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Position Paper

Recommendations for participation in leisure-time physical activity and competitive sports in patients with arrhythmias and potentially arrhythmogenic conditions
Part I: Supraventricular arrhythmias and pacemakers

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Received 26 October 2005 **Accepted** 4 January 2006

This document by the Study Group on Sports Cardiology of the European Society of Cardiology extends on previous recommendations for sports participation for competitive athletes by also incorporating guidelines for those who want to perform recreational physical activity. For different supraventricular arrhythmias and arrhythmogenic conditions, a description of the relationship between the condition and physical activity is given, stressing how arrhythmias can be influenced by exertion or can be a reflection of the (patho)physiological cardiac adaptation to sports participation itself. The following topics are covered in this text: sinus bradycardia; atrioventricular nodal conduction disturbances; pacemakers; atrial premature beats; paroxysmal supraventricular tachycardia without pre-excitation; pre-excitation, asymptomatic or with associated arrhythmias (i.e. Wolff–Parkinson–White syndrome); atrial fibrillation; and atrial flutter. A related document discusses ventricular arrhythmias, channelopathies and implantable cardioverter defibrillators. *Eur J Cardiovasc Prev Rehabil* 13:475–484 © 2006 The European Society of Cardiology

European Journal of Cardiovascular Prevention and Rehabilitation 2006, **13**:475–484

Keywords: athlete's heart, guidelines, atrial fibrillation, atrial flutter, pacemakers, recommendations, sports cardiology, supraventricular tachycardia, Wolff–Parkinson–White

Introduction

Patients with cardiac arrhythmias form an important proportion of those presenting for eligibility assessment to participate in competitive or leisure sports activity [1]. Although recommendations for competitive athletes have been published before [1,2], guidelines concerning recreational physical activity are scarce and have only so far addressed inherited arrhythmogenic conditions [3]. By including recommendations for leisure-time activities, this paper (and an accompanying article on ventricular arrhythmias in an upcoming issue of the journal) extends

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1741-8267

the more concise recommendations in competitive athletes from the Study Group on Sports Cardiology of the European Society of Cardiology [1] and the recently updated North American eligibility guidelines as formulated by the 36th Bethesda Conference [2].

Arrhythmias can occur in structurally normal hearts or reflect the physiological adaptation to sports participation itself. They can also be the expression of different cardiovascular abnormalities (ranging from genetic ion channel diseases, congenital anomalies to structural heart diseases) or be present in association with them; these situations are of specific prognostic importance because the main determinant for sports participation in patients with arrhythmias is the presence of heart disease [4–8]. It is not always unequivocal to detect and define an underlying pathology. Therefore, a careful diagnostic evaluation is mandatory.

Diagnostic evaluation: general framework

Cardiovascular preparticipation screening should be based on personal and family history, and on physical examination with the measurement of blood pressure. In contrast to American guidelines [9], a recent consensus document from the European Study Group on Sports Cardiology recommended the routine addition of an electrocardiogram (ECG), given its key role in the identification of cardiovascular diseases [10]. This section gives a brief summary of the diagnostic evaluation from an arrhythmic perspective, applying to both the sections on supraventricular and ventricular arrhythmias.

If the personal history reveals 'palpitations', it is essential to ask the patient to define them as accurately as possible: do they constitute (repetitive) premature beats or longer-lasting paroxysmal episodes, and is the heart rate during these episodes regular or not? In many cases, a patient will not complain about palpitations, but the history may reveal unspecific symptoms such as lightheadedness, presyncope or syncope, unexplained weakness, fatigue, chest pain, or dyspnoea. These symptoms may be the only indicator of arrhythmia, and should therefore be interpreted cautiously when noted in a physically active patient's history. Presyncope or syncope caused by neurocardiogenic (vasovagal) aetiology is prevalent in healthy, athletic individuals, and carries a good prognosis, but it should unambiguously be differentiated from an arrhythmic aetiology, which may carry a worse prognosis [11]. Vasovagal syncope usually occurs at rest. Exercise-related syncope is rarely neurocardiogenic, and should always lead to suspicion about an arrhythmic cause. The personal history should further enquire for performance-enhancing or other drugs that might be proarrhythmic or could affect the heart (such as antibiotics, antidepressants and others, but also smoking and alcohol consumption). Previous cardiovascular disease should be noted and other risk factors for coronary heart disease should be investigated in patients aged 35 years or older (see also the Recommendations in patients with ischaemic heart disease).

A thorough family history should enquire about sudden death (especially in youth and adulthood) or arrhythmogenic conditions known to have a genetic inheritance.

