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Understanding the ECG. Part 5: Pre-excitation

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Introduction

Pre-excitation occurs when part of the ventricle is depolarised earlier than normal, producing characteristic changes on the 12-lead electrocardiogram (ECG). When these changes are found in patients with paroxysmal arrhythmias, it is referred to as Wolff-Parkinson-White syndrome (WPW), although many people apply the same terminology to asymptomatic people. We now know that pre-excitation is caused by an accessory pathway (AP) - a small strand of muscle joining the atria to the ventricles - but for many years the cause of this ECG phenomenon was unknown (Hanon et al, 2005).

Pre-excitation is an important finding on the ECG, and should always be sought during systematic evaluation. Because the ventricles are activated abnormally, there may be significant changes in the appearance of the QRS complexes. These changes may result in misdiagnosis if the distinctive pattern of WPW is not recognised (Bennett, 2013). In addition, pre-excitation is associated with a small increase in the risk of sudden cardiac death (SCD), necessitating specialist evaluation when it is identified.

In this fifth article of the *British Journal of Cardiac Nursing's* ECG Interpretation Series, we take a detailed look at pre-excitation, APs and WPW. We discuss how electrical pathways were first discovered in the heart, and how they were connected to WPW. We also consider how they differ in their location and electrical properties. Finally, we relate this information to ECG appearance, acute presentation, and long term management of the patient with pre-excitation.

A brief history of pre-excitation

Electrical connections between the atria and ventricles were first discovered during the nineteenth century, and by 1906 the anatomy of the AV node, bundle of His and Purkinje fibres had been mapped out (Scheinman, 2005). A year later, Sir Arthur Keith and Martin Flack identified the sinus node in the heart of a mole, and our modern understanding of the structure of the conduction system was more or less complete (Silverman et al, 2006).

One area that remained unclear, however, was the existence of additional electrical connections, outside of this system. In 1893, Stanley Kent described strands of muscle fibres, linking the atria and ventricles, although their purpose was unknown and their existence disputed (Hanon et al, 2005). They later became known as bundles of Kent, although in the modern era we refer to them as APs. We now know that they cause pre-excitation on the ECG, but in the early years of the twentieth

century this relationship was not understood. It would be 40 years before a connection was made, and almost another 40 before it was proven (Scheinman, 2005).

The clinical signs of pre-excitation were first reported by Cohn and Fraser (1913). They described two patients with paroxysmal tachycardia that terminated with vagal stimulation. In both cases, the resting ECG was abnormal, with a wide QRS complex. Further sporadic reports were published over the next fifteen years, however it was Dr Louis Wolff, Sir John Parkinson, and Paul Dudley White that first identified it as a syndrome in 1930 (Wolff et al, 1930; Obeyesekere et al, 2012). They described eleven cases in which mostly young, healthy people presented with intermittent palpitations, and an abnormal resting ECG. The ECG abnormality was described as a 'bundle branch block', in association with a short PR interval (Wolff et al, 1930).

Three years later, Wolferth and Wood (1933) proposed that the ECG signs of pre-excitation were caused by early depolarisation of the ventricles by a Bundle of Kent. In other words, the electrical signal from the sinus node was passing down the Kent bundle, and reaching part of the ventricle before the signal travelling through the AV node. They further proposed that the bundle of Kent allowed an electrical impulse to re-enter the atrium, causing paroxysmal tachycardia. Both of these theories were correct, however a lack of proof led to many alternative theories during subsequent years. Finally, with the advent of cardiac surgery, doctors were able to electrically map an accessory pathway, and prove that it caused pre-excitation. In 1967, the first successful surgical procedure for WPW was performed, utilising this new understanding of the syndrome (Obeyesekere et al, 2012).

Accessory pathways

We now know considerably more about APs than these early pioneers. APs are minor congenital abnormalities that occur during fetal development. In most cases, the heart is otherwise normal in structure, but AP also occur in association with more serious congenital or inherited heart defects. These include Ebstein's anomaly, transposition of the great arteries, and hypertrophic cardiomyopathy (Triedman, 2009). Multiple pathways are possible, and are particularly common in Ebstein's anomaly (Chugh et al, 2008).

The vast majority of APs connect the atrial and ventricular myocardium at the annuli of the mitral and tricuspid valves (Rantner et al, 2012). Of these, approximately 60% are found in the left free wall of the heart, 25% in the septum, and 15% in the right free wall. Rarely, AP are found that connect to parts of the specialised conduction system. These may be atriofascicular (atria to right bundle branch), nodofascicular (AV node to right bundle branch), or nodoventricular (AV node to ventricle) (Chugh et al, 2008).

As well as varying in location, APs also exhibit a range of conduction properties. Some are capable of conducting very rapidly, while others are relatively slow. Around 60% of pathways are able to conduct in both directions, in other words from atria to ventricles (anterograde) as well as ventricles to atria (retrograde) (Thanavaro and Thanavaro, 2010). The remainder support retrograde conduction only, and therefore do not cause pre-excitation on the ECG. These accessory pathways are referred to as "concealed" because their presence is not apparent on the 12 lead ECG (Bennett, 2013).

APs have a longer refractory period than the AV node, however conduction through them is non-decremental. This means that they do not exhibit the 'filtering' effect of the AV node in the event of atrial arrhythmias (Chugh et al, 2008). APs that are capable of rapid anterograde conduction can

therefore conduct atrial fibrillation to the ventricles at much faster rates than the AV node will support (Mark et al, 2009).

