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Recommendations for participation in leisure-time physical activity and competitive sports in patients with arrhythmias and potentially arrhythmogenic conditions: Part 1: Supraventricular arrhythmias. A position statement of the Section of Sports Cardiology and Exercise from the European Association of Preventive Cardiology (EAPC) and the European Heart Rhythm Association (EHRA), both associations of the European Society of Cardiology

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Abstract

Symptoms attributable to arrhythmias are frequently encountered in clinical practice. Cardiologists and sport physicians are required to identify high-risk individuals harbouring such conditions and provide appropriate advice regarding participation in regular exercise programmes and competitive sport. The three aspects that need to be considered are: (a) the risk of life-threatening arrhythmias by participating in sports; (b) control of symptoms due to arrhythmias that are not life-threatening but may hamper performance and/or reduce the quality of life; and (c) the impact of sports on the natural progression of the underlying arrhythmogenic condition. In many cases, there is no unequivocal answer to each aspect and therefore an open discussion with the athlete is necessary, in order to reach a balanced decision. In 2006 the Sports Cardiology and Exercise Section of the European Association of Preventive Cardiology published recommendations for participation in leisure-time physical activity and competitive sport in individuals with arrhythmias and potentially arrhythmogenic conditions. More than a decade on, these recommendations are partly obsolete given the evolving knowledge of the diagnosis, management and treatment of these conditions. The present document presents a combined effort by the Sports Cardiology and Exercise Section of the European Association of Preventive Cardiology and the European Heart Rhythm Association to offer a comprehensive overview of the most updated recommendations for practising cardiologists and sport physicians managing athletes with supraventricular arrhythmias,

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and provides pragmatic advice for safe participation in recreational physical activities, as well as competitive sport at amateur and professional level. A companion text on recommendations in athletes with ventricular arrhythmias, inherited arrhythmogenic conditions, pacemakers and implantable defibrillators is published as Part 2 in Europace.

Keywords
Arrhythmia, athlete, atrial fibrillation, sports cardiology, supraventricular tachycardia, Wolf-Parkinson-White

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Introduction

The goal of the present article is to discuss the criteria to ensure safe participation in regular exercise programmes and competitive sports for individuals with arrhythmias or pro-arrhythmogenic conditions, based on updates from previous consensus documents.[1–3] This article also provides guidance for the appropriate evaluation of athletes with arrhythmias, assessment of risk, and the criteria for giving medical advice on eligibility or disqualification.

These recommendations assume that the arrhythmia diagnosis has already been made. The issues related to the modalities and setting of screening are beyond the scope of this text. Previous recommendations for sports participation from the Sports Cardiology Section of the European Society of Cardiology (ESC)[1–3] have been in place for over 10 years. The revision of these documents was prompted by a number of considerations, including our better understanding of the genetic basis for several arrhythmias, the improved diagnostic capability to identify pathologic substrates by imaging techniques, and long-term observational studies that have improved our knowledge of the natural history of arrhythmias in patients engaged in sports and exercise programmes. Easier access to medical information through the media has substantially improved the level of awareness of the general athletic population, which also has led to a change of the environment for providing recommendations.

Therefore, the European Heart Rhythm Association (EHRA) and the Sports Cardiology and Exercise Section of the European Association of Preventive Cardiology (EAPC), both associations of the ESC, have undertaken a combined effort to update previous reports and offer a novel, scientific and comprehensive set of recommendations to help examining physicians with appropriate criteria for managing athletes with arrhythmias. A companion text on recommendations in athletes with ventricular arrhythmias, inherited arrhythmogenic conditions, pacemakers and implantable defibrillators is published in Europace.[4]

As in other EHRA consensus documents, we opted for ranking of our recommendations using 'coloured hearts' (Table 1). This EHRA grading does not include definitions of the 'level of evidence'. Therefore, this categorization must not be considered directly similar to that used for official ESC guideline recommendations, which apply a classification (Class I–III) and level of evidence (A, B and C) to recommendations. A green heart indicates a 'should do this' recommendation or indicated treatment or procedure that is based on at least one randomised trial, or is supported

Table 1. Scientific rationale of recommendations.[a]

Definitions where related to a treatment or procedure	Consensus statement instruction	Symbol
Scientific evidence that a treatment or procedure is beneficial and effective. Requires at least one randomised trial or is supported by strong observational evidence and authors' consensus.	'Should do this'	
General agreement and/or scientific evidence favour the usefulness/efficacy of a treatment or procedure. May be supported by randomised trials based on a small number of patients or trials that are not widely applicable.	'May do this'	
Scientific evidence or general agreement not to use or recommend a treatment or procedure.	'Do not do this'	

[a]This categorization for our consensus document should not be considered as being directly similar to that used for official European Society of Cardiology (ESC) guideline recommendations which apply a classification (I–III) and level of evidence (A, B, and C) to recommendations.

by strong observational evidence that it is beneficial and effective. A yellow heart indicates general agreement and/or scientific evidence favouring a 'may do this' statement or the usefulness/efficacy of a treatment or procedure. A 'yellow heart' symbol may be supported by randomised trials based on a small number of patients or trials that are not widely applicable. Treatment strategies for which there is scientific evidence of potential harm and should not be used ('do not do this') are indicated by a red heart.

A general framework for evaluation and management of arrhythmias in athletes

When patients with known arrhythmias or with a potentially arrhythmogenic condition want to engage in sports activity, three principles should guide management. Addressing these three questions in a systematic way will lead to a logical decision about whether sports can be continued and to what extent. In many cases, there will not be an unequivocal answer to each question. Therefore, an open discussion with the athlete will be needed, in order to reach a balanced approach on how to proceed. The three aspects that need to be addressed are: (a) is there an increased risk of life-threatening arrhythmias by doing sports, and by doing sports at which level?; (b) how can we control symptoms due to arrhythmias that are not life-threatening, but that may hamper the performance of sports, and/or reduce the quality of life at rest and during sports activities; and (c) what is the impact of sports continuation on the natural progression of the underlying arrhythmogenic condition?