The initial evaluation includes a physical examination and 12-lead ECG [10]. Patients should also be encouraged to have an ECG taken during palpitations or other non-specific symptoms, and physicians have to discuss with them specific arrangements to enable documentation. Exercise testing and Holter monitoring will often be indicated; arrhythmia findings should always be correlated with symptoms and vice versa. Echocardiography can assess the presence or extent of structural heart disease. In some cases, it is advisable to obtain a blood count (e.g. unexplained sinus tachycardia), thyroid function markers (e.g. atrial fibrillation; AF), and electrolyte balance (in all patients with suspected or documented arrhythmias).

In case of a clinical suspicion of arrhythmias but no documentation, an external event recorder is indicated. An implantable loop recorder can be considered in rare cases of unexplained syncope. Head-up tilt testing may confirm the neurocardiogenic origin of syncope, although the testing has lower specificity in athletes compared with the general population [11].

An invasive electrophysiological study is indicated in some individuals when diagnostic uncertainty persists despite non-invasive evaluation or for prognostic reasons. Specific indications will be mentioned in the sections on the different arrhythmias below. In selected arrhythmias the diagnostic evaluation can be followed by ablation of the arrhythmogenic substrate [12].

Apart from the initial evaluation, regular follow-up should be performed in patients/athletes with arrhythmias. They should also be advised to present for immediate re-evaluation in the case of symptoms, certainly if these are exercise related. The importance of unspecific symptoms such as sudden exertional fatigue or dyspnoea needs to be discussed with them. Below, the intensity and frequency of the required diagnostic evaluation will be more specifically discussed for different arrhythmogenic presentations.

Arrhythmias and arrhythmogenic conditions

The following paragraphs will discuss different arrhythmias and arrhythmogenic conditions in detail. They will start with: (i) a description of the relationship between

the condition and physical activity. This will be followed by (ii) recommendations for leisure-time activities in patients with these conditions; and (iii) by recommendations concerning competitive or semi-competitive sports participation. It needs to be stressed, however, that there is no clear division between recreational and (semi)-competitive sports. Some patients may engage in high-intensity exercise during leisure-time activities. The physiological or pathophysiological implications may be similar [13]. Moreover, different sports activities will create varying cardiac stress, directly or through changes in autonomic tone, dehydration or electrolyte balance. Therefore, an individualized assessment of both the underlying condition and the type/intensity of the anticipated sports activity will be required in every patient. Classifications of sports depending on the amount of static and dynamic cardiovascular demand have been published elsewhere [1,14] The recommendations are summarized in Table 1.

Sinus bradycardia

Asymptomatic sinus bradycardia, enhanced sinus arrhythmia, wandering pacemaker and sinus pauses between 2 and 3 s are common in young athletes [13,15–18] and may persist later into life [19,20]. Junctional rhythm is often observed during periods of sinus bradycardia. These rhythm variations are part of the physiological adaptation to exercise. They are mainly functional, that is, caused by an increased vagal/sympathic balance, but also by intrinsic slowing of the sinus node rate, as evidenced after double autonomic blockade (i.e. the simultaneous administration of beta-blockers and atropine) [21].

Therefore, intense exercise training and its physiological adaptation mechanisms could aggravate pre-existing sinus dysfunction caused by other cardiac problems or induce them by itself and lead to symptoms. Sports participation is not formally contraindicated in these individuals, but its extent should be reassessed on the emergence of symptoms such as dizziness or syncope. These symptoms will present at rest and not during exercise. The prognostic significance of these arrhythmias is usually benign. Sinus bradycardia or pauses should always be related to clinical symptoms to assess their pathological significance (and vice versa). In particular, athletes with documented sinus pauses of 3 s or longer should be asked about symptoms. Diagnosis in patients with unspecific symptoms may require long-term ambulatory ECG recording. The cessation of sports activity may result in the resolution of symptoms and an improvement in rhythm after a 1–2 month period [16]. In these cases, the resumption of all sports activity can be advised. The resolution of sinus bradycardia may be incomplete, however, with remaining symptoms necessitating the implantation of a pacemaker in rare cases. The eligibility for sports participation in pacemaker patients is discussed below.

In asymptomatic patients a yearly follow-up could suffice. In those who were symptomatic before but became asymptomatic after a transient cessation of sports participation, a follow-up evaluation after 6 months is recommended. The patient should be instructed to consult earlier in the case of the re-emergence of symptoms.

