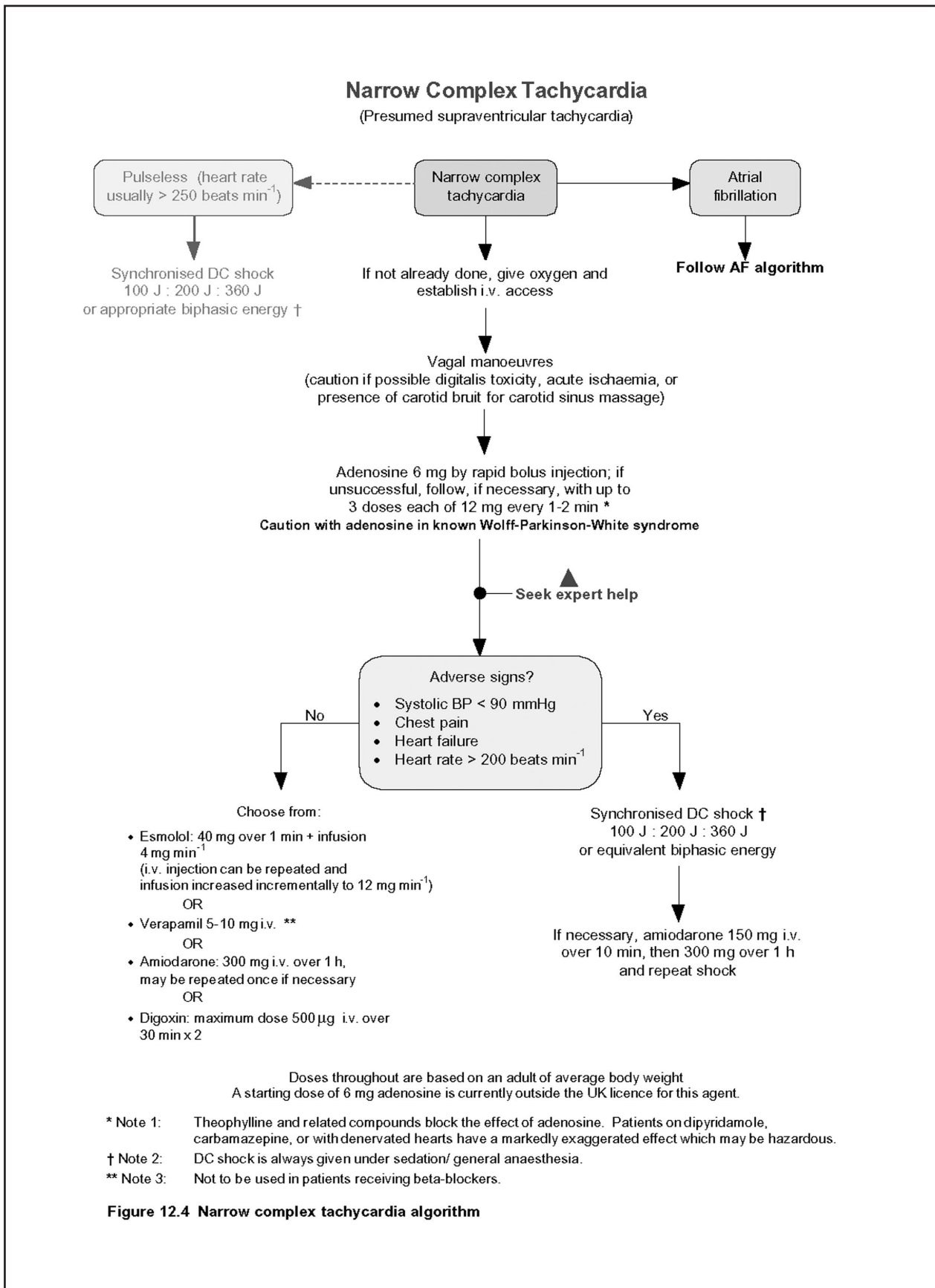
#### Examination

Evaluation of haemodynamic status is paramount in the assessment of patients presenting with any form of cardiac rhythm disturbance. Although significant haemodynamic impairment is not the hallmark of narrow complex tachycardia, patients with underlying structural heart disease may be particularly compromised by the heart rates reached during tachycardia. Although rarely of diagnostic use, it is of intellectual interest that in AVNRT, and in orthodromic AVRT, atrial contraction is against a closed atrioventricular valve, producing rhythmic cannon waves in the internal jugular venous pulse.<sup>8</sup>