The general view on the association between sports and arrhythmias is that exercise sets the stage for an arrhythmia in the context of an underlying and pre-existing condition (structural or electrical, inherited or acquired). Underlying disease is of specific prognostic importance since the main determinant for sports participation in patients with arrhythmias is the presence of heart disease.[5–9] It is not always unequivocal, however, to detect and define underlying pathology. Therefore, careful diagnostic evaluation is mandatory. How wide and how deep that evaluation is pursued depends on multiple factors, including the family history, the type of sports, amateur or professional setting, symptoms, etc.

Moreover, regular exercise programmes may induce or accelerate the progression of an underlying arrhythmic substrate. Typical examples are the promotion of atrial fibrillation (AF),[10–14] or of right ventricular (RV) dilatation and arrhythmias in the presence of an underlying desmosomal mutation,[15,16] and even without underlying mutations.[17–19] Conceptually, all the physiological structural and functional changes of the heart in the context of regular physical activity, known as athlete's heart, can contribute to the development of arrhythmias, whether at the atrial, nodal and ventricular level.[20] There is no clear border between physiology and pathophysiology, e.g. where atrial or ventricular dilatation becomes pro-arrhythmogenic. This explains why recommendations for sports participation in arrhythmogenic conditions are so complex. For many situations, we simply lack good prospective data for answering these questions in a definite way. Shared decision-making should consider these uncertainties (see below). Dissecting the answers to each of the three important questions mentioned above will allow a structured approach for the physician and rational discussion with the athlete/patient who wants to engage in sports. The guidance below will follow this framework when discussing different arrhythmogenic conditions. We hope that it will contribute to more rational shared decision-making, instead of emotional opinionated choices by athletes or physicians.

If the personal history reveals 'palpitations', it is essential to inquire with the patient to define these as accurately as possible: do these 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 like lightheadedness, pre-syncope or syncope, unexplained weakness, fatigue, chest pain or dyspnoea. These symptoms may be the only indicator of arrhythmia and therefore should be interpreted cautiously when noted in a physically active patient's history. Presyncope or syncope due to neurocardiogenic (vasovagal) aetiology is prevalent in healthy, athletic people and carries a good prognosis but it should unambiguously be differentiated from an arrhythmic aetiology which may carry an infaust prognosis.[21] 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 inquire for performance-enhancing or other drugs that might be pro-arrhythmic or could affect the heart (like antibiotics or antidepressants, 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 of 35 years or older. A thorough family history should inquire for sudden death (especially in youth and adulthood, but also sudden infant death) and/or arrhythmogenic conditions known to have a genetic inheritance.

In some cases, it is advisable to obtain a haematocrit (e.g. unexplained sinus tachycardia), thyroid function markers (e.g. AF) and electrolyte balance (in all

patients with suspected or documented arrhythmias). Also urine analysis to exclude illicit drug use may be considered in some situations.

Echocardiography can assess the presence or extent of structural heart disease, but may need to be supplemented by more sophisticated imaging techniques such as those extensively described elsewhere.[22]

Exercise testing and Holter monitoring will often be indicated to correlate symptoms with arrhythmia findings and vice versa. In some, recordings by consumer smart phones or watches may provide relevant information. There may be a need for an external event recorder or even an implantable loop recorder. An invasive electrophysiological (EP) study is indicated in some 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.

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 like sudden exertional fatigue or dyspnoea needs to be discussed with them.

Shared decision-making: cultural and ethical background of decision-making in athletes

The legitimacy of medical advice in individuals with cardiovascular (CV) abnormalities prior to participation in sports and/or intensive exercise programmes is substantiated by the scientific evidence that such individuals may be exposed to an increased risk for sudden cardiac death (SCD)/cardiac arrest (CA) or progression of the underlying disease. Conversely, proper advice regarding type/intensity of sport, or abstinence from competition in selected cases, may reduce the risk.[5,6,23–25]

Indeed, elite and professional athletes represent a special subset of the population, not only for their outstanding performances, perceived as models for achievement and success in our society, but also for the substantial economic interests that they attract, and the intense pressure to which they are exposed. The unpredictable nature of SCD/CA has historically necessitated a cautious approach regarding advising intense exercise and competitive sport in individuals with CV diseases or serious arrhythmias believed to convey a high-level of risk. Such attitude has inevitably affected several athletes who would never have experienced an adverse event during their career and has been viewed as an intrusion into their personal freedom, rather than a precautionary measure aimed to reduce the unforeseeable risk associated with sport participation.[3,26] Athletes, especially the youngest ones, are usually driven by the will to accept future risks, but achieve the immediate benefits conveyed by a successful athletic career. Several actors, including the sponsor, the media, the athletic association or college, and the relatives/partner, may contribute to the athlete's decision to pursue the competitive/professional career regardless of the severity of the risk. In this context, the role of the physician has historically been very prominent, in order to timely identify and manage the underlying CV pathological condition and properly advise on sport participation.[26,27] The societal landscape has changed, e.g. through medical information that can readily be accessed via the Internet. Therefore, at the present time, the approach to the eligibility/disqualification decision should not only be based on the physician competence and responsibility but should consider also the perspective of the patient/athlete, i.e. should ideally be shared decision-making.[28]

The role of the physician remains pre-eminent in order to correctly make a diagnosis in a timely manner, to assess the risk associated with the arrhythmia of the athlete, and to inform the athlete in a clear, comprehensive and plain fashion of the cardiac risks associated with the sport participation. We advocate an individualised approach, including discussion of symptoms, natural history of the disease, risk of SCD/CA, characteristics of the sport discipline also in relation to age of the athlete. On the other hand, the athlete's responsibility consists of providing comprehensive and truthful information regarding CV symptoms and/or limitation of physical performance, as well as a clear picture of the familial medical background. Any medical treatment and/or drug abuse should be known. The motivation of the athlete to pursue a specific sport discipline and the goal to be reached should be discussed, in order for the examining physician to give appropriate advice.