Atrioventricular nodal conduction disturbances

Like sinus bradycardia, the slowing of atrioventricular nodal conduction forms part of the physiological adaptations to exercise [15–17]. Athletes have a large prevalence of first-degree atrioventricular block or second-degree Wenckebach-type atrioventricular block (Mobitz type I), typically occurring at rest or during sleep [13,20,22–24]. Therefore, these findings in athletes or patients who want to perform leisure-time physical activity usually do not form exclusion criteria. Moreover, these physiological conduction disturbances should resolve during sympathetic stimulation or exercise [22]. In those cases, no further investigation and no therapy are indicated. All sports participation is allowed. Even if there are symptoms, these may resolve with the temporary discontinuation of sports, after which sports can be resumed, with a 6-monthly re-evaluation. Otherwise a yearly follow-up is appropriate.

In the case of second-degree atrioventricular block Mobitz type 2 or third-degree atrioventricular block, a more comprehensive diagnostic evaluation is warranted to exclude underlying structural heart disease, which will be more often present in these patients. Moreover, associated ventricular tachyarrhythmias should be excluded, by Holter, exercise testing and sometimes even an invasive electrophysiological study. These conduction disturbances may still be secondary to athletic activity [18,20]. However, as with for sinus arrest, ventricular pauses of 3 s or more or a resting heart rate of 40 bpm or less caused by conduction disturbances are rarely physiological, although they have been reported in high-level athletes [20,24]. In rare cases without structural heart disease, a deconditioning phase of 1–2 months can be considered, with the resumption of low-to-moderate dynamic or static sports activity when symptoms have disappeared. In the case of persisting or recurring symptoms, pacemaker implantation may be indicated.

In patients with underlying structural disease and second or third-degree atrioventricular block, pacemaker implantation is recommended.

Pacemakers

Patients with heart disease and a pacemaker can participate only in sports consistent with the limitations of the underlying heart disease. In the absence of heart disease, competitive or recreational sports participation is

Table 1 Recommendations for participation in competitive sports and leisure-time physical activity in patients with arrhythmias and potentially arrhythmogenic conditions: supraventricular arrhythmias and pacemakers. (For athletes with structural heart disease, see also the recommendations specific to the disease)

Arrhythmia	Evaluation	Criteria for eligibility	Recommendations	Follow-up
Sinus bradycardia, especially when marked ($\leq$ 30 bpm) or with sinus pauses $\geq$ 3 s	History, ECG, ET, Holter echo	(a) Asymptomatic, no cardiac disease (b) Symptomatic[a] (c) After >3 months from resolution of symptoms[a] and off therapy	(a) All sports (b) 1–2 month temporary interruption of sports (c) All sports	(a) Yearly (b) 1–2 months (c) Every 6 months
(a) AV block 1st and 2nd degree (type 1) (b) AV block 2nd degree (type 2) or 3rd degree	History, ECG, ET, Holter echo, sometimes EP study	(a1) Asymptomatic, no cardiac disease, with resolution during exercise (a2) Symptoms or cardiac disease (b1) Symptomatic[a] and no cardiac disease (b2) Persistence of symptoms or cardiac disease, ventricular arrhythmias during exercise, and if resting heart rate is >40 bpm	(a1) All sports (a2) + (b1) Consider pacemaker or 1–2 months temporary interruption of sports. If asymptomatic: low–moderate sports (b2) Pacemaker	(a1) Yearly (a2) + (b1) 1–3 months (b2) 6 months
Pacemaker	ECG, Echo, ET, Holter	(a) Evaluate underlying cardiac disease: specific recommendations (b) Evaluate tachyarrhythmias: specific recommendations	Low–moderate sports except those with risk of bodily collision Evaluate electromagnetic interference Evaluate rate-responsiveness	6 Months
Atrial premature beats	History, ECG, Holter, thyroid function	No symptoms, no cardiac disease	All sports	Not required
Paroxysmal supraventricular tachycardia (AVNRT, AVRT over a concealed accessory pathway, atrial tachycardia)	History, ECG, Echo, EP study if ablation is considered	Competitive sports: ablation recommended (a) If no recurrences for >1–3 months (b) If no ablation, only sporadic palpitations, no cardiac disease, no haemodynamic consequences even when during exercise. Leisure sports: no treatment, drugs or ablation depending on preference and haemodynamic tolerance of arrhythmia during sports	(a) Competitive sports allowed (leisure and low–medium training after 1 week, but individualized advice based on risk of arrhythmia recurrence during sports) (b) Sports without increased risk due to loss of consicousness allowed All sports allowed Cessation of activity on start of palpitations	(a) None (b) Yearly Yearly (unless ablated)
Ventricular pre-excitation (a) With history of AVRT, atrial tachycardia or AF (WPW syndrome) (b) Asymptomatic	History, ECG, Echo, non-invasive evaluation of antegrade refractory period, EP study	Competitive and recreational sports: (a) Ablation is mandatory (b) Ablation mandatory if increased risk of sudden death (competitive athletes: EP study; recreational athletes: non-invasive testing or EP study) or sports with increased risk. To be individually considered in others.	All sports if no recurrences after ablation for 1–3 months. Leisure and low to medium training after 1 week, but individualized advice based on risk of arrhythmia recurrence during sports. ECG control at 6 months and 1 year	Yearly
Atrial fibrillation	History, ECG, Echo, ET, Holter	(a) Secondary to reversible cause (b) First onset or very sporadic paroxysms (c) Paroxysmal or permanent, without major cardiac disease Note 1: caution for monotherapy with class 1 antiarrhythmic drugs Note 2: consider prophylactic flutter ablation ('hybrid therapy')	(a) All sports when cause corrected and stable sinus rhythm for >2 months (b) All sports when stable sinus rhythm for >3 months. 'Pill-in-the-pocket' approach for some (c) All sports when proven rate control with absence of haemodynamic impairment (indivualized therapy) Note: AF ablation still investigative in athletes. All sports if asymptomatic for $\geq$ 3 months (all) Classical indications for anticoagulation. In such case: no sports with bodily contact or high risk for trauma	(a) Yearly (b) Yearly (c) Every 6 months
Atrial flutter	History, ECG, Echo, EP study	Ablation mandatory	Competitive sports: all sports if asymptomatic for $\geq$ 3 months Leisure time sports: early resumption unless major haemodynamic impairment before Note: Evaluate occurrence of AF. See recommendations above	Yearly

