Minerva Medica 2024 October;115(5):533-64
DOI: 10.23736/S0026-4806.24.09519-3

GUIDELINES

Italian Cardiological Guidelines (COCIS) for Competitive Sport Eligibility in athletes with heart disease: update 2024

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ABSTRACT

Nearly 35 years after its initial publication in 1989, the Italian Society of Sports Cardiology and the Italian Federation of Sports Medicine (FMSI), in collaboration with other leading Italian Cardiological Scientific Associations (ANCE – National Association of Outpatient Cardiology, ANMCO – National Association of Inpatient Cardiology, SIC – Italian Society of Cardiology), proudly present the 2023 version of the Cardiological Guidelines for Competitive Sports Eligibility. This publication is an update of the previous guidelines, offering a comprehensive and detailed guide for the participation of athletes with heart disease in sports. This edition incorporates the latest advances in cardiology and sports medicine, providing current information and recommendations. It addresses various topics, including the details of the pre-participation screening in Italy and recommendations for sports eligibility and disqualification in competitive athletes with various heart conditions. This revised version of the Cardiological Guidelines for Competitive Sports Eligibility, recorded in the Italian Guidelines Registry of the Italian Minister of Health, stands as a crucial resource for sports medicine professionals, cardiologists, and healthcare providers, marked by its completeness, reliability, and scientific thoroughness. It is an indispensable tool for those involved in the care, management and eligibility process of competitive athletes with heart conditions.

(*Cite this article as:* Zeppilli P, Biffi A, Cammarano M, Castelletti S, Cavarretta E, Cecchi F, *et al.* Italian Cardiological Guidelines (COCIS) for Competitive Sport Eligibility in athletes with heart disease: update 2024. Minerva Med 2024;115:533-64. DOI: 10.23736/S0026-4806.24.09519-3)

KEY WORDS: Sports; Cardiology; Guidelines as topic; Death, sudden; Athletes.

Nearly 35 years after its initial publication in 1989, the Italian Society of Sports Cardiology and the Italian Federation of Sports Medicine (FMSI), in collaboration with other leading Italian Cardiological Scientific Associations (ANCE – National Association of Outpatient Cardiology, ANMCO – National Association of Inpatient Cardiology, SIC – Italian Society of Cardiology), proudly present the 2023 version of the Cardiological Guidelines for Competitive Sports Eligibility. This update supersedes the previous guidelines published in 2020, offering a comprehensive and detailed guide for the participation of athletes with heart disease in sports.[1-3] This edition incorporates the latest advances in cardiology and sports medicine, providing current information and recommendations. It addresses various topics, including contraindications and recommendations for athletes with various heart conditions and screening strategies.

For the first time, these guidelines have been recorded in the Italian Guidelines Registry of the Italian Minister of Health, underscoring the guidelines' quality and dependability. A notable feature of this update is the inclusion of the class of recommendation and level of scientific evidence, facilitating the guidelines' interpretation and application in clinical practice. Overall, this revised version of the Cardiological Guidelines for Competitive Sports Eligibility stands as a crucial resource for sports medicine professionals, cardiologists, and healthcare providers, marked by its completeness, reliability, and scientific thoroughness. It is an indispensable tool for those involved in the care, management and eligibility process of competitive athletes with heart conditions.

Class of recommendation

The indications for sports participation are delineated into classes of recommendation as follows:

• Class I: There is evidence and/or general agreement that a given competitive sport is safe, beneficial, useful, and effective. Indication: The athlete can participate in competitive sports.

• Class II: There is conflicting evidence and/or a divergence of opinion about the usefulness/effectiveness of the given competitive sport. Indication: The athlete may participate in competitive sports after an individualized evaluation.

• Class III: There is evidence or general agreement that the given competitive sport is potentially harmful. Indication: The athlete cannot participate in competitive sports.

Levels of evidence

• Level A: Data derived from multiple randomized clinical trials or meta-analyses.

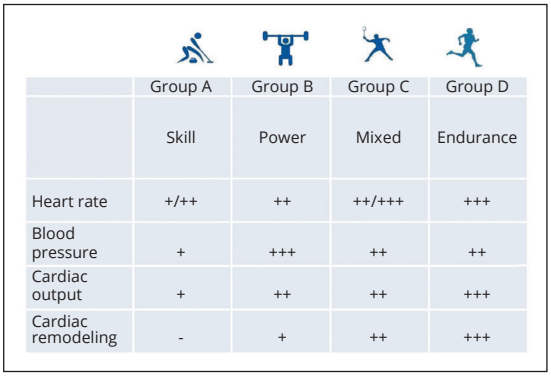
• Level B: Data derived from a single randomized clinical trial or large non-randomized studies.

• Level C: Consensus of the experts and/or small studies, retrospective studies, and registries.

Classification of sports

The classification of sports based on cardiovascular (CV) involvement is crucial for establishing eligibility criteria, particularly in individuals with heart disease or cardiac abnormalities. This classification is grounded in the acute physiological responses of the heart and blood vessels to exercise, the specific adjustments necessitated by each sport and discipline, and the long-term adaptations resulting from regular exercise or training.[4] This framework aids sports physicians in assessing the CV risks associated with participation in various sports disciplines. Systematic training leads to the remodeling of the CV system, termed adaptation.[5] The significant differences in stimuli over time, regarding type, duration, and intensity of training and competition, contribute to the diverse adaptations of the CV system. The acute responses to physical exercise are characterized by changes in heart rate (HR), blood pressure (BP), and peripheral resistance, as well as by the nature of the stimulus (*e.g.*, continuous or intermittent). Consequently, the proposed classification of sports activities considers the long-term CV adaptations related to physical activity, including changes in cardiac volumes, wall thicknesses, and mass (Figure 1).

Group A includes sports where excellence is attributed to the athlete's technical skills, prompting a response from the neuro-adrenergic system with moderate increases in HR and BP, such as golf, table tennis, bowling, sailing, and equestrian. The hemodynamic overload is minimal or nonexistent in these disciplines, so the heart does not experience significant morphological changes. Power sports (Group B) are marked by anaerobic energy utilization, leading to brief BP spikes but only modest HR increases. This anaerobic activity contributes to increased myocardial mass due to a slight thickening of the walls, with little to no increase in ventricular volumes. Examples include weightlifting, boxing, and sprint running. Endurance sports (Group D), such as marathon running, cross-country skiing, cycling, and swimming, involve continuous cardiac engagement. These sports are characterized by isotonic-dynamic muscular activity, predominantly powered by aerobic energy, which results in an increased cardiac output, primarily due to an increased preload. Consequently, as per Laplace's law, there is a predominant increase in all endocardial dimensions and a parallel, though not marked, increase in left ventricular wall thicknesses. Group C encompasses sports that combine aerobic and anaerobic metabolism, leading to intermittent increases in preload (and/or afterload) with intense periods of activity alternating with recovery phases. This is typical in team ball sports such as soccer, rugby, basketball, and volleyball. In these instances, the adaptive stimulus on the heart enlarges the endocardial dimensions alongside a parallel, albeit modest, increase in left ventricular wall thicknesses.



	Group A	Group B	Group C	Group D
	Skill	Power	Mixed	Endurance
Heart rate	+/++	++	++/+++	+++
Blood pressure	+	+++	++	++
Cardiac output	+	++	++	+++
Cardiac remodeling	-	+	++	+++

Figure 1.—Classification of sports activities based on cardiovascular involvement.

These adaptations also depend on the athlete's role. However, it is essential to recognize that the relationship between sports activity and the CV system does not always adhere to simple pathophysiological frameworks. The CV engagement and the consequent risk associated with various sports disciplines, while classifiable into reasonably homogeneous groups, vary according to the specific traits of the sport in question and external factors like concurrent illnesses, the athlete's psychological state, and environmental conditions.

Sports physicians must also acknowledge that some sports feature different specialties, each with distinct CV adaptations. A notable aspect in assessing CV risk in sports is the 'intrinsic risk' linked to certain sports activities performed in adverse environments (*e.g.*, underwater sports, mountaineering, motorsports). In these disciplines, the potential for pre-syncopal or syncopal episodes can pose a risk to athletes and specta-

tors, as seen in motorsports. Furthermore, it is plausible that CV risk is heightened in contact sports, where chest contusions or severe cardiac reflex stimulations, such as head injuries, can trigger the onset of arrhythmias.

Pre-participation screening in Italy

Since 1982, Italy has mandated that every athlete undergo a pre-participation sports medicine evaluation to secure eligibility for competitive sports.[6, 7] This screening aims to detect CV abnormalities that could increase the risk of sudden cardiac death during sports activities.[8] The comprehensive Italian pre-participation screening protocol encompasses the collection of personal and family medical histories, a physical examination, spirometry, a urine test, a 12-lead electrocardiogram (ECG), and exercise stress testing (EST) (Figure 2). Should any positive findings emerge, further second- and third-line CV diagnostic techniques should be employed to rule out CV diseases.[9, 10]

ECG alterations in mid to high-level competitive athletes are categorized into two main types: common/training-related and uncommon/training-unrelated.[11] This classification is not suitable for less-trained individuals or beginners in sports. The "uncommon" anomalies are subdivided into "major" and "minor," depending on the probability of underlying heart disease, which requires a comprehensive clinical and instrumental assessment. The decision to conduct further investigations for isolated minor alterations must be made on a case-by-case basis, guided by clinical suspicion (Figure 3).

