A broad complex tachycardia

History

Figure 1a illustrates the electrocardiogram (ECG) of a 30-year-old man who presented to the accident and emergency department with a 2-hour history of palpitations and breathlessness.

What is the diagnosis and how would you manage the patient (a) acutely and (b) in the long term?

The electrocardiogram in tachycardia

The ECG demonstrates pre-excited atrial fibrillation (AF) with a ventricular rate of 150–170/min and left axis deviation. The principal features are the presence of a delta wave most prominent in leads V1–V4. Since the delta wave is positive in V1, the accessory pathway responsible for pre-excitation on the ECG is left sided rather than right sided. In the latter case, the delta wave would be predominantly negative in V1 because conduction through the pathway carries activation away from the right side of the heart. The shortest RR interval is 280 msec on this 12-lead ECG. However, a separate rhythm strip of this patient in the ambulance revealed RR intervals as short as 240 msec. An RR interval of less than 250 msec in pre-excited AF is associated with an increased risk of sudden cardiac death as a result of ventricular fibrillation. Therefore this scenario constitutes a medical emergency and the patient requires prompt diagnosis and treatment.

Discussion

Wolff–Parkinson–White syndrome (WPW) was originally described in 1930 as the combination of a delta wave on surface ECG and paroxysmal palpitations (Wolff et al, 1930). WPW typically presents during infancy or the second and third decades and has a prevalence of 0.1–0.4% (Prystowsky, 1988).

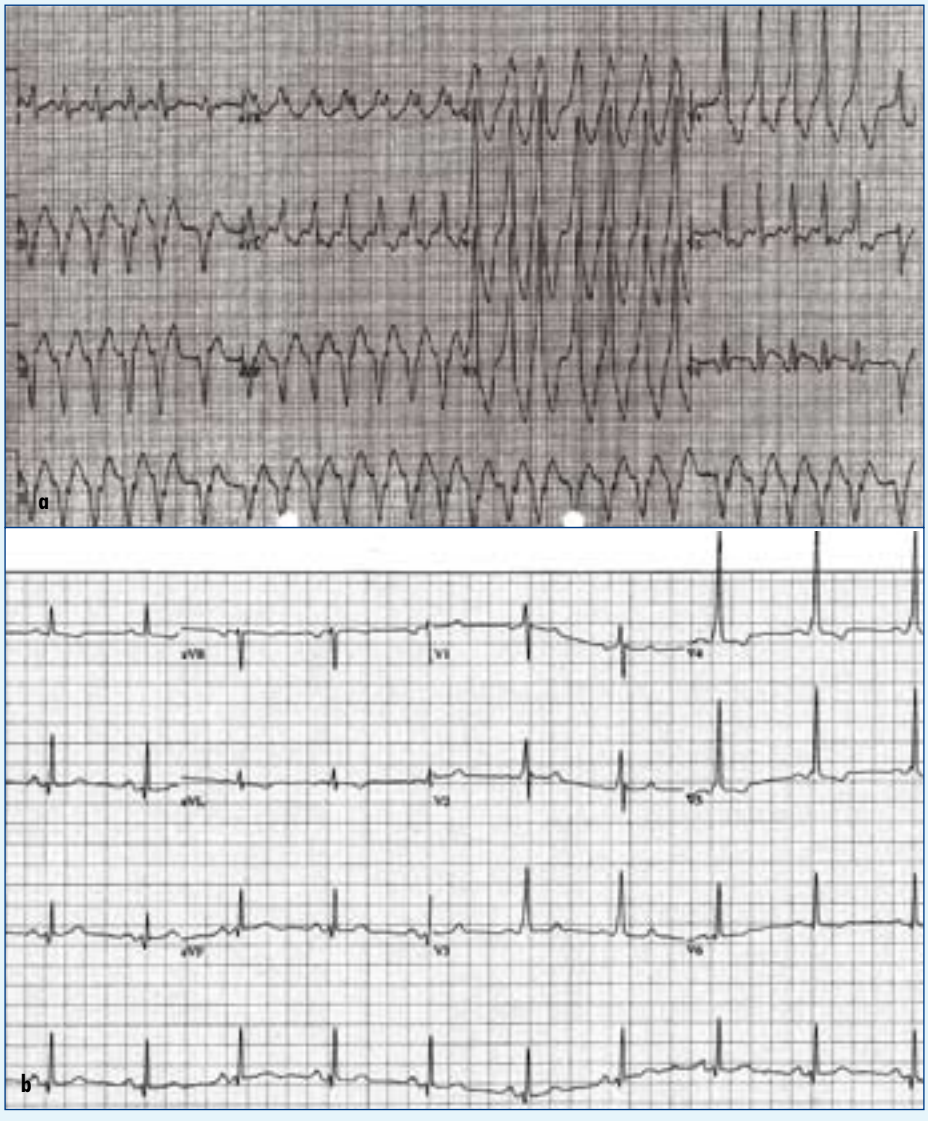
Pre-excitation is characterized by a PR interval of no more than 120 msec and slurring of the initial QRS upstroke known as a delta wave (Keating et al, 2003).

