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ESC Report

Recommendations for competitive sports participation in athletes with cardiovascular disease

A consensus document from the Study Group of Sports Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology

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Introduction

The rationale for offering an expert consensus document concerning the participation in competitive sports by individuals with cardiovascular (CV) disease is based on the widely accepted clinical perception, substantiated by scientific evidence,[1] that athletes with underlying (even clinically silent) CV disease have an increased risk for sudden cardiac death (SCD) or clinical deterioration in comparison with normal individuals, by virtue of their regular exercise training and sports participation. Therefore, the aim of the present recommendations is to provide careful guidelines to physicians and consultant cardiologists regarding the evaluation of athletes with CV abnormalities and to suggest sports activities that can be safely performed.

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These recommendations assume that the cardiac diagnosis has already been made, so that the issues directly related to screening for CV disease[2] are beyond the scope of this document.

Target of the recommendations

For the purpose of this document, the target of recommendations are competitive athletes, here defined as individuals of young and adult age, either amateur or professional, who are engaged in exercise training on a regular basis and participate in official sports competition. Official sports competition (local, regional, national, or international) is defined as an organized team or individual sports event that, placing a high premium on athletic excellence and achievement, is organized and scheduled in the agenda of a recognized Athletic Association.

A characteristic of competitive sports, regardless of the level of achievement, is the strong proclivity for participants to exert themselves physically until their limits and improve performance.[3]

Our interest in competitive sports is dictated by the awareness that competitive athletes (in particular, the elite and professional ones) represent a special subset of the society, not only for their outstanding performances, but also for the substantial economic interests they gather, as well as the intense pressure to which they are exposed by sponsors, Athletic Associations, and media.

Conversely, the present recommendations do not apply to individuals participating in a variety of recreational or leisure sports activities, from modest to vigorous, on either a regular or an inconsistent basis, not requiring systematic training or pursuit of excellence, nor the same pressure to prevail against others which is characteristic of competitive sports.

Nature of the recommendations

The present recommendations represent the consensus document of an international panel of experts appointed by the European Society of Cardiology (ESC), including clinical CV specialists with experience in exercise physiology, sports medicine, and clinical cardiology. The present recommendations are based on published scientific evidence, when available, and on personal experience and consensus of experts. However, in consideration of the scarcity of scientific investigations concerning the effect of regular sport activities on the pathophysiology and clinical course of several CV diseases, the panel acknowledges the difficulties inherent in formulating arbitrary recommendations, particularly for CV diseases in which scientific evidence is inconsistent. Therefore, caution is needed in applying the present document, and efforts should be made to tailor precise advice to each individual. The aim of the panel was to formulate indications which represent a reasonable balance between the risks and the benefits inherent with competitive sports participation, and not simply restrict sports activity that could conceivably be associated with increased risk. The present recommendations represent, therefore, a prudent, contemporary, and practical document for advising competitive sports activity in patients with CV disease.

Finally, these recommendations should be placed in perspective. The present document is based on the (scarce) scientific evidence relating the risk of death or disease progression of several CV diseases with exercise training and sports, and more information is expected to be available when a pre-participation screening programme will be widely implemented across the European countries.[2] Therefore, the recommendations formulated in this document are going to be updated when larger knowledge of the natural history of CV diseases in relation to sports participation will be available.

Implementation of the recommendations within the European countries

At present, a large heterogeneity (or lack) of regulations exists in the European countries, with only a few countries requiring medical clearance of competitive athletes and, in individuals with CV disease, having implemented guidelines which are considered the standard of medical care.[4] This panel believes that a common protocol for evaluation and management of competitive athletes with CV disease is now needed, in consideration of the unlimited opportunities for professional athletes to move across the European Union. Implementation of a unique and appropriate consensus document will be of relevant medical (and legal) value for physicians required to evaluate athletes with CV disease in the different European countries.

In the absence of binding requirements established by law, this panel recommends that the present recommendations represent the *standard of medical care* for evaluation of competitive athletes with CV disease. Adherence to these recommendations will have substantial and cost-effective impact on medical care, by enhancing the safety of athletic activities and reducing the legal controversies related to different (or lack of) regulations. This panel advises that implementation of the present recommendations will occur in the different European countries with keeping in mind the different legal and cultural backgrounds, possibly by legislative action, and with the support of the national scientific and sport organizations.

Role of the examining physician

Should the physician be the ultimate authority in determining whether an athlete with CV disease can participate in competitive sports? Alternatively, can the athlete with CV disease just sign an informed consent form and be engaged in a risky and potentially life-threatening sports activity?

Owing to the unique structure and pressures of competitive sports, individuals with CV disease may not always use proper independent judgement in assessing the overall risk associated with a competitive sports career. This panel believes that the examining physician (as well as the consultant cardiologist) has the ethical, medical, and legal obligation to exhaustively inform the candidate of the risks inherent in competitive athletic lifestyle and, when the CV risk appears to be disproportionately high, the physician should be responsible for the final decision, with the aim to prevent adverse clinical events and/or reduce the risk for disease progression. The safeguard of the athlete's health is the paramount objective of the physician, regardless of other considerations, such as the visibility and

economic revenues for sponsors or Athletic Association, which may be dependent on the athlete's competitive activity. These recommendations are intended, therefore, to support the physician's decision in such difficult instances and to offer medical protection to the athlete from the unsustainable hazard of a competitive sports activity.

Search methodology

We performed systematic MEDLINE searches of the English language literature up to 2004, and reviewed abstracts of all pertinent research and review articles, and also included selected articles suggested by experts in this field.

Classification of sports

A classification of the different sports is provided in *Table 1*. Sports activities are classified into two main categories (i.e. dynamic and static) and intensity is roughly divided into low, moderate, and high.[5] This classification is intended to provide a schematic indication of the CV demand associated with different sports, with an additional notification of those disciplines associated with increased risk of bodily collision and those associated with an enhanced risk if syncope occurs (which should be avoided in certain cardiac patients).

Recommendations for participation in competitive sports in athletes with congenital heart disease

General considerations

Patients with congenital heart disease (CHD) who participate in competitive sports may expose themselves to an upper limit of physical and mental stress.[6,7] Because the available literature regarding exercise and sports participation in patients with CHD is limited, a restrictive attitude seems wise. As a general recommendation, exercise physical tolerance in children with CHD is better than in adults with CHD, and dynamic exercise seems to be more suitable than static exercise.[8,9] Some lesions are not compatible with competitive sports, due to their morphologic severity/complexity and tendency to serious arrhythmias, including Eisenmenger syndrome, secondary pulmonary hypertension, univentricular hearts, coronary artery abnormalities, Ebstein anomaly, congenitally corrected transposition of the great arteries, and transposition of the great arteries corrected by the Mustard, Senning, or Rastelli procedure.

Arrhythmias

As survival has improved among patients with CHD, arrhythmias become a more common problem in the long-term course of these patients. Although SCD is a feared consequence of CHD, only few cases occur during exercise.[10] Patients who have undergone extensive atrial or ventricular surgery are at greater risk of arrhythmias due to scarring and ventricular dysfunction. Transventricular repair and repair late in life, both pre-dispose for arrhythmias and represent a possible reason for excluding these patients from competitive sports. In addition, the presence of ventricular dysfunction represents a serious risk for developing arrhythmias. These are important issues to consider in patients with repaired tetralogy of Fallot or corrected and uncorrected atrial septal defect (ASD), ventricular septal defect (VSD), or atrioventricular septal defect (AVSD).[11] For instance, in tetralogy of Fallot, a gradual widening of the QRS duration of $>$160 ms might indicate an increased risk of sustained

Table 1 Classification of sports

	A. Low dynamic	B. Moderate dynamic	C. High dynamic
I. Low static	Bowling	Fencing	Badminton
	Cricket	Table tennis	Race walking
	Golf	Tennis (doubles)	Running (marathon)
	Riflery	Volleyball	Cross-country skiing (classic)
		Baseball[a]/softball[a]	Squash[a]
II. Moderate static	Auto racing[a,b]	Field events (jumping)	Basketball[a]
	Diving[b]	Figure skating[a]	Biathlon
	Equestrian[a,b]	Lacrosse[a]	Ice hockey[a]
	Motorcycling[a,b]	Running (sprint)	Field hockey[a]
	Gymnastics[a]		Rugby[a]
	Karate/Judo[a]		Soccer[a]
	Sailing		Cross-country skiing (skating)
	Archering		Running (mid/long)
			Swimming
			Tennis (single)
			Team handball[a]
III. High static	Bobsledding[a,b]	Body building[a]	Boxing[a]
	Field events (throwing)	Downhill skiing[a,b]	Canoeing, Kayaking
	Luge[a,b]	Wrestling[a]	Cycling[a,b]
	Rock climbing[a,b]	Snow boarding[a,b]	Decathlon
	Waterskiing[a,b]		Rowing
	Weight lifting[a]		Speed skating
	Windsurfing[a,b]		Triathlon[a,b]

Adapted and modified after Mitchell et al.[5]
[a]Danger of bodily collision.
[b]Increased risk if syncope occurs.

ventricular tachycardia. A history of frequent and complex tachyarrhythmias is a reason for CHD patients to refrain from participation in competitive sports.

Ventricular function
Both left ventricular (LV) and right ventricular (RV) function may be impaired due to inadequate myocardial protection during surgical repair. Because impaired ventricular function is a trigger for arrhythmias and reduced exercise tolerance, the assessment of systolic and diastolic indexes of LV and RV function is mandatory.

Pulmonary vascular resistance
Lesions with longstanding left-to-right shunt, corrected or uncorrected, may have caused persistent pulmonary hypertension.[12] In addition, patients with mitral valve dysfunction are at risk for developing pulmonary hypertension. Assessment of pulmonary arterial pressure during exercise is indicated and an intermittent increase in systolic pressure to <35 mmHg may be safely tolerated.

Dysfunction of the valves
Like exercise, both valve stenosis and/or valve insufficiency result in ventricular and/or atrial overload and have to be accurately estimated before giving recommendations for competitive sports[13] (see also Acquired cardiac valve diseases).

Conduits and mechanical valves
Patients with conduit should be refrained from competitive sports and patients with mechanical valves on anticoagulation should avoid sports with a risk of bodily collision.

Functioning class
A score according to the New York Heart Association (NYHA) classification can be useful. Only patients in NYHA Class I are entitled for unrestricted participation in competitive sports.

Abnormal exercise blood pressure response
An abnormal increase in systolic blood pressure (BP) during exercise is found in patients with repaired coarctation of the aorta (CoA). Whether it is of significant long-term importance in competitive athletes with CoA is not completely known. An impaired rise or even fall in BP during exercise may be seen in patients with aortic stenosis and should lead to further investigations.

Prophylaxis of endocarditis
Patients with CHD participating in competitive sports follow the same recommendations regarding endocarditis prophylaxis as non-competitors.

Unrecognized CHD
Most of CHD lesions are diagnosed during childhood (provided a good health care system); nevertheless, late diagnosis of ASD, CoA, and LV outflow tract obstructions is not uncommon. A structured screening programme of all athletes would probably identify most of these cases.[2] Abnormal coronary arteries, in contrast, are unlikely to be diagnosed during life, despite extensive screening.