AF, Atrial fibrillation; AV, atrioventricular; AVNRT, atrioventricular nodal re-entrant tachycardia; AVRT, atrioventricular re-entrant tachycardia; ECG, 12-lead electrocardiogram; Echo, echocardiography; ET, exercise testing; Holter, 24-h ECG monitoring; EP, electrophysiological study; WPW, Wolff–Parkinson–White syndrome. Sport types, see Introduction. [a]Symptoms include presyncope, lightheadedness, exertional fatigue.

allowed in sports with minor to moderate cardiovascular demand [1,14]. Exercise testing and Holter ECG monitoring will help to programme an appropriate pacing rate responsiveness during exercise (and exclude inappropriate rate acceleration in other circumstances, e.g. horse-back riding) [25]. Bradyarrhythmias requiring pacing may be associated with tachyarrhythmias, both on an atrial level (mainly atrial tachycardia, flutter or fibrillation) and rarely on a ventricular level. Pre-implant screening should evaluate this possible association by Holter or event recording. Modern pacemaker diagnostics also allow the logging of atrial or ventricular high-rate episodes after implant, often with stored electrograms that facilitate evaluation. In the case of tachyarrhythmias, the appropriate recommendations apply. Individuals with a pacemaker should be restricted from sports with a risk of bodily impact, because of the possible damage to the electrodes or pacing unit (e.g. rugby, martial arts …) [26–29], or the risk of skin perforation (which may occur late after trauma) [30]. Other sports (such as soccer, basketball, baseball, …) can be allowed while wearing appropriate padding. Extreme ipsilateral arm movements should be avoided at least until complete fixation of the leads, namely 6 weeks. Sports with pronounced arm movements (such as volleyball, basketball, tennis, climbing, …) may also increase the risk of late lead damage as a result of subclavian crush (with insulation or conductor failure) [31–33]. The pacemaker should be implanted at the right or left side depending on arm dominance and the type of sports (e.g. on the left side in a right-handed tennis player). Electromagnetic interference is uncommon with modern devices, but it should be closely evaluated in specific athletic environments (such as starting gate electronic equipment or scoring equipment during fencing). Interference could lead to a temporary inhibition of pacing, which is of concern in pacemaker-dependent patients. Furthermore, myopotential inhibition may result in an inhibition of pacing, a problem that is more common with unipolar electrodes, although it can usually be corrected with appropriate reprogramming of the device [34,35]. Bipolar leads are less sensitive to this problem, but may have a reduced longevity.

Atrial premature beats

Atrial premature beats are a common finding in many individuals without any underlying cardiovascular abnormality [15]. There is some indication that physical activity may increase their frequency [13]. In most patients atrial premature beats pass unnoticed, although they may lead to subjective palpitations without associated haemodynamic impairment. Usually a careful history taking is indicative of their nature as isolated extrasystoles. Holter may be necessary to distinguish them from ventricular premature beats. Apart from a 12-lead ECG, physical examination and thyroid function tests, no more extensive cardiovascular assessment is indicated.

