

ABERRANT CONDUCTION AND WPW SYNDROME

Part IX of an educational series on ECG analysis and arrhythmia diagnosis

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Wide QRS complexes during normal sinus rhythm are usually due to either ventricular pre-excitation (i.e. where part of the ventricle is depolarised through an accessory pathway) or left or right bundle branch block (LBBB or RBBB). During tachycardias it is often difficult to distinguish between SVTs conducting with wide QRS complexes (either pre-existing bundle branch block or aberration) and ventricular tachycardia.

WOLFF-PARKINSON-WHITE SYNDROME

A syndrome consisting of 'bundle branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia' was first described by Wolff, Parkinson, and White in 1930. Kent and others, however, had already noted multiple bands of muscular tissue connecting the atria and outside the normal conducting system as early as 1893.

ECG features

The typical ECG findings in patients with this condition are:

- PR interval less than 120 msec during sinus rhythm
- QRS complex duration exceeding 120 msec with a slurred, slowly rising onset of the QRS in some leads (delta wave)
- secondary ST-T wave changes that are usually directed opposite to the major delta and QRS vectors.

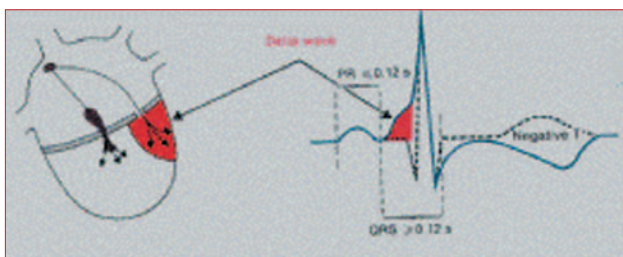


Fig. 1. The WPW pattern.

The short PR interval is due to the fact that conduction partially bypasses the AV node and the delta wave reflects that part of the ventricle depolarised through the accessory pathway. Because depolarisation is abnormal, repolarisation will be abnormal as well, resulting in the secondary ST-T wave changes.

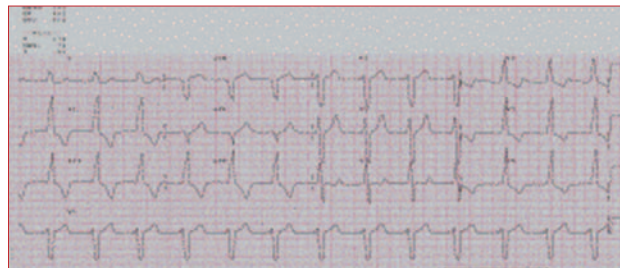


Fig. 2. Typical ECG manifestations in WPW syndrome. The QRS morphology in this case resembles a LBBB, but note the short PR interval and the normal R-wave progression across the right precordial leads (V1-V3), features not seen in 'regular' LBBB.

Clinical features

Orthodromic AV re-entry is the most common arrhythmia associated with the WPW syndrome. The tachycardia is regular, with narrow QRS complexes, reflecting antegrade conduction over the AV node and specialised conduction system and retrograde conduction over the accessory pathway. A retrograde P wave can often be seen after the QRS complex representing retrograde depolarisation of the atria over the accessory pathway.

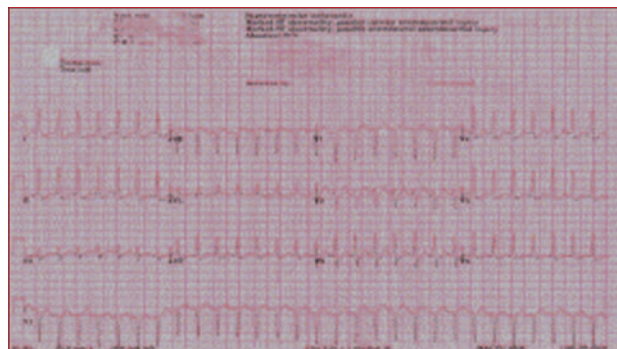


Fig. 3. Orthodromic tachycardia.

Antidromic re-entrant tachycardia is present when the accessory pathway is responsible for antegrade conduction, and the AV node or a second accessory pathway is responsible for retrograde conduction. The QRS complex is fully pre-excited on the ECG. These tachycardias are rare and often difficult to distinguish from ventricular tachycardias unless one has collateral evidence of pre-excitation during sinus rhythm.

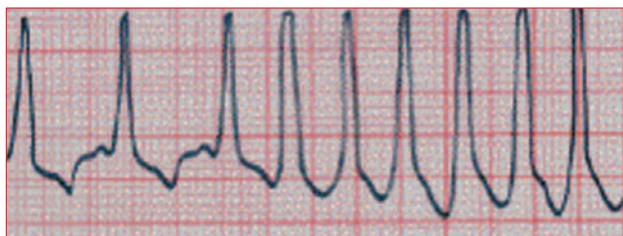


Fig. 4. Antidromic tachycardia.