Let's turn our attention to the ECG now, and consider the changes that occur due to a 'manifest' pathway - in other words, one that conducts in an anterograde direction, causing pre-excitation.

Recognising WPW on the ECG

The principal ECG features of pre-excitation are a short PR interval and a delta wave (Marks et al, 2009). A delta wave is a slurring and widening of the first part of the QRS complex (*figure 1*). The presence of a delta wave usually makes the overall QRS broad, and may also result in ST-segment and T-wave abnormalities. Because the activation of the ventricles is abnormal, the normal pattern of QRS complexes across the 12 lead ECG is also changed. These changes can mimic the ECG signs of myocardial infarction (Bennett, 2013). To understand why the ECG changes in pre-excitation, we need to think about how electrical conduction occurs in a patient with an APs.

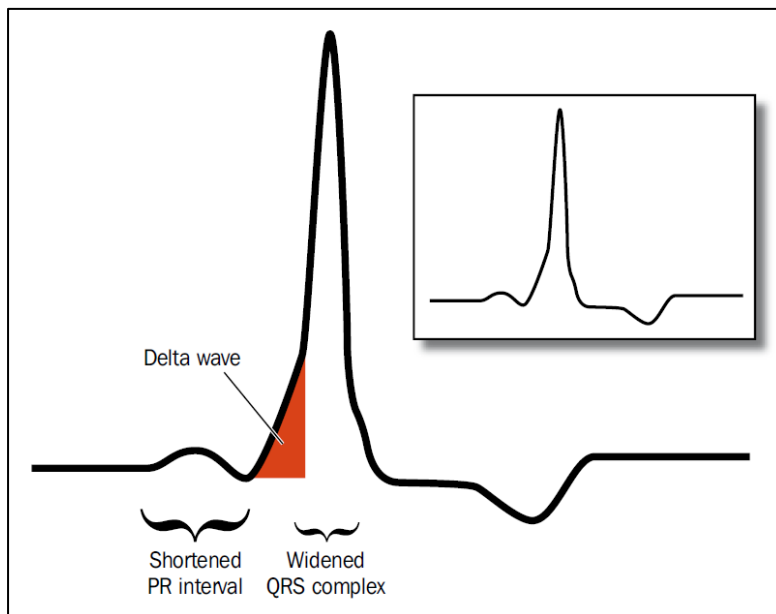


Figure 1. The principal ECG features of pre-excitation

In a normal heart, the AV node and bundle of His provide the only route of conduction between the atria and the ventricles (Klabunde, 2012). In a patient with pre-excitation, the AP provides a second route. These two routes have very different conduction properties. The AV node conducts slowly, and delays the electrical impulse on its passage to the ventricles, contributing to the normal PR interval of 120 to 200 milliseconds (Hampton, 2013). In contrast, an AP has no delaying properties (Fengler et al, 2007). An electrical impulse leaving the sinus node conducts down both the AV node and AP. Rapid conduction through the AP means that the impulse reaches the ventricle by this route while the signal travelling through the normal system is still delayed in the AV node. This shortens the PR interval (Garcia, 2015).

The early arrival of the electrical impulse in the ventricle also results in pre-excitation, which means the early depolarisation of part of the ventricle. This early activation creates the delta wave seen on the ECG (Bennett, 2013). While the electrical impulse is pre-exciting the ventricle, it is also traveling through the normal system. Once past the AV node, conduction through the rest of the system is very rapid, so the remainder of ventricular depolarisation proceeds normally (Mark et al, 2009). The latter part of the QRS complex is therefore normal in appearance. The overall result is a QRS that is a fusion of early depolarisation from the AP, and normal depolarisation via the AV node (Fengler et al, 2007). *Figure 2* illustrates conduction during pre-excitation.

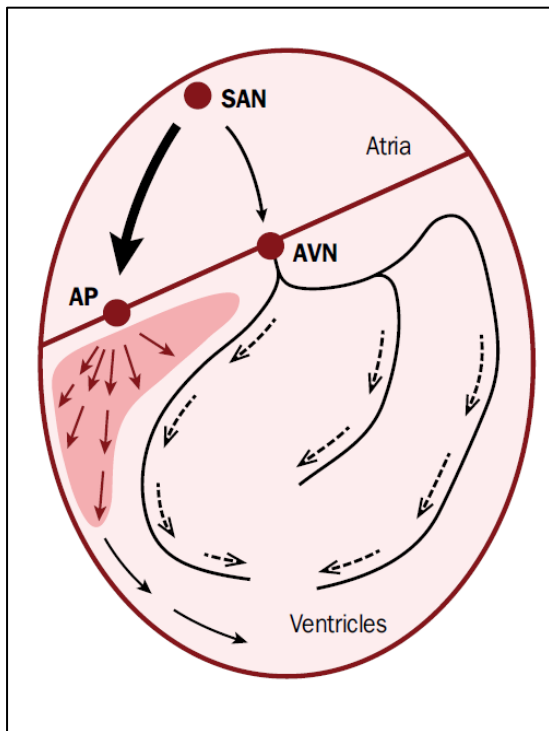


Figure 2. During sinus rhythm, conduction occurs via the AP and the AV node. The shaded area represents pre-excitation of the ventricle.

In the previous article in this series, we discussed how the pattern of QRS complexes seen on the 12 lead ECG is altered when bundle branch block occurs (Sampson, 2016). This alteration occurs because the spread of depolarisation through the ventricles occurs in an abnormal pattern, on account of the blocked bundle. An AP also changes the pattern of ventricular activation, and therefore the configuration of QRS complexes across the 12 lead ECG. The pattern produced depends on where the AP is within the heart, and can be used to predict the location of the pathway (Maden et al, 2015). This can be useful when planning catheter ablation, and in predicting the risk of the procedure (Rantner et al, 2012).