Evaluation

The evaluation should include a medical history, with special emphasis on surgical reports, a careful physical examination, electrocardiography, chest X-ray, and echocardiography, including an estimation of peak pulmonary artery pressure. We recommend a questionnaire to describe the symptomatic status according to the criteria of the NYHA. Treadmill or bicycle exercise testing with ergospirometry best evaluates the work capacity. The exercise testing should be standardized (e.g. the Bruce protocol) and include electrocardiogram (ECG) recording, maximal heart rate, BP, and possibly respiratory gas analysis with oxygen uptake (VO_2max).

Individualized supplementary investigations
Magnetic resonance imaging (MRI) can be extremely useful in describing both functional and anatomical features, especially in cases which are inadequately visualized by echocardiography. If an arrhythmia is mentioned by the patient or common in that particular lesion, both 24 h Holter ECG monitoring and exercise testing are indicated. In some situations, e.g. when an elevated pulmonary artery pressure is suspected and cannot be determined by other methods, cardiac catheterization may be indicated.

Follow-up and re-evaluation
It is recommended to closely follow the clinical course of the subject with CHD participating in competitive sports activities, and structured reassessment may be indicated, according to the clinical judgement, every 6 or 12 months in most patients. Finally, a complete reassessment is advisable every second or third year, according to the lesion and the individual clinical course.

Conclusion

Because physical activity and sports participation have positive effects on both physical and mental health, only those patients with CHD who are likely to deteriorate as a consequence of regular physical exercise and/or those in whom exercise may trigger serious atrial/ventricular tachyarrhythmias should be restricted from sports participation. Indeed, it should be considered that the haemodynamic balance in patients with CHD varies considerably, even among patients with the same lesion. This makes it impossible to state recommendations that are valid in all cases and support the relevance of the examining cardiologist to tailor the recommendation to each individual patient.

Recommendations

See *Table 2*.

Recommendations for participation in competitive sports in athletes with acquired cardiac valve diseases

Mitral valve stenosis

Mitral valve stenosis (MVS) is generally of rheumatic origin. This defect results in increased left atrial (LA) pressure, leading to pulmonary hypertension. The increase in heart rate and cardiac output associated with intensive exercise

Table 2 Recommendations for competitive sport participation in athletes with CHDs

Lesion	Evaluation	Criteria for eligibility	Recommendation	Follow-up
ASD (closed or small, unoperated) and Patent foramen ovale	History, NYHA functional class, PE, ECG, Echo, chest X-ray, ET	<6 mm defect, or 6 months post-closure, with normal pulmonary artery pressure, no significant arrhythmia or ventricular dysfunction	All sports In patients with PFO, percutaneous closure may be considered before regular scubadiving	Yearly
VSD (closed or small unoperated)	History, NYHA functional class, PE, ECG, Echo, chest X-ray, ET	Restrictive defect (left-to-right gradient >64 mmHg) or 6 months post-closure, no pulmonary hypertension	All sports	Yearly
AVSD	History, NYHA functional class, PE, ECG, Echo, chest X-ray, ET	No or only mild AV valve insufficiency, no significant subaortic stenosis or arrhythmia, normal maximal gas exchange measurements	All sports	Yearly. Complete reassessment every second year
Partial or complete anomalous pulmonary venous connection	History, NYHA functional class, PE, ECG, Echo, chest X-ray, ET, MRI	No significant pulmonary or systemic venous obstruction, no pulmonary hypertension or exercise-induced atrial arrhythmia	All sports	Yearly
Persistent ductus arteriosus (operated)	History, NYHA functional class, PE, ECG, Echo, chest X-ray, ET	6 months post-closure and no residual pulmonary hypertension	All sports	Not needed
Pulmonary stenosis (mild native or treated)	History, NYHA functional class, PE, ECG, Echo, chest X-ray, ET	Native or 6 months post-interventional/post-surgical; peak transvalvular gradient <30 mmHg, normal RV, normal ECG or only mild RV hypertrophy, no significant arrhythmias	All sports	Yearly
Pulmonary stenosis (moderate native or treated)	History, NYHA functional class, PE, ECG, Echo, chest X-ray, ET	Native or 6 months post-interventional/post-surgical; peak transvalvular gradient between 30 and 50 mmHg, normal RV, normal ECG or only mild RV hypertrophy	Low and moderate dynamic and low static sport (I A, B)	Every 6 months
Coarctation of the aorta (native or repaired)	History, NYHA functional class, PE, ECG, Echo, chest X-ray, ET, MRI	No systemic hypertension; peak pressure gradient between the upper and lower limbs of <21 mmHg, a peak systolic BP during exercise of <231 mmHg, no ischaemia on exercise ECG, no LV overload.	Low and moderate dynamic and static sport (I A,B + II A, B) If interposed graft avoid sport with a risk of bodily collision	Yearly. Complete reassessment every second year
Aortic stenosis (mild)	History, NYHA functional class, PE, ECG, Echo, chest X-ray, ET	Mean transvalvular gradient <21 mmHg, no history of arrhythmia, no syncope, dizziness, or angina pectoris	All sports, with exception of high static, high dynamic sports	Yearly
Aortic stenosis (moderate)	History, NYHA functional class, PE, ECG, Echo, chest X-ray, ET, 24 h Holter	Mean transvalvular gradient between 21 and 49 mmHg, no history of arrhythmia, no syncope, dizziness, or angina pectoris	Low dynamic and static sport (IA)	Every 6 months

Continued

Table 2 *Continued*

Lesion	Evaluation	Criteria for eligibility	Recommendation	Follow-up
Tetralogy of fallot	History, NYHA functional class, PE, ECG, Echo, chest X-ray, ET, 24 h Holter, MRI	Non or only mild RVOT obstruction, no more than mild pulmonary regurgitation, a normal or near normal biventricular function and no evidence of arrhythmia	Low and moderate static and dynamic sport (I A,B + II A, B)	Yearly. Complete reassessment every second year
		Moderate residual lesion with RV pressure <50% of systemic pressure, or residual VSD or moderate pulmonary regurgitation, but normal biventricular function	Low static and dynamic sport (IA) Patients with conduit should avoid sport with risk of bodily collision	
Transposition of the great arteries (arterial switch)	History, NYHA functional class, PE, ECG, Echo, chest X-ray, ET	No or only mild neo-aortic insufficiency, no significant pulmonary stenosis, no signs of ischaemia or arrhythmia on exercise ECG	All sports, with exception of high static, high dynamic sports	Yearly

ECG, 12-lead electrocardiogram; ET, exercise testing; Echo, echocardiography; PE, physical examination; 24 h Holter, 24 h Holter ECG monitoring. Follow-up includes medical record, NYHA functional class, PE, ECG, and Echo. Supplementary investigation will be performed dependent on lesion and symptoms.

can markedly increase the pulmonary arterial pressure and may eventually lead to acute pulmonary oedema.[14] Embolization by atrial thrombus represents a further complication, which usually occurs in the presence of atrial fibrillation (AF) and enlarged left atrium.[15] To date, the long-term effects on the pulmonary circulation and on the right ventricle of repeated pulmonary arterial pressure boosts as a result of chronic physical activity are not completely known.

Evaluation

Presence of MVS can be detected by characteristic auscultation and severity can be determined by ECG, echocardiography, chest X-ray, and exercise testing.

Echocardiography[16] allows assessment of valve opening area, presence of calcification, and papillary muscle function. The contribution of regurgitation should also be considered in the calculation of the valve opening area. Pulmonary systolic arterial pressure can be assessed by Doppler-echocardiography in the presence of tricuspid regurgitation, even during/after exercise. Exercise testing (or cardiopulmonary testing) can add information regarding the haemodynamic behaviour and occurrence of arrhythmias (particularly AF). Invasive testing, e.g. Swan–Ganz catheterization, is indicated only in selected cases, when accurate assessment of pressure in the pulmonary circulation is needed for therapeutic or legal purposes. Athletes who develop a pulmonary artery systolic pressure >80 mmHg during exercise are likely to develop severe adverse effects on RV function over time.

Classification

The severity of MVS can be categorized as follows:

(i) Mild = mitral valve opening area >1.5–2.5 cm^2, with pulmonary systolic arterial pressure <35 mmHg, and mean gradient ≤7 mmHg;
(ii) Moderate = mitral valve opening area between 1.0 and 1.5 cm^2, with resting pulmonary systolic arterial pressure between 35 and 50 mmHg, and a mean gradient between 8 and 15 mmHg;
(iii) Severe = mitral valve opening area <1.0 cm^2, with resting pulmonary systolic arterial pressure >50 mmHg, and a mean gradient >15 mmHg.

Patients with MVS and AF must receive anticoagulation treatment (provided there are no contraindications) to avoid risk of systemic embolism.

Recommendations

See *Table 3*.

For subjects with MVS associated with AF, see also section Arrhythmias.

Mitral valve regurgitation

The most frequent cause of mitral valve regurgitation (MVR) is the prolapse of leaflets. Other causes include post-rheumatic fever, infectious endocarditis, coronary heart disease (ischaemic cardiomyopathy), or connective tissue disease, e.g. Marfan's syndrome (MS) or dilated cardiomyopathy. MVR is responsible for a regurgitated blood into left atrium, which causes increased LV diastolic filling and raises LA pressure.

Table 3 Recommendations for competitive sport participation in athletes with valvular disease

Lesion	Evaluation	Criteria for eligibility	Recommendations	Follow-up
MVS	History, PE, ECG, ET, Echo	Mild stenosis, stable sinus rhythm	All sports, with exception of high dynamic and high static (IIIC)	Yearly
		Mild stenosis in AF and anticoagulation	Low-moderate dynamic, low-moderate static (I A,B + II A, B), No contact sport	Yearly
		Moderate and severe stenosis (AF or sinus rhythm)	Low dynamic and low static (IA) No contact sport	Yearly
MVR	History, PE, ECG, ET, Echo	Mild-to-moderate regurgitation, stable sinus rhythm, normal LV size/function, normal exercise testing	All sports	Yearly
		If AF, in anticoagulation	All sports, with exception of contact sport	Yearly
		Mild-to-moderate regurgitation, mild LV dilatation (end-systolic volume <55 mL/m^2), normal LV function, in sinus rhythm	Low-moderate dynamic, low-moderate static (I A,B + II A, B)	Yearly
		Mild-to-moderate regurgitation, LV enlargement (end-systolic volume >55 mL/m^2) or LV dysfunction (ejection fraction $<50\%$)	No competitive sports	
		Severe regurgitation	No competitive sports	
AVS	History, PE, ECG, ET, Echo	Mild stenosis, normal LV size and function at rest and under stress, no symptoms, no significant arrhythmia	Low-moderate dynamic, low-moderate static (I A,B + II A, B)	Yearly
		Moderate stenosis, normal LV function at rest and under stress, frequent/complex arrhythmias	Low dynamic and low static (IA)	Yearly
		Moderate stenosis, LV dysfunction at rest or under stress, symptoms	No competitive sports	
		Severe stenosis	No competitive sports	
AVR	History, PE, ECG, ET, Echo	Mild-to-moderate regurgitation, normal LV size and function, normal exercise testing, no significant arrhythmia	All sports	Yearly
		Mild-to-moderate regurgitation, proof of progressive LV dilatation	Low dynamic and low static (IA)	Yearly

Continued

Table 3 *Continued*

Lesion	Evaluation	Criteria for eligibility	Recommendations	Follow-up
		Mild-to-moderate regurgitation, significant ventricular arrhythmia at rest or under stress, dilatation of the ascending aorta	No competitive sports	
		Severe regurgitation	No competitive sports	
TVS	History, PE, ECG, ET, Echo	No symptoms	Low-moderate dynamic, low-moderate static (I A,B + II A, B)	Every second year
TVR	History, PE, ECG, ET, Echo	Mild-to-moderate regurgitation	Low-moderate dynamic, low-moderate static (I A,B + II A, B)	Yearly
		Any degree, with right atrial pressure >20 mmHg	No competitive sports	
Poly-valvular diseases	History, PE, ECG, ET, Echo	See most relevant defect		
Bioprosthetic aortic or mitral valve	History, PE, ECG, ET, Echo	Normal valve function and normal LV function, in stable sinus rhythm	Low-moderate dynamic, low-moderate static (I A,B + II A, B)	Yearly
		If AF and anticoagulation	No contact types of sports	Yearly
Prosthetic (artificial) aortic or mitral valve	History, PE, ECG, ET, Echo	Normal valve function and normal LV function and anticoagulation	Low-moderate dynamic, low-moderate static (I A,B + II A, B) No contact types of sport	Yearly
Post-valvuloplasty	History, PE, ECG, ET, Echo	See the residual severity of the MVS or MVR	Low-moderate dynamic, low-moderate static (I A,B + II A, B)	Yearly
Mitral valve prolapse	History, PE, ECG, ET, Echo	If unexplained syncope, or family history of sudden death, or complex supraventricular or ventricular arrhythmias, or long QT interval, or severe mitral regurgitation	No competitive sports	
		Absence of the earlier cited cases	All sports	Yearly

ECG, 12-lead electrocardiography; Echo, echocardiography; ET, exercise stress testing; PE, physical examination; sport type, see *Table 1*.