This delta wave is a fusion complex integrating slower depolarization of ventricular myocardium by an accessory pathway with normal more rapid activation via the specialized conduction tissue of the His–Purkinje system through the atrioventricular node (AVN). The greater the degree of ventricular activation via the pathway, the broader the QRS and more prominent the delta wave becomes. The conductivity of the accessory pathway, its anatomical location (*Figure 2*), and the effect of autonomic tone on AVN conduction determine this balance between activation by the pathway and via the AVN.

This explains why pre-excitation becomes manifest with pure AVN-blocking agents such as adenosine which therefore shift the balance of conduction to the accessory pathway. The electrocardiographic features may be confused with those of myocardial ischaemia, bundle–branch block and right ventricular hypertrophy.

Accessory pathway connections occur along the atrioventricular (AV) groove at the level of the tricuspid and mitral valve annuli (*Figure 2*).

Figure 1. a. Electrocardiogram at presentation. b. Electrocardiogram after initial treatment.



Dr Mehul Dhinoja *is Specialist Registrar in Cardiology and* **Dr Pier Lambiase** *is Clinical Lecturer in Electrophysiology in the Cardiology Department, The Heart Hospital, University College London, London W1M 8PH*

Correspondence to: Dr P Lambiase

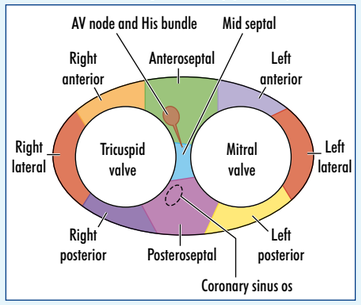
Tachycardia mechanisms

The commonest dysrhythmia is orthodromic reciprocating tachycardia (ORT) or AV re-entrant tachycardia which occurs in 84% of WPW presentations. This is followed by AF (51% incidence), antidromic reciprocating tachycardia (ART, 10% incidence) and ventricular fibrillation (<1% incidence) (Zardini et al, 1994).

ORT is usually initiated by a premature ventricular or atrial beat that establishes an AV re-entrant circuit as illustrated in *Figure 3a*. The ECG demonstrates regular, narrow QRS complexes. Retrograde P waves may be visible and can aid localization of the pathway. If a negative P wave is seen in lead I during ORT a left lateral accessory pathway should be suspected as this represents the atrial insertion of the pathway. However, P waves are often buried in the T wave and can be difficult to distinguish.

The narrow QRS complexes reflect rapid activation of the ventricles via the AVN and His–Purkinje system. However, functional delay in one of the bundle branches can produce broad QRS complexes. If the bundle–branch block is ipsilateral to the side of the pathway in the re-entrant circuit, the cycle length is prolonged in bundle–branch block allowing one to localize whether the accessory pathway is on the left or right side of the heart. ORT usually terminates with block in the AV node and thus a retrograde P wave which fails to conduct antegradely.

Figure 2. Locations of accessory pathways in the atrioventricular (AV) groove. The commonest site is left free wall (50–60%), followed by the posteroseptal region (20–30%) and right free wall (10–20%). The least common are the anteroseptal and midseptal pathways that are in proximity to the bundle of His (<10%) and thus carry the highest risk of AV block during ablation (up to 5%).



