

ATRIOVENTRICULAR REENTRANT TACHYCARDIA

Definitions

The anatomical basis for atrioventricular reentrant tachycardia (AVRT) is an abnormal connection (**accessory pathway**) between atrial and ventricular myocardium. One limb of the reentrant circuit is the AV node and the other is the anomalous connection. On rare occasions, the circuit constitutes of two accessory pathways.

The term **preexcitation** refers to activation of the ventricle, by an impulse arising in the atrium, earlier than would be expected if conduction occurred via the normal atrioventricular conduction pathway.

Wolff-Parkinson-White (WPW) syndrome refers to the presence of an overt (manifest) AP, thus resulting on the so-called pre-excitation, in combination with usually recurrent tachyarrhythmias (Figure 1).¹ The AP is located along the mitral or tricuspid annulus and during sinus rhythm a typical pattern in the resting ECG with the following characteristics is present: (1) short PR interval (≤ 120 ms), (2) slurred upstroke (or downstroke) of the QRS complex (“delta wave”), and (3) a wide QRS complex (> 120 ms). However, WPW patients with a *fasciculo-ventricular* pathway have a QRS width ≤ 120 ms.^{2,3} Occasionally, pre-excitation may not be fully apparent due to fusion of wavefronts progressing through the AP and the normal conduction system. In most cases, accessory pathways giving rise to the WPW pattern are seen in structurally normal hearts.

Pathophysiology and Classification of Accessory Pathways

APs are single or multiple strands of myocardial cells that bypass the physiological conduction system and directly connect atrial and ventricular myocardium.⁴ These AV connections are due to incomplete embryological development of the AV annuli, without complete separation between the atria and ventricles. There are different types of APs.⁵ The most common ones connect the atrium and the ventricle along the mitral or tricuspid annulus. Approximately 60% are located along the mitral valve and are referred to as left free wall APs, 25% insert along the septal aspect of the mitral or tricuspid annulus, and ~15% insert along the right free wall.⁶⁻⁸ Because ventricular muscle is lacking in the proximity of the anterior leaflet of the mitral valve, left-sided APs are usually limited to the region of the mitral annulus at the attachment of the mural (posterior) leaflet. APs can be located in the superoparaseptal area in close proximity to the His bundle and AV node.⁹

Overt pathways produce an abnormal ventricular activation, which leads to an altered pattern of repolarization. These manifestations may mimic myocardial infarction or

mask the ST-T changes of acute myocardial infarction (Figure 2).¹⁰ Secondary ST-T changes are most prominent in the leads with the delta waves and are discordant, that is, in a direction opposite to the delta wave. Concordant changes or the presence of reciprocal changes is abnormal and suggests myocardial ischemia. In patients with WPW syndrome, early repolarization patterns may be present and could be partly related to the early depolarization through the accessory pathway.¹¹

Accessory pathways exhibit rapid conduction. Approximately 50% of APs conduct both antegradely and retrogradely, and the majority of the others conducted only in the retrograde direction are labelled as “**concealed**” since there is no evidence of preexcitation on the ECG. A small percentage conduct only in the anterograde direction. Concealed APs give rise only to orthodromic AVRT, and occasionally have decremental properties.¹² They are not associated with an increased risk of sudden cardiac death. No gender predilection is found and these pathways tend to become clinically apparent at an earlier age than AVJRT; however, significant overlap exists¹³. Concealed APs are predominantly localized along the left free wall (65%), and less frequently at septal (30%) and right free wall locations.^{4,12}

Multiple APs occur in up to 12% of patients with preexcitation, and in up to 50% in patients with Ebstein’s anomaly.¹⁴

Characteristics suggestive of two or more Aps are:

1. Delta wave pattern not typical of any single location.
2. Changes in delta wave during AF or during right atrial vs. CS pacing.
3. Retrograde fusion during ORT.
4. Changes in retrograde atrial activation sequence during orthodromic AVRT, either spontaneous or during RFA.

Atypical APs (also called Mahaim fibres) are connections between the RA or the AVN and the RV into or close to the right bundle branch.¹⁵⁻¹⁷ Pathways with atypical characteristics can be atriofascicular, nodofascicular or nodoventricular, depending on their proximal and distal insertions.^{15,16} Left-sided atypical pathways have also been described but are extremely rare.¹⁸⁻²⁰ They usually contain accessory nodal tissue which results in decremental properties, and connect the atrium to the fascicles by crossing the lateral aspect of the tricuspid annulus, but posteroseptal locations can also be found in rare cases. Conduction is usually anterograde only, but concealed nodoventricular and nodofascicular fibres have also been described, and may give rise to incessant tachycardias.^{21,22} The baseline QRS of patients with atypical APs is normal or displays different degrees of manifest pre-excitation with LBBB morphology. Programmed or incremental atrial pacing results in manifest pre-excitation when there has been sufficient delay in the AH interval. Atypical pathways can participate in antidromic AVRT.

