**FÈDÈRATION INTERNATIONALE DE MÈDECINE DU SPORT /
THE INTERNATIONAL FEDERATION OF SPORTS MEDICINE**

FIMS Position Statement

Wolff-Parkinson-White syndrome and sport

March 2004

by
Prof. Dr. Hans-H. Dickhuth
Medical Clinic and Polyclinic, Dept. of Sportsmedicine
University of Freiburg, Germany

Priv. Doz. Dr. Christian Mewis
Medical Clinic and Polyclinic, Dept. of Cardiology
University of Tübingen, Germany

Priv. Doz. Dr. Andreas Niess
Medical Clinic and Polyclinic, Dept. of Sportsmedicine
University of Freiburg, Germany

Available on the FIMS web site: http://www.fims.org

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 [6].

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 [1,12.]

***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, plus12-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 Ia and Ic (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 <u>without</u> 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

should be done for subjects over 20 years old, possibly including EPT, before eligibility for competitive sports is given [2,4,14].

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 [4,14].
3. The following athletes can participate only in low-intensity static and dynamic sports [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 [4,5,7,9,14].

References

1. Al-Khatib SM, Pritchett LC. Clinical features of Wolff-Parkinson-White syndrome. Am Heart J 1999, 138: 403-413
2. American Heart Association (AHA).Cardiovascular preparticipation screening of competitive athletes. Circulation 1996, 94: 850-856
3. Calkins H, Yong P, Miller JM, Olshansky B, Carlson MD, Saul JP, Huang JL, Liem B, Klein LS, Moser SA, Bloch DA, Gillette PC, Prystowsky EN. Catheter ablation of accessory pathways, atrioventricular nodal reentrant tachycardia, and the atrioventricular junction. Final results of a prospective, multicenter clinical trial. Circulation 1999, 99: 262-270
4. Estes III NAM, Link MS, Cannom DS, Naccarelli GV, Prystowsky EN, Maron BJ, Olshansky B. Report of the NASPE policy conference on arrhythmias and the athlete. J Cardiovasc Electrophysiol 2001,12: 1208-1219
5. Flensted-Jensen E. Wolff-Parkinson-White syndrome: A long-term follow-up of 47 cases. Acta Med Scand 1969, 186: 65-74
6. Fitzpatrick AP, Gonzales RP, Lesh MD, Modin GW. New algorithm for the localization of accessory atriventricular connections using a baseline

electrocardiogram. JACC 1994, 23:107-116
7. Futterman L G, Myerburg R. Sudden death in athletes: An update. Sports Medicine 1998, 26: 335-350
8. Krahn AD, Klein GJ, Yee R. The approach to the athlete with Wolff-Parkinson-White-syndrome. In: *Sudden cardiac death in the athlete*. Estes III NAM, Salem DN, Wang PJ, Eds., Futura Publishing, Armonk, NY, 1998.
9. Leitch JW, Klein GJ, Yee R, Murdock C. Prognostic value of electrophysiologic testing in asymptomatic patients with Wolff-Parkinson-White pattern. Circulation 1990, 82: 1718-1723
10. Mitchell JH, Haskell WL, Raven PB. Classification of sports. JACC 1994, 24: 864-866
11. Munger TM, Packer DL, Hammill SC, Feldman BJ, Bailey J, Holmes DR, Gersh BJ. A population study of the natural history of Wolff-Parkinson-White syndrome in Olmsted County 1953-1989. Circulation 1993, 87: 866-873
12. Niess A, Mewis C, Dickhuth H-H.Wolff-Parkinson-White-Syndrom und Sport. Dtsch Z Sportmed 2001, 11: 325-326
13. Wellens HJ, Rodrigues LM, Timmermanns C, Smeets JLRM. The asymptomatic patient with Wolff-Parkinson White electrocardiogram. PACE 1997, 20: 2082-2086
14. Zipes DP, Garson R.Task force 6 : Arrythmias. Med Sci Sports Med 1994, S276- 283
15. Zipes DP, DiMarco JP, Gillette PC, Jackman WM, Myerburg RJ, Rahimtoola SH. ACC/AHA Task force report: Guidelines for clinical intracardiac electrophysiological and catheter ablation proceduresJ Am Coll Cardiol 1995, 26:555-573

Table 1: Long-term outcome of patients with WPW syndrome with and without symptoms (5, 9,11)

	Deaths	**Follow-up**
Munger et al. 1993	2/60 sympt. (3,3%)	36 years
Munger et al. 1993	0/53 asympt.	36 years
Flenstedt-Jensen 1969	2/47 sympt. (4,3%)	34 years
Leitch 1990	0/75 asympt.	4,3 years (Median)