### Acute Treatment of narrow complex tachycardia

The sinoatrial and atrioventricular nodes are richly innervated by the autonomic nervous system.<sup>9</sup> The AV node is a critical part of the re-entrant circuit in AVNRT and AVRT (Figure 1), in addition to being the only means of atrioventricular conduction in all forms of supraventricular rhythm disturbance in patients with a

## Cardiac Arrhythmias – Part III: Narrow Complex Tachycardia



**Figure 7.** Algorithm detailing the acute management of a patient presenting with a narrow complex tachycardia. In practice, esmolol is often unavailable and cautious intravenous metoprolol 5mg over 2 minutes repeated every 5min up to 15mg is an appropriate substitute. Reproduced with permission from the Resuscitation Council (UK) 2001 guidelines for peri-arrest arrhythmias.

## Cardiac Arrhythmias – Part III: Narrow Complex Tachycardia

structurally normal heart. Vagal manoeuvres (carotid massage, Valsalva manoeuvre, facial immersion in cold water) may therefore either terminate the arrhythmia (AVRT and AVNRT) or modify AV conduction sufficiently to allow interpretation of the underlying arrhythmia substrate (atrial flutter, atrial tachycardia). If such manoeuvres fail, then intravenous antiarrhythmic medication with specific effects on the AV node can be used in haemodynamically stable patients to diagnose and treat narrow complex tachycardia. Adenosine or nondihydropyridine calcium channel antagonists (verapamil and diltiazem) are the drugs of choice. It is important to record at least a rhythm strip during any physiological or pharmacological manoeuvres because the response may aid in the diagnosis even if the arrhythmia does not terminate (Figure 5). As a guide, a diagnosis of AVRT or AVNRT is favoured by termination of the tachycardia with a p wave after the last QRS complex of the tachycardia. Atrial tachycardia is favored by termination with a QRS complex while continuation of tachycardia despite AV nodal block effectively excludes AVRT and AVNRT and makes atrial tachycardia or atrial flutter highly likely (Figure 6).

Adenosine is an endogenous nucleoside with a half-life of elimination in blood of between 0.5 and 5 seconds.<sup>10</sup> Clinically relevant interactions can be seen with dipyridamole which potentiates the effects of adenosine by blocking cellular uptake, and with the methylxanthines which are adenosine receptor antagonists. Adenosine is contraindicated in patients with severe asthma<sup>11</sup> and there is only limited evidence to support its safe use in patients with lesser degrees of reversible airways obstruction.<sup>12</sup> Adenosine should be given at a starting dose of 6mg into a large peripheral vein followed by a 10ml rapid bolus of normal saline. Increasing the dose to 12mg is usually sufficient to produce a response.<sup>13</sup> Monitoring via a defibrillator is good practice as adenosine can initiate atrial fibrillation in the presence of AVRT and has been reported to precipitate other cardiac arrhythmias.<sup>14</sup>

As with all tachyarrhythmias, the presence of haemodynamic compromise merits urgent DC cardioversion under sedation if adenosine fails to cardiovert the patient to sinus rhythm. It may be appropriate to proceed immediately to cardioversion without pharmacological manipulation (Figure 7).

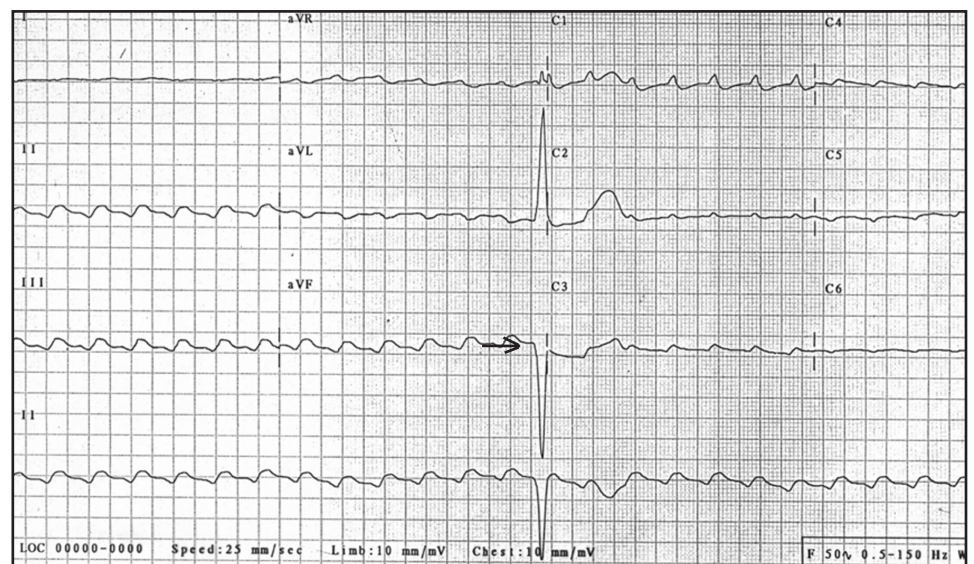
### Long term treatment of narrow complex tachycardia