Evaluation
MVR is detected by a characteristic auscultation. Severity of MVR can be assessed by Doppler-echocardiography,[17,18] ECG, and chest X-ray. In assessing the severity of MVR, it should be considered that LV end-diastolic size is usually increased in well-trained athletes and, therefore, the definition of LV dilatation should be adjusted. In athletes, the severity of MVR should be based on LV end-systolic volume, with the cut-off of 55 mL/m^2 useful to distinguish individuals with LV enlargement of clinical relevance. The extent of the LA enlargement should also be considered, because of the proclivity to AF. The 24 h Holter monitoring is recommended when arrhythmias are evident (or strongly suspected) and when MVR is due to prolapse of the leaflets.

Classification
There are several methods to classify MVR. The widely accepted PISA-method uses the width of the jet and the velocity to assess the degree of regurgitation. In addition, the vena contracta method is suitable for classification.[17]

(i) Mild = regurgitation width <0.3 cm;
(ii) Moderate = regurgitation width 0.3–0.6 cm;
(iii) Severe = regurgitation width >0.6 cm.

Recommendations
See *Table 3*.

Patients with AF must receive anticoagulation treatment and they should avoid sports with risk of bodily collision (see also section Arrhythmias).

Aortic valve stenosis

The most common cause for aortic valve stenosis (AVS) is a rheumatic or congenital lesion. Calcified degenerative stenosis is often associated with congenital abnormality of the aortic valve (e.g. bicuspid valve), especially when aortic stenosis is identified in young patients. Syncope can appear in a young athlete with a mild degree of AVS.[19] Nevertheless, symptoms such as angina and dyspnoea usually appear in a late stage of the disease. Occurrence of SCD is far more probable if one of these symptoms is present.[20]

Evaluation
AVS is often detected by a characteristic auscultation. Determination of the gradient and valve opening area should be carried out by Doppler-echocardiography. Exercise testing (by bicycle ergometry) is recommended to assess LV function, development of ST segment depression, BP behaviour, and possible arrhythmias. Given that the severity of AVS is often progressive, periodical evaluation is necessary.

Classification
Classification is based on the mean aortic valve gradient and aortic valve opening area (AVA).

(i) Mild = mean gradient ≤20 mmHg (AVA >1.5 cm^2);
(ii) Moderate = mean gradient between 21 and 49 mmHg (AVA 1.0–1.5 cm^2);
(iii) Severe = mean gradient ≥50 mmHg (AVA <1.0 cm^2).

Recommendations
See *Table 3*.

Aortic valve regurgitation

The commonest causes of aortic valve regurgitation (AVR) include congenital bicuspid aortic valve, rheumatic lesion, infectious endocarditis, MS, aortic dissection, systemic arterial hypertension, and rheumatoid spondylitis. AVR causes dilatation of the LV cavity with increases in LV diastolic and systolic volumes. Bradycardia can worsen the haemodynamic pattern, due to lengthening of the diastolic duration and increase of the regurgitant volume. Athletes with AVR in the chronic compensated phase are often asymptomatic and can remain so far for many years. As LV dysfunction proceeds, symptoms occur, typically including dyspnoea on exertion, arrhythmias and, in advanced cases, angina.[21]

Evaluation
AVR is often detected by a characteristic auscultation. LV dilatation can be evaluated by echocardiography. In consideration that LV cavity dimension is increased in healthy athletes as a consequence of training, this should be considered when assessing LV size in the presence of AVR. Exercise testing (or cardiopulmonary testing) can be helpful in the evaluation of exercise tolerance[22] and should be carried out up to the level that is consistent with the sports participation, enabling the patient tolerance to be specifically assessed. Because of possible progression of AVR over time, periodical evaluation is recommended.

Classification
The haemodynamic severity of AVR can be classified as follows:

(i) Mild = absence of peripheral signs of AVR and normal LV and atrial size and function; small dimension of the diastolic flow signal on Doppler-echocardiography.
(ii) Moderate = peripheral signs of AVR, mild-to-moderate enlargement of the LV, normal systolic function, moderate dimension of the diastolic flow signal on Doppler-echocardiography.
(iii) Severe = peripheral signs of AVR, marked dilatation of the LV and/or evidence of LV dysfunction; enlarged atrial size, and large dimension of the diastolic flow signal on Doppler-echocardiography.

Recommendations
See *Table 3*.

For athletes with Marfan's syndrome: see specific section.

Tricuspid valve stenosis

In most cases, tricuspid valve stenosis (TVS) is caused by rheumatic fever and is associated with MVS. In the presence of MVS and TVS, patients should be assessed with reference to the MVS. An isolated TVS is rare.[23] If the patient is asymptomatic (no dizziness, no dyspnoea, or peripheral oedema), participation in competitive sports may be possible.

Recommendations
See *Table 3*.

Tricuspid valve regurgitation

Tricuspid valve regurgitation (TVR) is often the consequence of RV dilatation. Rheumatic fever and infectious endocarditis are less common causes. Primary TVR leads to volume overload of the RV, increased venous pressure, and congestive symptoms.

The severity of TVR can be determined non-invasively by physical examination, chest X-ray, and echocardiography.

Recommendations
See *Table 3*.

Multi-valvular diseases

Multi-valvular diseases frequently occur in connection with rheumatic fever, myxomatous valvular diseases, or infectious endocarditis. These conditions can be diagnosed by physical examination and assessed quantitatively by Doppler-echocardiography. Multi-valvular diseases of mild severity may reciprocally worsen each other for their haemodynamic effects and, therefore, great caution is needed in these athletes with regard to participation in competitive sports.

Recommendations
See *Table 3*.

Post-operative patients with a prosthetic/bioprosthetic heart valve

Although patients are clinically improved by heart valve replacement, the long-term mortality after operative therapy is higher than in a control population. Furthermore, many patients with normal haemodynamic pattern at rest have abnormal values under physical stress. Therefore, exercise testing (possibly integrated with cardiopulmonary analysis) should be carried out up to the limit consistent with the sport the athlete wishes to pursue. Indeed, patients with mechanical valves (or bioprosthetic valves in selected cases) need systematic anticoagulation treatment, which further limits their potential for competitive sports participation. Patients with artificial valves should undergo periodic re-evaluation.[24]

Recommendations
See *Table 3*.

Athletes with a prosthetic or bioprosthetic valves who are receiving anticoagulation treatment should not participate in sports with a risk of bodily collision (*Table 1*).

Athletes post-valvuloplasty

Valvuloplasty is still performed in many patients with MVS, despite the likeness of restenosis and no clear advantage in comparison to valve replacement. Aortic valvuloplasty is only rarely performed in young patients with AVS. In patients after valvulotomy, recommendations for sports participation are based on the residual degree of severity of stenosis and/or regurgitation. Exercise testing should be carried out up to the level consistent with the level reached in the sport in which the patient participates.

Recommendations
See *Table 3*.

Mitral valve prolapse

Mitral valve prolapse (MVP) is mostly associated with myxomatous degeneration of the valve. It preferentially occurs in patients of tall stature and shows a familial cluster.[25] Ischaemic cardiomyopathy and hypertrophic obstructive cardiomyopathy are potential secondary etiologies.

Mitral regurgitation is often associated with MVP. In addition, rhythm disorders (i.e. brady- or tachyarrhythmias),[26] endocarditis, syncope, or embolism can also occur.

Evaluation
The typical auscultatory finding is a late-systolic click and a murmur due to late systolic or holosystolic regurgitation. Elongation and thickening of valve leaflets, degree of mitral regurgitation and LV dimension and function should be assessed by echocardiography. Evaluation should include exercise testing and/or Holter monitoring to assess the presence of arrhythmias. Yearly cardiologic evaluation is recommended, because the regurgitation can get worse by progressive degeneration of the leaflets.

Recommendations
See *Table 3*.

Prophylaxis against endocarditis

Infective endocarditis (IE) is an endovascular, microbial infection of intracardiac structures facing the blood, including infections of the large intrathoracic vessels. The early lesion is a vegetation of variable size, although destruction, ulceration, or abscess may follow. The increasing accuracy of echocardiography and therapeutic progress has contributed to the prognostic improvement in the last few years.

Patients with previous history of infective endocarditis, patients with prosthetic heart valves or acquired valve disease are considered high-risk patients and should receive antibiotic prophylaxis when exposed to risk of bacteraemia in accordance with the ESC recommendations.[27] Prophylaxis should be performed before dental, oral, respiratory, oesophageal, gastrointestinal, and genitourinary procedures. Dental hygiene is of relevance for prevention of IE.

As a general rule, all sports activity should be avoided when active infection with fever is present. Resumption of sport activity can be considered when the inflammatory process is completely extinguished, and systematic maintenance of endocarditis prophylaxis must be strictly observed.[27]

Recommendations for participation in competitive sports in athletes with cardiomyopathies, myocarditis, and pericarditis

Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is a primary cardiac disease, with hypertrophied and non-dilated left ventricle, in the absence of cardiac or systemic disease capable of producing LV hypertrophy of that magnitude.[28] Sports participation increases the risk for SCD in HCM patients,[28,29] and this disease is the most common cause of athletic field death in young athletes in the USA.[29]

Evaluation
Evaluation of athletes with suspected HCM includes personal and family history, physical examination, 12-lead ECG, and echocardiography.

12-Lead ECG. The majority (75–95%) of HCM patients show abnormal ECG patterns, commonly including markedly increased R- or S-wave voltages, deep and prolonged Q-waves, and deeply inverted T-waves.[30] ECG abnormalities may precede the LV hypertrophy and should raise suspicion of the disease in family members of HCM patients. However, trained athletes without evident CV disease may occasionally show similar ECG abnormalities[31] (see also Isolated abnormal ECGs).

