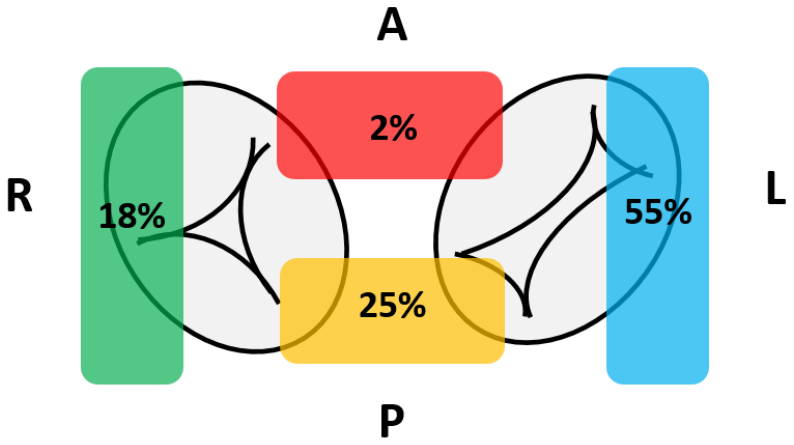
Atrioventricular re-entrant tachycardia (AVRT): what the general cardiologist needs to know

Mechanisms

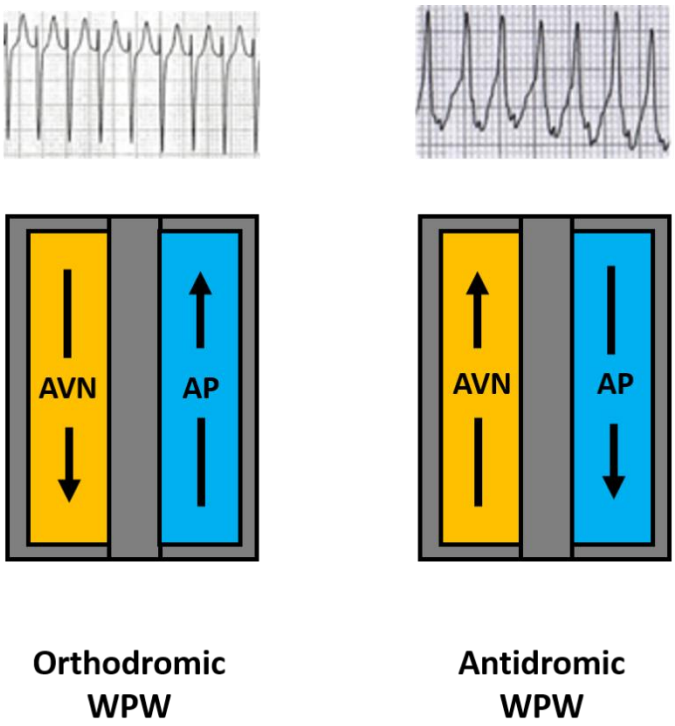
Atrioventricular re-entrant tachycardias (AVRT), otherwise known as Wolff-Parkinson White (WPW), refer to the formation of a circuit by the existence of a relatively fast-conducting aberrant accessory pathway (Bundle of Kent) [1]. This connects atria and ventricles at sites where they should be electrically isolated. An accessory pathway typically arises from a congenital defect in atrio-ventricular (AV) segmentation and in the development of the fibrous AV rings [2]. Hence, they breach the insulation typically provided by fibrofatty deposition in the AV groove and fibrous annulus of the mitral and tricuspid valves. They are found most frequently in the left lateral regions. Most of the pathways are constructed of functional myocardium with normal gap junctions, as opposed to histologically discrete cells. Impulse transmission initiated from the sinoatrial node (SAN) is ordinarily via both atrioventricular node (AVN)-His axis and accessory pathway simultaneously, albeit with differing velocities and with variable preference. This is in the context of patent pathways which conduct in an A:V fashion. However, concealed pathways also exist which solely conduct retrogradely (i.e. V:A). A separate example of a concealed pathway relates to the distinct type that occurs in the context of permanent junctional reciprocating tachycardia (PJRT) [3]. Nonetheless, it should be emphasised that most pathways have the ability to be bidirectional.