All sports participation, competitive and recreational, is allowed in the absence of structural heart disease.

Paroxysmal supraventricular tachycardia without pre-excitation

Paroxysmal supraventricular tachycardia (PSVT) is a generic term. The tachycardia may be caused by (i) atrioventricular nodal re-entrant tachycardia (AVNRT; most prevalent [36]); (ii) orthodromic atrioventricular re-entrant tachycardia with retrograde conduction over an accessory pathway (AVRT, sometimes called 'circus movement tachycardia'); or (iii) ectopic atrial tachycardia. When the accessory pathway is only capable of conducting from the ventricles to the atria and thus shows no pre-excitation on the sinus rhythm ECG, it is denoted as 'concealed' (meaning 'hidden'). When the accessory pathway also conducts antegradely, ventricular pre-excitation will be visible on the ECG during sinus rhythm. In this case the patient with PSVT has the 'Wolff–Parkinson–White syndrome' (i.e. pre-excitation plus tachycardia), which carries a risk of sudden death and will therefore be discussed below, given the different recommendations for athletic activity.

Exclusion of pre-excitation and underlying structural heart disease is paramount during the diagnostic work-up of an athlete with PSVT. In some patients, however, pre-excitation may be minimal and apparently absent despite antegrade conduction over an accessory pathway ('latent pre-excitation'). It can be unmasked on a 12-lead ECG by manoeuvres that slow conduction through the atrioventricular node during sinus rhythm, such as carotid sinus massage or the administration of adenosine intravenously; prolongation of the pulse rate interval without a change in the QRS morphology or transient atrioventricular block rule out pre-excitation. An invasive electrophysiological study in a patient with documented PSVT excluding pre-excitation and excluding atrial flutter (see below) is not necessary for the differential diagnosis of the exact aetiology unless ablation is considered because the recommendations for sports participation are not dependent on the type of underlying arrhythmia (AVNRT, AVRT over concealed accessory pathway or atrial tachycardia). In some patients with atrial tachycardia there may be more than one focus, or this may be a hallmark of underlying cardiovascular disease. Atrial tachycardia may also be associated with AF. Therefore, diagnostic work-up in athletes in whom the diagnosis of atrial tachycardia is made should evaluate these aspects.

PSVT is generally considered to be a benign arrhythmia, not associated with sudden death if no structural heart disease is present. However, during physical activity and

sympathetic stimulation the rates of both AVNRT and AVRT over a concealed accessory pathway increase. Exercise will also increase the ventricular rate during atrial tachycardia as a result of the facilitation of AV nodal conduction. This may lead to symptoms of haemodynamic compromise such as dizziness, acute fatigue or syncope, even in the absence of structural heart disease.

If the patient wants to perform competitive athletic activity, definitive treatment by ablation is therefore recommended. Moreover, drug treatment may not be tolerated, may be illegal, or may even be dangerous during competitive sports. When the ablation procedure is successful, the risk of recurrence is very low (< 3%) and usually occurs within the first months. Therefore, if no recurrence has developed after 1–3 months, competitive sports activity can be resumed for all types of sports and further follow-up is not required. Leisure-time and low to medium intensity training practice can generally be resumed after 1 week (cf. healing of the puncture sites) provided that there is no particular risk of arrhythmia recurrence (e.g. a history of exercise-related major (pre)syncope, findings at electrophysiological study, ease of ablation,…). In case the PSVT is only sporadic and is not associated with haemodynamic consequences even when it develops during exercise, or in case ablation is not successful, sports activity is allowed when there is no increased risk from the loss of consciousness (such as in pilots, motorsports drivers, parachute jumpers, divers). Exercise should be stopped as soon as palpitations arise but can be resumed after the cessation of palpitations. A yearly follow-up is necessary.

In the case the patient only wants to perform leisure-time and lower-level sports and there are no associated symptoms during tachycardia, participation is allowed and he/she only needs to be instructed to stop physical activity as soon as palpitations arise. Prophylactic drug treatment with beta-blockers or calcium antagonists can be considered, although it has limited efficacy, will not be well tolerated, and may need to be continued life-long. Class 1 drugs generally play no role in the management of regular PSVT. Therefore, in a recreational athlete with recurrences of PSVT despite maintenance treatment with beta-blockers or calcium antagonists, ablation should also be considered as a definitive treatment. Ablation can also be the patient's preference as first choice therapy after discussion with the physician of its ability for long-term cure, its high efficacy (> 98% for AVNRT and AVRT, > 85% for atrial tachycardia) and relatively rare incidence of complications in experienced centres. Complications mainly consist of atrioventricular block (requiring pacemaker implantation, < 1%) or perforation with cardiac tamponade. In selected cases with an increased risk of complete atrioventricular block (such as para-Hisian accessory pathways or certain forms of atypical AVNRT) cryo-ablation can be an alternative to radiofrequency ablation although its superiority over radiofrequency energy has not been shown.