Atrial fibrillation or atrial flutter is observed in up to 40% of patients with the WPW syndrome and can be potentially life threatening. The ventricular response during these arrhythmias depends on the refractory periods of the accessory pathway and the AV node. Patients with very short accessory pathway refractory periods may conduct very fast over the accessory pathway, resulting in rates in excess of 300 beats per minute. This may result in ventricular fibrillation and sudden cardiac death. The QRS is usually bizarre and not typical LBBB or RBBB.

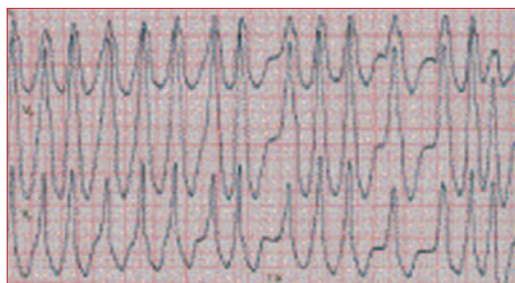


Fig. 5. Atrial fibrillation in WPW syndrome.

Treatment of WPW syndrome

Termination of acute episode. In narrow complex tachycardias (orthodromic tachycardia), vagal manoeuvres may be tried followed by intravenous adenosine as first drug of choice. Intravenous verapamil may also be used. An external cardioverter-defibrillator should be available immediately as adenosine may lead to atrial fibrillation which can conduct rapidly over the accessory pathway resulting in very fast ventricular rates and subsequent ventricular fibrillation.

In wide complex, regular tachycardias (antidromic tachycardia) it is best to regard it as ventricular tachycardia and to treat it as such, unless one has collateral evidence of pre-excitation. **NB: never give IV verapamil or digitalis!**

For atrial fibrillation, drugs that prolong the refractoriness of the accessory pathway (e.g. procainamide) must be used. As the ventricular rates are often very fast, resulting in a haemodynamically unstable patient, synchronised DC cardioversion is often necessary.

Prevention. It is not always possible to predict which drugs may be most effective for an individual patient. Procainamide in combination with a beta blocker can be beneficial. Drugs which prolong the refractoriness in both

the accessory pathway and AV node, e.g. class Ic drugs, sotalol, or amiodarone can be effective. **Radiofrequency catheter ablation of the accessory pathway may be regarded as first-line therapy in patients with WPW syndrome as it is curative and obviates long-term drug control.**

ABERRANT VENTRICULAR CONDUCTION

The basic cause of aberrant conduction is the premature arrival of a stimulus before the conduction system has completely recovered from its refractory state from the previous beat. The beat then conducts with either a RBBB or LBBB pattern. The factors that influence refractoriness are rate and preceding cycle lengths.

When a stimulus reaches the RBB during phase 3 of the action potential, the membrane potential is reduced and conduction is compromised, resulting in aberrant conduction, particularly if the refractory period is abnormally prolonged and the stimulated rate rapid. This phenomenon is often called tachycardia-dependent bundle branch block. When cycle length varies widely from beat to beat, such as in atrial fibrillation, aberrancy is commonly observed (long-short interval, commonly referred to as *Aschman's phenomenon*).

Aberrant conduction can also result from retrograde concealed conduction. This occurs when a VPB activates the RBB in a retrograde manner. The RBB is subsequently in the refractory period for the next sinus beat, resulting in aberrant conduction through the ventricle. The morphology of the QRS complex during aberrant conduction usually demonstrates a typical LBBB or RBBB.

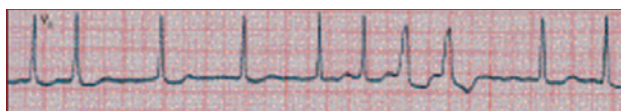


Fig. 6. Aberrant conduction during atrial fibrillation demonstrating the Aschman Phenomenon. (long-short interval preceding the aberrant conducted beats).

IN A NUTSHELL

- Symptomatic ventricular pre-excitation through an accessory pathway is known as the Wolff-Parkinson-White (WPW) syndrome.
- The usual arrhythmia in WPW syndrome is a narrow QRS atrioventricular re-entry tachycardia, but a rarer, wide QRS (antidromic) tachycardia may also occur.
- Atrial fibrillation occurring in the setting of WPW may be potentially life-threatening and it is therefore advisable for all patients with this syndrome to be referred to an arrhythmia specialist.

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