Echocardiography. Classically, HCM is diagnosed when LV wall thickness is $\geq$13 mm, but a more substantial LV wall thickening may be found, usually with asymmetric distribution and sharp transition between contiguous segments.[28] LV hypertrophy becomes evident during adolescence, in association with body growth,[28] but in a few individuals, it may develop in midlife or beyond. LV end-diastolic cavity dimension is normal or even reduced, with an abnormal and sometimes bizarre shape. Diastolic LV filling (by Doppler-echocardiography)[32] and tissue doppler imaging (TDI) are abnormal in the majority of HCM patients and may precede the development of LV hypertrophy. Other alterations include malformation of the mitral valve, with elongation of the leaflets, or anomalous insertion of papillary muscles.[28]

In contrast, distribution of LV hypertrophy is symmetric in athletes and maximum LV wall thickness does not exceed 15–16 mm.[33] The LV cavity is enlarged (i.e. end-diastolic diameter $\geq$55 mm) with a normal shape, a normally positioned mitral valve, and no outflow tract obstruction.[33] LV filling (by Doppler-echocardiography)[32] and relaxation (by TDI) are normal. Most importantly, serial echocardiographic studies demonstrate reduction in LV wall thickness after complete deconditioning.[34]

Family screening is mandatory in borderline cases, and identification of the disease in a family member is diagnostic for HCM. Additional criteria include peak oxygen consumption (with $VO_2max > 50$ mL/kg/min being more consistent with athlete's heart[35]) and gender, because women athletes do not usually show LV wall thickening >12 mm.[36]

MRI is indicated, when echocardiography is inadequate, in identifying an atypical pattern of hypertrophy or apical HCM.

Molecular genetics. A variety of mutations of genes encoding structural and regulatory proteins of the cardiac sarcomere cause familial HCM.[37,38] However, genetic testing is still not available in the current clinical practice because of the substantial genetic heterogeneity of the disease, and the complex, time consuming and expensive techniques needed.

Recommendations
See *Table 4*.

Isolated abnormal ECGs. Special attention should be paid to athletes with ECG abnormalities (such as markedly increased QRS voltage, diffuse T-wave inversion, deep Q-waves in precordial leads) suggestive for HCM, in the absence of familial incidence of HCM and in the absence of LV hypertrophy. Evaluation of these athletes should include complete family screening, personal history, echocardiography, and 24 h Holter ECG monitoring. When SCD or HCM in the family are excluded, and in the absence of symptoms, arrhythmias and LV hypertrophy, and with a normal diastolic filling/relaxation, there is no reason for restricting athletes from competitive sports, but periodical clinical and diagnostic follow-up is recommended.

Dilated cardiomyopathy

Dilated cardiomyopathy (DCM) is a myocardial disease characterized by LV dilatation and impaired systolic function. DCM includes disorders which are familial or genetic in origin, or secondary to infection or inflammation, exposure to toxic substances, metabolic disorders, or of idiopathic origin.[39] Although not frequent, DCM represents a cause of SCD in athletes.

Evaluation
The assessment of athletes with suspected DCM includes personal and family history, physical examination, 12-lead ECG with exercise testing, echocardiography, and 24 h Holter monitoring.

Exercise testing and 24 h Holter monitoring. In young DCM patients, exercise performance may be only mildly impaired and arrhythmias are present at a very early stage of the disease, including supraventricular and ventricular tachyarrhythmias, as well as major conduction delays.

Echocardiography. The LV cavity is enlarged with respect to the LV walls, which are normal or only mildly thickened. LV shape becomes more spherical; the mitral annulus eventually enlarges with distortion of leaflets and resultant valvular regurgitation.[39] Most importantly, LV systolic function is diminished (with ejection fraction <50%), segmental wall motion abnormalities may be present, and stroke volume is usually reduced.

In contrast, the physiologic LV enlargement present in athletes (mostly engaged in aerobic disciplines such as cycling, cross-country skiing, rowing, long-distance running[40]) is characterized by a normal systolic function, no segmental wall motion abnormalities, and normal diastolic filling and relaxation (by Doppler-echocardiography and TDI). In case of borderline ejection fraction (i.e. $\geq$50, <60%), it may be useful to test LV function during exercise (by echocardiography or radionuclide imaging). Absence of significant improvement of systolic function at peak exercise is in favour of a pathological dilatation.

Recommendations
See *Table 4*.

Arrhythmogenic RV cardiomyopathy/dysplasia

Arrhythmogenic RV cardiomyopathy/dysplasia (ARVC) is a primary myocardial disease characterized histologically by fibro-fatty replacement of the RV myocardium, and clinically by life-threatening ventricular tachyarrhythmias in young individuals.[41] Sudden death may occur in young ARVC individuals in association with exercise, and this

Table 4 Recommendation for competitive sport participation in athletes with cardiomyopathies, myocarditis, and pericarditis

Lesion	Evaluation	Criteria for eligibility	Recommendations	Follow-up
Athletes with definite diagnosis of HCM	History, PE, ECG Echo		No competitive sports	
Athletes with definite diagnosis of HCM but low risk profile	History, PE, ECG, Echo, ET, 24 h Holter	No SD in the relatives, no symptoms, mild LVH, normal BP response to exercise, no ventricular arrhythmias	Low dynamic, low static sports (I A)	Yearly
Athletes with only gene abnormalities of HCM, without phenotype changes	History, PE, ECG, Echo	No symptoms, no LVH, no ventricular arrhythmias	Only recreational, non-competitive sport activities	Yearly
Athletes with definite diagnosis of DCM	History, PE, ECG, Echo		No competitive sports	
Athletes with definite diagnosis of DCM but low risk profile	History, PE, ECG, Echo, ET, 24 h Holter	No SD in the relatives, no symptoms, mildly depressed EF ($\geq$40%), normal BP response to exercise, no complex ventricular arrhythmias	Low-moderate dynamic and low static sports (I A,B)	Yearly
Athletes with definite diagnosis of ARVC	History, PE, ECG Echo		No competitive sports	
Athletes with active myocarditis or pericarditis	History, PE, ECG, Echo		No competitive sports	
Athletes after resolution of myocarditis	History, PE, ECG, Echo, ET	No symptoms, normal LV function, no arrhythmias	All competitive sports	First control within 6 months[a]
Athletes after resolution of pericarditis	History, PE, ECG, Echo, ET	No symptoms, normal LV function, no arrhythmias	All competitive sports	First control within 6 months[a]

DCM, dilated cardiomyopathy; Echo, echocardiography; EF, ejection fraction; ET, exercise testing; 24 h Holter; 24 h Holter ECG monitoring; LVH, left ventricular hypertrophy; PE, physical examination; SD, sudden death; sport type, see *Table 1*.

[a]Subsequent controls according to the individual case.

disease represents the most common cause of SCD in young athletes in Italy.[42]

Evaluation
Diagnosis of ARVC is based on the criteria previously proposed by an expert consensus panel.[41]

12-Lead ECG. In clinical practice, the ECG is of particular value in raising suspicion for ARVC, in consideration that ECG abnormalities are present in >50% of ARVC patients. The most common abnormalities include prolonged QRS duration >110 ms (with RBBB pattern) and inverted T-waves in the right precordial leads, evidence of epsilon wave and either isolated PVCs or VT (typically with LBBB pattern and vertical axis).

Echocardiography. In ARVC patients, echocardiography (or MRI) shows an enlarged RV cavity, segmental morphologic abnormalities (with thinning, bulging and aneurysms in the RV wall), and wall motion abnormalities. MRI may identify areas of altered signal intensity consistent with fibro-fatty replacement. Cine-MRI may be decisive for assessing wall motion anomalies. An enlarged right ventricle, in association with an enlarged left ventricle, may also be found in elite athletes (mostly engaged in endurance disciplines, such as cycling, rowing/canoeing),[40] but in these instances, RV wall thickness is normal and no segmental wall motion abnormalities are present.

Recommendations
See *Table 4*.

Myocarditis

Myocarditis is defined as an inflammatory process of the myocardium, with histological evidence of myocyte degeneration and necrosis of non-ischaemic origin, associated with inflammatory infiltration.[43]

Evaluation
The assessment of athletes with suspected myocarditis includes medical history, physical examination, 12-lead ECG, and echocardiography. Additional testing may be required according to the specific case.

History. The clinical picture usually starts with upper respiratory or gastrointestinal symptoms, but palpitations, fatigability, exertional dyspnoea, or syncope may be the clinical onset. Evidence of flu-like illness, or epidemiological circumstances supporting viral infection should be assessed.

12-Lead ECG. The ECG abnormalities include frequent and/or complex ventricular and/or supraventricular arrhythmia, ST-segment alteration (usually depression; rarely elevation), T-wave inversion and, occasionally, LBBB or AV blocks.[44]

Echocardiography. Global LV enlargement and dysfunction can be evident in certain cases;[45] however, localized wall motion abnormalities (usually in the apex), mildly enlarged LV cavity, and borderline systolic dysfunction are common. Modest pericardial effusion may be present, associated with increased reflectivity of pericardial leaflets.

Histology. Biopsy is not usually performed in the routine diagnostic course and may be reserved for selected circumstances, when needed for therapeutic or legal purposes.

Recommendations
See *Table 4*.

Pericarditis

Pericarditis is defined as an inflammatory process of the pericardium, which may also affect the subepicardial layers of the myocardium.

Evaluation
The assessment of athletes with suspected pericarditis includes medical history, physical examination, 12-lead ECG, and echocardiography.

History. Pericarditis usually starts with upper respiratory or gastrointestinal symptoms, but clinical presentation may include chest pain, increased fatigability, or exertional dyspnoea. The onset of the disease may also be concealed, and the clinical course characterized by only transient fever, without significant cardiac symptoms.

12-Lead ECG. In patients with pericarditis, a spectrum of ECG abnormalities may be present, including most commonly ST–T wave alterations mimicking ischaemic heart disease (IHD) and ventricular or supraventricular tachyarrhythmias.

Echocardiography. Often a pericardial effusion is present at the onset of the disease, with increased reflectivity and separation of the pericardial leaflets.

Recommendations
See *Table 4*.

Recommendations for sports participation in patients with Marfan's syndrome (MFS)

MFS is an autosomal dominant connective tissue disorder affecting 1:5000 subjects.[46] MFS is caused by fibrillin 1 gene defects (*FBN1*) and by mutation of transforming growth factor beta receptor 2 (TGFßR2) in a minority of patients (OMIN #154705). More than 600 mutations have been detected to date, the majority private.[47] Penetrance is complete, but the involvement of different organs/tissues is variable, with large phenotypic heterogeneity. The classical phenotype includes osteo-skeletal, CV, ocular, skin, pulmonary, and nervous anomalies. The primary cause of mortality in young people and competitive athletes is aortic root dilatation, dissection, and rupture.[48]

Evaluation

The evaluation of an athlete with suspected MFS includes family and personal history, physical examination, echocardiography, and genetic screening.[49–51] Clinical diagnosis of MS is based on the Ghent criteria,[52,53] namely the combination of two major criteria plus the involvement of a third organ/system.