Accessory pathways are a congenital disorder. Rarely, atrioventricular connections can be iatrogenic following a Fontan operation.²³ Rare, familial forms of preexcitation associated with left ventricular hypertrophy and multisystem disease (mutations in the *PRKAG2* gene, Danon and Fabry disease and others) have also been described.²⁴ A missense mutation in the gene that encodes the gamma 2 regulatory subunit of AMP-activated protein kinase (*PRKAG2*) has been associated with ventricular preexcitation and conduction disease.²⁵ A particular mutation in *PRKAG2* has also been associated with Mahaim (nodoventricular fibers).²⁵ A novel form of WPW syndrome is associated with microdeletion in the region of gene *BMP2*, that encodes the bone morphogenetic protein-2, a member of the transforming growth factor (TGF- β) gene superfamily, and affects the development of annulus fibrosus. It is characterized by variable cognitive deficits, dysmorphic features, and prolonged AV conduction on atrial pacing.²⁵ Atrioventricular AP with high-risk characteristics, multiple AP, and the coexistence of AP and fasciculoventricular fibers can be found in patients with HCM secondary to underlying storage disease, metabolic disease, genetic syndromes, and in patients with isolated HCM.²⁶

Presentation

Patients present with symptoms of paroxysmal tachycardia or with preexcited AF. AVRT is the most common tachycardia associated with the WPW syndrome. WPW predisposes to development of AF which may or may not be eliminated following ablation of the pathway.^{27,28} It is more frequent in patients with WPW syndrome than in the general population, and may be the presenting arrhythmia in affected patients²⁹. These patients are typically young and have no structural heart disease. During AF, antegrade conduction over the accessory pathway results in a wide QRS complex tachyarrhythmia which may be mistaken for ventricular tachycardia. AF with fast ventricular response over an overt AP with a short anterograde refractory period is a potentially life-threatening arrhythmia in patients with WPW syndrome due to potential degeneration into VF.³⁰

Up to 50% of patients with WPW syndrome may be asymptomatic,³¹ and diagnosis is made after an incidental ECG. Loss of antegrade conduction capacity occurs with time in up to 30% of patients,³¹ but when preexcitation remains, the frequency of tachycardia increases with age.³²

Diagnosis

Patients with concealed pathways or Mahaim fibers, have completely normal resting ECG. Antegradely conducting pathways may produce the typical WPW pattern or may be latent. Latent pathways, when conducting antegradely, may be revealed by infusion of adenosine or

sometimes isoprenaline. In the presence of a WPW pattern, localization of the AP may be accomplished from the 12-lead, surface ECG (Figures 3 and 4).

Orthodromic AVRT refers to a re-entrant tachycardia that uses the AV node-His bundle axis as the antegrade limb and the AP as the retrograde limb (Figure 5). Orthodromic AVRT accounts for >90% of AVRTs, and 20-30% of all sustained SVTs.^{33,34} The rate usually is 170-220 bpm. During tachycardia, the QRS is narrow unless there is a functional or underlying BBB. ST segment depression often is present and has been shown to usually not be due to myocardial ischemia.³⁵ Ipsilateral bundle branch block during AVRT results in an increase in VA time, which can also increase the CL of the tachycardia, although no change in rate may also be seen due to a compensatory reduction in AH interval. A contralateral bundle branch block has no effect on the tachycardia as it is not a part of the tachycardia circuit.

Antidromic AVRT refers to a re-entrant circuit that uses the AP as the antegrade limb, and the AV node-His bundle axis (or, rarely, another AP) as the retrograde limb (Figure 6). Antidromic AVRT occurs in 3-8% of patients with WPW syndrome.³⁶ In patients with spontaneous antidromic AVRT, multiple APs (manifest or concealed), which may or may not serve as the retrograde limb of the AVRT may be present. Antidromic AVRT is a wide QRS complex (fully pre-excited) tachycardia. In the presence of AT, AFL, AF or AV nodal re-entry tachycardia, the QRS complexes can also be pre-excited when the AP acts as a **bystander** and is not a critical part of the re-entry circuit.