Some exceptions to this ideal scenario may be mentioned, such as the case of an athlete with full judgment and complete understanding wishing to participate in competitive sports against strongly founded medical advice. In this case, it is recommended that the entire medical assessment and final advice should be reported in writing, of which a copy may be handed to the athlete. A special case is when the cardiac risk is disproportionately high and/or in case of a minor athlete; in such scenarios, the physician should not only exhaustively inform the candidate-athlete, but also share the information with competent parties (i.e. the relatives or guardian) in order to ensure that full understanding is achieved and the proper decision will be made.

Finally, we should acknowledge that this process is conducted differently according to the cultural and societal background of shared values, and the medical competencies existing in each country.[29] The weight of the physician's advice and the athlete's autonomy is also influenced by different legal systems. Therefore, shared decision-making should always be viewed in the context of the existing medical and legal rules.

Arrhythmias and arrhythmogenic conditions

The following paragraphs present different arrhythmias and arrhythmogenic conditions in detail. 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 and, hence, this needs to be gauged in each individual case.[30] Classifications of sports depending on the amount of dynamic and static CV demand have been published elsewhere (Figure 1).[22]

Sinus bradycardia

Sinus bradycardia as low as 30 bpm at rest or even lower during sleep, sinus arrhythmia, sinus pauses and junctional escape rhythms are common in young athletes.[30,31] These rhythm variations are part of the athlete's heart and reflect both functional and structural adaptations. These arrhythmias usually have a benign prognosis. However, sinus bradycardia may persist in veteran athletes,[32,33] and progression into sinus node disease with an indication for pacemaker implantation has been reported in a small proportion of former competitive endurance athletes.[34] Sinus bradycardia and high heart rate variability have been quoted as evidence for high vagal tone in athletes.[35] However, there is an exponential decay-like relationship between heart rate and its variability, making heart rate variability an unreliable marker of cardiac autonomic nervous activity in athletes.[36,37] Double autonomic blockade suggests intrinsic sinus node remodeling in athletes.[38] Recently, exercise-induced downregulation of the pacemaking ion channel HCN4 has been demonstrated as an alternative explanation for sinus bradycardia in athletes.[39]

Clinical evaluation includes a careful history to determine whether sinus bradycardia is related to symptoms like dizziness or (pre-)syncope. Exercise testing may be useful for verifying a normal rate response. Symptomatic athletes and asymptomatic athletes with extreme bradycardia at rest (e.g. <30 bpm on ECG) should have long-term ambulatory ECG monitoring and a cardiac work-up to exclude structural heart disease.[40]

Cessation of sports activity may result in resolution of symptoms and improvement in rhythm after a 1–2 month period.[41] In these cases, resumption of all sports activity can be advised under close monitoring.

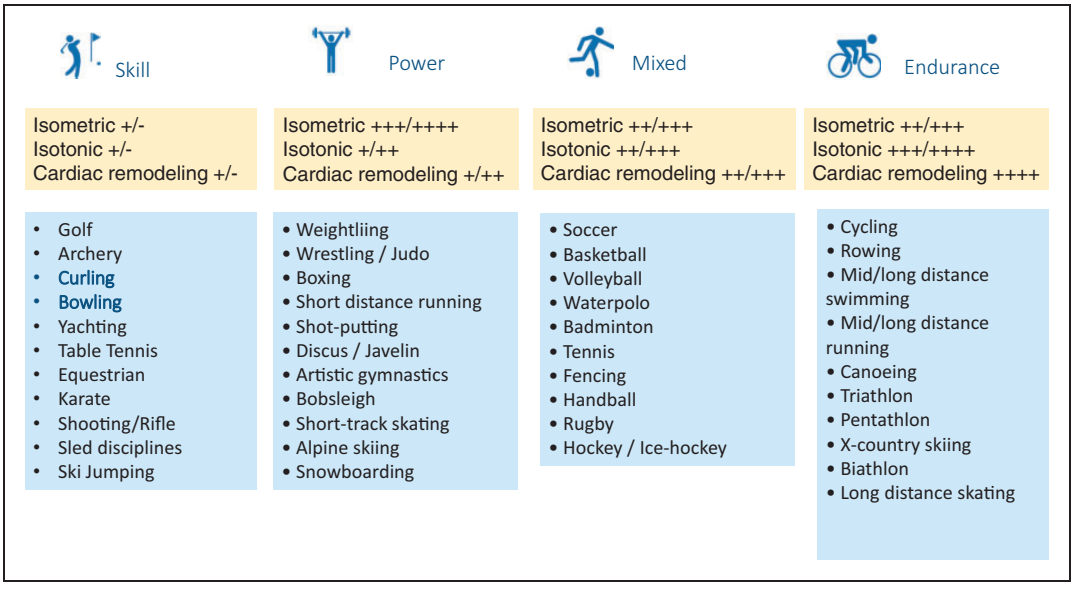

Figure 1. Different sports disciplines have variable impact on the cardiovascular system. Therefore, each athlete with an arrhythmogenic condition should be evaluated in the context of the expected stress on the cardiovascular system, and its potential impact on arrhythmias. Adapted from Pelliccia et al. with permission from Oxford University Press.[22]
− minimal/no change
+ minor/mild increase.
++ moderate/moderate increase.
+++ intensive/strong increase.

