Electrocardiographic features of Wolff-Parkinson-White syndrome

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Wolff-Parkinson-White syndrome (WPW) is the commonest form of ventricular pre-excitation. It is characterised by the presence of an accessory pathway between the atria and the ventricles which provides an alternative route for ventricular activation. This bypass tract avoids the atrioventricular node (AVN) permitting premature ventricular activation hence the term pre-excitation. Historically Wolff, Parkinson, and White first described the condition in 1930 in a series of 11 healthy young adults with a functional bundle branch block, an abnormally short PR interval and paroxysms of tachycardia or atrial fibrillation.

The ECG features are characteristic and important to recognise. Although some patients may remain asymptomatic throughout their lives, others are prone to tachyarrhythmias that may be life threatening. It is seen in around 2.4% of patients presenting with a regular narrow complex tachycardia to the emergency department where it can be successfully treated with the restoration of sinus rhythm. Atrial fibrillation associated with WPW is less common. In this situation rapid and irregular ventricular responses are seen, which may degenerate into ventricular fibrillation.

This article consists of two case histories followed by a review of the literature covering diagnostic and urgent initial therapeutic issues.

CASE HISTORIES

Case one
A 14 year old boy presented to the emergency department complaining of palpitations and dizziness. He had no medical history of note. A 12 lead ECG was performed that demonstrated a narrow-complex tachycardia with retrograde P waves (fig 1). He was haemodynamically stable and treated with adenosine. He reverted to sinus rhythm and the 12 lead showed evidence of pre-excitation (fig 2). He was referred for risk stratification by electrophysiological studies. He was found to have an accessory pathway with both antegrade and retrograde properties and underwent successful radiofrequency ablation.

Case two
A 52 year old woman presented to the emergency department with a three hour history of chest pain and shortness of breath. She had a medical history of palpitations. On admission her rhythm strip showed atrial fibrillation with a very fast ventricular response (fig 3).

Figure 1 Narrow-complex tachycardia with retrograde P waves visible deforming early part of ST segment.

Figure 2 Pre-excitation with shortened PR interval and “delta wave”—that is, slurred upstroke of QRS complex, most visible in rhythm strip.

Figure 3 AF in WPW. Note irregularly irregular rhythm with delta wave visible in QRS. Note fast ventricular response.
examination she was unwell, sweating, and clammy. She was tachycardic and haemodynamically compromised with a systolic blood pressure of 80 mm Hg. A 12 lead ECG (fig 3) showed an irregularly irregular rhythm with broad complexes at a rate of 220. She underwent DC cardioversion and reverted to sinus rhythm. A 12 lead ECG demonstrated no pre-excitation. She was referred for electrophysiological studies. The accessory pathway was identified and found to be capable of fast antegrade conduction. She was successfully treated with radiofrequency ablation.

DISCUSSION

In the normal heart electrical impulses generated from the sino-atrial node in the right atrium pass to the AVN. Physiological slowing occurs at this point before conduction to the His-Purkinje system and the ventricular muscle. The PR interval represents the time taken for the atria to depolarise and the wave of depolarisation to travel down the AVN and through the His-Purkinje system. In pre-excitation syndromes the accessory pathway permits the impulse to bypass the AVN (thus avoiding the associated delay) and permitting early depolarisation of the ventricular myocardium (fig 4A). This is reflected on the ECG as a shortened PR interval. Non-specialised ventricular myocardium conducts at a slower rate. Its direct depolarisation in pre-excitation syndrome generates an initial slurring or “delta wave” at the start of the QRS complex (fig 2). Simultaneous activation of the ventricles occurs as normal through the AVN and His-Purkinje system and hence the remainder of the QRS complex appears relatively normal. The mechanism of ventricular depolarisation in sinus rhythm in patients with WPW is a combination of pre-excitation and normal conduction giving rise to a short PR interval and a delta wave followed by a relatively normal QRS complex (fig 2).

The accessory pathway is congenital in origin and results from a failure of complete separation of the atria and ventricles. It consists of a thin filamentous structure situated anywhere along the atroventricular groove and the left lateral pathway is the most common. In around 10% of cases multiple pathways exist. The incidence of associated congenital abnormalities ranges from 7% to 20%.

Tricuspid valve lesions are the most common. The prevalence of WPW syndrome in the population is about 0.3% with an associated risk of sudden death of around 0.5% to 4%.