Ventricular pre-excitation (Wolff–Parkinson–White syndrome)

Wolff–Parkinson–White syndrome (WPW) is defined as the presence of paroxysmal arrhythmias in a patient with ventricular pre-excitation, caused by an accessory pathway with antegrade conduction. The prevalence of pre-excitation in the general population varies from 0.1 to 0.3% and it seems not to differ in athletic populations [37,38]. Accessory pathway-dependent arrhythmias include AVRT, either orthodromic or antidromic. WPW patients, however, may also develop other arrhythmias such as AF, which could lead to ventricular fibrillation (VF; and sudden death) as a result of rapid antegrade conduction over the accessory pathway. It has been estimated that one-third of patients with WPW syndrome may develop AF. There is still discussion on how far WPW patients have an increased risk of AF *per se* (e.g. caused by secondary degeneration of AVRT), but exercise has been reported to be associated with an increased risk of VF development in this context [39]. Moreover, athletes may have an increased risk of AF (see below) even after they have ceased high-level sports competition. Therefore, sports activity in the presence of overt pre-excitation may expose the athlete to an increased risk of sudden death if the accessory pathway has the potential for fast antegrade conduction. The risk of sudden death in patients with pre-excitation varies in population-based studies from 0.15 to 0.20%, but has been reported to be approximately 2% in symptomatic patients [39].

Evaluation of the athlete with ventricular pre-excitation should exclude an associated structural cardiac disease, such as hypertrophic cardiomyopathy or Ebstein anomaly, by physical examination, 12-lead ECG and echocardiogram, which may be underlying.

Pre-excitation and documented paroxysmal supraventricular tachycardia or atrial fibrillation

The risk of the development of VF secondary to AF is dependent on the antegrade refractory period of the accessory pathway. The refractory period is modulated by autonomic tone. Although non-invasive measurements may indicate a long refractory period (e.g. when there is intermittent pre-excitation during sinus rhythm on ECG or Holter or when pre-excitation suddenly disappears during sinus tachycardia on an exercise test), the conditions during competitive events cannot be fully reproduced under laboratory conditions. Moreover, WPW patients with documented PSVT or AF have an increased risk of rapidly conducted AF and sudden death. The majority of sudden deaths occur during exercise or under emotional stress [39].

Therefore, ablation of the accessory pathway is mandatory in competitive and recreational athletes with pre-excitation and documented arrhythmias. In the case of only sporadic episodes of palpitations, good haemodynamic tolerance (even during exercise) or anticipation of an ablation procedure with increased risk (e.g. anteroseptal accessory pathway), management can be guided by an assessment of the antegrade conduction characteristics of the accessory pathway by non-invasive testing or an invasive electrophysiological study (see below). When they reveal a long refractory period and thus a low risk of sudden death, the continuation of sports activity is allowed without ablation (with instructions to stop sports on the recurrence of palpitations and a yearly re-evaluation). However, when arguments are present for an increased risk of sudden death, ablation is mandatory.

Resumption of leisure-time and low-to-medium intensity training practice can generally be resumed after 1 week (cf. healing of the puncture sites) provided that there is no particular risk of arrhythmia recurrence (e.g. a history of exercise-related major (pre)syncope, findings at electrophysiological study, ease of ablation, …). An individualized approach is warranted. Resumption of competitive sports is possible after 1 to 3 months, but with further ECG control at 6 months and 1 year (given the very small risk of the late recurrence of pre-excitation).

Asymptomatic pre-excitation

Asymptomatic pre-excitation in the absence of structural heart disease carries a small but definite risk of sudden cardiac death. Sudden death may be the first manifestation of the WPW syndrome in approximately half of WPW patients, and it usually presents during exercise or emotional stress [39].