A follow-up evaluation after 6 months is recommended. The athlete should be instructed to consult earlier in the case of re-emergence of symptoms. Although resolution of sinus bradycardia may be incomplete, and symptoms may persist, pacemaker treatment is rarely needed in athletes.[34,40] The eligibility of sports participation in athletes with pacemakers is discussed in Part 2 of these recommendations.[4]

In asymptomatic patients with extreme bradycardia at rest (e.g. <30 bpm on ECG) a yearly follow-up could suffice. In those who were symptomatic before but became asymptomatic after 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 re-emergence of symptoms.

Consensus statement: Sinus bradycardia	Symbol
Athletes with symptomatic sinus bradycardia should be restricted from training and competition while being evaluated for structural heart disease.	
In the absence of symptoms, athletes with sinus bradycardia and related bradyarrhythmias can participate in all recreational or competitive sports unless otherwise excluded by an underlying structural heart disease.	

Atrioventricular (AV) nodal conduction disturbances

Slowing of AV nodal conduction forms part of the physiological adaptations to exercise.[30,42,43] Athletes have a large, though variable, prevalence of asymptomatic 1st degree AV block or 2nd degree Wenckebach type AV block (Mobitz type I), typically occurring at rest or during sleep.[30,42,44,45] It is not known whether the proportion is different in male and female athletes. Most of the 1st degree AV blocks exhibit a moderate PR interval extension of ≤280 ms.[41] Such findings in athletes or individuals who want to perform leisure time physical activity do not involve exclusion criteria for any sports. These vagally induced conduction disturbances should, however, resolve during sympathetic stimulation or exercise.[42] If so, no further investigation and no therapy are indicated.

Even if symptoms of dizziness or syncope occur, these may resolve with temporary discontinuation of sports, after which sports can be resumed. A re-evaluation is recommended within a few months in such cases. Otherwise a yearly follow-up is appropriate, especially in those athletes with extreme PR prolongation (>300 ms), since data on the natural progression in these athletes are not available.

In the case of 2nd degree AV block Mobitz type 2, or of 3rd degree AV block, a more comprehensive diagnostic evaluation is warranted to exclude underlying structural heart disease (which may be present more often in these athletes) and an EP study to evaluate infra-His block.[46] Also, associated ventricular tachyarrhythmias should be excluded, by Holter, exercise testing and even EP study. Such conduction disturbances may still be secondary to athletic activity.[33,47] If the conduction disturbances resolve during sympathetic stimulation or exercise, a high vagal tone as the cause is more likely. However, ventricular pauses of ≥3 s or a resting heart rate ≤40 bpm due to conduction disturbances are rarely observed in leisure-time athletes, even if such disturbances have been reported in high-level athletes.[45,48] In cases without structural heart disease, a deconditioning phase of 2 months may be considered, with resumption of low-moderate sports if the AV conduction defect has resolved. In cases of persisting or recurring symptoms (dizziness, (pre)syncope, exercise intolerance), pacemaker implantation, finally, may be indicated.

In patients with underlying structural disease and 2nd degree type 2 AV block or 3rd degree AV block, pacemaker implantation is recommended according to the present pacemaker guidelines.

Consensus statement: AV nodal conduction disturbances	Symbol
Athletes with asymptomatic 1st degree AV block or Wenckebach type 2nd degree AV block at rest and normalisation during exercise can participate in all sports	
Symptomatic athletes with Wenckebach type 2nd degree AV block while awake, should temporarily refrain from sports. If exclusion of a structural cause and normalisation within 2 months, low-moderate sports can be resumed. After 4 weeks with tolerable low-moderate sports, higher intensity sports may be undertaken. Follow-up with Holter recordings is advised.	
In athletes with extreme PR prolongation 1st degree AV block (>300 ms), more intensified follow-up is warranted (e.g. every 6 months).	
2nd degree AV block Mobitz type 2 or 3rd degree AV block requires exclusion of underlying structural heart disease.	
In 2nd degree AV block Mobitz type 2 or 3rd degree AV block without structural heart	

(continued)

Continued

Consensus statement: AV nodal conduction disturbances	Symbol
disease, a deconditioning phase of 2 months can be considered.	
In 2nd degree AV block Mobitz type 2 or 3rd degree AV block with structural heart disease, a pacemaker is recommended.	

Atrial premature beats (APBs)

Infrequent APBs (<3/h) are a common finding, both in athletes and in the general population.[49] Their frequency is independently associated with age, height, history of cardiovascular disease, natriuretic peptide levels and also with physical activity and exercise training.[30,50,51] Frequent APBs (>30/h) may indicate a higher risk of AF, stroke and death in the general population.[52] APBs may serve as a triggering factor for AF also in endurance athletes.[43]

APBs may pass unnoticed but may also lead to symptoms like palpitations, usually as isolated extrasystoles. Ambulatory Holter monitoring may be necessary to distinguish them from ventricular premature beats. Apart from a 12-lead ECG, physical examination and thyroid function tests, no further more extensive CV assessment is indicated. A full cardiac work-up may be considered in symptomatic athletes with a high burden of APBs.

Participation in any sports, competitive and recreational, is allowed in the absence of structural heart disease.

Consensus statement: APBs	Symbol
Participation in any sports, competitive and recreational, is allowed in the absence of structural heart disease.	

Paroxysmal supraventricular tachycardia (PSVT) without pre-excitation

PSVT is a generic term denoting rapid and regular supraventricular tachycardias (SVTs) with sudden onset and termination. PSVT includes aetiologically (a) atrioventricular nodal re-entrant tachycardia (AVNRT; most common); (b) atrioventricular re-entrant tachycardia (AVRT) involving an accessory pathway; or (c) ectopic atrial tachycardia (AT). PSVT has an estimated prevalence of 2.3/1000 overall.[53] PSVT is diagnosed based on a typical history and the ECG during tachycardia. As PSVT occurs sporadically, event recorders are useful for arrhythmia documentation. Sometimes athletes notice tachycardia episodes suggestive of PSVT on pulse watches. PSVT generally is considered a coincidence in athletes. A recent study, however, reported that athletes present more frequently with atypical subforms of AVNRT (44–45% versus 80–84%, $p<0.001$).[54] This is possibly related to cardiac remodelling with dilatation of the cardiac cavities leading to changed conduction properties in the septal area.