Exercise testing can be conducted *via*:

• Step test: In line with the 1982 Ministerial Decree, the step test is a practical alternative to the competitive certification screening protocol. It is designated for male participants under 40 and female participants under 50. The procedure entails continuous 12-lead ECG monitoring throughout the 3-minute test, extending monitoring at least until the second minute of recovery,

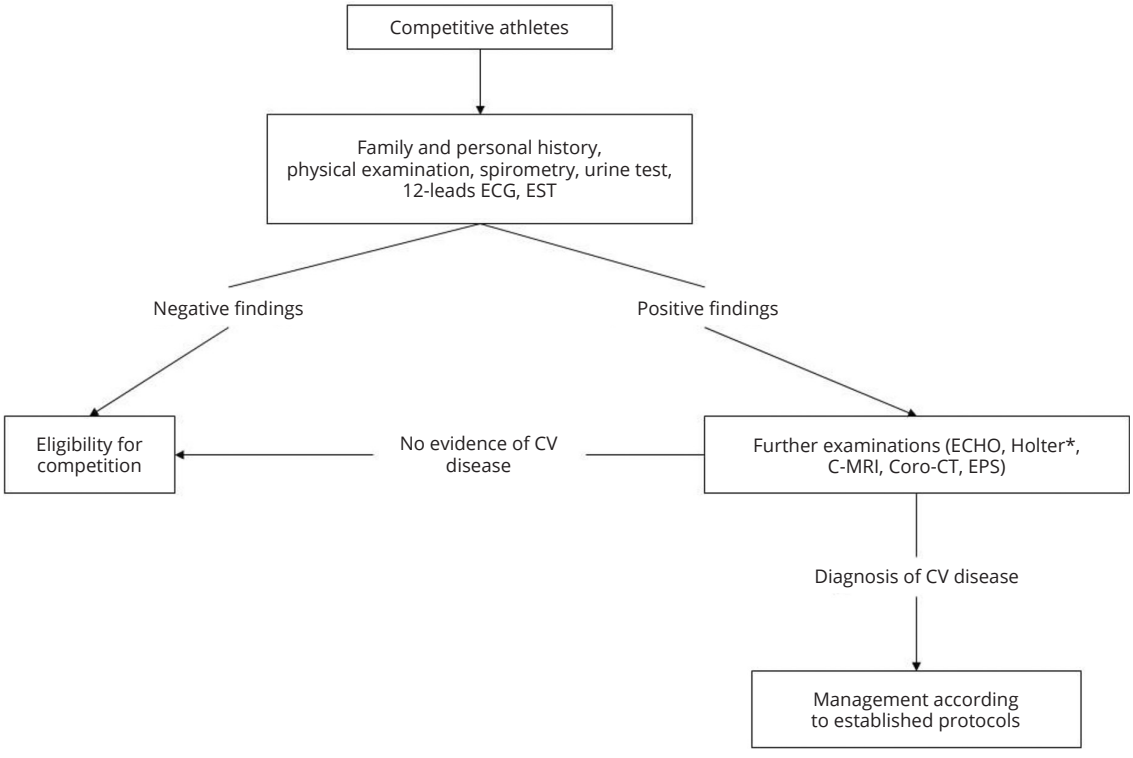


Figure 2.—The Italian preparticipation screening flow chart.
*Holter: 12-lead 24-hour ambulatory ECG monitoring, including a training session.

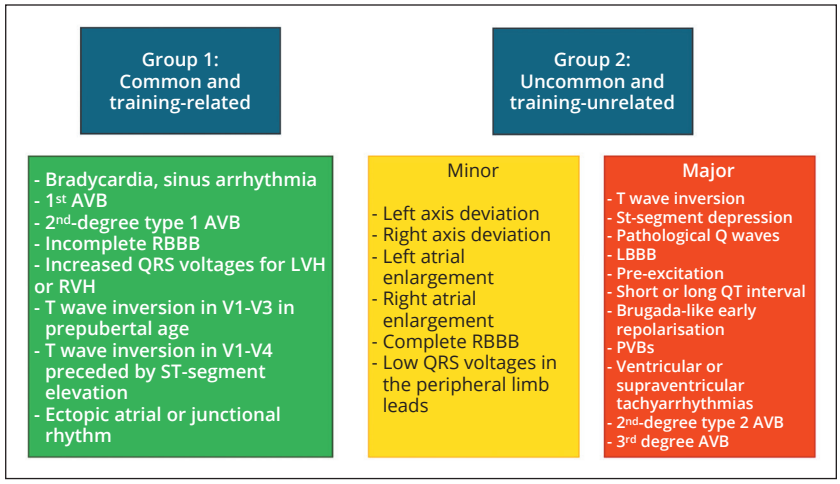


Figure 3.—Electrocardiographic findings in competitive athletes.
AVB: atrioventricular block; RBBB: right bundle branch block; LVH, left ventricular hypertrophy; RVH, right ventricular hypertrophy; LBBB: left bundle branch block; PVBs, premature ventricular beats.

following the methodology described by Montoye *et al.*[12]

• EST: This test is recommended for male participants aged 40 and above and female participants aged 50 and above. However, clinical conditions might necessitate its use for males under 40 and females under 50. It assesses various factors, including typical St-T segment variations, symptoms, BP, chronotropic response to exercise, the potential induction of arrhythmias, cardiorespiratory fitness, and the interaction between sinus and atrioventricular bradyarrhythmias during physical exertion. Additionally, it examines the QT interval and delta wave behaviors in Wolff-Parkinson-White syndrome. The protocols, either stepwise or ramp, are designed to reach maximal HR and are halted solely due to the athlete's inability to continue or for clinical reasons such as symptoms or ECG changes.[13] Monitoring is recommended to continue until at least the fourth minute of recovery.

In analyzing these tests, it is crucial to weigh the athlete's CV risk and the CV demands of the chosen sport. The risk assessment for the athlete can employ parameters from the European Society of Cardiology (ESC) guidelines, including the Systematic Coronary Risk Evaluation (SCORE) model, particularly SCORE2/SCORE2-OP, to estimate the 10-year risk for fatal and non-fatal CV events.[14] This aids sports physicians in evaluating the athlete's risk level. Today, various diagnostic tools are available to detect the presence and quantify the extent of coronary artery disease.[15] Coronary Computed Tomography Angiography (CCTA) is recognized as the gold standard anatomic test for intermediate-risk subjects.[16, 17] The Swedish Cardiopulmonary Bioimage (SCAPIS) Study, which included over 25,000 individuals without known coronary heart disease, found CCTA-detected atherosclerosis in 42.1% of participants: the onset of atherosclerosis was delayed by an average of 10 years in women, and atherosclerosis was more prevalent among older individuals.[18] Thus, a modern prevention strategy (especially for competitive master athletes over 50 years old) could take into consideration a broader indication of CCTA in the future.[4] However, given the Italian legislative context and its focus on protecting vulnerable individuals from sports-related consequences, a widespread "screening" is currently impractical for the national health system due to the lack of prospective data. Therefore, although EST has a lower diagnostic specificity for myocardial ischemia than CCTA, especially in asymptomatic individuals, screening with CCTA is not feasible for the athletic population. Consequently, the decision to conduct a CCTA for screening should be carefully made and linked to the risk assessed through SCORE2/SCORE2-OP (Figure 4).

Arrhythmias

The recommendations for sports eligibility and disqualification in athletes with brady- and tachyarrythmias are summarized in Table I, II, III.

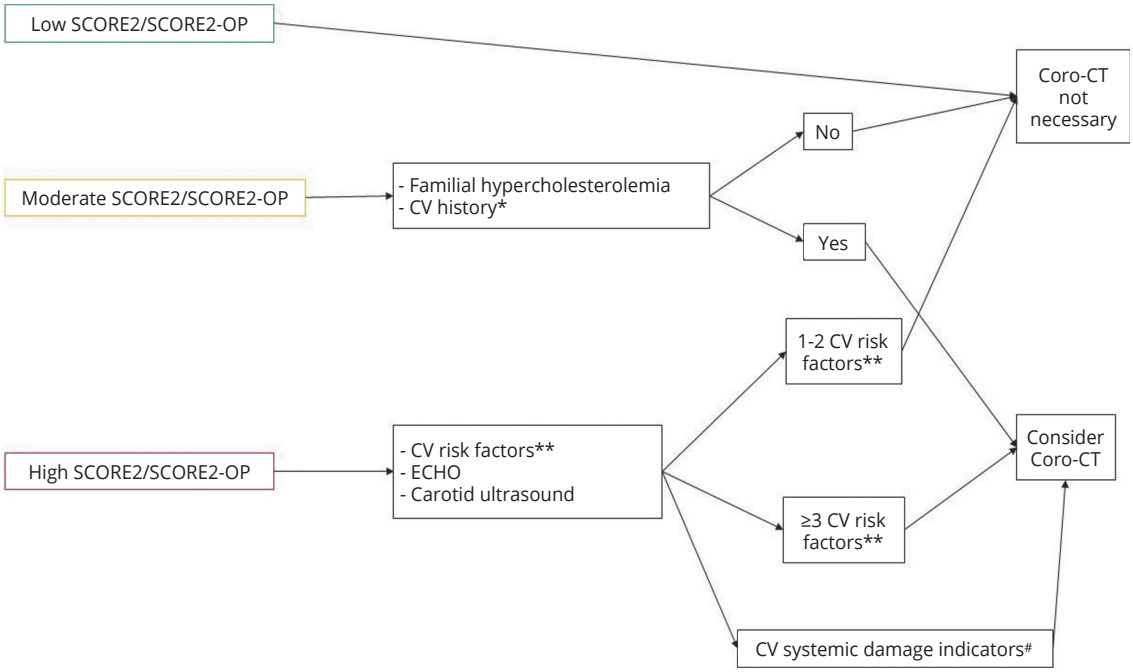


Figure 4.—Indications on when to perform a coronary CT angiography in the sports eligibility evaluation of an athlete with a negative exercise stress testing.
*CV history: CAD familiar history, coronary revascularization familiar history, SCD familiar history (1st-grade relatives who died suddenly <55 aged if male or <65 aged if female); **CV risk factors: male sex, smoking habits, abnormal Low-Density-Lipoprotein (LDL) (>55 mg/dL if <70 age, >100 mg/dL if >70 age), high BP, impairing fasting glycemia (IFG) (110-126 mg/dL), obesity (Body Mass Index >30 kg/m^2, waist circumference ≥102 cm if male, ≥88 if female); #CV systemic damage indicators: eccentric LV hypertrophy, atherosclerotic plaques at carotid ultrasound (>1.5 mm thickened), 3rd-grade kidney failure (Glomelural Filtration Rate <60 mL/min), long-standing diabetes mellitus (more than 10 years).

Because of increased vagal tone, athletes often exhibit sinus bradycardia and/or AV nodal atrioventricular blocks.[3] Similarly to sedentary individuals, athletes can experience arrhythmias unrelated to sports activities. Arrhythmias such as sporadic premature ventricular beats (PVB) are common and typically lack a pathological foundation. Conversely, some arrhythmias may arise from congenital anomalies of the conduction system (*e.g.*, Wolff-Parkinson-White Syndrome) or organic heart disease (such as coronary heart disease - CHD or cardiomyopathies). Identifying organic heart disease during screening is a primary goal in preventing malignant arrhythmias in athletes.

Objectives and eligibility criteria

The conditions analyzed in this document for granting competitive sports eligibility include:

- symptoms of a suspected arrhythmic nature (syncope, pre-syncope, and palpitations);
- documented arrhythmias;
- heart disease predisposing to malignant arrhythmias;
- individuals treated with transcatheter ablation;
- individuals with pacemakers (PMK) or implantable cardioverter-defibrillators (ICD).

Regarding heart diseases predisposing to malignant arrhythmias, this chapter discusses conditions occurring in the absence of structural heart disease, such as Wolff-Parkinson-White (WPW) syndrome and ion channel diseases. Please refer to the relevant chapters for structural pathologies (cardiomyopathies, ischemic heart disease, etc.).