The re-entrant circuit in ART is the reverse of that for ORT (*Figure 3b*). The ventricles are fully pre-excited and the ECG demonstrates broad QRS complexes similar to those in ventricular tachycardia. Indeed if one sees chest lead concordance in the context of a broad complex tachycardia and Q waves in the inferior leads one should also consider a left posterior accessory pathway-mediated antidromic tachycardia – particularly in a young patient with no preceding history of ischaemic heart disease.

AF in WPW has the greatest risk of causing haemodynamic compromise and ventricular fibrillation. The ECG demonstrates a rapid, irregularly irregular broad QRS complex tachycardia usually with full pre-excitation as in this case. However, there may be occasional normal QRS complexes as a result of dominant antegrade conduction down the AVN–His–Purkinje system. This intermittent pre-excitation is usually indicates that the pathway is at a lower risk of conducting at sufficient rates to trigger ventricular fibrillation. Note that in *Figure 1b*, the delta wave is less prominent in sinus rhythm *vs* AF since the AVN conducts to a greater extent *vs* the pathway at normal heart rates. This is a function of the relative refractory periods of the AVN and AP. AF is thought to occur in WPW syndrome via retrograde activation in the atria modifying the electrophysiological substrate since the atria are usually structurally normal.

Treatment

Acute management

Drug therapy

This is targeted towards the AVN, the accessory pathway or both (*Table 1*). *Figure 4* demonstrates a suggested pharmacological protocol to follow in the acute treatment of accessory pathway-mediated tachycardia.

Haemodynamically unstable patients are relatively rare but should be treated by synchronized DC cardioversion (DCC) (*Figure 4*).

ORT is usually terminated with intravenous AVN-blocking drugs such as adenosine, beta blockers (propranolol, metoprolol), verapamil or diltiazem. If this fails, intravenous procainamide can be administered at 25–50 mg/min to a maximum of

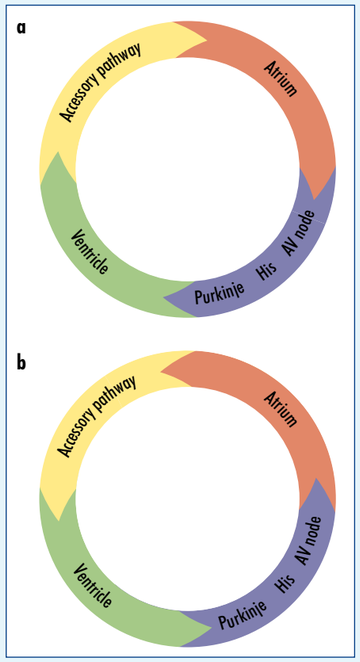


Figure 3. a. Orthodromic reciprocating tachycardia. b. Antidromic reciprocating tachycardia. AV = atrioventricular.

10 mg/kg with blood pressure monitoring. Chronic drug prophylaxis can include AVN-blocking drugs or those affecting the accessory pathway.

Conversely, procainamide is first-line therapy in ART and pre-excited AF since AVN-blocking drugs can potentially accelerate conduction down the accessory pathway and initiate ventricular fibrillation. Therefore, one should avoid agents such as verapamil, digoxin and adenosine in this situation. Chronic drug therapy, if necessary, should not increase the risk of rapid heart rates during AF, so digoxin and verapamil should be avoided. Drugs such as

Table 1. Sites of primary antiarrhythmic effect

AV node	Accessory pathway	Both
Digoxin	Quinidine	Flecainide
Beta blockers	Procainamide	Propafenone
Adenosine	Disopyramide	Sotalol
Verapamil		Amiodarone

AV = atrioventricular

procainamide, quinidine and disopyramide suppress conduction across the accessory pathway in both directions and are very effective. Flecainide and propafenone also slow conduction through the AVN and are also effective prophylactic drugs. The utility of amiodarone is limited because of its side effects, particularly in young patients.