When an accessory pathway is only capable of conducting from the ventricles to the atria, there is no pre-excitation on the sinus rhythm ECG ('concealed pathway'). PSVT with pre-excitation is associated with a risk of sudden death and will, therefore, be discussed separately below since recommendations for athletic activity are different.

PSVT without pre-excitation is considered a benign arrhythmia provided structural heart disease is excluded, which is an essential step in the diagnostic work-up.[55] In rare cases, incessant SVT may induce cardiomyopathy.[56] During physical activity and sympathetic stimulation the heart rate increases during PSVT. This may lead to symptoms of haemodynamic compromise such as dizziness, fatigue or syncope, even in the absence of structural heart disease. Underlying CV disease is more common in AT compared with AVNRT or AVRT, and AT may also be associated with AF.

Given its association with sudden death, it is important to exclude the presence of pre-excitation in athletes with PSVT. Pre-excitation may be intermittent which usually indicates low risk properties of the pathway. However, exceptions occur. Minimal or 'latent' pre-excitation can be unmasked on a 12-lead ECG during sinus rhythm by vagal manoeuvres or intravenous administration of adenosine that slows conduction through the AV node; prolongation of the PR interval without a change in the QRS morphology, or transient AV block, rules out latent pre-excitation.

The underlying mechanism of PSVT may be suggested by characteristics of the ECG during arrhythmia but a definite diagnosis can only be established by an invasive EP study. However, recommendations for sports participation in PSVT without pre-excitation are not dependent on the type of underlying arrhythmia. Thus, in this patient group, an EP study is not

necessary for diagnostic purposes unless ablation is considered.

In a patient with PSVT who wants to perform competitive athletic activity, curative treatment by ablation is often recommended. Drug treatment is commonly not tolerated by athletes, may be illegal or may even be dangerous during competitive sports. If the PSVT is only sporadic and not associated with haemodynamic consequences even when it develops during exercise, or in case ablation is not desired or not successful, sports activity is allowed when there is no increased risk from a potential loss of consciousness (such as in pilots, motorsports drivers, parachute jumpers, divers). Exercise should be stopped as soon as palpitations recur but can be resumed after the cessation of palpitations. A yearly follow-up is recommended.

In patients who want to perform only leisure-time and low to medium intensity sports and are only paucisymptomatic during tachycardia, participation is allowed without definitive ablative treatment. Physical activity should be stopped as soon as palpitations arise. Prophylactic drug treatment with beta-blockers or calcium antagonists can be considered, although this treatment has limited efficacy and may not be tolerated. Class 1 drugs generally play a marginal role in the chronic management of PSVT. Therefore, ablation should also be considered as a definitive treatment in recreational athletes with recurrent PSVT despite drug treatment or as a first choice based on the patient's preference after discussion with the physician. In general, all patients with PSVT should be educated on how to safely perform vagal manoeuvres (such as carotid sinus massage or Valsalva manoeuvre) to facilitate termination of ongoing arrhythmia.

Ablation of PSVT is today performed with high procedural success rates ranging from 84% (AT) to 99% (AVNRT) and low procedural risk (<1% for AV block and tamponade).[57] After successful ablation the risk of recurrence is low with re-ablation rates ranging from 4.5% in AVNRT to 10.0% in AT.[57] Ablation outcome is equally safe in athletes as it is in non-athletes with similar acute success rates, although athletes with AVNRT may experience a higher longer-term recurrence rate (10% vs 4%; $p = 0.03$).[54] Recurrence usually occurs within the first months after ablation with 50% occurring within 35 days.[58] Therefore, if no recurrence has developed after 1 month, 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 (i.e. after healing of the puncture sites) unless there are special circumstances (e.g. a history of exercise-related (pre)syncope or special findings at EP study).

Consensus statement: PSVT	Symbol
General recommendations in patients with a PSVT (AVNRT, AVRT conducted via a concealed accessory pathway, AT):	
PSVT patients should be evaluated by history, ECG, echo and other diagnostic procedures, where appropriate, to establish the diagnosis and to exclude (latent) pre-excitation and structural heart disease.	
Patients should be educated on how to safely perform vagal manoeuvres (e.g. Valsalva and carotid sinus massage) for arrhythmia termination.	
In patients with a PSVT, physical activity should be stopped as soon as the arrhythmia arises.	
An EP study is not necessary for diagnostic purposes unless ablation is considered.	
Recommendations in patients with a PSVT (AVNRT, AVRT conducted via a concealed accessory pathway, AT) who participate in competitive sports:	
If the PSVT is only sporadic, not associated with haemodynamic consequences and if ablation is not desired or successful, sports activity is allowed given there is no increased risk from a potential loss of consciousness. In these patients a yearly follow-up is recommended.	
Ablation therapy should be considered for curative arrhythmia treatment.	
After successful ablation, training can be resumed after 1 week (i.e. after healing of the puncture sites), and competitive activity after 1 month, in most cases.	
Recommendations in patients with a PSVT (AVNRT, AVRT conducted via a concealed accessory pathway, AT) who participate in leisure time low to medium intensity sports:	
Patients who are largely asymptomatic during tachycardia can participate in sports, except those sports with risk from a potential loss of consciousness (such as in pilots, motorsports drivers, parachute jumpers, divers).	
Ablation therapy should be considered for curative arrhythmia treatment if drug treatment fails to provide symptomatic improvement, or as a first choice based on the patient's preference after discussion with the physician.	