Eligibility is determined based on the risk that:

- An arrhythmia may cause significant hemodynamic repercussions due to excessively high or low HR during sporting activity, recovery, and/or at rest;
- an arrhythmia may lead to pre-syncope, syn-

TABLE I.—*Recommendations for eligibility to competitive sports in athletes with sinus bradycardia, sinus arrhythmia, sinoatrial blocks, and atrioventricular (AV) blocks.*

Recommendation	LOE	COE
Sinus bradycardia, sinus arrhythmia, sinoatrial blocks		
Eligibility is granted under all the following conditions: • Absence of any heart disease that is incompatible with the sport; • No symptoms related to bradycardia, such as syncope, pre-syncope, asthenia (weakness), dyspnea (shortness of breath), or intolerance to effort; • For individuals who, after first-level investigations, show no signs suggesting intrinsic sinus node dysfunction (demonstrated by normal sinus response during effort and sinus pauses lasting less than 3 seconds); • In highly trained athletes, particularly those engaged in aerobic sports, eligibility may be considered even if resting HR is below 40 bpm and sinus pauses exceed 3 seconds, as long as there are no bradycardia-related symptoms and EST confirms normal chronotropic competence; • In cases where the diagnosis remains uncertain, a period of detraining lasting 2-3 months may be recommended to observe if the bradycardia normalizes, aiding in the decision-making process regarding eligibility.	I	B
Atrioventricular (AV) blocks and normal QRS		
Eligibility is granted under all the following conditions: • In the absence of any heart disease that is incompatible with sport; • In the absence of symptoms correlated with bradycardia; • In the presence of a narrow QRS complex or incomplete RBBB; • In cases of first-degree AV block, when the PR interval normalizes during hyperventilation and physical effort; • In grade II AV block, Luciani-Wenckebach type (or Mobitz 1), in the event of normalization of AV conduction with an increase in HR (as documented during ECG monitoring or EST).	I	B
Eligibility can be considered: • In cases of advanced and complete AV block documented during periods of vagal hypertonicity (typically observed during the night) in the absence of pauses exceeding 3 seconds, which disappears after a period of detraining; • In congenital AV block with a tight QRS junctional rhythm greater than 40 bpm and a normal increase during effort (eligibility limited to Group A sports).	II	C
Eligibility must be denied: • In individuals with mutations in genes associated with conduction disorders; • In cases of total congenital or acquired AV block, eligibility may be considered for sports other than those categorized in Group A; • If EPS, when applicable, indicates an infra- or sub-Hisian delay; • Subjects exhibiting symptoms attributable to bradycardia.	III	B

TABLE II.—*Recommendations for eligibility to competitive sports in athletes with intraventricular conduction disturbances.*

Recommendation	LOE	COE
Intraventricular conduction disturbances		
Eligibility may be granted for athletes with intraventricular conduction disorders, specifically in cases of RBBB and left anterior hemiblock, in the absence of a cardiac disease that is incompatible with sports participation.	I	B
Eligibility could be considered for athletes with LBBB, RBBB + LAH, and RBBB + LPH, after excluding cardiomyopathy or congenital or acquired CAD, for participation in Group A sports. An individualized assessment is recommended for eligibility in other sports	II	C
Eligibility must be denied, with exceptions subject to individual evaluation, in cases involving LBBB or bifascicular blocks under the following circumstances: • In the presence of a family history of SCD; • In the presence of a family history of Lenegre disease, Brugada syndrome, or ion channel diseases; • In the presence of suspected syncope or presyncope of cardiac origin, when the arrhythmic nature of the syncope has not been definitively excluded; • In the presence of heart disease incompatible with the sports practice or mutations related to genes associated with conduction disorders.	III	C

TABLE III.—*Recommendations for eligibility to competitive sports in athletes with supraventricular premature beats, paroxysmal supraventricular tachycardias, atrial fibrillation and atrial flutter, and ventricular pre-excitation.*

Recommendation	LOE	COE
Supraventricular premature beats		
Eligibility can be granted to athletes with SPBs without major symptoms.	I	C
Paroxysmal supraventricular tachycardias		
Eligibility is granted under all the following conditions: • Absence of major symptoms such as syncope or pre-syncope; • The arrhythmia is sporadic and does not have a cause-and-effect relationship with the sports activity, and the sports activity itself does not pose inherent risks; • The arrhythmia, whether spontaneous or induced during an EPS, at rest and/or during exertion, does not exhibit a high frequency (exceeding the expected maximal heart rate for the individual's age).	I	C
Eligibility could be considered: • In the absence of major symptoms such as syncope or presyncope; • If your resting HR is slightly higher than the normal sinus rate and if, during exertion, the heart rate does not exceed the maximum heart rate for age.	II	C
Atrial fibrillation and atrial flutter in the absence of WPW		
Eligibility is granted under all the following conditions: • There is an absence of heart disease incompatible with competitive sports and major symptoms; • Any identifiable triggering cause (such as hyperthyroidism, alcohol, drugs, illicit substances, etc.) has been identified and removed; • There is no cause-effect relationship between sports activities and the arrhythmia; • The arrhythmic episode is infrequent, has no high frequency, and has limited duration.	I	C
Eligibility could be granted to athletes with permanent AF or limited atrial flutter for low or moderate CV commitment sports activities, provided there is no heart disease and significant symptoms, and the HR during EST and ECG monitoring does not exceed the maximum HR for their age.	II	C
Eligibility must be denied for high-risk sports involving trauma if the athlete is taking anticoagulant therapy.	III	B
Ventricular pre-excitation		
Eligibility is granted under all the following conditions: • In the absence of symptoms and for symptomatic athletes with pre-excitation patterns such as Mahaim, if they meet the same eligibility criteria provided for episodes of supraventricular tachycardia; • If an EPS induces pre-excited AF with a minimum R-R interval >250 milliseconds at baseline, during isoproterenol infusion, or EST; • If during EPS, 1:1 conduction through the anomalous pathway is interrupted with an atrial pacing cycle length >250 milliseconds (at rest) and/or the effective refractory period of the anomalous pathway is >250 milliseconds at rest, during isoproterenol infusion, or during EST; • If AV reentry tachycardia is not inducible during EPS at rest and during isoproterenol infusion or physical exertion.	I	C
Eligibility must be denied: • In individuals with documented episodes of tachycardia from AV reentry or symptoms suggestive of AV reentry tachycardia; • In cases where re-entry AV tachycardia is inducible during an EPS; • In instances where, during EPS, AF is inducible with a minimum R-R interval between pre-excited beats ≤ 250 milliseconds at rest and/or during EST or isoproterenol infusion. • For prepubertal children, it is reasonable to adopt less restrictive eligibility criteria than those used for adults, considering the theoretical risk of allo-induced AF. Specifically in EPS with a minimal R-R interval between pre-excited beats ≤210 milliseconds at rest.	III	B

cope, and/or cardiac arrest, potentially resulting in sudden death;

• sporting activity may unfavorably affect the anatomical and electrophysiological substrate of the arrhythmia, worsening or accelerating a potential pathology or adversely altering the characteristics of the arrhythmia itself.

The judgment of suitability may vary depending on the sport practiced, considering the different CV demands and the inherent risk of each sporting discipline. In sports with intrinsic risk, even a benign loss of consciousness can result in serious adverse events for the athlete and/or spectators. Athletes with non-physiological arrhythmias and/or significant symptoms (such as syncope or tachycardia), where arrhythmogenic heart disease or an arrhythmia incompatible with sports is suspected, must be suspended from competitions until the completion of the investigations.

Bradycardia and conduction disorders (activation delays)

Sinus bradycardia, sinus arrhythmia, sinoatrial blocks

Sinus bradycardia is prevalent among athletes engaged in aerobic sports. Even significant sinus bradycardia can be observed in elite athletes without indicating any pathology. Typically, the lowest frequencies and/or asystolic pauses occur during sleep, with pauses generally lasting less than 3 seconds. However, in highly trained athletes, these pauses may exceed this duration. An EST normalizes bradycardia in healthy individuals, with the maximal HR being achieved according to the formula (220 bpm - age).[19-22]

AV blocks and normal QRS

First-degree atrioventricular (AV) block is common in trained athletes engaged in aerobic sports. Second-degree type 1 AV block (Luciani-Wenckebach phenomenon) frequently occurs in highly trained athletes, rarely observed in basal ECG but commonly during sleep. Nocturnal, advanced AV block and asystolic pauses may occur but are generally less than 3 seconds. AV block in healthy athletes typically normalizes during an EST. In such instances, further evaluations are not usually necessary. However, in selected cases, ambulatory ECG monitoring and an echocardiogram may be required to rule out structural heart disease. Mobitz 2 AV block and persistent third-degree AV block necessitate a more comprehensive evaluation.[23]

Intraventricular conduction disturbances

An incomplete right bundle branch block (RBBB) is a common benign finding that typically does not prompt further investigation. Complete RBBB is less common but generally is not linked with heart disease, though it does require at least an echocardiogram for evaluation.

In contrast, left bundle branch block (LBBB) and RBBB associated with left anterior hemiblock (LAH) or left posterior hemiblock (LPH) are rare and warrant additional investigations. In certain cases, advanced-level diagnostics such as CMR or electrophysiological studies (EPS) may also be required.[24]

Supraventricular arrhythmias in the absence of WPW

Supraventricular premature beats

The identification of supraventricular premature beats (SPBs), particularly when frequent and/or repetitive, necessitates the exclusion of illicit drug use and hyperthyroidism.[25]

Paroxysmal supraventricular tachycardias

These arrhythmias are caused by a circuit within the AV node (AV node reentry tachycardia - AVNRT) or involve a concealed pathway (AV reentry tachycardia - AVRT).[25]

Atrial fibrillation and atrial flutter in the absence of WPW

Paroxysmal and persistent atrial fibrillation (AF) is the most common arrhythmia in adults, whether or not heart disease is present. Notably, AF is a rare finding in young athletes, and it must be investigated deeply. Factors such as hyperthyroidism, alcohol abuse, and illicit drug use may increase the risk of AF. Some authors suggest that sports activity, particularly among older athletes and those engaged in endurance sports, may contribute to AF; however, this hypothesis is still debated.[26-28]

For athletes with AF, an echocardiogram, ambulatory ECG monitoring, and an EST are recommended to rule out heart disease. If there is a suspicion that supraventricular tachycardia is triggering AF, EPS may be beneficial. Permanent AF typically precludes eligibility for competitive sports involving medium to high CV demand. Atrial flutter (whether paroxysmal, persistent, or permanent) is rare in athletes without heart disease but can lead to elevated HRs during exercise, making such arrhythmias generally incompatible with athletic activity at medium-high CV demand.[29]

Ventricular pre-excitation

WPW pattern and WPW syndrome

The WPW pattern, characterized by a short PR interval and a delta wave, is caused by an anom-

alous pathway (Kent bundle) with anterograde conduction that connects the atria to the ventricles, bypassing the AV node. WPW may spontaneously disappear with ageing. Individuals with the WPW pattern may experience various arrhythmias (WPW syndrome), including:

- atrio-ventricular orthodromic re-entrant tachycardia (AVRT);
- antidromic AVRT (which is very rare);
- pre-excited AF. The latter, in the presence of a short refractory period of the Kent bundle, may progress to ventricular fibrillation, leading to sudden death.[30]

Many asymptomatic individuals do not have reentry arrhythmias because the anomalous pathway lacks retrograde conduction. Therefore, asymptomatic individuals, particularly the young, are not assured to remain symptom-free over time, necessitating thorough evaluation. The risk of sudden death is generally low, especially in asymptomatic patients.