The simplest ECG classification of WPW is into type A or type B, according to the polarity of the QRS complex in lead V1. If V1 is predominantly upright, it is type A (*figure 3*). Type A is associated with left sided pathways. If the QRS is predominantly negative, it is type B (*figure 4*). In type B, the pathway is usually located in the septum or right side of the heart (Bennett, 2013). If you look at *figure 4*, you will also see that there are deep Q waves in leads III and aVF. The unusual appearance

of these leads is due to the abnormal activation of the ventricles, and not because the patient has suffered a myocardial infarction. This 'pseudo-infarct' pattern is relatively common in WPW, and can cause confusion and misdiagnosis, especially in patients presenting with chest pain (Mark et al, 2009).

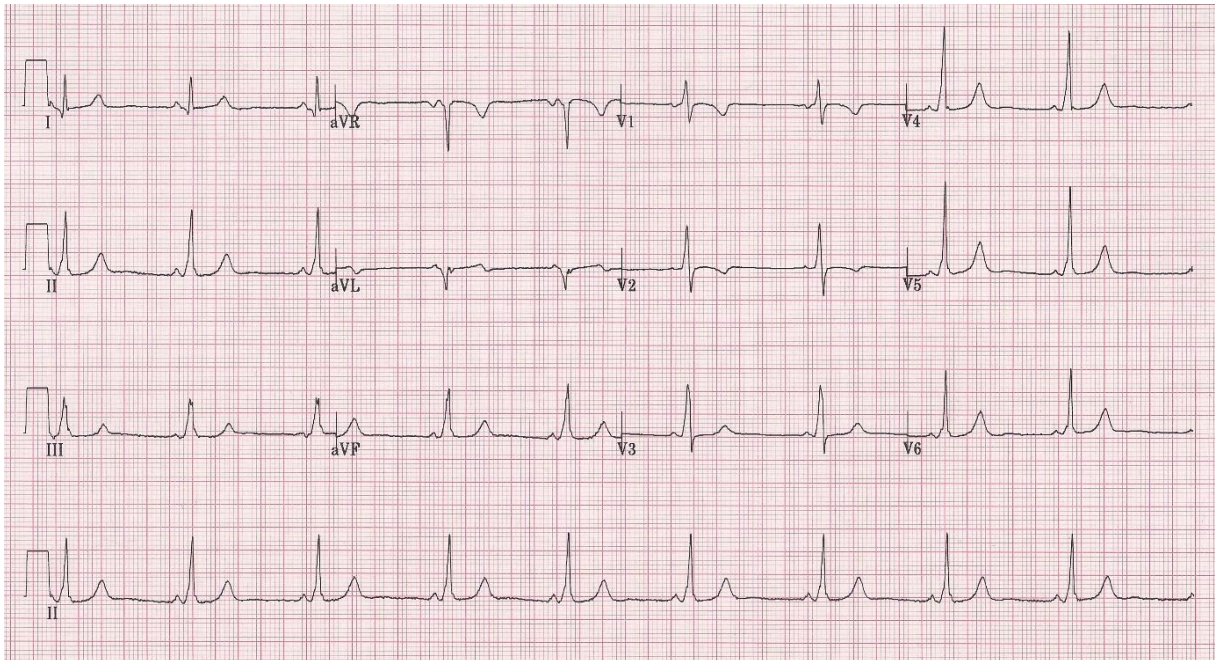


Figure 3. Type A WPW. The QRS in V1 is more positive than negative. This suggests a left-sided accessory pathway.