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

Symptomatic pre-excitation due to PSVT or AF. The Wolff-Parkinson-White (WPW) syndrome is defined as the presence of supraventricular arrhythmias in patients with ventricular pre-excitation (during sinus rhythm) due to an accessory AV pathway with antegrade conduction. Regular arrhythmias involving the accessory pathway include AVRT, either orthodromic (antegrade AV conduction over the AV node and retrograde over the accessory pathway) or antidromic (antegrade AV conduction over the accessory pathway and retrograde over the AV node). However, WPW patients may also incidentally develop AF which could lead to ventricular fibrillation (VF) and sudden death due to rapid antegrade conduction over the accessory pathway. Most WPW patients have normal cardiac anatomy, but in all patients with ventricular pre-excitation an associated structural cardiac disease (like hypertrophic cardiomyopathy or Ebstein anomaly) should be ruled out by physical examination, 12-lead ECG and imaging.

WPW patients with PSVT have an increased risk of AF due to secondary degeneration of AVRT and to sports-related atrial remodeling (see below). Athletes may have an increased risk for AF even after they have ceased high-level competitive sports. The majority of WPW-related sudden deaths occur during exercise or under emotional stress.[59,60] Therefore, sports activity in the presence of overt pre-excitation may expose WPW patients to an increased risk for sudden death if the accessory pathway has the potential for fast antegrade conduction. Ablation of the accessory pathway is often recommended in the general population and is mandatory in both competitive and recreational athletes with pre-excitation and documented arrhythmias.[61–65] The safety and efficacy of catheter ablation of the accessory pathway is well established. Ablation does not necessarily prevent future AF, especially in older patients, but this is then no longer associated with the risk of VF due to pre-excited AF.[64,66]

If palpitations are rare and associated with very good haemodynamic tolerance (even during exercise) and/or ablation may be associated with increased risk (e.g. AV block in patients with an anteroseptal accessory pathway) or if the athlete refuses ablation, management can be guided by assessment of the antegrade conduction characteristics of the accessory pathway by non-invasive testing or an invasive EP study. If non-invasive testing demonstrated intermittent pre-excitation at rest, abrupt loss of pre-excitation during exercise, or disappearance of pre-excitation after administration of a low dose of class 1 drugs, this indicates a long refractory period and an accessory pathway with low risk of fast pre-excited AF. An invasive EP study may be considered to guide ablation therapy based on the presence or absence of findings indicating an accessory pathway with increased risk of sudden death (see Table 2).[59,67–70] In the case of a long refractory period and hence low risk for sudden death, continuation of sports activity is allowed without ablation on the understanding that sporting activity should be interrupted in the event of recurrence of palpitations during sports. Sports in which the potential loss of consciousness could be fatal should be discouraged.

Table 2. Findings during an invasive electrophysiological study indicating an overt accessory pathway with increased risk of sudden death.[56,67–69]

Inducibility of AVRT or AF
A pre-excited R-R during AF $\leq$250 ms at baseline
A pre-excited R-R $\leq$220 ms during isoproterenol infusion
An antegrade refractory period $\leq$250 ms at baseline
Presence of multiple accessory pathways
Septal location of the accessory pathway (mainly posteroseptal and midseptal)

AF: atrial fibrillation; AVRT: atrioventricular re-entrant tachycardia.

After successful ablation, training practice can generally be resumed after 1 week (healing of the puncture sites) provided that there is no particular risk of arrhythmia recurrence (difficult ablation) and no other (pro-arrhythmic) structural heart disease. Follow-up with ECG recordings is warranted at 1 week, 1 month, 6 months and 1 year to exclude late recurrence of pre-excitation. After successful ablation, resumption of competitive sports is possible when the likelihood of accessory pathway conduction and risk are negligible (i.e. absence of pre-excitation on ECG, difficulty of ablation, prior history, structural heart disease), usually after 1–3 months.

Consensus statement: Pre-excitation and documented PSVT or AF (= WPW syndrome)	Symbol
In WPW patient imaging should be performed to rule out/detect structural heart disease.	
Ablation of the accessory pathway is recommended in competitive and recreational athletes with pre-excitation and documented arrhythmias, especially if the accessory pathway has a short refractory period ($\leq$250 ms at rest or $\leq$220 ms during isoproterenol infusion).	
After successful ablation, resumption of competitive sports is possible after ECG exclusion of recurrent conduction and when the likelihood of accessory pathway recurrence and risk are negligible (based on the difficulty of ablation, prior history, structural heart disease), usually after 1–3 months	

(continued)

Continued

Consensus statement: Pre-excitation and documented PSVT or AF (= WPW syndrome)	Symbol
If palpitations are rare and associated with very good haemodynamic tolerance (even during exercise) and/or an ablation may be associated with increased risks (e.g. AV block in patients with an anteroseptal accessory pathway), assessment of the antegrade conduction characteristics of the accessory pathway by non-invasive testing or by an invasive EP study may be considered to guide further therapy.	
Antiarrhythmic 'nodal' drugs in WPW-patients with pre-excited AF (intravenous adenosine as well as intravenous or oral digoxin and nonhidropyridine calcium channel antagonists) can accelerate conduction over the accessory pathway and are therefore potentially harmful.[64, 66]	

Asymptomatic pre-excitation. Asymptomatic pre-excitation is generally discovered by chance when performing a routine ECG. It may be due both to Kent bundles (WPW pattern) and, rarely, to Mahaim bundles, which are AV fibres with decremental conduction.