The arrhythmic risk in individuals with the WPW pattern should be assessed through EPS, either transesophageal or endocavitary, based on the inducibility of reentry arrhythmias and the evaluation of the anterograde refractory period of the Kent bundle. EPS can be bypassed in asymptomatic patients exhibiting intermittent delta waves during an EST and may be postponed in asymptomatic children under 12 years of age (Table IV).

Mahaim-type pre-excitation

Mahaim-type pre-excitation, a rare condition, is characterized by an anomalous pathway with decremental properties that typically connects the right atrium to the right ventricle or the AV node to the right ventricle. The presence of a delta wave on the ECG can lead to many cases being mistakenly identified as asymptomatic WPW syndrome, with correct diagnosis often only achieved during EPS. Most individuals with Mahaim-type pre-excitation are asymptomatic. AVRT, displaying an LBBB morphology, is the most commonly induced or spontaneous arrhythmia associated with this condition. The risk of high-rate AF is very low.[31, 32]

Premature ventricular beats

Sporadic PVBs within 24 hours are common in the general population, whereas frequent PVBs, either singularly or in pairs, are relatively rare. The presence or absence of heart disease primarily influences the prognosis of single or paired PVBs.[33] In cases of underlying heart disease, the risk is associated with the type of the disease itself. Primary cardiomyopathies and myocarditis are the most common pathological conditions found in athletes, especially those under 35 years of age with PVBs.

Athletes with PVBs, identified incidentally (during baseline ECG or the EST), require a level II evaluation, including echocardiography, maximal exercise testing, and ambulatory ECG monitoring. Level III tests such as CCTA or CMR might be necessary in selected cases (Figure 5). Characteristics suggesting a higher likelihood of underlying heart disease, and thus the need for level III investigations, are detailed in Table IV.[34]

TABLE IV.—*Characteristics of premature ventricular beats that increase or decrease the probability of an underlying heart disease.*

	Greater risk of heart disease	Less risk of heart disease
Family history	+	-
History of syncope/pre-syncope	+	-
ECG abnormalities	+	-
PVB's morphology	Intermediate axis or higher (especially if with BBD pattern in V1 and QRS duration >130ms)	Lower axis (especially if with BBS pattern in V1), BBD with QRS duration <130ms
Morphology numbers	>1	1
Ergometric Test	Increase/persistence of PVBs	PVBs reduction
Complexity (close couples, TVNS, "R on T")	+	-
Maximum Ergometric Test reproducibility/ repeated Holter ECGs	+	-

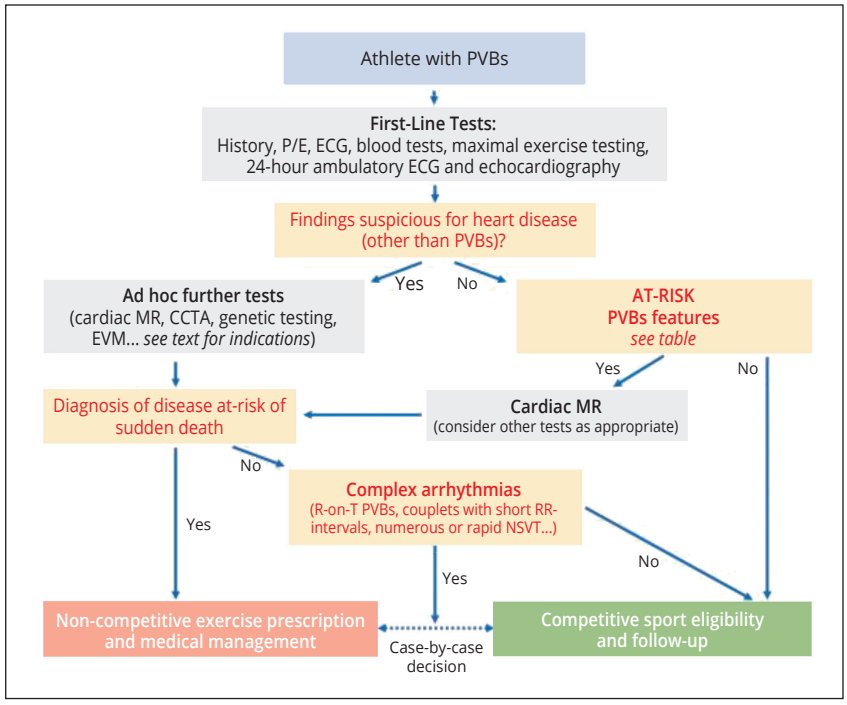


Figure 5.—Diagnostic flow chart for evaluating an athlete with premature ventricular beats.

The diagnostic process should be driven by clinically motivated suspicion.

The importance of thoroughly evaluating family history and critically analyzing resting ECG is emphasized. Resting ECG can reveal not only pathological signs like bundle branch blocks but also alterations that, while possibly non-specific, are significant in diagnosing certain arrhythmogenic pathologies, such as a T-wave inversion in V1-V3 indicating arrhythmogenic cardiomyopathy (ACM), or in the inferolateral leads, which might suggest hypertrophic cardiomyopathy (HCM).

For athletes presenting new findings of PVBs with risk characteristics, eligibility should be suspended until the necessary diagnostic investigations are complete. This approach also allows the evaluation of the arrhythmia's reproducibility post-detraining and helps prevent the exacerbation of any acute pathologies, such as myocarditis.

Interestingly, the number of PVBs in 24 hours does not correlate with prognosis.[35] Paradoxically, individuals with heart disease, such as HCM, may have few PVBs in 24 hours yet remain at significant risk.[36] Conversely, non-cardiac patients can exhibit a high number of PVBs within the same timeframe. However, when PVBs exceed 10,000/24 hours in non-cardiac patients, left ventricular (LV) ejection fraction (EF) reduction may occur over time. In the absence of heart disease, PVBs usually disappear during exercise. Nevertheless, their persistence during effort does not necessarily indicate heart disease. Similarly, the suppression of the arrhythmia during maximal EST does not definitively rule out underlying heart disease. Analyzing the morphology of PVBs can also provide insights into their origin.[37-39] Furthermore, detraining is a viable solution in diagnosing PVB in athletes when CV diseases are ruled out.[40]

Benign ventricular tachycardias (VTs), including fascicular VT and right or left ventricular outflow tract (RVOT or LVOT) VT, are noteworthy. Fascicular VT, related to a ventricular reentry phenomenon within the left ventricular conduction system, exhibits an ECG pattern similar to RBBB plus left anterior hemiblock.[41] RVOT and LVOT-VT, associated with an automatic focus in the outflow tract of the respective ventricle, typi-

cally do not feature short RR intervals. Physical exercise often triggers these arrhythmias. Assessing these conditions requires EST, echocardiography, and ambulatory ECG monitoring to exclude organic heart disease and document the arrhythmia's onset mode and characteristics.

Sports activity in patients with implantable pacemaker

For individuals planning to practice sports, the device should be implanted in the contralateral side relative to the dominant arm.[42] A leadless PMK is preferred for patients who participate in sports.[43] Sports eligibility for individuals with a PMK should be assessed at least 2-3 months post-implantation, considering the dependency on the PMK for those involved in sports with a risk of trauma.[44]

Sports activity in patients with implantable defibrillators

Many patients with ICDs have structural heart disease, which inherently disqualifies them from participating in competitive sports. For patients not requiring an anti-bradycardic device, a subcutaneous ICD is recommended. Eligibility for sports participation in patients with an ICD should be evaluated at least 3 months post-implantation.[45, 46]

Ion channel diseases

Ion channel diseases represent a significant category of genetic disorders that affect the functioning of potassium (K+), sodium (Na+), and calcium (Ca++) channels. These dysfunctions can either enhance or diminish the channels' function, leading to alterations in the monophasic potential that predispose individuals to both supraventricular and ventricular arrhythmias.

The variety of ion channel diseases is constantly increasing. Long QT (LQT) syndrome, Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia (CPVT) are the most recognized types. The recommendations for sports eligibility and disqualification for athletes with channelopathies are summarized in Table V.

Long QT syndrome

Long QT syndrome (LQTS) is a genetic disorder that causes an abnormal QTc interval prolongation. The most prevalent forms, constituting 90% of cases, are LQT1, LQT2, and LQT3, associated with dysfunctions in the IKs, IKr, and INa channels, respectively.

LQTS places individuals at risk for malignant ventricular arrhythmias, which can be triggered by stress (LQT1), emotional stimuli (LQT2), or sinus bradycardia (LQT3).

A diagnosis of LQTS can be made with a QTc of 480 milliseconds or more, including in asymptomatic individuals or those with a Schwartz score of 3.5 or higher. Syncope, coupled with a QTc of at least 460 milliseconds, warrants suspicion of LQTS. Genetic testing can offer additional insights, boasting a sensitivity exceeding 70%.

Participation in any form of competitive sports is contraindicated for individuals diagnosed with LQTS, even in the absence of documented ventricular arrhythmias.[47]

Brugada ECG patterns and Brugada Syndrome

Brugada syndrome (BrS) is a genetic disorder associated in approximately 20-30% of cases with a sodium channel dysfunction, characterized by a distinct ECG pattern in the V1-V3 precordial leads, also noted in the II and III intercostal spaces.[48] This pattern, identified as Type-1 BrS ECG, features a J point elevation of ≥2 mm and "coved" ST elevation followed by a downward sloping ST segment and negative T wave. Additionally, Type-2 and Type-3 BrS ECG patterns, such as a "saddle-back" ST elevation >2 mm in V1-V2 followed by a positive T wave for Type-2 and ST elevation <2 mm for Type-3, can be observed in individuals with Brugada Syndrome and intermittent BrS type 1 ECG. However, only the Type-1 pattern is diagnostic, whether observed in the basal state, in II and III intercostal space, during fever, or following class 1C drugs. Types 2 and 3 BrS ECG alone are insufficient for diagnosing Brugada Syndrome.[49]

The presence of a Brugada ECG pattern in asymptomatic individuals does not equate to a diagnosis of the syndrome, which is defined

TABLE V.—*Recommendations for eligibility to competitive sports in athletes with ion channel disease.*