Anatomical distribution of accessory pathways in AVRT

Categorisation

There are various ways to classify WPW. Initially, it can be useful to distinguish orthodromic from antidromic WPW, with 95% of cases being the former [4]. In these instances, an atrial ectopic beat is propagated by the AVN-His axis in the usual antegrade fashion but there is subsequent retrograde conduction via the accessory pathway. Alternately, a ventricular ectopic beat is propagated retrogradely as most pathways have the ability to be bidirectional. This results in a narrow complex tachycardia (NCT). In rarer instances, the accessory pathway is the anterograde limb with use of the AVN retrogradely. In view of this, there is early ventricular activation (termed 'pre-excitation') with faster conduction than via the normal AVN-His axis. However, the term describes the existence of anomalous conduction even if the velocity is no faster than usual. Because subsequent ventricular depolarisation is propagated directly via myocardial cells rather than the specialised conduction system, it will manifest as a broad complex tachycardia (BCT). Indeed, it becomes particularly challenging to distinguish from ventricular tachycardia.



Mechanisms implicated in orthodromic and antidromic WPW

Factors modifying pre-excitation

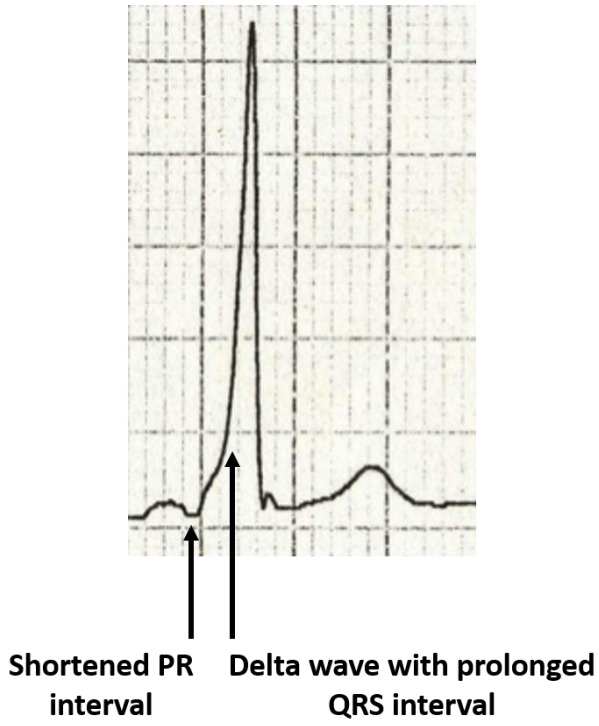
The degree of ventricular pre-excitation in the context of antidromic WPW is affected by several factors. Firstly, the anatomical location of the accessory pathway has important influence [5]. The closer the accessory pathway is to the SAN or site of atrial ectopy, the greater the degree of pre-excitation. Hence, pathways originating from the right free-wall conventionally present with a shortened PR interval and wide pre-excited QRS complexes, whilst left free-wall pathways mimic normal electrophysiology more closely. Invasive pacing at a region in close proximity to the pathways may enable clearer manifestation of pre-excitation. If there is existence of atrial fibrosis or enlargement, pre-excitation may not be evident in those with a left free-wall pathway due to the prolonged duration for impulse propagation. Intrinsic properties of the pathway itself, namely conduction velocity and length, can affect conduction times over the pathway. For instance, there is a co-association of WPW with Ebstein anomaly, where the septal and posterior leaflets of the tricuspid valve are displaced towards the right ventricular apex [6]. In this context, pathways may be longer despite propagating at higher velocity. Lastly, pre-excitation is also determined by the relative conduction over the normal AVN-His axis. If there is enhanced AVN blockade, for example, manifestation of pre-excitation is enhanced.