In many asymptomatic subjects, the absence of symptoms is only a temporary situation.[71] Among subjects with pre-excitation, over 90% are asymptomatic during childhood, about 65% when adolescents and about 40% in the over-30-years age group.[61,71–76] The absence of symptoms can also be due to absence of retrograde conduction over the AV node (necessary for antidromic AVRT) or absence of retrograde conduction over the Kent bundle (a necessary feature for the appearance of orthodromic AVRT), but the latter does not rule out potentially fast antegrade conduction.[77,78] In general, the risk of SCD in patients with asymptomatic pre-excitation is low,[79] and depends on the anterograde conduction properties of the Kent bundle. Two studies involving 386 WPW patients followed for 10 years found that 15% developed AF.[80,81] Sudden death is the first manifestation of the WPW syndrome in about half of WPW patients who die suddenly, and it usually presents during exercise or emotional stress.[59]

The risk of sudden death can be evaluated non-invasively or by EP study, as discussed above.[60,63,68,78,79,82–85] An EP study can be avoided in patients with intermittent pre-excitation, which most often (but not necessarily)[86,87] is associated with longer anterograde refractory periods.[67] In asymptomatic Mahaim pre-excitation the risk of sudden death is negligible, but the differential diagnosis with WPW can usually only be made by an EP study. The risk in asymptomatic children is controversial,[79,88,89] but a recent meta-analysis showed that it is very low in the absence of heart disease. As a consequence, an EP study generally is delayed in asymptomatic children aged <12 years, although one study suggested that prophylactic assessment and ablation reduces the risk of sudden death.[88]

Consensus statement: Asymptomatic pre-excitation	Symbol
Sport eligibility may be granted in asymptomatic patients with intermittent pre-excitation (rest and effort).	
In asymptomatic competitive athletes with persistent pre-excitation, an EP study is recommended in adults and in children aged 12 years or more.	
In asymptomatic recreational athletes with pre-excitation, risk assessment may first be considered via non-invasive testing (repetitive ECG ± anti-arrhythmic drug administration, Holter and/or exercise test to show intermittent pre-excitation or block at longer cycle length).	
If the EP study reveals one or more of the risk factors as described in Table 2, participation in sports should be denied unless catheter ablation of the accessory pathway has been performed.	
In asymptomatic subjects with borderline EP parameters, the decision to allow participation in sports should take into account parameters such as haemodynamic impact of arrhythmia, ease of induction of sustained AF, absence of structural heart disease, and type of sports.	

Atrial fibrillation

Prevalence and pathophysiology. AF is the most common sustained arrhythmia, also in lifelong athletes. While moderate physical activity is a cornerstone of AF prevention,[90–94] AF is more prevalent in active and former competitive athletes and those performing high-intensity endurance sports compared to the general population.[10–14] All this leads to a U-shaped relationship of physical activity and AF.[95–97] The risk is increased by age and underlying predisposing conditions like hypertension, with direct effects of exercise presumably through altered autonomic tone (both increased sympathetic activity during exercise and increased vagal tone at rest), volume load during exercise, atrial dilatation, fibrosis and atrial hypertrophy as shown in animal models and humans.[51,98–101] Nevertheless, the slightly increased risk of AF in endurance athletes does not reduce the significant overall CV

benefit of sports participation. In some patients, AF may develop secondary to PSVT with or without overt pre-excitation or to AT, due to an underlying cardiomyopathy, silent myocarditis or other structural heart disease.[14] The inducing role of performance-enhancing drugs, like anabolic steroids, in the development of the AF substrate or in triggering the arrhythmia has been described.[102–104]

Prognostic and symptomatic relevance. AF by itself is not a life-threatening arrhythmia in the absence of underlying structural heart disease or underlying pre-excitation and these factors should always be excluded before allowing sports activity. Rapid conduction through the AV node during physical activity may lead to symptoms of haemodynamic impairment like dizziness, syncope, fatigue and impaired physical performance.[65] Athletes with AF may also have intermittent atrial flutter (AFL), which could be associated with higher ventricular rates during exercise (see next section).

Required minimum assessment. All athletes presenting with AF should have an assessment including ECG, echocardiogram, stress testing and thyroid function test. A full medical history and a query for drug use (especially performance-enhancing drugs) should be included. Diagnostic assessment should further focus on the underlying substrate (i.e. hypertension) and/or structural heart disease.

If a clear primary cause is present, sports participation should be temporarily stopped and can be resumed after correction of the cause and after restoration of a normal heart rate. 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. The patient should be instructed to stop physical activity on the emergence of palpitations or other major symptoms. If adequate rate-control is secured and there are no symptoms of haemodynamic impairment, competitive sports participation is possible.

Recommended management for symptomatic improvement. As in all patients with AF, a treatment strategy of either rate or rhythm control needs to be chosen. Some athletes may choose to avoid any therapy and may tolerate either short lived episodes of AF, or even permanent AF.

Rate control, although an option, will not be the ideal strategy in most competitive athletes. The therapeutic goal of rate control is difficult to reach in athletes since beta-blockers will not be well tolerated (or are even prohibited in the case of competitive athletes) and digoxin or calcium channel blockers alone may not be potent enough to slow heart rate during exertional AF. Often a combination of individually titrated bradycardic agents is needed. When the heart rate during AF is acceptable at maximal physical performance or during stress testing (e.g. evaluated during in-office exercise test while in AF, or with Holter or long-term monitoring during training/competition) or the episodes of AF are rare and short, and there are no signs of haemodynamic impairment, sports activity can be resumed.

In cases of rhythm control, a strategy of antiarrhythmic drug treatment or catheter ablation are an option. Antiarrhythmic drugs are often poorly tolerated. Caution should specifically be mentioned for 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 AFL, which may conduct 1-to-1 to the ventricles during situations with high sympathetic tone (see below).[105,106] Impregnation of the ventricles with the class 1 drug can lead to broad QRS complexes (resembling ventricular tachycardia) and profound negative inotropic effects leading to cardiogenic shock and even sudden death. Therefore, if drug therapy is the choice, prophylactic ablation of the typical flutter circuit should be considered in athletes in whom therapy with class 1 drugs is indicated. Such 'hybrid' therapy of class 1 drugs and ablation of the flutter circuit (i.e. cavo-tricuspid isthmus) might also obviate the need for maintenance therapy with bradycardic agents.[107,108] In some athletes with paroxysmal AF class 1 drugs can be used only for sporadic acute reconversion therapy (the 'pill-in-the-pocket' approach). It is prudent to instruct these patients to refrain from sports after intake as long as the arrhythmia persists and until two half-lives of the antiarrhythmic drug have passed.[109] Other antiarrhythmic drugs are rarely a viable treatment option in athletes due to their potential side effects.