Recommendation	LOE	COE
Long QT syndrome		
All subjects diagnosed with LQT syndrome, whether due to positive genetic testing or the accumulation of clinical parameters, must be disqualified, except in the specific cases listed below. This restriction also applies to individuals diagnosed with short QT syndrome. It is important to note that the disqualification does not directly represent an indication for an ICD implantation, nor should an ICD be implanted solely to obtain sports eligibility.	III	B
Eligibility may be considered in subjects with mutations in genes associated with asymptomatic LQT syndrome who have a negative or borderline phenotype while on beta-blocker therapy. The evaluation process should begin with a detailed analysis of the type of sport and considerations related to the current pharmacological treatment, particularly beta blockers. The decision must be highly individualized, considering the specific type of LQTS (LQT1, LQT2, LQT3, etc.), family history, QTc values at rest, during recovery after maximal EST and findings from ECG monitoring. Eligibility decisions should be made by centers with great expertise in LQTS.	II	C
Brugada ECG pattern and Brugada Syndrome		
Eligibility is granted under all the following conditions: • In asymptomatic subjects with type 2 or 3 patterns; • In the absence of a family history of SCD and other risk factors; • In cases with type 1, without risk factors, and with a negative EPS.	I	C
Eligibility can be considered in asymptomatic athletes with a spontaneous low-risk type 1 pattern, without a family history of SCD and/or BrS, etc.	II	C
Eligibility should be denied in subjects with a doubtful-high risk score (as per the Sieira and Shanghai Score table), except in cases evaluated on an individual basis and in centers or by experts with high competence.		
Eligibility must be denied: • In symptomatic athletes experiencing syncope of a probable arrhythmic nature or cardiac arrest; • In athletes with a spontaneous or drug-induced type 1 pattern; • Disqualification is not equivalent to an indication for an ICD implantation, nor should ICD implantation be indicated solely to obtain eligibility.	III	B
Catecholaminergic polymorphic ventricular tachycardia (CPVT)		
Eligibility is normally denied, with exceptions to be evaluated on an individual basis, in individuals who are carriers of a genetic defect but have no documented arrhythmias.	II	C
Eligibility must be denied in all cases with documented arrhythmias, whether symptomatic or asymptomatic, even if the individual has an ICD.	III	B

by both the ECG pattern and the potential for malignant arrhythmias or risk of sudden cardiac death (SCD). The risk of SCD is low in asymptomatic individuals with Type-1 patterns, especially those with drug-induced Type-1 patterns. Malignant arrhythmias in BrS typically manifest at rest, during bradycardic phases, with no evidence suggesting that sports activity increases the risk of SCD.[50] Risk stratification for SCD in asymptomatic individuals with the Brugada Type-1 pattern relies on a multiparametric evaluation considering several potential risk factors,[51-54] such as family history of sudden juvenile death, fragmented QRS, early ventricular repolarization in the inferolateral leads without ST-segment elevation, associated conduction disorders, and the manifestation of a Type-1 pattern or increased ST-segment elevation post-exercise. The inducibility of sustained ventricular arrhythmias during EPS may be nonspecific but gains significance when combined with other risk factors. Individually, these risk factors have a low positive predictive value, indicating a minimal chance of serious events. The predictive value increases with the coexistence of multiple risk factors. Conversely, factors such as the absence of spontaneous Type-1, only drug-induced Type-1, absence of syncope, and negative EPS have a high negative predictive value, suggesting a negligible risk of sudden death for asymptomatic individuals with drug-induced Type-1 without other risk factors.[55-57]

Catecholaminergic polymorphic ventricular tachycardia

Catecholaminergic polymorphic ventricular tachycardia (CPVT) is associated with genetic defects leading to an excess of intracellular calcium. This ion imbalance causes repetitive-type

polymorphic VT, triggered by adrenergic stimulation, rendering participation in competitive sports incompatible.[58]

Congenital heart disease

The advancement in clinical care has led to an increase in patients diagnosed with congenital heart disease (CHD), representing a diverse and heterogeneous group in terms of disease characteristics, natural history, surgical or percutaneous treatments, and outcomes. The positive effects of physical activity in countering impaired exercise capacity and sedentary lifestyles, which patients with CHD are often inclined towards due to a perceived higher risk of exercise-induced CV events, are well-established.[59] Although the benefits of customized exercise prescriptions are clear and should be recommended for all CHD patients, participation in competitive sports may be permitted in selected cases, subject to evaluation in centers equipped with multidisciplinary expertise.

Every athlete with CHD should provide a comprehensive medical and surgical history, including information about the primary CHD diagnosis, comorbidities, surgical and percutaneous interventions, medications, and nutritional supplements. Assessments should always encompass details of cardiac symptoms, functional class, type of sport, training volume and intensity, and competition level. Physical examinations should thoroughly review heart rhythm, additional heart sounds, murmurs, arterial BP, oxygen saturation, indicators of heart failure, and the presence or absence of central cyanosis.[60] Essential to the serial evaluation of athletes with CHD are resting and exercise ECG, echocardiography, and 24-hour ambulatory ECG monitoring, including a training session and any other necessary examinations.

Transthoracic echocardiography, a critical examination, facilitates the assessment of biventricular dimensions and function, heart valve conditions, obstructions, and intracardiac shunts. Furthermore, understanding the specific pathophysiology of CHD and any interventions undertaken is crucial; hence, collaborative assessments by a multidisciplinary team are recommended to identify subtle clinical changes. Echocardiographic reports should detail left and right ventricular dimensions and volumes, LV wall thickness, biventricular systolic function – including EF, regional wall motion abnormalities, global longitudinal strain (GLS) when available – and diastolic function.[61] It should also include systolic pulmonary artery pressure and aortic dimensions in alignment with European guidelines.[62]

In some CHD cases, the RV plays the most significant role, and residual anatomic and hemodynamic abnormalities are common post-surgical or percutaneous correction. Cardiopulmonary exercise testing (CPET) proves exceptionally useful for CHD athletes by evaluating baseline fitness, tracking disease progression, assessing training effects, and revealing significant hemodynamic lesions less apparent at rest.[63, 64] Among the cardiopulmonary indexes examined during CPET, peak V_{O2} stands out as one of the most reliable predictors of morbidity and mortality in CHD patients.[65] Additionally, CPET and EST can evaluate the BP response to exercise, ischemia, arrhythmias, conduction diseases, and oxygen saturation levels both at rest and peak exercise.

This comprehensive data collection is vital for the individual assessment of each case, determining whether the hemodynamic impact of rest and effort falls within normal or acceptable limits for competitive sports participation.

For more details on the specific most common CHD, please refer to Table VI, VII, VIII.

Bicuspid aortic valve

The presence of a bicuspid aortic valve (BAV) is the most common CHD in both general and athletic populations, with a prevalence of 0.77-1.3% and a male-to-female ratio of 2.1:1.[66] The etiology of BAV remains unclear, and its clinical presentation can vary widely. According to the International BAV Nomenclature and Classification Consensus, there are three BAV types: fused BAV, 2-sinus BAV, and partial-fusion BAV, each with specific phenotypes, with or without raphe. Additionally, three hypothetical BAV prognostic groups have been identified: 1) typical valvulo-aortopathy; 2) complex valvulo-aortopathy; and 3) undiagnosed or uncomplicated BAV.[67]

TABLE VI.—*Recommendations for eligibility to competitive sports in athletes with atrial and ventricular septal defect.*

Recommendation	LOE	COE
Atrial septal defect		
Subjects with small, non-significant atrial septal defect (ASD) ostium secundum or with patent foramen ovale (PFO) or pulmonary vein anomalous drainage can practice all sport types except scuba diving with breathing equipment due to the increased risk of paradoxical embolism/ decompression disease, on the contrary apnea is allowed.	I	B
In subjects with a repaired (both surgical and percutaneous) ASD: • It is mandatory to re-evaluate the patients with ECG, ECHO, EST and 24-hour ECG monitoring, including a training session 6 months after the intervention. The athlete can be cleared for all sports if the right ventricle is normal and there are no significant arrhythmias; • If the athlete practices competitive scuba diving with breathing equipment, it is mandatory to perform a transthoracic echocardiography or a transcranial Doppler with bubble test to rule out a residual shunt at rest and/or during Valsalva maneuver.	I	C
In the presence of persistent right ventricular (RV) dilatation and/or dysfunction, residual pulmonary hypertension, supraventricular tachyarrhythmias and/or symptomatic sinus-atrial dysfunction (see arrhythmias chapter), competitive sport is not recommended. The athlete can be re-evaluated over time in the presence of a favorable course.	III	B
Ventricular septal defect		
Subjects with small, non-significant ventricular septal defect (VSD), not associated with other congenital cardiac anomalies, can practice all sports types. Usually, during infancy, those VSDs show a spontaneous closure.	I	B
In subjects with a repaired (both surgical and percutaneous) VSD: • It is mandatory to re-evaluate the patients with ECG, ECHO, EST and 24-hour ECG monitoring, including a training session 6 months after the intervention. The athlete can be cleared for all sports in the absence of residual shunt and/or significant arrhythmias; • If the VSD has been closed with a device, it is suggested to follow-up those individuals with ECHO, EST, and 24-hour ECG monitoring to rule out the presence of progressive conduction dysfunction or valve dysfunction, which can occur more frequently in perimembranous VDSs.	I	B
Athletes with residual significant VDS and/or with pulmonary hypertension, left ventricular (LV) dilatation and/or reduced LV ejection fraction cannot compete.	III	B

TABLE VII.—*Recommendations for eligibility to competitive sports in athletes with patent ductus arteriosus and aortic coarctation.*

Recommendation	LOE	COE
Patent ductus arteriosus		
Subjects with small, non-significant patent ductus arteriosus (PDA), not associated with other congenital cardiac anomalies, can practice all sports types.	I	B
In subjects with a repaired (both surgical and percutaneous) PDA: • It is mandatory to re-evaluate the patients with ECG, ECHO and EST 3 months after the intervention. The athlete can be cleared for all sports without a residual shunt.	I	B
Aortic coarctation		
Subjects with non-significant aortic coarctation, not associated with other congenital cardiac anomalies, can practice only skill sports. Power sports are contraindicated.	I	C
The practice of mixed or endurance sports should be discussed case-by-case in centers dedicated to congenital heart disease. After surgical or percutaneous correction, it is mandatory to re-evaluate the athlete with EST with particular attention to peak arterial pressure and ambulatory blood pressure monitoring 3 months after the intervention. Subjects with a complete recovery can participate in all sports types, with the potential exception of power sports.	II	B
Even in subjects with non-significant aortic coarctation or after a successful repair, the rare but potential risk of aortic rupture after thoracic traumatism should be evaluated after an angio-MR or CT to rule out the presence of lumen reduction, kinking, aneurysms only for contact sports with low risk of severe thoracic traumatism.	II	C

Typical valvulo-aortopathy represents the most common presentation of BAV, often asymptomatic in childhood but requiring monitoring due to an increased risk of valve complications (such as aortic valve stenosis, regurgitation, or a combination of both) and aortic com-

TABLE VIII.—*Recommendations for eligibility to competitive sports in athletes with complex congenital heart disease after surgical correction.*

Recommendation	LOE	COE
Tetralogy of Fallot		
Every subject should be evaluated case-by-case in specialized centers, athletes with repaired Tetralogy of Fallot (TOF) can compete in skill sports if the following criteria are present: • Mild tricuspid and pulmonary regurgitation with normal pulmonary artery systolic pressure (<35 mmHg), evaluated by ECHO and/or CMR; • Normal or mildly dilated right ventricle (RV) with preserved systolic function, preferably evaluated with CMR, to rule out also RV fibrosis; • Absence of residual shunt with normal left ventricular systolic function; • Indexed aortic root dimension within normal limits (<2.1 cm/m2) with none/mild aortic regurgitation; • QRS duration <160 msec without supraventricular or ventricular arrhythmias at 24-hour ECG monitoring and/or EST; • Normal effort tolerance (at least 80% of predicted VO2 max and VE/VCO2 within limits).	I	B
In subjects with surgically repaired TOF with optimal results, it might be possible to allow them to compete in mixed team sports after a complete evaluation in dedicated centers and with strict clinical follow-up.	II	C
Transposition of the great arteries		
Subjects who underwent the previous physiological correction (Mustard and Senning procedure cannot compete in any sport.	III	B
Subjects with a repaired transposition of the great arteries (TGA) with the arterial switch and preserved functional capacity, absence of major arrhythmias, normal coronary arteries tree (evaluated with CCTA) and the absence of induced ischemia at stress echocardiography or myocardial perfusion scintigraphy, can compete in skill sports under strict clinical surveillance.	I	C
In subjects with surgically repaired TGA with optimal results, it might be possible to allow to compete in power and mixed team sports after a complete evaluation in dedicated centers and with strict clinical follow-up.	II	C

plications (including dilatation and dissection of the aortic root and/or ascending aorta) in adulthood. Complex valvulo-aortopathy, while rare, is generally associated with genetic syndromes like the Shone complex and Marfan syndrome or with other congenital heart defects (*e.g.*, subvalvular aortic stenosis, aortic coarctation, or ventricular defects) and necessitates early intervention.[68] Both groups have a heightened risk of infective endocarditis compared to the general population.