Diagnosis

Over 40% of individuals aged greater than 30 years with a WPW pattern on resting ECG are asymptomatic [7]. WPW can only be described as a syndrome if there are concurrent symptoms such as palpitations. In addition to Ebstein anomaly, there is an association with other congenital disorders such as hypertrophic cardiomyopathy (HCM), coronary sinus diverticulum and tuberous sclerosis [8]. However, there is no strong hereditary predisposition.

The presence of a manifest accessory pathway may be confirmed incidentally on the ECG of an asymptomatic patient. In this case, it would demonstrate a shortened PR interval, widened QRS complex and slurring of the initial segment caused by early ventricular depolarisation (delta wave) that is classically associated with antidromic WPW. Clinical presentation for acute episodes is similar to that described above for other NCT, with palpitations, breathlessness or chest pain. ECG on admission will demonstrate a NCT, but it is typically challenging to discriminate AVRT from other differentials. Rate is usually in the region of 140-240 bpm, and characteristically, associated with a shortened RP interval.

Once the patient has reverted into normal sinus rhythm, either spontaneously or with therapy, the subsequent ECG can provide useful indicators regarding anatomical location of the accessory pathway. Generally, a useful rule is that negative delta waves point away from the earliest site of ventricular activation, i.e. the insertion point of the pathway [9]. Hence, if the pre-excited QRS complex is predominantly positive in V1, it usually originates from left-sided pathways (Type A). If it is negative in the inferior leads, the pathway is left paraseptal or left inferior. Left posterior pathways produce positive deflections in the inferior leads. Right-sided pathways result in predominantly negative complexes in V1 (Type B). As with AVNRT, triggering of entrainment via electrophysiological testing is also beneficial in confirming the existence of a re-entry circuit.



Classic delta wave associated with ventricular pre-excitation in antidromic WPW

Management strategies

For an acute episode, treatment is consistent with that of other NCTs and involves transient AVN blockade using vagal manoeuvres and/or adenosine. If there is an established history of WPW, intravenous flecainide may be preferred. If there is any suggestion of adverse features, synchronised electrical cardioversion is warranted without the need for preceding anticoagulation. Sudden death is exceptionally rare, but when it does occur, is usually related to the co-existence of atrial fibrillation (AF) with 1:1 antegrade conduction along the accessory pathway resulting in pre-excitation and ventricular fibrillation [10]. In these

patients, conventional AVN blockade is of direct detriment by enhancing propagation along the accessory pathway.

If symptoms are infrequent or have a clear onset, 'pill in the pocket' use of flecainide as with AVNRT is entirely appropriate, assuming that ventricular dysfunction and significant coronary disease have been excluded. The definitive treatment option for patients with WPW syndrome is catheter ablation. Nonetheless, flecainide, amiodarone or sotalol can be utilised as interim options if symptoms are frequent, intrusive or unlikely to respond to simple vagal manoeuvres. The success rate of ablation is > 98%, with minimal morbidity and mortality [11]. Left-sided accessory pathways are usually ablated using a retrograde aortic approach, though trans-septal access may also be achieved. In contrast, right-sided pathways require access via the venous system, and are associated with slightly higher rates of recurrence. Superior paraseptal and peri-Hisian accessory pathways are associated with risk of iatrogenic AVN blockade, and detailed electrode mapping pre-intervention is therefore of paramount importance.

Evidence for ablation in patients with truly asymptomatic WPW is more controversial [12]. Holter monitoring and exercise testing is generally helpful, with clear and abrupt loss of pre-excitation suggestive of lower dysrhythmic risk [13]. These cohorts may simply require periodic cardiology follow-up with advice regarding symptom awareness. Co-existent structural heart disease confers the propensity for unfavourable haemodynamics and ablation is generally favoured. In asymptomatic individuals, a randomised controlled trial observed less arrhythmic events (5% versus 60%) in patients who had ablation compared to those that had no treatment [14]. However, no guidelines are currently available to guide a definitive management strategy.

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