In competitive athletes with asymptomatic pre-excitation, an electrophysiological study is warranted to evaluate the risk of sudden death. Inducibility of AVRT or AF [40], a pre-excited R-R less than 240 ms at baseline or less than 220 ms during isoproterenol infusion, an antegrade refractory period less than 250 ms at baseline [41], the presence of multiple accessory pathways, or a septal location (mainly posteroseptal and midseptal) [39], are electrophysiological parameters considered to be associated with an increased risk of sudden death [42]. In these asymptomatic athletes, ablation of the accessory pathway is mandatory given its high success rate and low incidence of complications. Moreover, the efficacy and safety of antiarrhythmic drug treatment has never been proved in WPW patients in general or athletes in particular. Moreover in others, ablation can be considered because approximately 3.5% of non-inducible patients may become symptomatic during follow-up, but the risks of ablation should be weighed against the benefits, after extensive discussion with the athlete, and a decision should be made on an individual basis. For athletes who refuse ablation, or in case the procedure is associated with high risk (such as anteroseptal accessory pathways), competitive sport activities are allowed when the electrophysiological study demonstrated the absence of parameters of risk (as specified above) and when they are involved in sports in which there is no increased risk from the loss of consciousness (such as pilots).

In recreational athletes, risk assessment can first be pursued via non-invasive testing, looking for intermittent pre-excitation on ECG or Holter, for the disappearance of pre-excitation after the administration of a low dose of class 1 drugs or for its abrupt disappearance during exercise testing [42]. The sensitivity of non-invasive screening for fast antegrade conduction is good but its specificity is low [41], meaning that approximately half of the patients will need a subsequent electrophysiological study to rule out an increased risk of sudden death. When inducible or having an accessory pathway with a short antegrade refractory period, ablation is mandatory. In others, the decision to ablate or not should depend on individual assessment. In patients performing sports activity in which there is an increased risk from the loss of consciousness (pilots) ablation is mandatory even when not inducible.

Of note, in children younger than 12 years, the risk of AF-induced VF and sudden death is very low. Generally, a conservative approach is recommended in this age group, although a recent study [43] suggested that prophylactic assessment and ablation reduces the risk of sudden death. The benefit/risk ratio of this approach on a large scale needs further study.

Atrial fibrillation

There are indications that AF is more prevalent in active and former competitive athletes and those performing recreational endurance sports compared with the general population [23,44–47]. The reasons for this association are probably complex: physical activity and increased sympathetic activity may act as the trigger for AF in patients with underlying predisposing conditions (such as hypertension), whereas an increased vagal tone may predispose to AF development at rest. In some patients, AF may develop secondary to PSVT or atrial tachycardia with or without overt pre-excitation, as a result of an underlying cardiomyopathy, silent myocarditis or other structural heart disease [46]. There are also indications that cardiac structural adaptation to endurance training (such as atrial dilatation and hypertrophy) contribute to the atrial substrate for AF [44,47], although another study found no relationship between left atrial diameter and the incidence of supraventricular arrhythmias [48]. As

with sinus bradycardia and AV conduction disturbances, it may therefore be likely that sports participation accelerates the development and progression of AF from other underlying causes. The role of performance-enhancing drugs, such as anabolic steroids, in the development of the AF substrate or in triggering the arrhythmia is largely unknown [49]. Atrial fibrillation can be categorized as 'first onset', 'paroxysmal', 'persistent' (i.e. requiring chemical or electrical cardioversion) or 'permanent' (i.e. accepted chronic AF) [50]. Usually, one can expect progression in the frequency and duration of AF episodes over time unless a specific aetiological cause can be discerned (such as hyperthyroidism, illegal drug use, myocarditis).

If such a primary cause is present, competitive or leisure-time sports participation should be temporarily suspended and can be resumed after correction of the cause and a stable sinus rhythm is present for over 2 months. Diagnostic assessment should further focus on underlying hypertension or structural heart disease. Therefore, sports participation will also be guided by these underlying conditions. AF is not a life-threatening arrhythmia without cardiac disease except in patients with the WPW syndrome (for recommendations see above). However, rapid atrioventricular conduction through the AV node during physical activity may lead to symptoms of haemodynamic impairment such as dizziness, syncope or sudden extreme fatigue. Therefore, in the absence of primary disorders or major cardiac disease, recommendations for competitive or leisure-time sports participation will largely depend on the ventricular rate during AF episodes, especially during high sympathetic tone. A careful history, and, if possible, ECG recording during such circumstances (exercise test, Holter, long-term event recorder) will guide treatment. If there is a fast ventricular response or symptoms of haemodynamic impairment, bradycardic treatment is mandatory for sports continuation. The patient should be instructed to stop physical activity on the emergence of palpitations or other major symptoms. The therapeutic goal of rate control, however, is difficult to reach in athletes because beta-blockers will not be well tolerated (or are even prohibited in the case of competitive athletes), and digoxin or calcium antagonists alone will not be potent enough to slow the heart rate during exertional AF. Often a combination of bradycardic agents is needed, with individually titrated therapy avoiding sinus bradycardia at rest or chronotropic incompetence during exercise. When the heart rate during recurrent paroxysms of AF (or during permanent AF) is acceptable at maximal physical performance in any given athlete and there are no signs of haemodynamic impairment, sports activity can be resumed.