Patients with AVNRT, AVRT and atrial flutter have arrhythmias which are curable.<sup>15</sup> Our understanding of the electrophysiological mechanisms of these arrhythmias has facilitated the development of catheter ablation strategies. Cure rates in excess of 98% are reported for both AVNRT and AVRT and 95% for atrial flutter, with a serious complication rate of less than 1%.<sup>11</sup> Most physicians treating patients who present to hospital with a narrow complex tachycardia will not have immediate access to an electrophysiology laboratory. Effective pharmacological therapy is important to alleviate symptoms in patients awaiting referral to an electrophysiologist and may be a definitive therapy in patients deemed unsuitable or unwilling for an interventional procedure.

For patients with frequent, recurrent sustained episodes of AVNRT, standard therapy includes verapamil, diltiazem, beta blockers and digoxin. Flecainide is suitable for patients with structurally normal hearts who are unresponsive to AV node active medication. Sotalol or amiodarone may also be used.<sup>11</sup>

Antiarrhythmic medication is being increasingly replaced by catheter ablation for management of patients with accessory pathway mediated tachycardia. Either the AV node or the accessory pathway or both may be targeted by a drug. Conduction through the AV node may be modified by beta blockers, verapamil or diltiazem. Conduction through the accessory pathway can be slowed by class I agents such as flecainide, procainamide, disopyramide and propafenone as well as by class III agents such as sotalol and amiodarone. Drugs which target the

**Figure 6.** 12 lead electrocardiogram and lead II rhythm strip in a patient with atrial flutter following the administration of 9mg of adenosine. There is a single QRS complex during the 10s recording indicated by the arrow, confirming the underlying diagnosis to be typical atrial flutter.



## Cardiac Arrhythmias – Part III: Narrow Complex Tachycardia

accessory pathway rather than the AV node are recommended for patients with coexisting atrial tachycardia, flutter or fibrillation to reduce the risk of promoting transmission of the high atrial rates to the ventricle via the accessory pathway.<sup>11</sup> However, it is this patient group which must be considered for urgent radiofrequency ablation of the accessory pathway to eliminate the future risk of pre-excited atrial fibrillation and potentially ventricular fibrillation.

### Atrial Flutter

The long term management of atrial flutter requires anticoagulation in addition to addressing the arrhythmias itself. The risk of thromboembolism following DC cardioversion for atrial flutter may be as high as 7% with most events occurring in patients either not receiving warfarin or subtherapeutically anticoagulated.<sup>16</sup> The current consensus is that the guidelines for anticoagulation for patients with atrial fibrillation be extended to those with atrial flutter.<sup>11</sup> Electrical cardioversion should only be considered if the INR is between 2 and 3, the arrhythmia is less than 48 hours duration or a transoesophageal echocardiogram (TOE) has excluded intracardiac thrombus.

The majority of episodes of atrial flutter occur as part of an identifiable precipitant for example, an acute exacerbation of chronic obstructive pulmonary disease or following surgery. Effective control of ventricular response rate using diltiazem, verapamil or beta-blockers, and

anticoagulation is an appropriate strategy in the stable, recovering patient. Although flecainide or amiodarone may be chosen to attempt cardioversion to sinus rhythm, the lengthening of the atrial refractory period may result in a slowing of the atrial rate from 300bpm to 220bpm, thereby permitting 1:1 atrioventricular conduction via the AV node, and paradoxically resulting in an acceleration of the tachycardia. Control of ventricular response rate is an acceptable long term strategy for management of atrial flutter. However, with a 95% success rate for ablation therapies, referral to an electrophysiologist should be considered.

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