Table 5 Recommendations for competitive sport participation in athletes with MS

Phenotype	Genotype	Criteria for eligibility	Recommendations	Follow-up
Adult with full phenotype; adolescent with incomplete phenotype; children/adolescent without phenotype	Positive		No competitive sports	
Athletes (adults) with full phenotype	Not available		No competitive sports	
Athletes (adolescents) with incomplete phenotype	Not available	Positive family history	No competitive sports	
Athletes (adolescents) with incomplete phenotype	Not available	Negative family history	Continued sport participation with follow-up	Yearly
Athletes (children/adolescent) without phenotype	Not available	Positive family history	Continued sport participation with follow-up	Yearly

Particular care should be paid in evaluating tall children and adolescents engaged in certain sports such as basketball and volleyball. The hypermobility of their joints, as well as their stature and osteo-skeletal aptitude are favouring prerequisites. Once they are involved in a successful sports activity, the finding of aortic root dilatation requiring to interrupt athletic activity may raise severe psychological problems.

Recommendations

See *Table 5*.

In examining patients with MFS, the physician (and consultant cardiologist) should be committed to avoid the risk of aortic dissection and to protect the aortic root from accelerated dilation. The following suggestions may help cardiologists in their recommendations:

(i) In children, offspring of patients with MFS, who present a major skeletal phenotype, an integrated educational activity can be proposed to parents, in order to dissuade children from pre-competitive commitments and direct their hobbies or physical training towards non-competitive and moderate intensity activities.

(ii) In youths committed to pre-competitive activity:
  (a) in case of positive family history, uncertain phenotype but positive FBN1 mutation: competetive sports participation should be strongly discouraged, and interests should be re-addressed to non-risky activities;
  (b) in case of positive family history, uncertain phenotype and negative FBN1 mutation: youths may continue their activity but should undergo periodical CV monitoring;
  (c) in case of negative family history ($\sim$30% of MFS is caused by *de novo FBN1* or TGFßR2 mutations), unknown genotype, uncertain phenotype (especially in youths), the decision may prove particularly difficult. If clinical suspicion is strong, even in the absence of completion of the Ghent criteria, the cardiologist should be cautious and discourage the candidate from competitive sports participation.

(iii) In young competitive athletes, when Ghent criteria are reached, the cardiologist should strongly discourage any competitive sports activity.

In patients with MFS, a low-intensity, leisure physical activity could be appropriate for the osteo-skeletal problems. However, these patients should avoid contact sports (*Table 1*), because of the risk of damaging the aorta and injuring the eyes. It is also wise to avoid strenuous physical activities to prevent any increase in aortic wall stress. Patients with MFS in the absence of aortic dilation but with MVP can perform leisure, non-contact sports with moderate intensity, such as running, cycling, swimming, and tennis (see also MVP). Patients with MFS and mechanical heart valves under anticoagulant therapy have an increased risk of haemorrhages and regular monitoring of the anticoagulation is mandatory.

Recommendations for participation in competitive sports in athletes with systemic hypertension

Hypertension is defined as systolic BP $\geq$140 mmHg and/or diastolic BP $\geq$90 mmHg, measured by conventional techniques in the seated subject according to established guidelines.[54–57] Isolated systolic hypertension corresponds to an elevated systolic BP with normal diastolic BP. Subjects with elevated BP in the clinic and normal out-of-office BP have white-coat or isolated clinical hypertension. The current threshold for an elevated 24 h ambulatory BP is 125/80 mmHg; the threshold for daytime ambulatory BP and home BP is 135/85 mmHg.[57]

Risk stratification

The severity of hypertension does not only depend on the BP level, but also on the presence of other CV risk factors, target organ damage, and CV and renal complications, i.e. the overall CV risk.[54,55] The current risk stratification is based on the presence of selected risk factors, target organ damage, and/or of associated clinical conditions, as outlined in *Table 6*. The terms low, moderate, high, and very high added risk, in comparison with healthy normotensive subjects without risk factors, are calibrated to indicate an approximate absolute 10-year risk of CV disease of $<$15,

Table 6 Stratification of risk to quantify prognosis in patients with systemic hypertension

Other risk factors and disease history	Clinic BP (mmHg)		
	Grade 1: SBP 140–159 or DBP 90–99	Grade 2: SBP 160–179 or DBP 100–109	Grade 3: SBP ≥180 or DBP ≥110
No other risk factors[a]	Low added risk	Moderate added risk	High added risk
One or two risk factors[a]	Moderate added risk	Moderate added risk	Very high added risk
Three or more risk factors[a] or TOD[b] or diabetes	High added risk	High added risk	Very high added risk
Associated clinical conditions[c]	Very high added risk	Very high added risk	Very high added risk

TOD, target organ damage; SBP, systolic blood pressure; DBP, diastolic blood pressure. Low, moderate, high, and very high added risk indicate an approximate 10 year risk of fatal and non-fatal CV disease of <15, 15–20; 20–30, and >30%, or of fatal CV disease of <4, 4–5, 6–8, and >8%.

[a]Risk factors used for stratification: BP level (grades 1–3); gender and age (men >55 years; women >65 years); smoking; dyslipidaemia (total cholesterol >250 mg/dL or LDL-cholesterol >155 mg/dL or HDL-cholesterol <40 mg/dL in men and <48 mg/dL in women); abdominal obesity (men ≥102 cm; women ≥88 cm); first-degree family history of premature CV disease (men <55 years; women <65 years).

[b]Target organ damage: hypertension-induced LV hypertrophy; ultrasound evidence of arterial wall thickening or atherosclerotic plaque; slight increase in serum creatinine (men 1.3–1.5 mg/dL, women 1.2–1.4 mg/dL); presence of micro-albuminuria.

[c]Associated clinical conditions: cerebrovascular disease; IHD; heart failure; peripheral vascular disease; renal impairment; proteinuria; advanced retinopathy (haemorrhages, exsudates, papiloedema).

15–20, 20–30, and >30%, respectively, according to the Framingham criteria, or an approximate absolute risk of fatal CV disease of <4, 4–5, 6–8, and >8% according to the European SCORE system.[56]

With regard to LV hypertrophy, it should be noted that sports activity itself may induce hypertrophy; the pattern of hypertrophy and assessment of diastolic LV function may help to distinguish between hypertensive heart disease and athlete's heart.[58,59]

Evaluation

Diagnostic procedures comprise repeated BP measurements according to established guidelines,[54–57] medical history, physical examination, laboratory, and instrumental investigations, of which some should be considered part of the routine approach in all subjects with high BP, and some are recommended in the hypertensive athlete. For instance, echocardiography and exercise testing (with ECG and BP monitoring), which are not always included in the evaluation of the hypertensive patient, are warranted as routine tests in the hypertensive athlete. Additional tests, such as stress echocardiography/myocardial scintigraphy and/or 24 h Holter ECG monitoring, may be recommended depending on the patient's symptoms, CV risk profile, associated clinical conditions, and the results of the first set of investigations.

Recommendations

General recommendations

Athletes with hypertension should be treated according to the general guidelines for the management of hypertension.[54,55] Appropriate non-pharmacological measures should be considered in all patients. Antihypertensive drug therapy should be started promptly in patients at high or very high added risk for CV complications (*Table 6*). In patients at moderate added risk, drug treatment is only initiated when hypertension persists after several months despite appropriate lifestyle changes. Drug treatment is not considered mandatory in patients at low added risk. The goal of antihypertensive therapy is to reduce BP to at least <140/90 mmHg and to lower values if tolerated in all hypertensive patients, and to <130/80 mmHg in diabetics. Current evidence indicates that patients with white-coat hypertension do not have to be treated with antihypertensive drugs, unless they are at high or very high risk (*Table 6*), but a regular follow-up and non-pharmacological measures are recommended.[60]

Choice of drugs

Several drug classes can be considered for first-line antihypertensive therapy: diuretics; beta-blockers; calcium channel blockers; angiotensin converting enzyme (ACE)-inhibitors, and angiotensin II receptor blockers.[54,55] However, in endurance athletes, diuretics and beta-blockers are not recommended because they may impair exercise performance and capacity and/or cause electrolyte and fluid disturbances.[61,62] In addition, they are on the doping list for some sports, in which weight loss or control of tremor are of paramount importance. Calcium channel blockers and blockers of the renin–angiotensin system are the drugs of choice for the hypertensive endurance athlete[63] and may be combined in case of insufficient BP control. However, the combination of an ACE inhibitor and an angiotensin II receptor blocker is currently not advocated. If a third drug is required, a low-dose thiazide-like diuretic, possibly in combination with a potassium sparing agent, is recommended. There is no unequivocal evidence that antihypertensive agents would impair performance in static sports.

Recommendations for sports participation

See *Table 7*.

Recommendations for participation in competitive sports in athletes with hypertension are based on the risk stratification and with the understanding that the general recommendations for the management of hypertension are observed as described earlier, and the clinical condition is stable. In patients with secondary hypertension, clinical and diagnostic evaluation for sports participation is

Table 7 Recommendations for competitive sport participation in athletes with systemic hypertension (and other risk factors) according to the CV risk profile

Lesion	Evaluation	Criteria for eligibility	Recommendations	Follow-up
Low added risk	History, PE, ECG, ET, Echo	Well controlled BP	All sports	Yearly
Moderate added risk	History, PE, ECG, ET, Echo	Well controlled BP and risk factors	All sports, with exclusion of high static, high dynamic sports (IIIC)	Yearly
High added risk	History, PE, ECG, ET, Echo	Well controlled BP and risk factors	All sports, with exclusion of high static sports (III A–C)	Yearly
Very high added risk	History, PE, ECG, ET, Echo	Well controlled BP and risk factors, no associated clinical conditions	Only low-moderate dynamic, low static sports (I A–B)	6 months

ET, exercise testing; Echo, echocardiography. PE, physical examination, including repeated BP measurements according to guidelines.[54–57] Sport type, see Table 1.

postponed to after the removal of the cause of hypertension, if possible. Patients with polycystic kidney disease or with CoA should avoid sports with danger of bodily collision.

Recommendations for participation in competitive sports in athletes with ischaemic heart disease (IHD)

IHD accounts for most exercise-related SCD, especially in individuals >35 years of age.[64] The risk for triggering coronary events increases transiently during vigorous physical activity due to several mechanisms, including sympathetic drive and release of catecholamines, platelet adhesion/activation[65] (with risk of thrombotic complications), electrolyte disturbances such as an increased potassium level (trigger for ventricular tachyarrhythmias), and heart-related complications (such as sub-endocardial ischaemia and necrosis).[66]

In younger athletes (<35 years), congenital CV abnormalities are more frequently implicated. Non-coronary anomalies may also result in acute ischaemic episodes in athletes. Abuse of certain drugs, such as cocaine, as well as risk factors for IHD, such as obesity and diabetes, may trigger myocardial ischaemia and SCD.[67–69] Physical inactivity is also considered a major risk factor for IHD, whereas regular physical training reduces the risk of SCD during vigorous exertion.[70]

The benefits of regular physical activity and sports participation are believed to outweigh the increased risk for coronary events triggered by acute, intensive exercise. However, physical activity and sports participation should be individually tailored in patients with IHD.

Athletes with evidence of IHD

These include athletes with unstable angina, stable angina, after coronary artery bypass grafting/percutaneous coronary intervention (CABG/PCI), after myocardial infarction (MI) or with silent ischaemia.

Unstable angina is here defined as recent onset angina, progressive angina (by frequency, intensity, and duration), angina at rest, or angina developing in close relation to an MI.

Stable angina is defined as angina (chest discomfort or related symptoms) experienced during/after physical exercise, temperature changes, an emotional period, a meal, or hyperdynamic circulation (such as occurs in anaemia, fever, hyperthyroidism). For a given patient, stable angina occurs with repeated progressive exercise at approximately the same rate-pressure product (i.e. ischaemia threshold).