In patients with a single episode of AF or with only very sporadic paroxysms, the resumption of all sports activity without treatment can be considered when stable rhythm is present for more than 3 months and when the AF episodes did not lead to major haemodynamic impairment.

Caution should specifically be exercised with the use of class 1 antiarrhythmic drugs in monotherapy in patients with AF. These drugs may prevent AF recurrences. They can, however, convert AF into slow atrial flutter, which may conduct one-to-one to the ventricles during situations of high sympathetic tone [51,52]. Impregnation of the ventricles with the class 1 drug will lead to broad QRS complexes (resembling VT) and profound negative inotropic effects leading to cardiogenic shock and even sudden death. Class 1 drugs can be initiated for the prevention of AF episodes after previous evaluation has shown adequate ventricular rate control during exercise. Prophylactic ablation of the flutter circuit must be considered in athletes in whom therapy with class 1 drugs is indicated. The 'hybrid' therapy of class 1 drugs and the ablation of flutter may obviate the need for maintenance therapy with bradycardic agents [53,54].

In some athletes with paroxysmal AF, class 1 drugs can be used only for acute reconversion therapy (the 'pill-in-the-pocket' approach) [55]. It is prudent to instruct these patients to refrain from sports as long as the arrhythmia persists and until one to two half-lifes of the antiarrhythmic drug have passed.

Given the difficulties of treating athletes with AF, as mentioned above, non-pharmacological options such as pulmonary vein isolation [56] or other extensive left atrial ablation approaches [57] have to be considered, especially in patients in whom AF is induced by focal atrial tachycardia. Mid-term results suggest freedom from AF between 50–80% in patients with persistent and paroxysmal AF, respectively, but there are no long-term data available nor data in this specific patient population. Moreover, possible complications such as pulmonary vein stenosis, cardiac tamponade, stroke and left atrial oesophageal fistula have to be discussed with the patient before an ablation strategy is chosen. After a successful ablation procedure and the absence of symptomatic recurrences for 3 months or more, resumption of all sports activity seems warranted, but the athletes should be followed up closely (i.e. every 6 months).

In competitive and leisure-time athletes with unsuccessful rhythm control or under rate control therapy, anticoagulation may be necessary depending on the presence of classic risk factors for thromboembolic events [58]. Anticoagulation therapy excludes these individuals from sports with a risk of bodily collision or trauma.

Atrial flutter

Atrial flutter is uncommon in the young healthy population. It is usually the manifestation of a counterclockwise

(or rarely clockwise) re-entrant circuit around the tricuspid valve. In athletes with atrial flutter, the presence of structural heart disease such as cardiomyopathy should be excluded. Atrial flutter may be present together with AF or may develop after the administration of class 1 antiarrhythmic drugs for AF ('class-1c flutter'). Flutter may convey an increased thromboembolic risk (dependent on risk factors as for AF). The arrhythmia may be life threatening during exertion because of one-to-one conduction to the ventricles under high sympathetic tone. As discussed under 'Atrial fibrillation', one-to-one conduction is facilitated with class 1 or class 3 drugs as a result of slowing of the flutter circuit, especially when no concomitant rate control therapy is initiated.

Catheter ablation of the isthmus is a highly effective and safe therapy [59,60], and is therefore recommended as first-line therapy in both competitive and leisure-time athletes. Recurrence rates are low and rare beyond 3 months. Non-competitive sports participation can be allowed early after ablation unless symptoms of major haemodynamic impairment were present during exercise before ablation, and the resumption of competitive sports activity is possible after a 3-month period free of flutter.

Many patients with successful flutter ablation will develop paroxysmal or persistent AF during follow-up [60]. A history of endurance sports has been identified in multivariate analysis as an independent risk factor for AF development after flutter ablation, indicating common functional (autonomic nervous) or structural mechanisms in their pathogenesis [47]. The recommendations of 'Atrial fibrillation' are therefore also valid for these athletes, both concerning arrhythmia management and anticoagulation therapy. In the presence of combined atrial flutter and AF, isthmus ablation is certainly recommended, with the continuation of drug therapy for AF ('hybrid therapy') [53,54].

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