FIMS Position Statement

Wolff-Parkinson-White syndrome and sport

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Definition and pathophysiology

Ventricular pre-excitation according to Wolff-Parkinson-White (WPW, prevalence approximately 3:1000) is characterized by the premature excitation of the ventricle by an accessory pathway (the bundle of Kent). As a consequence of the related abnormal stimulation of the ventricular myocardium, the surface ECG shows a -wave at the beginning of the QRS complex, a PQ time shortened to < 0.12 s, as well as changes in the ST-segment and the T-wave. Ventricular excitation usually occurs in combination with normal AV-nodal and accessory pathways, whereby the QRS morphology in the different leads results from the fusion of both excitation impulses. The excitation of larger ventricular areas results in more pronounced deformation of the QRS complex. The extent of pre-excitation varies from patient to patient, sometimes markedly, and there are even greater variations in the same individual. Pre-excitation can be amplified by decreasing AV-nodal conduction velocity, i.e. using the Valsalva maneuver, during sleep or under the influence of medication. Vagolytic or adrenergic excitation can decrease pre-excitation through improved AV-nodal conduction velocity. Accordingly, the -wave can disappear during physical exertion.

Possible locations of accessory pathways include almost every position in the right or left AV area. If the categorization according to ECG morphology into type A (sternal positive type – left-side pathway) and B (sternal negative type – right-side pathway) pre-excitation reveals inaccuracies, it is possible to apply different algorithms, which are based on the polarity of the -wave and the QRS vector, to better predict the localization of the accessory pathway.

Arrhythmia in pre-excitation syndrome

By definition, WPW syndrome is characterized pre-excitation accompanied by tachycardia. Possible symptoms include palpitations, thoracic discomfort, dyspnea, dizziness, and syncope.

Orthodrome re-entry tachycardia: In 90% of all AV re-entry tachycardia (AVRT) occurring in WPW, anterograde conduction takes place via normal AV pathways, and retrograde conduction via the accessory pathway. In this case, we speak of an orthodromic AVRT with frequencies between 150 and 250 beats/min, and a QRS complex with mostly narrow morphology. However, a bundle-brunch block broadening of the QRS complex, in this case caused by frequency-dependent aberrant conduction, can also be observed in orthodromic AVRT. The negative P-wave in leads II, III and aVF follows the QRS complex through retrograde atrium excitation. Its identification can be made difficult by fusion with the ST-segment.

A special form of this condition is concealed WPW syndrome. Here the conductivity of the accessory pathway is limited to retrograde direction. Therefore, WPW syndrome can only be assumed in the case of the occurrence of AVRT with negative P-waves that follow the QRS complex.
Antidrome re-entry tachycardia: Anterograde conduction takes place through accessory pathways in less than 10% of all cases, whereby the normal pathway represents the retrograde side. This form of tachycardia is known as antidrome AVRT. A surface ECG shows a broadened and deformed QRS complex, often without the demarcation of a P-wave. This finding cannot be distinguished from ventricular tachycardia.

Atrial fibrillation in WPW syndrome: In 20% of WPW cases with arrhythmia, atrial fibrillation is observed, which can be induced by an initial re-entry mechanism. If high-frequency conduction takes place during atrial fibrillation, then there is a great risk of very rapid ventricular rate, ventricular fibrillation, and the danger of sudden cardiac death. The expected ventricular frequency results from the duration of the effective anterograde refraction period (ERP) of the accessory pathway. Values < 250 ms must be considered a potential threat. This is equivalent to a time value of < 250 ms for the shortest RR interval of preexistent ventricular complexes during atrial fibrillation. Indirect indications of a longer ERP are pre-excitation during a stress test or after intravenous administration of Ajmalin (50 mg), although these would not be conclusive.

Diagnosis

Basic diagnosis: History, physical examination, plus 12-channel, long-term and stress ECG, as well as echocardiography to assess pre-excitation, for documentation of arrhythmic occurrences, exclusion of structural cardiac disease, and for follow-up during and after therapy.

Electrophysiological testing (EPT): Determination of the location and functional characteristics of the accessory pathway.

Treatment

High-frequency (HF) catheter ablation of the accessory pathway allows the curative treatment of WPW syndrome and is considered the best treatment for symptomatic patients, particularly those at high risk. The success rate is 88-99%; the rate of complication is 1 % in experienced centers (> 100 WPW ablations per year).

Prophylactic treatment with drugs has become less and less important in the face of HF catheter ablation. Beta-blockers can be used for low-risk WPW patients (decreasing the AV conduction velocity). Alternatives are class la and lc (Ajmalin, Propafenon), and class III antiarrhythmics (Sotalol, Amiodarone). These drugs influence the conductivity of the accessory pathway. They reduce the risk of atrial fibrillation, and are therefore of great importance to patients with an increased risk of sudden cardiac death. However, HF catheter ablation remains the primary therapeutic goal for this group 3,14,15.

WPW syndrome and sport participation

1. Asymptomatic subjects with pre-excitation syndrome without tachycardia have a very low risk of sudden cardiac death 8,9,11,13. Therefore, unlimited participation in sports with regular observation is justifiable for this group 4,14. However, a more precise evaluation

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should be done for subjects over 20 years old, possibly including EPT, before eligibility for competitive sports is given.\textsuperscript{2,3,14}

2. Athletes with tachycardia should undergo an EPT. If an accessory pathway exists, the recommended treatment is HF ablation. Since this procedure is carried out quickly and with a low risk of complications in experienced centers, an exclusively invasive diagnostic procedure without HF ablation does not appear justifiable. In addition, drug treatment appears to be the less attractive option due to the possible impairment of performance and doping problems (with beta-blockers). The resumption of competitive sports can be permitted 3-6 months after successful HF ablation. Participation in competitive sports is also justifiable for athletes with short episodes (5-10 s) of non-symptomatic AVRT, which is neither induced nor intensified by physical stress.\textsuperscript{4,14}

3. The following athletes can participate only in low-intensity static and dynamic sports:\textsuperscript{10}

(a) those who were neither successfully treated by ablation or medication and who show simultaneously signs of a) repeated AVRT along with pre-syncopic conditions, syncope, or significant palpitations, or

(b) episodes of atrial fibrillation with a frequency of > 240/min through the accessory pathway and/or report pre-syncopic or syncope conditions.\textsuperscript{4,5,7,9,14}

References

6. Fitzpatrick AP, Gonzales RP, Lesh MD, Modin GW. New algorithm for the localization of accessory atrioventricular connections using a baseline
Table 1: Long-term outcome of patients with WPW syndrome with and without symptoms (5, 9, 11)

<table>
<thead>
<tr>
<th>Patients</th>
<th>Deaths</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Munger et al. 1993</td>
<td>2/60 sympt. (3,3%)</td>
<td>36 years</td>
</tr>
<tr>
<td>Munger et al. 1993</td>
<td>0/53 asympt.</td>
<td>36 years</td>
</tr>
<tr>
<td>Flenstedt-Jensen 1969</td>
<td>2/47 sympt. (4,3%)</td>
<td>34 years</td>
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<tr>
<td>Leitch 1990</td>
<td>0/75 asympt.</td>
<td>4,3 years (Median)</td>
</tr>
</tbody>
</table>

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