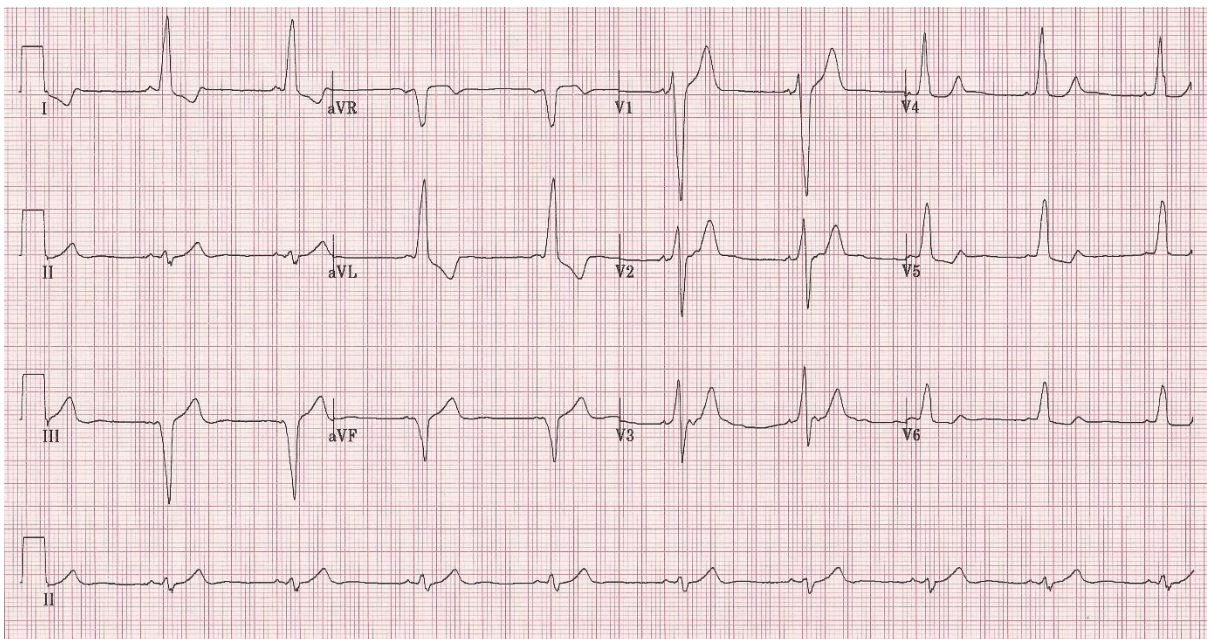
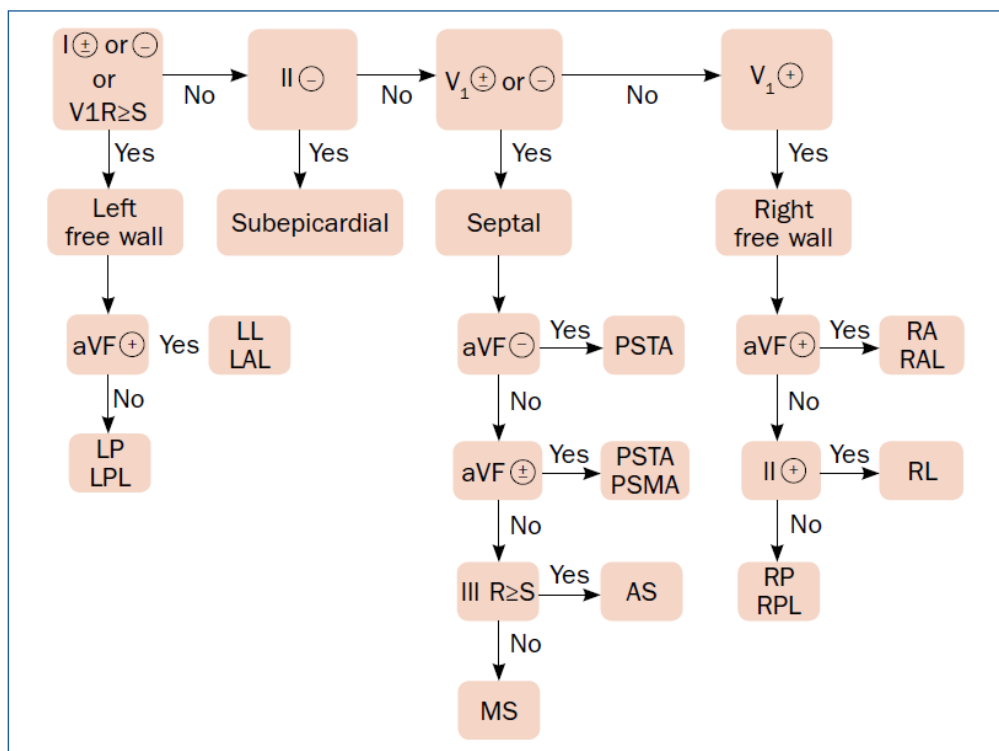


Figure 4. Type B WPW. The QRS is predominantly negative in V1. Use of the Arruda algorithm suggests that this is a right free wall pathway.

The major limitation of the type A / type B classification is lack of precision, in particular a failure to distinguish between septal and right sided pathways. To address this problem, a number of algorithms have been developed that attempt to predict with greater accuracy the location of the AP. None are completely accurate, and all are complex (Bennett, 2013). Their predictive value is especially low when the degree of pre-excitation is small, other ECG abnormalities are present, or in the case of multiple pathways (Rantner et al, 2012). A recent study compared three of the more commonly used algorithms, and found that all predicted pathway location with around 70% accuracy (Maden et al, 2015). The authors concluded that the algorithm by Arruda et al (1998) was the most useful in clinical practice. This algorithm uses evaluation of delta wave polarity in leads I, II and V1, as well as the R/S ratio in lead V1, to define the pathway as either left free wall, subepicardial, septal or right free wall. Further analysis of leads aVF, II and III then allow the user to predict a more precise location, for example a left free wall pathway may be left lateral, left anterolateral, left posterior or left posterolateral (Arruda, 1998). The full algorithm is shown in *figure 5*.



Key to pathway abbreviations					
Left free wall pathways		Septal pathways		Right free wall pathways	
LL	Left lateral	PSTA	Posteroseptal tricuspid annulus	RA	Right anterior
LAL	Left anterolateral	PSMA	Posteroseptal mitral annulus	RAL	Right anterolateral
LP	Left posterior	AS	Anteroseptal	RL	Right lateral
LPL	Left posterolateral	MS	Midseptal	RP	Right posterior
				RPL	Right posterolateral

Figure 5. Algorithm for locating an accessory pathway by Arruda et al (1998). The first part of the delta wave should be evaluated for orientation, as well as the size of the R and S waves in V1. The positive and negative symbols refer to the delta wave orientation: + is a positive delta wave, - a negative one, and +/- one that is isoelectric (does not deviate up or down from the baseline).

Acute presentation

WPW affects between one and three people in every thousand, however the majority are asymptomatic (Tischenko et al, 2008). Friedman (2009) suggests that around 50% of WPW is discovered incidentally, for example when an ECG is performed during routine screening. When symptoms do occur, they are due to paroxysmal arrhythmia, and include palpitation, dizziness and loss of consciousness (Mark et al, 2009). In rare cases, patients can present with cardiac arrest due to ventricular fibrillation (VF) (Pappone et al, 2012). The arrhythmias commonly associated with WPW are atrioventricular re-entrant tachycardia (AVRT) and atrial fibrillation (AF).