Silent ischaemia is defined by unequivocal evidence of ischaemia on stress testing or Holter monitoring, but without clinically evident symptoms. In these patients, coronary angiography shows evidence of coronary atherosclerosis.

Evaluation

The candidate athlete with IHD should be systematically evaluated under the following criteria:

History: to assess symptoms consistent with stable or unstable angina, presence of risk factors for IHD, as well as the type of sports in which the athlete participates, and family history of IHD/SCD.
Resting ECG and provocative testing: with symptom-limited exercise testing (by treadmill or bicycle) for evaluation of ischaemia-threshold, symptoms, ST–T changes, BP and heart rate response, exercise capacity, and arrhythmias. Exercise testing or pharmacological stress testing with SPECT may show (ir)reversible perfusion defects of the myocardium, and exercise or pharmacological stress testing with echocardiography (or MRI) may show reversible regional wall motion abnormalities.
Echocardiography: to assess global LV function, regional wall motion abnormalities, and/or associated structural cardiac anomalies.
Coronary angiography: is mandatory in individuals with IHD willing to participate in competitive sports. Luminal coronary stenosis/occlusion, coronary flow disturbances or abnormal coronary anatomy should be evaluated.
24 h Holter monitoring (including a training session): to assess arrhythmias or silent ischaemic changes.

Risk stratification

On the basis of the results of diagnostic testing the risk may be stratified[71] as follows.

Table 8 Recommendations for competitive sport participation in athletes with IHD

Lesion	Evaluation	Criteria for eligibility	Recommendations	Follow-up
Athletes with definite diagnosis of IHD and high probability of cardiac events	History, ECG, ET, Echo, coronary-angiography		No competitive sports allowed	
Athletes with definite diagnosis of IHD and low probability of cardiac events	History, ECG, ET, Echo, coronary-angiography	No exercise induced ischaemia, no symptoms or major arrhythmias, not significant (<50%) coronary lesions, EF >50%	Only low-moderate dynamic and low static sports (I A,B)	Yearly
Athletes without evidence of IHD but with high risk profile (>5% global SCORE)	History, ECG, ET	If positive provocative ECGs, further testing are needed (stress echo, scintigraphy, and/or coronary angiography) to confirm IHD. If positive, consider as athletes with diagnosis of IHD	Only low-moderate dynamic and low static sports (I A,B)	Yearly
		If negative provocative ECGs	Individual based decision; avoid high static sports (IIIA–C)	Yearly
Athletes without evidence of IHD and low risk profile	History, ECG, ET optional	Negative ECG	All competitive sports	Every 1–3 years

ECG, 12-lead electrocardiogram; ET, exercise testing or other provocative testing; sport type, see *Table 1*.

- Low probability for exercise-induced adverse cardiac events if all the following criteria are present:
  - ejection fraction >50% on echocardiography or on SPECT;
  - normal exercise capacity according to age and gender on exercise testing;
  - absence of exercise-induced ischaemia on ECG/stress testing at lower steps;
  - absence of frequent, complex ventricular tachyarrhythmias at rest and during stress testing;
  - absence of significant coronary stenosis (i.e. >70% of major coronary arteries, or >50% of left main stem) on coronary angiography.
- High probability for exercise-induced adverse cardiac events if one or more of the following criteria are present:
  - ejection fraction <50% on echocardiography or on SPECT, or
  - exercise-induced ischaemia (>1 mm ST depression in two leads) on exercise testing at lower steps, or
  - exercise-induced pathological dyspnoea (angina equivalent), or syncope, or
  - frequent, complex ventricular tachyarrhythmias at rest and/or during stress testing, or
  - significant coronary stenosis of major coronary arteries (i.e. >70%) or left main stem (>50%) on coronary angiography.

Specific comments

- Athletes with clinical unstable angina have a high risk for future CV events.
- Post-CABG/PCI athletes, who do not show evidence of myocardial ischaemia on stress testing, are allowed to resume physical activity under supervision of a sports physician after completion of an out-patient cardiac rehabilitation programme. Before entering sports activity, however, they need to be risk-stratified as specified earlier.
- The incidence of SCD in symptomatic or asymptomatic post-MI individuals is equal. Coronary angiography in SCD survivors and athletes after MI must be performed before they resume or initiate sports activity. In general, these athletes should be risk-stratified as specified earlier.
- Silent ischaemia increases the risk for cardiac arrest during physical stress similarly to symptomatic IHD. After establishing presence of true ischaemia, the patient should be risk-stratified as outlined earlier.

Recommendations
See *Table 8*.

Athletes without evidence of IHD, but with one or more risk factors for IHD

In asymptomatic subjects without evidence of IHD, but in the presence of known risk factors, assessment of the risk profile is needed. The risk for IHD can be estimated from the presence of major risk factors including age, sex, BP, smoking, and total cholesterol level, according to the SCORE-system[72] or as outlined in *Table 6*.

The high-risk profile for developing a fatal CV event is defined by:

- the presence of multiple risk factors, resulting in a 10-year risk for a fatal CV event of >5% at present, or when extrapolated to age 60, on the SCORE-chart; or
- markedly raised levels of total cholesterol (>8 mmol/L, or 320 mg/dL), LDL-cholesterol (>6 mmol/L, or 240 mg/dL) or BP >180/110 mmHg; or
- diabetes mellitus type 1 or type 2 with microalbuminuria;

- individuals with a family history of premature CV disease in more than one first-degree relative are added to this group for the purpose of our recommendations.

The low-risk profile is defined as a 10-year risk for a fatal CV event $<5\%$, according to the SCORE-chart, due to the absence of major risks factors.

Evaluation
Athletes with a high-risk profile should be further evaluated to rule out silent ischaemia, by history, physical examination and ECG with maximal exercise testing. In case of: negative exercise testing, the absolute risk of a major cardiac event during physical activity is considered to be low in these asymptomatic patients without evidence of IHD. For positive exercise testing, the risk for future coronary events in these individuals, although asymptomatic, is increased. They need further evaluation by stress-echocardiography/myocardial scintigraphy and/or coronary angiography to assess the presence of (even silent) IHD. Finally, athletes with evidence of ischaemia should be stratified as athletes with IHD.

Exercise testing is not routinely recommended in healthy asymptomatic athletes without classical risk factors and age <35 years for men and <45 years for women.

Recommendations
See *Table 8*.

Recommendations for participation in competitive sports in athletes with arrhythmias and potentially arrhythmogenic conditions

General considerations

Cardiac arrhythmias may occur not only in association with several CV abnormalities, such as genetic ionic channel diseases, anomalies of the conducting system, or structural heart diseases, but also without evidence of a morphologic substrate. The main prognostic determinant to be assessed in athletes with arrhythmias is the presence of heart disease.[48,73–75]

The evaluation of athletes with arrhythmias, either documented or suspected, includes personal history, searching for any hints of substance abuse such as smoking, alcohol, drugs, or doping. Risk factors for IHD should be investigated particularly in adult/senior athletes. The history should explore previous CV disease and identify symptoms such as palpitations, pre-syncope or syncope, unexplained weakness, chest pain, or dyspnoea. A thorough family history should search for SCD (especially in youth and adulthood) and/or potentially arrhythmogenic conditions. The initial evaluation also includes physical examination and ECG, exercise testing, 24 h Holter monitoring, and echocardiography. In some cases, it is advisable to obtain a blood count, thyroid function markers, and electrolyte balance. When initial testing fails to demonstrate the arrhythmia, the external event recorder or loop recorder can be eventually considered; finally, an electrophysiologic study is indicated when the arrhythmia is paroxysmal and/or associated with haemodynamic impairment.

Sinus bradycardia

Asymptomatic sinus bradycardia, sinus bradyarrhythmia, wandering pacemaker (PM), and sinus pauses are common in young athletes.[76] A number of studies suggest that these arrhythmias in athletes are the consequence of increased vagal tone and withdrawal of sympathetic tone. Occasionally, marked sinus bradycardia (i.e. $\leq$40 b.p.m.) at rest, or sinus pauses $\geq$3 s can be found in asymptomatic, well-trained endurance athletes. These arrhythmias are usually benign, and evaluation may be limited to history, physical examination, and ECG. Treatment is usually not necessary.[4]

If marked bradycardia is associated with symptoms, such as lightheadedness, pre-syncope/syncope (see also section of syncope), or exertional fatigue, 24 h Holter monitoring and exercise testing are recommended. If structural heart disease is suspected, echocardiography (or another imaging technique) is mandatory. In selected cases, a deconditioning period of 1–2 months could be useful to clarify the clinical significance of extreme bradyarrhythmias.

Recommendations
See *Table 9*.

Atrioventricular blocks

In athletes, the prevalence of first-degree atrioventricular (AV) block or second-degree AV block of the Wenckebach type (Mobitz type I) is high.[77,78] The AV block typically occurs during sleep or at rest. In asymptomatic athletes without structural heart disease (as assessed by echocardiography) and with resolution of the AV block during exercise (as assessed by 24 h Holter monitoring and/or exercise testing), no further investigations and no therapy are indicated.

Rarely, Mobitz type II or third-degree AV block may be observed in athletes, which require a more comprehensive clinical and diagnostic evaluation. If these findings are associated with symptoms or with structural heart disease, PM implantation is recommended.

Recommendations
See *Table 9*.

Supraventricular premature beats and tachycardia

Supraventricular arrhythmias may be associated with symptoms, such as palpitations, fatigue, chest discomfort or may present with dyspnoea, lightheadedness, or syncope. The patient should be encouraged to have an ECG taken during the arrhythmia, which may clarify the diagnosis. The 24 h Holter monitoring is indicated in these instances. An electrophysiologic study is indicated in patients with paroxysmal palpitations if catheter ablation may represent a therapeutic option for resolution of the arrhythmia.[79]

Supraventricular premature beats
Premature atrial beats are a common finding in many individuals including athletes.[76,78] A careful history, physical examination, and ECG should be performed. In the absence of structural heart disease and thyroid dysfunction, with no or only mild symptoms (such as occasional palpitations), no further evaluation or therapy is required.

Recommendation. See *Table 9*.