Uncomplicated BAV may remain asymptomatic throughout life, with no LVOT obstruction or mild/non-progressive valvulo-aortopathy (mean gradient <20 mmHg and/or mild aortic regurgitation), normal aortic root and ascending aorta dimensions (<40mm), and normal LV dimensions, volumes, and systolic function.[69] Athletes with uncomplicated BAV are eligible to compete in any sport, provided they are under clinical and echocardiographic surveillance. Additionally, it is essential to exclude LV hypertrophy, LV systolic or diastolic dysfunction, pathological increase in systolic BP at rest and peak exercise, and the absence of exercise-induced arrhythmias or ischemia. In cases of aortic dilation (>40 mm or >2 Z-scores), it is crucial to investigate for connective tissue disease or a genetic syndrome association and to avoid power sports. Athletes with BAV and aortic valve stenosis (mean gradient >20 mmHg) are generally disqualified from all competitive sports, with exceptions possibly made for skill sports after assessing peak and mean gradients *via* stress echocardiography. Furthermore, all individuals with BAV should be screened for coronary artery anomalies.

Coronary artery anomalies

Coronary artery anomalies (CAAs) encompass congenital deviations in the origin, course, or termination of the coronary arteries. Despite their rarity and often asymptomatic presentation, CAAs are significant due to their potential association with exercise-induced CV events, including SCD.[70] Among CAAs, the most concerning is the anomalous origin of the left main or left anterior descending coronary artery from the right coronary sinus, especially when it involves an interatrial/intramural course characterized by features such as an acute angle takeoff, a slit-like orifice, or an ostial ridge. While the origin of the

right coronary artery from the left coronary sinus is deemed less dangerous, it requires thorough examination before clearing an individual for competitive sports. Conversely, the origin of the circumflex artery from the right coronary sinus or the right coronary artery with a retroaortic course is generally considered to have a benign prognosis (Table IX).

CAAs may go unnoticed until an athlete experiences effort-related symptoms like chest pain, thoracic discomfort, near-syncope or syncope, cold sweating, palpitations, dizziness, and breathlessness. Transthoracic echocardiography, particularly focusing on the parasternal short axis at or slightly above the aortic valve level to view the coronary ostia and their initial segment, is the initial diagnostic step.[61, 71] Subsequent CCTA and CMR confirm suspicions and provide detailed anatomical insights into the anomaly. Invasive functional testing is reserved for specific cases and should only be performed in centers with specialized expertise.[72, 73] Myocardial bridges, which involve cardiac muscle overlying a coronary artery segment, represent a relatively frequent finding among CAAs, detectable in autopsy series and CCTA in athletes. Most myocardial bridges remain clinically silent. However, significant symptoms such as exertional chest tightness and exercise-induced ischemia can occur with long (>1 mm) and deeply tunneled (≥3mm) segments of the anterior descending coronary artery during EST and CPET. Such bridges have been linked to SCD.

General principles of management of athletes with valvular heart disease

In athletes diagnosed with valvular heart disease (VHD), the physiological remodeling due to exercise can intersect with the pathophysiological hemodynamic effects of VHD. Thus, it is crucial to quantify valve dysfunction and assess chronic structural remodeling to exclude adverse cardiac remodeling. Additionally, the surge in adrenergic activity during competitive sports and increased hemodynamic preload and afterload in a structurally remodeled heart may lead to exercise-induced arrhythmias. Echocardiography stands as the foundational examination for VHD, offering detailed insights into valve anatomy and

TABLE IX.—*Recommendations for eligibility to competitive sports in athletes with coronary artery anomalies.*

Recommendation	LOE	COE
Adolescents and young adult athletes with an anomalous origin of the circumflex artery from the right coronary sinus or from the right coronary artery with a retroaortic course, without evidence of exercise-induced ischemia at maximal EST or CPET or other stress imaging exams, can compete in all sports type with a yearly follow-up. Differently, in master (>40-year-old) athletes, the risk of additional atherosclerotic coronary artery disease can impair the practice of competitive sports due to the increased risk of exercise-induced myocardial ischemia.	I	B
In asymptomatic athletes with an anomalous origin of the right coronary artery from the left coronary sinus with an interatrial course but with less malignant features of the coronary artery anomaly, the possibility of practicing competitive sports must be evaluated in selected centers on a case-to-case basis, including evaluation with stress imaging tests and even invasive functional tests.	II	C
The anomalous origin of the left coronary artery from the right coronary sinus with an interarterious/intramural course is a contraindication to all types of competitive sports and, in symptomatic athletes, is an indication of surgery. The majority of authors are favorable to proceeding to surgery, even in asymptomatic athletes, due to the increased risk of myocardial ischemia and SCD. If this anomaly is not surgically corrected, all moderate-intensity activities are not recommended, and pharmacological therapy with a beta-blocker can be started.	III	B
In symptomatic athletes with an anomalous origin of the right coronary artery from the left coronary sinus and interarterious course or asymptomatic athletes with high-risk characteristics (in the presence of an acute angle take-off, a slit-like orifice or an ostial ridge), competitive sports are not recommended.	III	B
Athletes with an incidental diagnosis of myocardial bridge or coronary fistula without evidence of exercise-induced myocardial ischemia (at CPET and/or stress echocardiography/CMR and/or exercise stress radionuclide imaging test or CCTA) can be cleared for all competitive sports.	I	B
Athletes with a long (>1cm) and deeply tunneled (≥3mm) myocardial bridge or with coronary fistula and a significant shunt, with evidence of induced myocardial ischemia or exercise-induced significant ventricular arrhythmias, cannot participate in competitive sports. In selected cases, athletes can be re-evaluated in selected centers six months after surgical correction without exercise-induced myocardial ischemia.	III	B

dysfunction and quantifying the hemodynamic impact of the lesion, as per current guidelines.

For athletes, the exercise test is essential to determine functional capacity, detect effort-related symptoms, assess BP response to exercise, and identify myocardial ischemia or exercise-induced arrhythmias.[72] This evaluation may extend to exercise stress echocardiography in ambiguous or selected cases. CMR plays an expanding role in the quantification of valve dysfunction in uncertain cases and in detecting myocardial fibrosis associated with chronic, significant valve dysfunction.[74]

Generally, aortic, mitral, and/or tricuspid regurgitations are better tolerated. Athletes with mild valve regurgitation can engage in all competitive sports, subject to annual surveillance. Those with moderate aortic or mitral regurgitation are assessed individually for participation in low-intensity (skill) sports. Severe aortic or mitral regurgitation precludes competition in any sport, necessitating evaluation for potential surgical intervention.

Post-surgical valve repair, athletes may return to competitive sports across all categories after a minimum six-month recovery, provided annual follow-ups confirm:

- normal or slightly dilated left ventricular dimensions with a normal LV EF;
- absence or mild valve regurgitation;
- mild tricuspid regurgitation with normal systolic pulmonary artery pressure;
- no complex supraventricular or ventricular exercise-induced arrhythmias on EST and 24-hour ambulatory ECG monitoring, including a training session. Conversely, athletes with functional biological prostheses are restricted to skill sports.

Valve stenosis is less well-tolerated, limiting athletes with mild mitral stenosis or, in specific cases, moderate stenosis in sinus rhythm to skill sports only.

Mitral valve prolapse

The prevalence of mild primary regurgitation among athletes is notable but does not hinder participation in competitive sports. However, secondary mitral regurgitation, indicative of systolic ventricular dysfunction, disqualifies athletes from competitive sports. Degenerative mitral regurgitation, especially bileaflet mitral valve prolapse (MVP), as seen in Barlow's disease, warrants careful examination. Accurate diagnosis of MVP is crucial to avoid overdiagnosis, requiring anatomical criteria such as mitral valve leaflet billowing, leaflet thickness, systolic prolapse of ≥2 mm above the annular plane in at least one leaflet, and the presence of mitral regurgitation on color Doppler.[75]

Among MVP variants, Barlow's disease and fibroelastic deficiency are distinct, with Barlow's disease particularly linked to arrhythmias. High-risk characteristics of arrhythmogenic MVP, associated with SCD regardless of mitral regurgitation severity, necessitate a thorough evaluation of athletes with MVP. This includes personal and family medical histories, physical examination, ECG, echocardiography (transesophageal if necessary), EST, and 24-hour ambulatory ECG monitoring. Follow-up intervals should be annual or every six months, as appropriate, for those with MVP.

High-risk characteristics include:

- negative T-waves in the inferior leads;
- polymorphic and/or complex RBBB ventricular arrhythmias;
- mitral annular disjunction coupled with myocardial fibrosis identified by CMR;
- symptoms such as syncope or near-syncope.

If any of these high-risk features are present, competitive sports are contraindicated, and further diagnostic testing may be required (class IIC). A flow chart[75] has been proposed for managing athletes with MVP (Figure 6). Tricuspid valve prolapse does not impact the ability to compete in sports, but concurrent prolapse of the aortic valve or aortic root dilation necessitates evaluation for potential Marfan Syndrome.