Atrioventricular re-entrant tachycardia

AVRT is the most common arrhythmia affecting people with WPW (Tischenko et al, 2008). The arrhythmia is triggered by an ectopic beat, and may be orthodromic, or antidromic, depending on the path the electrical impulse takes. In orthodromic AVRT, the electrical impulse conducts normally through the AV node, but is blocked in the AP due to its longer refractory period. By the time the impulse has entered the ventricles, the AP has recovered, and the impulse is conducted back into the atria, recreating a re-entry circuit (*figure 6*). This results in a regular, narrow complex tachycardia with P waves that are hidden, or occur after the QRS complex (*figure 7*) (Aehlert, 2011). The delta wave is not seen during orthodromic AVRT because the electrical signal is entering the ventricles only via the AV node, using the AP for return to the atria. In the acute setting, AVRT is treated by slowing conduction through the AV node, which interrupts the re-entry circuit (Medi et al, 2009). This is commonly achieved using Valsalva manoeuvres, carotid sinus massage or adenosine (Pitcher and Nolan, 2015). The orthodromic type accounts for around 90% of AVRT (Fengler et al, 2007).

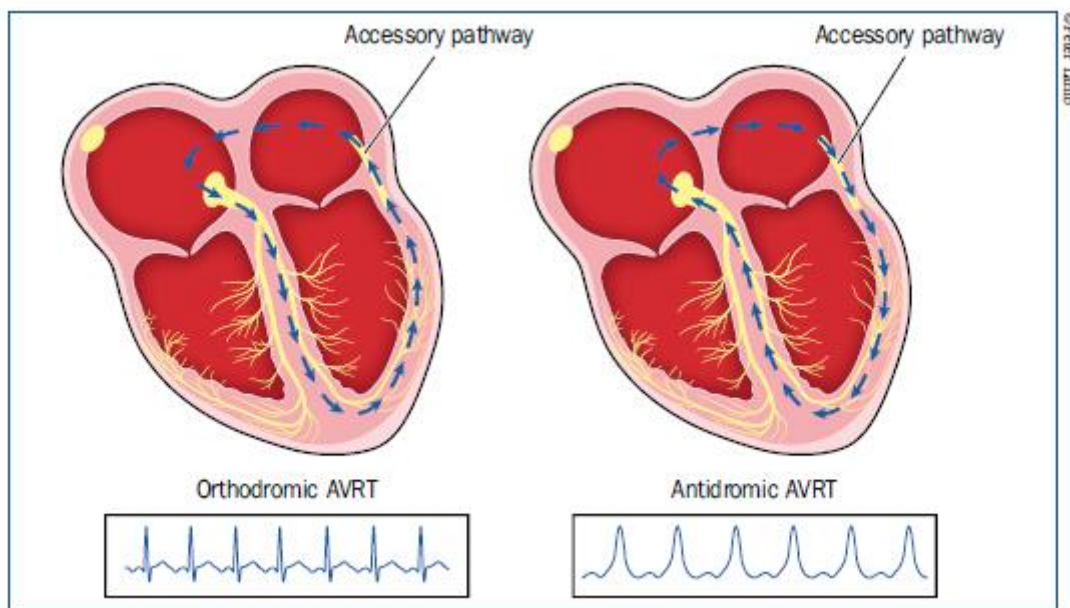


Figure 6. Orthodromic and antidromic AVRT

In the less common antidromic type, the electrical impulse travels in the reverse direction. In other words, it conducts down the AP to the ventricle, and returns to the atria via the AV node (*figure 6*). Because conduction to the ventricle is solely via the AP, the result is a regular, broad complex tachycardia (*figure 8*). This rhythm has the same appearance as ventricular tachycardia (VT), and it can be very difficult to tell the two rhythms apart (Bennett, 2013). The acute management of antidromic AVRT is identical to the orthodromic type, in other words slowing AV node conduction. Before any treatment is given, however, clinicians need to be sure that the presenting rhythm is orthodromic AVRT, and not VT (Mark et al, 2009). This is difficult, although AVRT is more likely in younger patients, in the absence of structural heart disease, and in patients who are known to have an accessory pathway. Previous ECGs should be obtained and evaluated. If in doubt, the rhythm should be treated as VT (Whinnett et al, 2012).



Figure 7. Orthodromic AVRT results in a regular, narrow complex tachycardia in which P-waves are often not seen. In this example, there appears to be an inverted P-wave in the ST-segment. On the ECG, it is often impossible to distinguish AVRT from other forms of SVT, for example AVNRT.

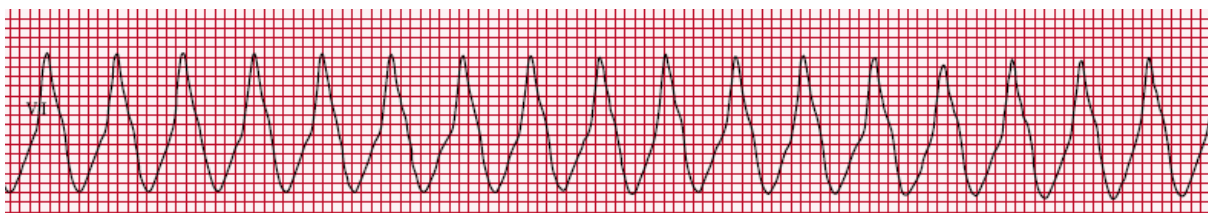


Figure 8. Antidromic AVRT. The QRS is wide because the impulse is entering the ventricles via the AP, and returning via the normal conduction system. It is difficult to distinguish antidromic AVRT from VT.