Table 9 Recommendations for competitive sport participation in athletes with arrhythmias and arrhythmogenic conditions

Lesion	Evaluation	Criteria for eligibility	Recommendations	Follow-up
Marked sinus bradycardia (<40 b.p.m.) and/or sinus pauses ≥3 s with symptoms	History, ECG, ET, 24 h Holter, Echo	a) If symptoms[a] are present b) After >3 months from resolution of symptoms[a]; off therapy	a) Temporary interruption of sport b) All sports	 Yearly
a) AV block first and second degree, type 1	History, ECG, ET, 24 h Holter, Echo	a) If no symptoms[a], no cardiac disease, with resolution during exercise	a) All sports	Yearly
b) AV block second degree, type 2 or advanced		b) In the absence of symptoms, cardiac disease, ventricular arrhythmias during exercise, and if resting heart rate is >40 b.p.m.	b) Low-moderate dynamic, low-moderate static sports (I A,B + II A, B)	
Supraventricular premature beats	History, ECG, thyroid function	No symptoms[a], no cardiac disease	All sports	Not required
Paroxysmal supraventricular tachycardia (AVNRT or AVRT over a concealed accessory pathway)	History, ECG, Echo, EP study	Ablation is recommended: a) After catheter ablation: if no recurrences for >3 months, and no cardiac disease b) If ablation is not performed and AVNRT is sporadic, without cardiac disease, without hemodynamic consequences and without relation with exercise	a) All sports b) All sports, except those with increased risk[b]	Yearly
Ventricular pre-excitation (WPW syndrome) and: a) Paroxysmal AV reentry tachycardia b) AF or flutter c) Asymptomatic pre-excitation pattern	a, b,c) History, ECG, Echo, EP study	a, b) Ablation is mandatory After catheter ablation: if no recurrences, no cardiac disease c) Ablation is recommended but not mandatory.	a, b) All sports c) Asymptomatic athletes at low risk and not ablated: all sports, except those with increased risk[b]	Yearly
AF (paroxysmal, permanent)	History, ECG, Echo, ET, 24 h Holter	a) After paroxysmal AF: if no cardiac disease, no WPW, and stable sinus rhythm >3 months b) Permanent A F in the absence of cardiac disease, and WPW: assess heart rate and LV function response to exercise	a) All sports b) Assessed on individual basis	a) Yearly b) Every 6 months
Atrial flutter	History, ECG, Echo, EP study	Ablation is mandatory; after ablation: if no symptoms[a] for >3 months, no cardiac disease or WPW, and off therapy;	All sports	Yearly

Continued

Table 9 *Continued*

Lesion	Evaluation	Criteria for eligibility	Recommendations	Follow-up
PVBs	History, ECG, Echo (ET, 24 h Holter, in selected cases invasive tests)	In the absence of: cardiac disease or arrhythmogenic condition[c], family history of SD, symptoms[a], relation with exercise, frequent and/or polymorphic PVBs and/or frequent couplets with short RR interval	All sports	Yearly
Nonsustained ventricular tachycardia	History, ECG, Echo (ET, 24 h Holter, in selected cases invasive tests)	In the absence of: cardiac disease or arrhythmogenic[c] condition, symptoms[a], family history of SD, relation with exercise, multiple episodes of NSVT with short RR interval	All sports	Every 6 months
Slow ventricular tachycardia, fascicular ventricular tachycardia, RV outflow tachycardia	History, ECG, Echo, ET, 24 h Holter (in selected cases EP study)	In the absence of: cardiac disease or arrhythmogenic[c] condition, family history of SD, symptoms[a]	All sports, except those with increased risk[b]	Every 6 months
Syncope	History, ECG, Echo, ET, 24 h Holter; tilting test	a) neurocardiogenic b) arrhythmic or primary cardiac	a) All sports (except those with increased risk[b]) b) see specific cause	Yearly
Long QT syndrome	History, ECG, (24 h Holter, genetic testing)	Positive long QT syndrome	No competitive sports	
Brugada syndrome	History, ECG, provocative test	Positive Brugada syndrome	No competitive sports	
Implanted PM	ECG, Echo, ET, 24 h Holter	Normal heart rate increase during exercise, no significant arrhythmias, normal cardiac function	Low-moderate dynamic and low static sports (I A,B), except those with risk of bodily collision	Yearly
Implantable cardioverter defibrillator	ECG, Echo, ET, 24 h Holter	No malignant VTs, normal cardiac function, at least 6 months after the implantion, or the last ICD intervention	Low-moderate dynamic and low static sports (I A,B), except those with risk of bodily collision	Yearly

For athletes with structural heart disease, see the recommendations of the disease.
ECG, 12-lead electrocardiogram; Echo, echocardiography; ET, exercise testing; 24 h Holter, 24 h Holter monitoring; EP, electrophysiologic; Sport types, see *Table 1*.
[a]Symptoms include pre-syncope, lightheadedness, exertional fatigue
[b]Increased risk if syncope occurs (see Classification of Sports).
[c]Arrhythmogenic conditions include cardiomyopathies, IHD and channelopathies.

Paroxysmal supraventricular tachycardia

Paroxysmal supraventricular tachycardia may be caused by AV nodal re-entrant tachycardia (AVNRT), orthodromic AV re-entrant tachycardia (AVRT) due to an accessory pathway, or an ectopic atrial tachycardia. AVNRT is the most common form of supraventricular reciprocating tachycardia in the general population, including athletes.[80] Although typically occurring in the third and fourth decade of life with a preference for women, this paroxysmal arrhythmia may be observed at any age. The arrhythmia is caused by a re-entry mechanism involving the AV node, perinodal atrium, and slow and fast pathways (which connect the AV node to the atrium). Adequate evaluation of the athlete with AVNRT includes a history with emphasis on the characteristics of the arrhythmia onset, and symptoms during tachycardia. An electrophysiologic study aimed to define the re-entry mechanism is recommended.

The catheter ablation has become the preferred therapy, especially in athletes, because antiarrhythmic drug therapy is a lifelong treatment with limited efficacy. Ablation of the slow AV nodal pathway is the elective procedure, which can be performed with success rates of $>95\%$, and a complication rate (permanent AV block) of <1–2% in experienced centres. In selected cases with an increased risk for complete AV nodal block (i.e. para-His accessory pathway), cryo-ablation can be an alternative approach.

AVRT over a concealed accessory pathway is based on a typical electrophysiologic mechanism, i.e. the accessory pathway is able to conduct only in the retrograde way (concealed pathway). Therefore, there is no evidence of the accessory pathway on the standard ECG during sinus rhythm. Also, for the AVRT over the concealed pathway, catheter ablation is the elective therapy.

Recommendations. See *Table 9*.

Ventricular pre-excitation (Wolff—Parkinson—White Syndrome)

Wolff–Parkinson–White syndrome (WPW) is defined as the presence of paroxysmal arrhythmias in a patient with overt ventricular pre-excitation. Prevalence of pre-excitation in the general population varies from 0.1 to 0.3% and it is not different in the athletic population.[81,82] The tachyarrhythmias related to WPW syndrome include AV re-entry tachycardia (either orthodromic or antidromic), AF and, rarely, ventricular fibrillation.

Evaluation of the athlete with ventricular pre-excitation includes history, physical examination, ECG, and echocardiography (to exclude an associated structural cardiac disease, such as HCM or Ebstein anomaly).

WPW and paroxysmal AV re-entry tachycardia

The most common form of paroxysmal supraventricular tachycardia in the WPW syndrome is the orthodromic tachycardia with a narrow QRS complex. This AVRT involves the node-Hissian pathway anterogradely and the accessory pathway retrogradely. Occasionally, an orthodromic tachycardia may have anterograde conduction over the bypass tract and a wide QRS tachycardia is documented.

Athletes with WPW syndrome and documented symptomatic paroxysmal AV re-entry tachycardia should be preferentially treated with radiofrequency ablation of the accessory pathway.

WPW and AF or flutter

It has been estimated that one third of patients with WPW syndrome may develop AF. AF or atrial flutter in the presence of ventricular pre-excitation may result in a rapid activation of the ventricles through the accessory pathway which may degenerate into ventricular fibrillation and SCD. The risk of SCD in WPW patients/athletes varies in population-based studies from 0.15 to 0.2%, whereas in symptomatic WPW patients, the risk appears to be higher (2.2%).[80] The incidence of SCD is higher in patients/athletes with an accessory pathway characterized by short refractoriness and is triggered by an episode of AF/flutter. Therefore, symptomatic patients/athletes with ventricular pre-excitation and AF or flutter should undergo catheter ablation.

Asymptomatic pre-excitation on ECG

It is generally considered that asymptomatic athletes with pre-excitation on 12-lead ECG, in the absence of structural heart disease, have a low but definite risk of SCD. Assessment of the risk profile in these athletes is based on an electrophysiologic study and includes measurement of refractoriness of the accessory pathway and induction of AF to assess the shortest pre-excited RR interval. A short pre-excited RR interval (currently used criteria are <240 ms during induced AF and <220 ms during effort or isoproterenol infusion), the presence of multiple accessory pathways, or even easy induction of AF[83] are considered to be associated with an increased risk of SCD.

At present, given the high success rate and low incidence of complications, catheter ablation of the accessory pathway has become the first choice treatment for (even asymptomatic) athletes with pre-excitation. Therefore, these athletes should be fully advised of this preferential therapeutic option and decision should be made on an individual basis.

For those athletes who refuse the ablation, or when the procedure is associated with a high risk, competitive sports activities may nevertheless be allowed if the electrophysiologic study shows absence of risk criteria (as specified earlier) and, specifically, a short refractoriness at baseline and during effort, or isoproterenol infusion. In all other instances, catheter ablation should be performed.

To be noticed, in children <12 years of age, the risk of AF and/or SCD is considered virtual, and assessment of the risk may be postponed.

Recommendations. See *Table 9*.

Atrial fibrillation

The prevalence of AF in competitive athletes is not well known, although is supposed to be higher than in the general population. In $\sim$40% of athletes with AF, a possible substrate, such as WPW syndrome, cardiomyopathy, or silent myocarditis can be found.[82,84] Use of doping substances, such as anabolic steroids, can also possibly cause AF in athletes.[85]

Pulmonary vein catheter ablation is not yet established as a routine procedure in focal AF, due to its limited long-term

success rate (50–80%) and considerable complications (pulmonary vein stenosis, tamponade, peri-procedural stroke in 3–10%). In athletes with unsuccessful rhythm control or in athletes under rate-control therapy, anticoagulation may be necessary, depending also on the presence of risk factor for thrombo-embolic events.

Recommendations
See *Table 9*.

For athletes with AF and ventricular pre-excitation: see ventricular pre-excitation. Anticoagulant therapy excludes individuals from sports with a risk of bodily collision or trauma (see *Table 1*).

Atrial flutter

Atrial flutter is uncommon in the young healthy population. The electrophysiologic substrate for atrial flutter of the common type is a counter-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, because it is often the basis of this arrhythmia. Atrial flutter may convey an increased thrombo-embolic risk and may be life-threatening due to potential 1:1 conduction to the ventricles.

Catheter ablation of the isthmus is a highly effective and safe procedure[86] and is recommended as first-line therapy in athletes. Anticoagulation therapy in atrial flutter follows the same recommendations as in AF. In the presence of combined atrial flutter and fibrillation, isthmus ablation is recommended, followed by drug therapy for AF ('hybrid therapy').

Recommendations
See *Table 9*.

Athletes with structural heart disease and atrial flutter can participate in competitive sports consistent with the limitation of the disease, only after successful catheter ablation and in the absence of recurrence of arrhythmia for >3 months. For athletes with atrial flutter and ventricular pre-excitation: see ventricular pre-excitation. Athletes who require anticoagulation therapy should not participate in sports with danger of bodily collision or trauma (*Table 1*).

Premature ventricular beats

Premature ventricular beats (PVBs) are a frequent finding in the athletic population.[76,78] The main factor for determining prognosis and recommendation for sports participation is the presence of heart disease.[87] Unfortunately, large randomized clinical trials in athletes with PVBs are uncommon. However, available studies suggest that PVBs in the absence of CV abnormalities are not associated with an increased risk of malignant ventricular arrhythmias and convey a good outcome.[88] However, PVBs may also be the initial and unique manifestation of clinically silent arrhythmogenic conditions at risk of SCD (such as ARVC, HCM, myocarditis); therefore, athletes with PVBs require careful evaluation including history, physical examination, echocardiography, exercise testing, and 24 h Holter monitoring. The family history is relevant for excluding the presence of juvenile SCD or familial arrhythmogenic cardiac conditions, as well as the presence of symptoms, such as palpitations or syncope, particularly during exertion. In athletes with suspected heart disease, further testing (such as MRI, coronaro-angiography, endomyocardial biopsy) may be required in the individual case to rule out underlying disease and establish appropriate management. In selected cases, re-evaluation after 3–6 months of deconditioning may be useful.[89]

Recommendations
See *Table 9*.