Cardiomyopathies, myocarditis, pericarditis and non-ischemic left ventricular scar

Hypertrophic cardiomyopathy

HCM has a prevalence of approximately 1:500 and is characterized by either segmental or diffuse LV hypertrophy without cavity dilation and in the absence of other cardiac or systemic causes. A di-

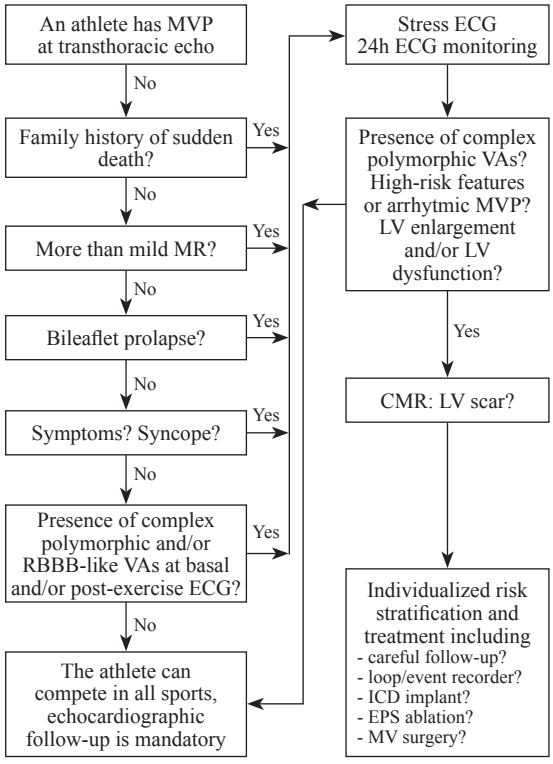


Figure 6.—Proposed algorithm for evaluating an athlete with mitral valve prolapse [From Cavarretta *et al.*[75]].

agnosis of HCM is established with an LV wall thickness of ≥15 mm. In cases where LV hypertrophy is milder, with LV wall thickness between 13-14 mm, a diagnosis can still be made if there is a positive family history, symptoms, ECG abnormalities, and/or pathogenic genetic variants.[76]

Initially, pathological studies indicated HCM as one of the leading causes of SCD among athletes, accounting for up to 40%. However, more recent findings report a significantly lower percentage (6-16%). Studies have also shown that tailored exercise programs can be safe for HCM patients.[77] For instance, a study with a 7-year median follow-up of 88 HCM patients found no difference in symptoms and adverse events between those who ceased sports activities post-diagnosis and those who continued, with no adverse events reported among the 27 continuing sports activities.[78] Another study involving 53 low-risk HCM athletes, including professionals, documented only two events during the follow-up.[79] An international registry of athletes with ICDs noted that only one of the 65 athletes with HCM experienced an appropriate shock during follow-up.[80] A recent survey also highlighted a similar incidence of adverse events between sedentary and physically active HCM patients.[81]

These findings suggest that "low-risk" HCM patients face a reasonably low probability of adverse events during low-to-moderate intensity exercise. Therefore, determining eligibility for competitive sports might be feasible, considering the risks and therapy, with evaluations best conducted in specialized centers (Table X).

The guidelines consider HCM patients as "low risk" in the absence of the following criteria:

1. age under 30 years, as the disease is not considered stable;
2. history of cardiac arrest or sustained VT;
3. symptoms such as chest pain, near-syncope, syncope, or recurrent/exercise-related palpitations;
4. history of SCD before 40 years in first-degree relatives;
5. moderate to severe LV hypertrophy;
6. severe left atrial enlargement;
7. LV EF below 50%;
8. LVOT obstruction (intraventricular gradient ≥ 30 mmHg at baseline, post-Valsalva maneuver, or during exercise testing);
9. moderate to severe mitral regurgitation;
10. exercise-induced myocardial ischemia or abnormal BP response to exercise (less than a 20 mmHg increase at peak exercise);

TABLE X.—*Recommendations for eligibility to competitive sports in athletes with hypertrophic cardiomyopathy.*

Recommendation	LOE	COE
Adolescents and young adults (<30 years old) with definite HCM diagnosis, even if at low-risk, and older patients with one or more risk markers should not engage in competitive sports activities.	II	B
Adult patients (≥30 years old) with a definite diagnosis of HCM at low-risk and genotype positive-phenotype negative patients may engage in competitive sports activities after an accurate evaluation in experienced centers, considering the discipline that the patient wishes to practice. For patients with an ICD, see the appropriate chapter.	II	C

11. AF, paroxysmal supraventricular tachycardia, frequent and polymorphic PVBs, non-sustained VT (≥3 beats, >120 bpm);

12. QRS duration ≥120 ms (excluding post-myomectomy);

13. late-enhancement >15% of myocardial mass on CMR (to be performed in specialized centers);

14. apical aneurysm, especially when associated with apical LGE;

15. ESC 5-year risk score >4%.

Arrhythmogenic cardiomyopathy

ACM is characterized by the progressive loss of myocardium and its replacement with fibro-fatty tissue. Often inherited, it is most frequently linked to mutations in desmosomal genes, though in about half of the cases, both family history and genetic testing yield negative results. ACM's phenotypic range is broad, spanning from the classic variant with predominant RV involvement to biventricular disease – which is the most common – and variants with predominant or exclusive LV involvement. Most patients exhibit ventricular arrhythmias, while severe ventricular dysfunction leading to heart failure is rare.[82]

Diagnosing ACM involves a combination of criteria across different categories: 1) morpho-functional abnormalities; 2) tissue characterization (*via* CMR or endomyocardial biopsy); 3) ECG abnormalities; 4) ventricular arrhythmias; 5) family history or genetic testing. The 2020 "Padua criteria" sought to refine the 2010 Task Force Criteria, especially by defining specific criteria for diagnosing LV involvement.[83]

ACM is a primary cause of SCD during exercise. Retrospective studies that include patients with the classic RV phenotype and those with ACM and an ICD have indicated that competitive sports can increase the risk of 1) disease development in gene mutation carriers; 2) disease progression in individuals already meeting the definitive diagnostic criteria; and 3) life-threatening ventricular arrhythmias. For the phenotype with prevalent or exclusive LV involvement, there is substantial evidence that competitive sports heighten the arrhythmic risk, though data on the likelihood of disease development in gene mutation carriers or phenotype exacerbation is lacking.[84] Nonetheless, the consensus among experts is to apply similar recommendations across the various phenotypic variants (Table XI).

Dilated cardiomyopathy

DCM is a myocardial disorder marked by LV or biventricular dysfunction, potentially accompanied by ventricular dilation, that cannot be attributed to abnormal hemodynamic load or myocardial ischemia. Its origins vary, encompassing genetic factors and acquired conditions such as myocarditis, adverse drug reactions, alcohol use, and peripartum events, though the cause remains unidentified in a substantial number of patients. Clinical manifestations of DCM are diverse, ranging from asymptomatic cases to mild phenotypes with LV dilatation and mild dysfunction, necessitating differentiation from an athlete's heart.[76]

While DCM is not a leading cause of SCD during exercise, there is a recognized risk of arrhythmias or hemodynamic deterioration associated with sports in individuals diagnosed with DCM. Nonetheless, it is feasible to consider those with DCM who lack the following risk factors as low-risk (Table XII):

1. symptoms, particularly syncope;

2. a family history of SCD before age 40 in first-degree relatives;

TABLE XI.—*Recommendations for eligibility to competitive sports in athletes with arrhythmogenic cardiomyopathy.*

Recommendation	LOE	COE
Patients with a definite diagnosis of ACM, according to the 2020 Padua criteria, including ICD carriers and family members with a borderline diagnosis of ACM, should not engage in competitive sports activities.	III	B
Phenotype negative-genotype positive individuals may engage in competitive skill discipline after accurate evaluation and counseling in experienced centers. The same recommendation applies to individuals with a family history of ACM in I or II-degree relatives with an identified gene mutation in the affected relative who did not undergo the genetic testing.	II	C
Individuals with a family history of ACM in I or II-degree relatives without identified gene mutation in the affected relative may be eligible for competitive sports activities provided an accurate clinical evaluation in experienced centers has excluded any sign of the disease.	II	C

TABLE XII.—*Recommendations for eligibility to competitive sports in athletes with dilated cardiomyopathy.*

Recommendation	LOE	COE
Patients with DCM with symptoms or with one or more risk markers should not engage in competitive sports activities.	III	C
Adult patients (≥30 years) with a definite diagnosis of HCM at low-risk and genotype positive-phenotype negative patients (excluding mutations in high-risk genes, see above) may engage in competitive sports activities after an accurate evaluation in experienced centers taking into account the discipline that the patient wishes to practice. For patients with an ICD, see the appropriate chapter.	II	C

3. AF, paroxysmal supraventricular tachycardia, frequent and polymorphic PVBs, non-sustained ventricular tachycardia (≥3 beats, >120 bpm);

4. moderate to severe LV dysfunction (LV ejection fraction <45%);

5. segmental LV akinesia or dyskinesia;

6. insufficient improvement in LV EF (<15%) or an abnormal BP response (less than a 20 mmHg increase at peak exercise) during EST;

7. extensive areas of LGE on CMR;

8. pathogenic or likely pathogenic mutations in genes associated with a high risk of adverse outcomes (such as LMNA, SCN5A, PLN, TMEM43, FLNC, RBM20, DSM).

Excessive trabeculation of the left ventricle (left-ventricular noncompaction)

LVNC is a congenital condition marked by inadequate development of the LV compact layer and an increased thickness of the non-compact layer. Its clinical manifestation can be diverse, potentially including progressive left ventricular dysfunction, LV hypertrophy, ventricular arrhythmias, and embolic events.[85]

It is important to distinguish simple hypertrabeculation of the LV, where the compact layer maintains normal thickness without additional disease characteristics, from LVNC. Such hypertrabeculation might simply be an adaptation to the increased hemodynamic load from athletic activity and should not be classified as pathological.[86]

Patients with LVNC are deemed low risk when they do not meet any of the following criteria:

1. symptoms, particularly syncope;

2. a family history of SCD;

3. significant atrial or ventricular arrhythmias;

4. LV dysfunction, characterized by an EF below 45% with a reduced (<15%) increase during exercise;

5. LGE on CMR.

Myocarditis and pericarditis

Myocarditis is an inflammation of the myocardium characterized by histologic evidence of myocardial necrosis and lymphocytes infiltration, distinct from myocardial ischemia. Causes can be viral, toxic, or immune-mediated. It is a notable cause of SCD in athletes, with a prevalence of 6-10% in post-mortem studies. During its acute phase, cessation of sports activity is a recognized therapeutic measure. Generally, the prognosis for individuals presenting with chest pain ("infarct-like") or other mild symptoms is favorable, though the healing process may lead to LV scar formation, potentially serving as an arrhythmogenic long-term substrate.[87]

Pericarditis, the inflammation of the pericardium, with or without effusion, is relatively common in young individuals. Viral infection is the most frequent cause among healthy young people. The prognosis for viral or idiopathic pericarditis is usually positive, but a subset of patients might experience recurrent episodes, especially those presenting with a temperature >38 °C, significant pericardial effusion, or resistance to non-steroidal anti-inflammatory drugs (NSAIDs). The risk of recurrent pericarditis peaks 6-12 months following the initial episode. As with myocarditis, athletes with acute pericarditis are advised to rest, minimizing mechanical stress on the inflamed pericardium.[88]

Myocarditis and pericarditis are deemed healed when the following criteria are met (Table XIII):

1. absence of symptoms;

2. negative inflammatory biomarkers and troponin;

3. normal ECG;

4. no frequent or complex ventricular arrhythmias on EST and 24-hour ambulatory ECG;

5. no LV dysfunction;

TABLE XIII.—*Recommendations for eligibility to competitive sports in athletes after an episode of myocarditis or pericarditis.*

Recommendation	LOE	COE
Temporary suspension of competitive sport is recommended in individuals with a definite or probable diagnosis of myocarditis or pericarditis.	III	B
Individuals with clinically healed myocarditis may resume sports activity after 3-6 months. In case of persistent LGE on follow-up CMR, see the paragraph on "non-ischemic LV scar".	I	C
Individuals with clinically healed pericarditis may resume sports activity after one month if the disease is mild and characterized by rapid improvement, not earlier than three months in the other cases.	I	C
Individuals with constrictive pericarditis should not engage in competitive sports activity.	III	C

6. absence of myocardial or pericardial edema on CMR;

7. normal results on EST or CPET.