Atrial fibrillation

For reasons that are poorly understood, AF is more common in people with accessory pathways than in similar people without them (Thanavaro and Thanavaro, 2010). AF may occur due to degeneration of AVRT, or may occur spontaneously. If the AP is capable of anterograde conduction, AF may be conducted down the pathway, as well as through the AV node. This is a potentially dangerous situation, given that accessory pathways do not have the delaying properties of the AV node (Fengler et al, 2007). In pathways that conduct rapidly, heart rates in excess of 300 beats per minute are possible (Mark et al, 2009). Heart rates this fast cause severe haemodynamic compromise, and can degenerate into VF. Rapid conduction of AF, with subsequent degeneration to VF, is the mechanism of SCD in individuals with WPW (Bennett, 2013).

On the ECG, pre-excited AF can be recognised by a rapid, irregular, broad complex tachycardia (*figure 9*). The QRS complex is a fusion of the impulses passing down the accessory pathway and the

AV node. This results in subtle beat to beat variation in QRS morphology, caused by variation in the degree of conduction via the two routes (Thanavaro and Thanavaro, 2010). Unlike polymorphic VT, the QRS complexes do not twist around the baseline. Another differential diagnosis for this ECG appearance is AF conducted with aberrancy (e.g. bundle branch block), however this usually results in a stable QRS morphology (Mark et al, 2009).

The acute treatment of pre-excited AF depends on presenting symptoms. Stable patients can be treated with anti-arrhythmic drugs such as sotalol, flecainide and amiodarone (Bennett, 2013). Drugs that block or slow AV conduction should be avoided as they can increase conduction down the AP, resulting in higher heart rates, and a greater risk of VF. Drugs to avoid include beta-blockers, diltiazem, verapamil, digoxin and adenosine (Hashimi et al, 2014). If the patient is haemodynamically unstable, immediate DC cardioversion is recommended (Pitcher and Nolan, 2015). VF warrants immediate defibrillation and cardiopulmonary resuscitation (Soar et al, 2015).

Any patient who has been treated for AVRT or pre-excited AF should have a 12 lead ECG recorded during sinus rhythm to check for pre-excitation. Referral should be made to a cardiologist specialising in electrophysiology. This will ensure that the patient undergoes expert evaluation, and that appropriate long term treatment is offered (Whinnett et al, 2012).

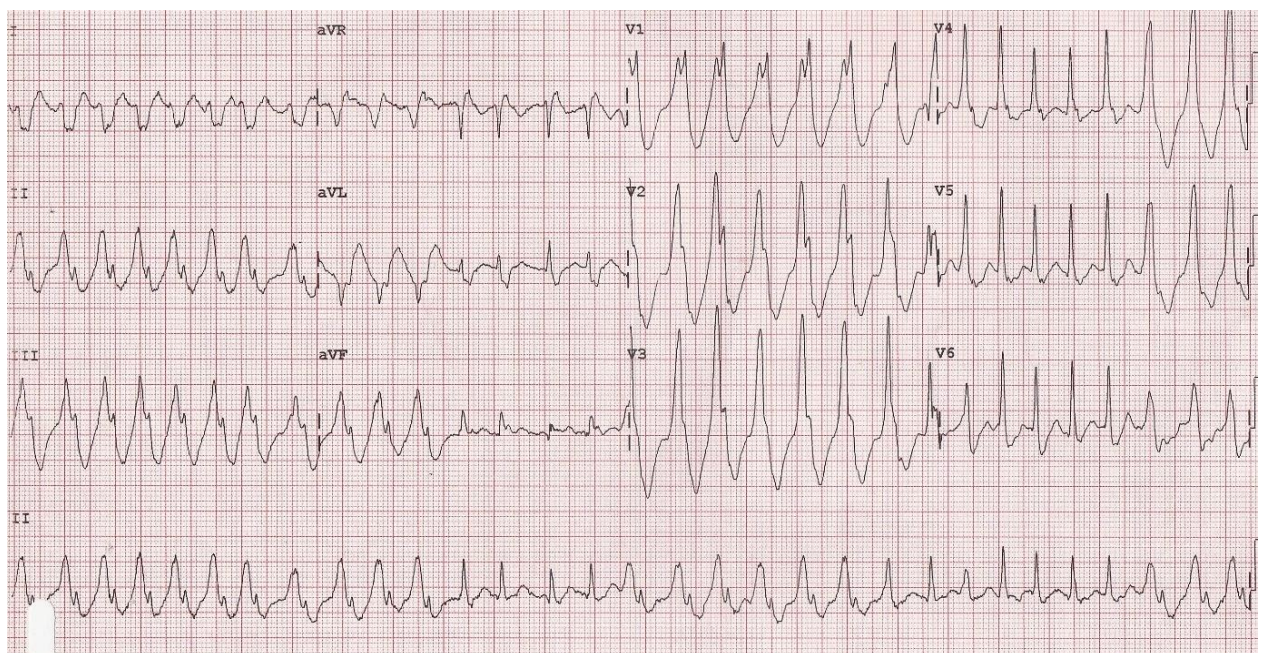


Figure 9. Pre-excited AF. Note the irregular rhythm, and slight variation in QRS morphology. QRS complexes 12 to 15, 23, and 25 to 28 are narrow, suggesting that they have conducted through the AV node only. Intermittent pre-excitation is relatively common.