Non-sustained ventricular tachycardia

Non-sustained ventricular tachycardia (NSVT), defined as ventricular tachycardia of three or more consecutive beats $\geq$100 b.p.m. and lasting $<$30 s, is an uncommon arrhythmia in healthy subjects. NSVT requires extensive clinical assessment, including echocardiography (or other imaging tests), exercise testing, and 24 h Holter monitoring to rule out underlying disease and to evaluate the mechanism of the arrhythmia. Further tests, such as an electrophysiologic study and other invasive testing, should be individually based according to the suspected cardiac lesion. Particular attention should be paid to identify those athletes with polymorphic/bi-directional NSVT which is triggered by exercise or occurs during exercise testing ('catecholaminergic ventricular tachycardia') which carries a high risk of SCD.

Recommendations
See *Table 9*.

Slow ventricular tachycardia (idio-ventricular accelerated rhythm)

This is a focal automatic ventricular rhythm with a heart rate $<$100 b.p.m., due to enhanced ventricular automation, which is favoured by bradycardia. Echocardiography, exercise testing, and 24 h Holter monitoring are indicated for clinical evaluation.

Recommendations
See *Table 9*.

Benign idiopathic ventricular tachycardia: fascicular ventricular tachycardia and RV outflow tachycardia

Fascicular ventricular tachycardia[90] and automatic RV outflow tachycardia, also known as 'RV outflow tract' (RVOT) ventricular tachycardia[91] are distinct entities which are not usually associated with heart disease, are haemodynamically well tolerated, and have a benign prognosis. Both VTs are usually induced by physical exercise. Fascicular VT originates from the distal ramifications of the left posterior bundle in the inferior part of the interventricular septum, is characteristically paroxysmal and presents with right bundle branch block QRS morphology and superior axis. RVOT originates from an automatic focus in the RVOT, may be either paroxysmal or repetitive (so called 'repetitive monomorphic VT') and presents with left bundle branch block QRS morphology and inferior axis. Clinical assessment including echocardiography, exercise testing, and 24 h Holter monitoring is recommended to exclude the presence of heart disease and to analyse the VT characteristics, because overlapping forms between idiopathic RVOT–VT and ARVD have

been described. An electrophysiologic study may be necessary for the differential diagnosis from VT due to ARVC (see Cardiomyopathies) and supraventricular tachycardia with aberrant conduction.

Catheter ablation of the substrate is a reasonable therapeutic option for the two VTs, with high rate of success and few complications, and the athlete should be fully advised on this therapeutic option. When catheter ablation is refused or not possible, the risk must be stratified according to the presence of heart disease, the RR interval of the VT and the presence of symptoms, such as dizziness, presyncope and syncope.

Recommendations
See *Table 9*.

Malignant ventricular tachycardia

Malignant VTs include sustained ventricular tachycardia, polymorphic ventricular tachycardia, *torsades de pointes*, and ventricular fibrillation. These VTs are associated with haemodynamic deterioration and may lead to cardiac arrest. IHD is the most common cause of malignant VT in adult individuals; whereas in young people, a spectrum of pathologic conditions, including HCM, ARVC, and congenital coronary anomalies, have been reported. Affected individuals need a thorough clinical assessment and therapeutic options for SCD prevention, such as implantable cardioverter defibrillator (ICD). In athletes with documented malignant VT, competitive sports are contraindicated. A possible exception is ventricular arrhythmias occurring in the context of acute and transient myocardial lesions, such as myocarditis, commotio cordis, and acute electrolytic depletion, when the cause has proven to be completely resolved.

Symptoms of possible arrhythmic origin: syncope

Syncope is characterized by sudden and momentary loss of consciousness and postural tone, due to abrupt reduction of global brain blood flow, with spontaneous and complete recovery within a short time.[92] Syncope may be neurocardiogenic (vasovagal, sino-carotid, environmental, or situational), orthostatic, or of cardiac origin (either arrhythmic or structural), or due to primary brain blood flow dysfunction. In young athletes, syncope is most often neurocardiogenic and seems to have a fair prognosis.[92,93]

Evaluation of athletes with syncope should differentiate true syncope from other conditions causing loss of consciousness (epilepsy, transitory ischaemic attack, drop attack, hypoglycaemia), and assess the presence of cardiac disease. Initial evaluation includes history (aimed at a clear description of the syncope), physical examination with BP measurement in the recumbent and standing positions, and ECG. The 24 h Holter monitoring is necessary in case of suspicion of an arrhythmic cause of the syncope; exercise testing is indicated if syncope is related to exercise, as well as in subjects with suspected IHD.[92] The head-up tilt test is needed to confirm the neurocardiogenic origin of the syncope (although this test has lower specificity in athletes compared with the general population[93]). An electrophysiologic study is indicated when syncope is associated with palpitations as the likely expression of paroxysmal tachycardia.

Recommendations
See *Table 9*.

In athletes with neurocardiogenic syncope, the restriction from sports with intrinsic risk (*Table 1*) is dictated by the awareness that any transient loss of consciousness will convey adverse effects for the athlete and the people nearby. In athletes with syncope of arrhythmic or mechanic origin, recommendation will be granted depending on the type of arrhythmia and/or on the associated abnormal CV condition.

Arrhythmogenic disorders: ion channel disease

Sudden arrhythmic death in the athlete may be caused by inherited cardiac ion channel defects (channelopathies) which include the long QT syndrome, Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia.

Long QT syndrome

Long QT interval is defined as heart rate corrected QT interval (according to the Bazett's formula) measured in lead II exceeding 440 ms in males and 460 ms in females.[94] Congenital long QT syndrome has been associated with genetically defective cardiac K^+ and Na^+ channels which result in prolongation of ventricular repolarization and predispose to *torsade de pointes* and ventricular fibrillation. To exclude potential causes of acquired long QT interval, therefore, electrolyte depletion (i.e. hypokalaemia) or chronic intake of drugs capable to prolong repolarization (such as certain antibiotics, antihistaminics, etc.) should be first investigated. Athletes with borderline QT lengthening should be evaluated with exercise testing and 24 h Holter monitoring. Genetic testing is mandatory when definitive diagnosis for genotype-related risk stratification and therapy is required. Congenital long QT syndrome is a contraindication for any type of sports, even without documented major arrhythmic events.

Brugada syndrome

The Brugada syndrome is a genetic condition characterized by a peculiar ECG pattern in the anterior precordial leads V1–V3, i.e. ST-segment elevation >2 mm, with 'coved type' (either spontaneous or induced by pharmacological sodium channel blockade) in association with related arrhythmic events (syncope, cardiac arrest).[95] A cardiac Na^+ channel gene (*SCN5A*) mutation has been detected in up to 30% of cases. This syndrome is associated with risk of SCD due to malignant ventricular arrhythmias (sustained VT, VF), which usually occur at rest and often at night, as a consequence of increased vagal stimulation and/or withdrawal of sympathetic activity. Increased vagal tone as a consequence of chronic athletic conditioning may eventually enhance the propensity of athletes with Brugada syndrome to die at rest, during sleep, or during the recovery after exercise. Therefore, although no relation between exercise and arrhythmias has been found, subjects with definite diagnosis of Brugada syndrome should be restricted from competitive sports. Whether healthy genetic carriers of the Brugada syndrome without phenotypic expression should be restricted from sports participation is at present uncertain.

Catecholaminergic ventricular tachycardia
Catecholaminergic ventricular tachycardia is characterized by exercise-induced polymorphic ventricular tachycardia (most often with 'bi-directional pattern') which can degenerate in ventricular fibrillation. The disease has been linked to mutations of the ryanodine receptor and calcequestrin genes leading to abnormal calcium release from the sarcoplasmic reticulum. Unlike long QT syndrome and Brugada syndrome, this condition is not associated with abnormalities of the basal ECG and remains unrecognized unless the athlete undergoes exercise testing.

Catheter ablation in athletes

Catheter ablation of focal and re-entry tachyarrhythmias has become a therapeutic strategy with high success rate (>95%) and minimum risk of side effects (<1%).[96,97] Lethal complications are uncommon (<1%) and usually occur when ablation is required in the left heart. The occurrence of AV blocks is rare (<1%), and limited to ablation of AV nodal re-entry tachycardia and anteroseptal accessory pathways.

In athletes, indications for catheter ablation differ from those in the general population, because the procedure is not only aimed to eliminate disabling symptoms, but also to allow resumption of competitive sports activity. When cardiac abnormalities which are *per se* responsible for non-eligibility are excluded, catheter ablation is recommended in athletes with the following conditions:

(i) WPW syndrome, either symptomatic or asymptomatic, with electrophysiological evidence of short anterograde refractoriness of the AV accessory pathway;
(ii) supraventricular re-entry tachycardia, either paroxysmal (frequent and sustained episodes with heart rate faster than maximum heart rate by age) or incessant and iterative (with the exception of episodes with slow heart rate);
(iii) typical atrial flutter, either common or non-common;
(iv) symptomatic fascicular ventricular tachycardia or RV outflow ventricular tachycardia.

Three months after successful catheter ablation, patients can resume competitive sports, provided that the ECG shows no signs of ventricular pre-excitation in case of WPW and that they are asymptomatic and without recurrence of tachycardia. Repeated electrophysiologic assessment may be required in selected cases when the outcome of the procedure is considered uncertain.

Patients with PM

Patients with heart disease and PM implantation can participate only in sports consistent with the limitations of the arrhythmia and the underlying heart disease. Athletes with a PM and no signs of heart disease will be allowed to participate in competitive sports with only minor CV demand, provided that exercise testing and 24 h Holter monitoring show an appropriate increase in the paced heart rate during exercise, and no occurrence of significant arrhythmias. However, subjects with PM should be restricted from sports with a risk of body impact, because of the possible disruption of the electro-catheters and damage to the pacing unit. Furthermore, the possible risk of electromagnetic interferences should be closely evaluated.

Recommendations
See *Table 9*.

Patients with ICD

Most patients with ICD have a cardiac disease which represents *per se* a contraindication for competitive sports. Indeed, the efficacy of the ICD to interrupt malignant ventricular arrhythmias during exercise remains to be established. Because ICD patients may benefit from low-intensity and supervised exercise programmes,[98] it seems wise that individuals with ICD and no evidence of structural heart disease (or with mild morphologic abnormalities) and preserved cardiac function can be allowed to participate in sports with only low dynamic or static demand, which do not pose a risk of trauma to the device, and do not specifically trigger malignant VTs (such as *torsade de pointes* in congenital long QT syndrome and polymorphic catecholaminergic ventricular tachycardia).

Sports participation can be allowed at least 6 months after the ICD implantation, or after the most recent arrhythmic episode requiring defibrillator intervention (including pacing, antitachycardia pacing, or shock). Furthermore, to reduce the risk of inappropriate shocks related to sinus tachycardia induced by exercise, the cut-off heart rate for the ICD needs to be appropriately set by exercise testing and 24 h Holter monitoring.

Recommendations
See *Table 9*.

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