The most frequently encountered tachycardia in WPW syndrome is an atrioventricular re-entrant tachycardia (AVRT). The arrhythmia uses the AVN and accessory pathway to form a re-entry circuit triggered by an appropriately timed ectopic. This re-entry circuit maybe classified as either orthodromic or antidromic depending on whether the ventricles are activated via the normal conduction system or the accessory pathway. Orthodromic AVRTs account for most tachycardias in WPW syndrome (70%). Conduction occurs down the AVN and retrogradely up the accessory pathway producing a narrow complex tachycardia (fig 4B). The QRS morphology is normal (that is, with no delta wave) during the arrhythmia because ventricular activation has occurred through the normal pathway. The rate is usually between 140 and 250 beats per minute (fig 1). Inverted P waves may be visible deforming the ST segment indicating that atrial depolarisation occurs later than ventricular depolarisation. In a patient with a narrow complex tachycardia the presence of such late P waves is frequently the only ECG evidence that the patient has an accessory pathway rather than a much more common atrioventricular non-re-entrant tachycardia (AVNRT) (fig 5).

Antidromic tachycardias occur when the accessory pathway conducts antegrade (atrioventricular) and the impulse returns to the atria via the AVN (fig 4C). The resulting QRS is broadened reflecting the abnormal ventricular activation (fig 6). These broad complex tachycardias can be difficult to distinguish from a ventricular tachycardia and are less common, occurring in around 10% of patients with WPW syndrome.

It is important to note that if the accessory pathway only conducts retrogradely (ventriculo-atrial) the resting ECG will be normal in sinus rhythm with no evidence of pre-excitation. This is known as a concealed pathway.

For reasons that are not clear atrial fibrillation is relatively common (20%) in WPW syndrome compared with the normal population. In atrial fibrillation with pre-excitation activation of the ventricles is predominantly via the accessory pathway (fig 4D). This causes the expected irregular rhythm but the QRS is widened with a bizarre morphology. Occasional activation of the ventricles occurs via the AVN resulting in a capture beat with a normal QRS (fig 3). If the accessory pathway is capable of conducting rapidly potentially life threatening arrhythmias may result. The ventricular response may be in the region of 180–220.

Treatment of AVRTs essentially consists of interrupting the re-entrant circuit. Stable symptomatic patients can safely undergo vagal manoeuvres to block the AVN conduction and

**Definition of WPW syndrome**

- Symptoms suggestive of recurrent tachycardias in addition to the following ECG characteristics:
  - Shortened P-R interval of <0.12 s
  - Slurred slow rising onset to QRS known as the delta wave
  - A prolonged QRS complex >0.11 s

![Series of four stylised drawings of the heart.](Image)
hence break the circuit. Adenosine is used to prolong AVN refractoriness but it should be used with full resuscitation facilities. This is because atrial fibrillation with a rapid ventricular response maybe induced in as many as 12% of patients. A haemodynamically compromised patient with an AVRT or AF must undergo electrical cardioversion. Flecaïnide is a useful alternative in the stable patient with AF or an antidromic broad complex tachycardia. Calcium channel blockers, digoxin, and amiodarone must be avoided in the management of the patient with AF. This is because of the potential to increase accessory pathway conduction by increasing AVN block with the inherent danger of precipitating ventricular fibrillation.

Significant advances have been made in the field of electrophysiological studies. Radiofrequency ablation has radically changed the management of these patients. This is particularly important given that the long term safety of many anti-arrhythmics has not been fully elucidated. The long term success rates of such procedures are now thought to be approaching 95%. The risk of serious side effects is low and occurs at a rate of less than 1%. Radiofrequency ablation was previously limited to patients with episodes of atrial fibrillation or frequent or disabling symptoms. WPW syndrome is a condition that primarily affects younger people in whom long term anti-arrhythmic prophylaxis is undesirable. There is now evidence to suggest that as its safety improves there may be a place for the risk stratification of all patients.

CONCLUSION
WPW syndrome is the commonest form of ventricular pre-excitation. It is not infrequently encountered in the patient presenting to the emergency department with tachyarrhythmias. After restoration of sinus rhythm prompt referral to cardiology is essential for risk stratification through electrophysiological studies. Recent advances in diagnostic and therapeutic procedures in this field mean that the long term prognosis for these patients is good.

Contributors
FPM initiated the idea with WJB. LK reviewed the literature and wrote the manuscript. FPM edited the manuscript and will act as guarantor of the paper.

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