Long term management of WPW

The long term management of WPW depends on the degree of symptoms, as well as the risk of SCD. Although some people report no symptoms, and others only mild or infrequent episodes, some individuals suffer frequent visits to the Emergency Department, and describe severe impairment of their quality of life (Walfridsson et al, 2009). A loss of confidence is common, and a fear that an episode will occur in a situation where medical attention is not readily available - for example on an aeroplane flight, or foreign holiday (Wood et al, 2010). The individual's usual lifestyle can be severely curtailed as a result, with patients declining to travel or even giving up work and social activities. Symptomatic individuals are also thought to be at higher risk of SCD, compared to those who have no symptoms (Obeyesekere et al, 2012). In these individuals, there is therefore a pressing argument for definitive treatment to reduce the risk of death, as well as to improve quality of life. In patients without symptoms, the case for treatment is much less clear cut (Triedman, 2009).

Treatment of WPW

Until the late 1980s, the only treatment choices for individuals with symptomatic WPW were drugs or surgery (Scheinman, 2005). Although drugs are fairly effective at reducing symptoms, they only prevent them entirely in 50-60% of people (Tischenko et al, 2008). They have no effect on the risk of SCD (Triedman, 2009). Surgery, while highly effective, requires thoracotomy and is therefore associated with unacceptable risk and recovery factors for many patients.

Fortunately, a third option has been available for the past 30 years in the form of catheter ablation. Using a minimally invasive approach, catheters placed inside the heart chambers are used to locate and modify the AP, usually under local anaesthesia (Lee and Linker, 2014). This procedure has a high success rate, low risk of complication, and is the recommended treatment for symptomatic WPW in current guidelines (Blomström-Lundqvist et al, 2003).

A typical ablation for WPW starts with the placement of three or four catheters via the femoral veins. Usual catheter positions include the high right atrium, right ventricular apex, adjacent to the bundle of His, and within the coronary sinus (CS) (*figure 10*). Because the CS runs around the AV groove into the left side of the heart, the CS catheter allows the measurement of electrical activity in the left side of the heart, without actually entering the left sided chambers (Joseph and Rajappan, 2011).

Once catheters have been placed, an electrophysiological (EP) study is performed. This is basically an electrical test of the heart. The conduction properties of the normal conduction system and AP are measured, and then rapid pacing is used to try to induce the clinical arrhythmia (Chugh et al, 2008). This process provides information about the location of the AP, its role in the clinical arrhythmia, and its ability to support rapid conduction. The faster the AP conducts, the greater the risk of SCD should AF occur (Triedman, 2009). Once the EP study is concluded, an ablation catheter is moved to the insertion point of the AP in either the atrium or ventricle. In the case of right sided pathways, this is relatively straightforward as the venous system gives direct access to the right side of the heart. For left sided pathways, the left heart must be accessed either by trans-septal puncture (using a needle to pierce the septum between right and left atrium) or a catheter must be advanced via the femoral artery and aorta into the left ventricle (Lee et al, 2013).

Once the ablation catheter is located at the insertion of the AP, radiofrequency (RF) energy is delivered (Joseph and Rajappan, 2011). If the correct point has been identified, and if catheter

contact is adequate, the cells underlying the catheter tip are destroyed and conduction through the AP ceases. On the surface ECG, pre-excitation should disappear. It may take multiple attempts to accurately locate the pathway, and therefore a number of ‘burns’ may be delivered before the AP is successfully ablated (Chugh et al, 2008). Each burn lasts up to 60 seconds, and patients may experience sharp chest pain during energy delivery, despite the use of opiates and benzodiazepines (Joseph and Rajappan, 2011; Lee and Linker, 2014). If long term success is achieved, the patient will be free of AVRT. The risk of AF is reduced, although not removed, however rapid conduction to the ventricles can no longer occur, so the risk of sudden death is reduced to normal population levels (Thanavaro and Thanavaro, 2010).

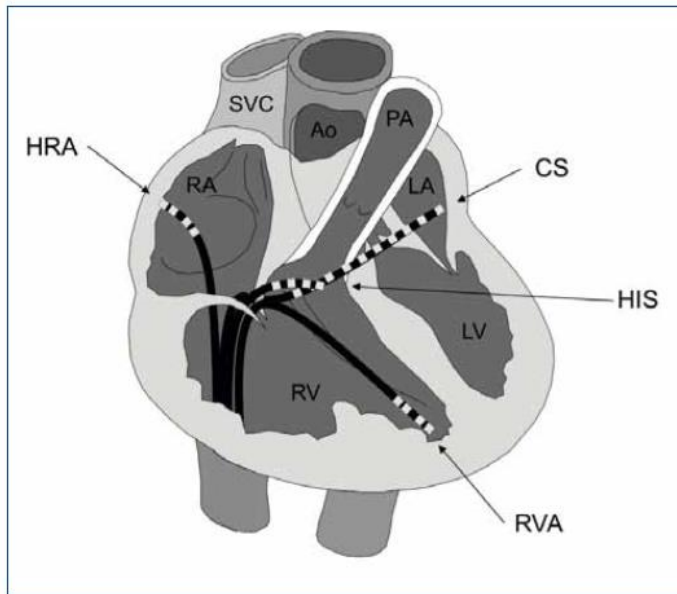


Figure 10. Typical catheter placement during an EP study.