For many athletes, catheter ablation by pulmonary vein isolation (PVI) is a viable treatment option, especially in those with symptomatic paroxysmal AF and without structural heart disease. It is reasonable to offer athletes PVI as first-line therapy, but left atrial size and operator's experience should be considered.[110] Long-term results (i.e. 3–5-year follow-up) generally suggest freedom from AF between 50–80% in patients with persistent and paroxysmal AF respectively, a rate which seems comparable to that in non-athletes.[111–113] However, there are few long-term data available in athletes after PVI, also not on any pro-arrhythmia in the form of left AFLs. After a successful ablation procedure and absence of symptomatic recurrences for $\geq$1 months, resumption of competitive sports activity seems warranted.

Oral anticoagulation (OAC) may be warranted in athletes with AF depending on the CHA_2DS_2-VASc

score.[65] However, many athletes will most likely have a low thromboembolic risk and may therefore not be candidates for OAC. Sports with direct bodily contact or prone to trauma should be avoided in patients on OAC.[114]

Patients with AF should be followed up at least once a year. A common question is whether sports can be resumed at the same level after successful ablation. Detraining can reduce AF in animal models,[98,101] but the role of detraining for AF in humans remains unknown. If it is considered that physical activity contributed to the pathogenesis of AF, continuation of the same sports stimulus may continue the disease process and lead to atrial structural abnormalities beyond the pulmonary veins. Therefore, shared decision-making on the desirability of continuing at the pre-ablation sports activity level is important.

Consensus statement: AF	Symbol
Regular physical activity is recommended to prevent AF.	
Athletes with AF should undergo a work-up including ECG, echocardiogram, stress test, and thyroid function test. Alcohol or drug abuse should be excluded.	
Athletes without structural heart disease, in whom AF is well-tolerated and short-lived may participate in sports without therapy.	
AF ablation should be considered to prevent AF recurrence in athletes with recurrent symptomatic AF and/or in those who do not want drug therapy.	
In patients with a pill-in-the-pocket approach, patients should refrain from sports participation as long as AF persists, and until two half-lifes of the antiarrhythmic drug have passed.	
After successful AF ablation and absence of symptomatic recurrences, resumption of competitive sports activity after 1 month is warranted.	
Prophylactic cavo-tricuspid isthmus ablation to prevent flutter should be considered in AF patients who want to engage in intensive exercise and in whom class I drug maintenance monotherapy is initiated.	
In case of AF-related symptoms, or in case of increased ventricular rate while in AF during exercise, rhythm or rate control should be instituted.	
In athletes with AF and the indication for oral anticoagulation, the bleeding risk of the specific sport should be considered before further sports participation.	

Atrial flutter

AFL is uncommon in young athletes.[115] However, exercise-induced enlargement of the atria may lead to both left and right atrial remodeling in the aging athlete,[116] and may predispose to a counterclockwise (or rarely clockwise) re-entrant circuit around the tricuspid valve ('cavo-tricuspid isthmus-dependent AFL'). In former competitive endurance athletes, AFL was more prevalent than AF.[34] AFL often precedes, but also coexists with AF.[13,117,118] AFL caused by re-entrant circuits not involving the cavo-tricuspid isthmus is uncommon in athletes and is usually related to prior cardiac surgery or AF ablation procedures.[55] In athletes with documented AFL the presence of structural heart disease such as cardiomyopathy should be excluded. AFL may develop as a consequence of administration of class 1c antiarrhythmic drugs for AF ('class-1c flutter').

The arrhythmia may be life-threatening during exertion due to 1-to-1 conduction to the ventricles under high sympathetic tone. This form of conduction is facilitated with class 1c antiarrhythmic drugs, especially when no concomitant rate control therapy is initiated.[65] Like AF, AFL conveys an increased thrombo-embolic risk. Oral anticoagulation should be initiated according to guidelines, based on the CHA_2DS_2-VASc score. Bleeding risk, especially in contact sports, has to be discussed with the athlete.[65]

Catheter ablation of the cavo-tricuspid isthmus is a highly effective and safe therapy and therefore is recommended as first-line therapy in both competitive and leisure-time athletes.[119,120] Using bidirectional isthmus block as an end-point, the recurrence rate is as low as 7% and does not increase with the duration of follow-up.[121] Therefore, it is recommended as first-line therapy in both competitive and leisure-time athletes. In the general population, 34–58% of patients developed AF after cavo-tricuspid isthmus ablation during an average follow-up of 14–26 months, with AF increasing with follow-up duration.[120,121] A history of endurance sports has been identified as an independent risk factor for AF after AFL ablation.[13] In the presence of combined AF and AFL, isthmus ablation is recommended, especially if continuation of drug therapy for AF ('hybrid therapy') is considered,[107,108] given the risk of life-threatening 1-to-1 conduction.

In the absence of structural heart disease, non-competitive sports participation can be allowed early after ablation unless symptoms of major haemodynamic impairment were present during exercise before ablation. Resumption of competitive sports activity is possible after a one-month period free of

AFL unless symptoms of major haemodynamic impairment were present during exercise before ablation.

Consensus statement – AFL	Symbol
Catheter ablation of the isthmus is recommended as first-line therapy in both competitive and leisure-time athletes for cavo-tricuspid isthmus-dependent AFL.	
In the absence of structural heart disease, non-competitive sports participation can be allowed 1 week after ablation (i.e. after healing of the puncture sites) unless symptoms of major haemodynamic impairment were present during exercise before ablation.	
Resumption of competitive sports activity is possible after a 1-month symptom-free period after ablation.	

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