Long-term management

All symptomatic patients with WPW, particularly those with rapid tachycardias, should be considered for curative ablation of the accessory pathway. The success rates are over 90% with minimal risks (Schluter et al, 1991; Plumb, 1995). There is some debate regarding the management of the asymptomatic patient with evidence of pre-excitation on resting ECG as there remains the concern that such a person may develop rapidly conducted pre-excited AF and ventricular fibrillation. The current consensus is that the asymptomatic patient with intermittent pre-excitation is at minimal risk and requires no therapy. However, patients with multiple accessory pathways, recurrent AV re-entrant tachycardia or AF and pre-excited AF (spontaneous or induced at electrophysiological study) with an RR interval of less than 250 msec are at higher risk and should be considered for ablation. The decision to proceed with ablation should be considered in the context of the patient's symptom status, risk of sudden cardiac death and risks of the procedure.

Conclusions

WPW is one of the commonest causes of tachycardia in the under 40-year-old population. Pre-excited AF is a medical emergency and should be treated promptly with intravenous procainamide or DCC. All patients with supraventricular tachycardias should be evaluated by an electrophysiologist to undertake risk assessment and plan therapy. **BJHM**

Conflict of interest: none.

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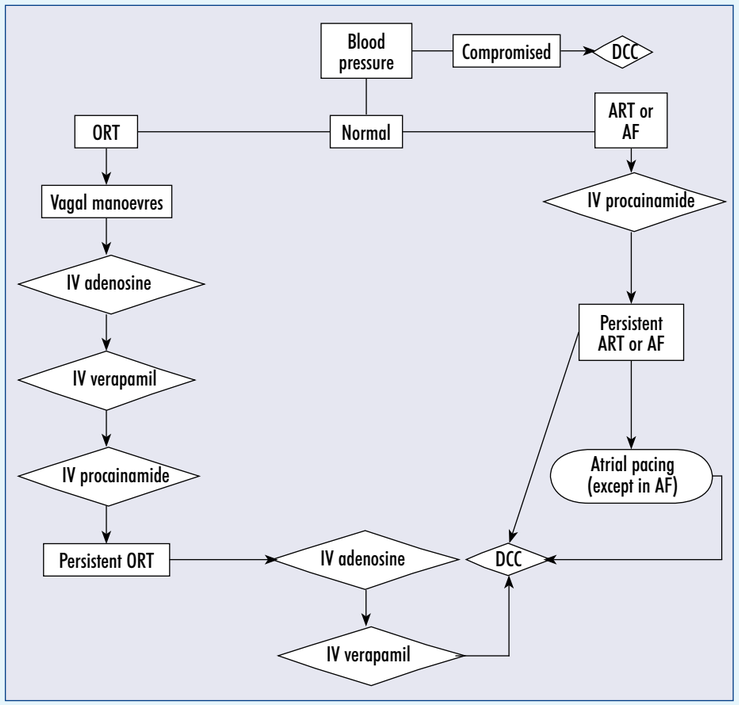
Figure 4. Proposed protocol for tachycardia management in Wolff–Parkinson–White syndrome. In patients presenting de novo with orthodromic re-entrant tachycardia (ORT) it may not be possible to conclude that an accessory pathway is present in which case verapamil can be used. If ORT persists, the introduction of adenosine in the presence of verapamil is usually effective in blocking atrioventricular node conduction and terminating the tachycardia. AF = atrial fibrillation; ART = antidromic re-entrant tachycardia; DCC = direct current cardioversion; IV = intravenous.



KEY POINTS

- Wolff–Parkinson White syndrome is defined as the combination of palpitations and evidence of a delta wave on surface electrocardiogram (ECG).
- The direction of re-entry is defined according to the direction activation passes through the atrioventricular node (AVN) – orthodromic re-entrant tachycardia (ORT) (activation passes from atrium to ventricle via the AVN) and antidromic (retrograde activation of the node). Therefore the surface ECG will be maximally pre-excited as all activation passes via the accessory pathway from atrium to ventricle.
- If the accessory pathway only conducts retrogradely and no delta wave is visible on surface ECG, the pathway is concealed and manifests as a narrow complex supraventricular tachycardia (ORT).
- Pre-excited atrial fibrillation is a medical emergency and requires prompt cardioversion either pharmacologically or with DC energy.
- Patients with palpitations and evidence of pre-excitation on resting ECG should be referred to an electrophysiologist for assessment.