The success rate for ablation of WPW is widely quoted as 95%, with 5% of patients needing a repeat procedure (Obeyesekere et al, 2012; Tischenko et al, 2008; Triedman, 2009). Complications are infrequent, occurring in 2-4% of people. A meta-analysis by Spector et al (2009) studied the outcomes of AP ablation using RF, and found similar rates of success and complication. The risk of procedure-related death in this analysis was 1 in 1000. The commonly reported complications of AP ablation are shown in *table 1*.

Type of complication	Frequency
AV block	0.8%
Vascular access complications	0.7%
Stroke, TIA or embolic event	0.4%
Tamponade	0.4%
Permanent pacemaker	0.3%
Pericardial effusion	0.2%
Total	2.8%

Table 1. Complications of catheter ablation for WPW (Spector et al 2009).

Although some of these risks are universal, for example vascular access problems, in other cases the degree of risk depends on the location of the pathway. Left heart ablation is associated with a small risk of stroke that is not relevant to right sided procedures. The risk of AV block and pacemaker implantation is highest in antero- and mid-septal pathways, which are close to the normal conduction system (Chugh et al, 2008). This risk may be reduced by the use of cryoablation instead of RF. Cryoablation allows the electrophysiologist to apply a reversible freeze to determine whether catheter placement is correct, and whether there is any adverse effect on normal conduction (Avitall and Kalinski, 2015). If a mild freeze halts AP conduction without inducing AV block, lower temperatures can be applied, resulting in a permanent lesion. The downside of this approach is a slightly higher rate of repeat ablation, however this may be preferable to a higher risk of pacemaker implantation (Andrade et al, 2013).

Asymptomatic pre-excitation

In the absence of symptoms, patients with pre-excitation derive no benefit from medication. Whether they will benefit from ablation depends largely on the perceived risk of SCD. Longitudinal studies of WPW populations suggest that the risk of sudden death is approximately 3-4% over a lifetime, or 0.1% per year (Obeyesekere et al, 2012). Asymptomatic patients are generally considered lower risk than those with symptoms of arrhythmia. Nonetheless, 40-50% of patients presenting with cardiac arrest due to WPW have no previous symptoms, suggesting that lack of symptoms is not a reliable indicator of risk.

A more useful indicator appears to be loss of AP conduction at higher heart rates (Refaat et al, 2014). A simple exercise test can be used to determine this. If pre-excitation disappears at higher sinus rates, the pathway cannot support rapidly conducted AF, and is deemed relatively safe (Triedman, 2009). If exercise testing is not helpful, an EP study can provide further information. Failure to induce an arrhythmia during a study suggests lower risk, however the effective refractory period (ERP) of the pathway is considered a more reliable indicator. An ERP of 250ms or more indicates that the pathway cannot conduct at the speeds that result in degeneration to VF, and the pathway is considered low risk (Tischenko et al, 2008).

Although risk stratification is useful in defining patients who are more likely to benefit from ablation, a number of other factors must also be considered. Firstly, research suggests that 15-21% of asymptomatic patients will develop symptoms over the following two decades (Tischenko et al, 2008). Patients may prefer to be treated now, rather than wait to see if this happens. Secondly, patients may have a strong preference for taking the small but one-off risk of ablation, rather than the small but indefinite risk of SCD. Thirdly, the presence of pre-excitation on the ECG may bar people from certain activities, for example professional flying or competitive sport (Chevalier et al, 2013; Link, 2009). Finally, the location of the pathway must be considered. Ablation of a pathway close to the His bundle carries a higher risk of AV block and pacemaker dependency, which will be especially undesirable for young or athletic patients. Thorough assessment of the individual, as well as the pathway, is therefore essential.

Conclusion

Pre-excitation is an important finding on the ECG, and is associated with an increased risk of paroxysmal arrhythmias and sudden death. For this reason, a search for the signs of pre-excitation is an essential part of any systemic evaluation of the ECG. All patients found to have pre-excitation should be referred to an electrophysiologist for expert appraisal and risk assessment. For those with symptoms, catheter ablation is recommended by European guidance and has a good success rate, with a low rate of complication. For asymptomatic patients, the decision to treat is more difficult, and should be based on risk stratification of the AP, as well as patient preference, occupation and lifestyle.

Next month, we turn our attention to the QRS axis, discussing how it can be evaluated on the ECG, and what it can tell us about the condition of the heart and conduction system.

Key points

- Pre-excitation is caused by an accessory pathway, a strand of myocardium that joins the atria to the ventricles, providing an alternative route of conduction for the electrical impulse. It is often associated with paroxysmal atrioventricular re-entrant tachycardia (AVRT) or atrial fibrillation (AF), in which case it is referred to as Wolff-Parkinson-White syndrome (WPW). It is a congenital condition, often found in otherwise normal hearts.
- The ECG features of WPW are a short PR interval, delta wave, wide QRS, and ST and T-wave abnormalities. Changes in ventricular activation also alter the pattern of QRS complexes, and may mimic myocardial infarction. QRS pattern may be helpful in predicting pathway location.
- WPW is associated with a 3-4% lifetime risk of sudden cardiac death (SCD). The mechanism for sudden death is rapid conduction of AF to the ventricles, with subsequent degeneration to ventricular fibrillation (VF). Patients that present with pre-excited AF should not be treated with AV nodal blocking agents, as this can increase the heart rate and risk of VF.
- Catheter ablation is the first line treatment for symptomatic WPW, and has a 95% success rate with a complication rate of 2-4%. The risk of some complications is universal, the risk of others depends on the location of the accessory pathway.
- The treatment of asymptomatic patients is more difficult, and depends on the risk of SCD. Pathway conduction properties and location should be considered, as well as patient age, preference, occupation and leisure pursuits.

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