Permanent junctional reciprocating tachycardia (**PJRT**) represents a rare form of a long RP tachycardia using a concealed AP. Usually these APs are located in the posteroseptal region and are associated with retrograde decremental conduction properties.³⁷ The incessant nature of PJRT may result in tachycardiomyopathy that usually resolves after successful treatment by RF catheter ablation, particularly in younger patients.^{37,38}

Left ventricular dysfunction may be related to electrical asynchrony and subsequent **abnormal conduction-induced cardiomyopathy** in patients, especially children, with asymptomatic preexcitation.³⁹⁻⁴¹

Therapy

Acute

The acute management of patients with AVRT is as discussed in the Chapter on the management of SVT in general (Figures 7 and 8 and Table 1). However, adenosine should be used with caution because of potential induction of fast AF⁴²⁻⁴⁴. AF with fast ventricular conduction could also induce VF, therefore electrical cardioversion should always be available. During orthodromic and antidromic AVRT, drug therapy could be directed at one of the components of the circuit, the AVN (beta-blockers, diltiazem, verapamil, etipamil),⁴⁵⁻

⁴⁸ or the AP (ibutilide, procainamide, propafenone, flecainide).^{49,50} Antidromic AVRT is associated with malignant WPW syndrome due to very fast conducting AP,³⁶ and drugs acting mainly on the AP should be probably preferred. In addition, in case of antidromic AVRT with APs representing both the anterograde and retrograde limb, drugs acting on the AVN are ineffective. In drug refractory antidromic AVRT, amiodarone may also be considered.⁵¹⁻⁵³

In patients presenting with **pre-excited AF** urgent correction is usually required, and the threshold for the use of electrical cardioversion is lower. Conduction of electrical impulses can occur preferentially via the AP due to its shorter RP when compared to the AVN.⁵⁴ Thus, any AV node-modulating agents (adenosine/verapamil/beta-blockers/digoxin) should be avoided since they may contribute to a risk of ventricular fibrillation (VF).^{55,56}

Pharmacological cardioversion of preexcited AF or delayed AP conduction can be achieved with ibutilide (Figure 9 and Table 2).⁵⁷ Drugs such as procainamide, propafenone or flecainide that affect conduction over the accessory pathway may also be used, even if they may not restore sinus rhythm.⁵⁸⁻⁶⁰ However, class Ic agents should be used with caution since they do exert an effect on the AV node. In pre-excited AF, i.v. amiodarone may not be as safe as previously thought, since enhanced pathway conduction and VF have been reported, and should not be considered.⁶¹⁻⁶⁴ Procainamide appears to be safer in this setting.⁶⁵

Chronic therapy

If ablation is not desirable or feasible in patients with pre-excitation and symptomatic antidromic AVRT, and in whom structural or ischaemic heart disease has been excluded, class IC drugs act mainly on the AP and can be used in antidromic tachycardia.^{66,67} In case of preexcited AF caution should be taken not to transform it into atrial flutter and induce 1:1 conduction. Apart from Class IC drugs, beta-blockers, diltiazem, or verapamil may also be considered in case of orthodromic tachycardias if no signs of pre-excitation are observed on the resting ECG.^{68,69}

Catheter ablation

The treatment of choice for patients with recurrent AVRT or preexcited AF is catheter ablation (Figures 10 and 11, and Table 1). In patients with overt pre-excitation this can be accomplished during sinus rhythm, with the target being an AP potential or V electrogram preceding the delta wave as much as possible. At the successful ablation site the AV interval is minimum and a pathway potential may be found.⁷⁰ For **left-sided APs** two approaches are available: an antegrade transseptal and a retrograde aortic approach. There is evidence that the transseptal approach in experienced hands results in reduced radiation and procedure time.⁷¹

With **septal APs** close to the AV node, the electrocardiogram typically displays a positive delta wave in leads aVF, and aVL, and a narrow positive delta wave in lead V1 that has a

prominently negative QRS complex.⁹ RF ablation of para-Hisian APs without creating AV block is possible,⁷² but with cryoenergy the incidence of AV block is lower.⁷³ However, the recurrence rate of AP conduction is significantly higher after cryo-ablation.⁷⁴ In some patients with anteroseptal pathways, the successful ablation site can be in the right coronary cusp and noncoronary cusp of the aortic valve, which lie in close anatomical proximity to the anterior septum.⁷⁵

Epicardial APs occur in <10% of all cases and can be approached from the coronary sinus or the pericardial space (<1%).

In patients with **concealed** or latent pathways ablation is usually performed during ventricular pacing or AVRT by targeting the site of the earliest retrograde atrial activation or earliest retrograde AP potential. Bracketing of the pathway by the CS electrodes may be useful in identifying its approximate position.

In general, the local electrogram parameters of greatest importance in predicting the success or failure of radiofrequency catheter ablation of accessory AV connections are electrogram stability, the presence of an accessory AV connection potential, and the timing of ventricular activation relative to the QRS complex (for manifest accessory AV connections) or retrograde continuous electrical activity (for concealed accessory AV connection).⁷⁶

Ablation of the AP has a high acute success rate ($\geq 95\%$) and is associated with a low complication rate depending on pathway location.^{74,77,78} Major complications include cardiac tamponade (0.13–1.1%), and complete AV block (0.17–2.7%) in patients in whom ablation of septal APs is attempted, and the procedure carries a very small, but non-negligible mortality risk (0.1%).^{79,80} Risk factors associated with early recurrence include the presence of multiple APs and radiofrequency consolidation time <90 sec.⁸¹

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