For individuals who fulfill these criteria but exhibit persistent LGE on follow-up CMR, refer to the "non-ischemic LV scar" section.

Non-ischemic left ventricular scar

Non-ischemic LV scar (NILVS) is identified by segmental LV LGE on CMR, particularly in the subepicardial and/or mid-myocardial layers of the LV wall, without accompanying morpho-functional indicators of a specific disease. This type of myocardial fibrosis can arise from various conditions, including healed myocarditis and genetically determined heart diseases like the left-dominant ACM variant or Anderson-Fabry disease. The theory that intense exercise might induce non-ischemic myocardial scarring in otherwise healthy athletes remains unproven.[89]

CMR is the sole method for diagnosing NILVS, requiring considerable expertise for interpretation in athletes. Especially in cases of mild or borderline abnormalities, or in the absence of other clinical anomalies, it's crucial to rule out false positives, such as those related to the septal perforator branch of the left anterior descending artery, pericardium, or artefacts.[90]

The initial evaluation step for athletes with NILVS involves excluding cardiomyopathies that could deteriorate over time and have familial implications. Nonetheless, even with an unresolved NILVS origin, myocardial fibrosis is considered a potential substrate for life-threatening ventricular arrhythmias, particularly during sports activities.[39, 91]

There's currently no definitive evidence for risk stratification in athletes with NILVS. The consensus among experts categorizes the following conditions as "high-risk" (Table XIV):

- polymorphic PVBs or repetitive (couplets or non-sustained ventricular tachycardia) ventricular arrhythmias, especially during exercise;
- LV dysfunction (EF ≤ 50% *via* echocardiography or <45% *via* CMR);
- significant (non-training-related) ECG abnormalities;
- LGE extending across ≥3 segments or involving the septum.

Athletes deemed low-risk and eligible for sports should undergo annual clinical monitoring (or every six months for adolescents and young adults), considering NILVS might signify the onset of cardiomyopathy.

TABLE XIV.—*Recommendations for eligibility to competitive sports in athletes with non-ischemic left ventricular scar.*

Recommendation	LOE	COE
Athletes with NILVS associated with LV dysfunction and/or major ECG abnormalities and/or significant ventricular arrhythmias should not engage in competitive sports activity.	III	C
Athletes with NILVS involving less than 3 segments of the LV free wall with normal systolic function, normal ECG and no significant ventricular arrhythmias (at maximal EST and 24-hour ambulatory ECG monitoring, including training sessions) may engage in competitive sports activity after an accurate evaluation in experienced centers, taking into account the discipline that the patient wishes to practice.	II	C
Patients with larger NILVS (3 or more segments), even without other risk markers, may not engage in competitive sports activity, except for skill disciplines, after an accurate evaluation in experienced centers.		

Arterial hypertension

Arterial hypertension (AH) is a significant CV risk factor commonly detected during pre-participation screenings in athletes, with a varying prevalence of 3% to 24% based on the cohort and sport type.[92] In adults, AH is defined by systolic and diastolic values ≥140 and ≥90 mmHg, respectively, with children's and adolescents' cut-offs placed at the 95th percentile of arterial BP.[93] Proper measurement following a standardized protocol is crucial for diagnosis, and for sports that asymmetrically engage the upper limbs, such as tennis, BP measurement on the non-dominant arm is recommended.[94] To avoid an incorrect diagnosis, it is recommended to perform BP monitoring, re-evaluate the patient over a long period (3-6 months), and ask for a home BP diary.[94]

Upon diagnosis, it is essential to explore potential causes and assess overall risk. Secondary causes in athletes are rare (0.2%), and often related to external factors like high sodium intake, energy drinks, or anti-inflammatory drugs.[92] AH elevates the risk of myocardial infarction and ventricular arrhythmias, particularly during sudden BP spikes in physical activity. Comprehensive risk assessment is vital, considering AH rarely acts as the sole CV disease risk factor.

For athletes with AH, differentiating between exercise-induced physiological adaptations and potential organ damage, such as myocardial hypertrophy, is crucial.[95, 96] An ECG can help with differential diagnosis. Athletes' ECGs typically display bradycardia, high QRS voltages, right branch delay, positive T waves, and early repolarization. On the other hand, hypertensive patients' ECGs show P-wave alterations, QRS abnormalities with positive Sokolow index, left axis deviation, and intraventricular conduction delay. Echocardiography is the most useful clinical investigation. In hypertensive patients, the increased wall thickness is not associated with an increased ventricular dimension, while the opposite usually occurs in athletes. Additionally, hypertensive patients generally have diastolic dysfunction, increased filling pressures, and left atrial dilatation.

Exercise-induced BP response varies based on multiple factors, with a gradual systolic BP increase and stable or slightly reduced diastolic BP being normal.[97, 98] Athletes, especially those in endurance or mixed sports, may show an elevated BP response without associated organ damage or metabolic issues. Isolated systolic AH, common in those over 60 due to increased vascular stiffness and systolic hypertension in younger individuals, require careful evaluation and management.[99]

Athletes' BP exceeding 220/85 mmHg for males and 200/80 mmHg for females during stress testing warrants further evaluation.[100] It is recommended that athletes use ACE inhibitors or sartans to control their BP.[93] If they are unable to use these drugs, dihydropyridine calcium antagonists should be used instead. Non-dihydropyridines such as diltiazem and verapamil have the same effectiveness as dihydropyridines. Doxazosin may be used for those who are unresponsive to the above drugs, but it is less efficient. Beta-blockers should be avoided due to their systemic effects. If necessary, nebivolol should be used as the first choice.

Pre-participation screening for athletes with AH (Table XV) should include regular risk profile re-evaluations, annual EST, echocardiograms every 3 to 5 years, depending on age, and routine blood and urine tests.[101]

Coronary artery disease

CAD is the primary cause of death and morbidity worldwide, with coronary atherosclerosis often being a common pathological finding in individuals over 35 years of age.[102] Primary prevention plays a crucial role, as CAD can remain asymptomatic for extended periods until a situation arises that may increase oxygen demand and/or cause plaque instability. Secondary prevention is equally vital for enhancing patient prognosis and quality of life.[102] Exercise-induced cardiac ischemic events are infrequent, evidenced by a study of a large North American cohort (10.9 million marathon/half marathon participants) where none of the athletes with CAD showed signs of plaque rupture or thrombosis, indicating a mismatch in oxygen demand rather than physical activity being a direct cause.[103] Conversely, research from a Belgian team suggested that lifelong endurance sports participation could lead

TABLE XV.—*Recommendations for sports participation in subjects with arterial hypertension.*

Recommendation	LOE	COE
Individuals with well-controlled systolic BP and low/moderate CV risk can participate in all types of sports as long as their systolic BP during EST is below 220 mmHg for males and 200 mmHg for females.	I	B
Individuals with normal resting BP but excessive response to exercise, without CV risk factors and signs of organ damage, can participate in all sports as long as their BP values are normal during 24-hour BP monitoring and home- measurements, SBP during EST is below 250 mmHg.	I	C
Individuals with low to moderate CV risk who experience abnormal BP responses to exercise may be able to participate in sports if they can maintain well-controlled BP levels at rest and during exercise through the use of medication. After six months, their ability to participate in sports should be reassessed, and their BP should be checked regularly during this period. Additionally, the individuals must agree to adhere to their medication regimen properly.	II	C
Individuals with moderate global CV risk may be able to participate in sports as long as their BP is well-controlled during maximal EST and 24-hour monitoring, except for sports that involve a great isometric and power component.	II	C
Hypertensive individuals with not well-controlled resting BP (systolic BP> 160 mmHg) must not participate in sports.	III	C
Individuals with systolic BP ≥ 250 mmHg during EST must not participate in sports.	III	C

to less favorable coronary plaque composition compared to maintaining a healthy lifestyle.[104]

Given these insights, a comprehensive pre-evaluation of athletes is now deemed essential, identifying three potential categories (Figure 7):

1. individuals with no history of acute coronary syndrome (ACS-) and no prior coronary revascularization (REV-), labelled as "ACS- REV-";

2. those without a history of ACS but who have undergone revascularization (REV), termed "ACS- REV+";

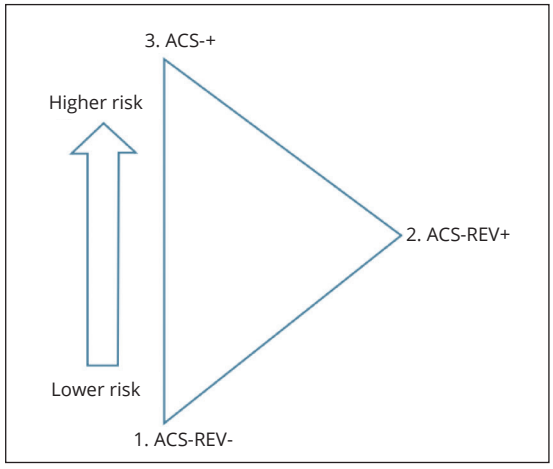


Figure 7.—Different types of athletes: 1) athletes with a negative history of acute coronary syndrome (ACS-) and with no previous coronary revascularization (REV-) considered as ACS-REV- ; 2) athletes with a negative history of acute coronary syndrome (ACS-) and with previous coronary revascularization (REV+) considered as ACS- REV+; 3) athletes with a positive history of acute coronary syndrome (ACS+).

3. individuals with a history of ACS, regardless of revascularization status, categorized as "ACS+".

This classification underscores the importance of tailored assessments and interventions to manage risks associated with CAD in athletes, highlighting the nuanced relationship between extensive physical activity and CV health.

For all athlete categories, the evaluation process typically encompasses:

1. assessment of the type and intensity of the sport;

2. risk assessment using parameters from the European Society of Cardiology, including the "SCORE2/SCORE2-OP to estimate the 10-year risk of CV disease in Europe;[14]

3. clinical and diagnostic evaluations, notably an exercise test;

4. additional diagnostic testing for selected individuals based on initial findings.

ACS- REV-

For "ACS- REV-" athletes, after a thorough risk assessment evaluation utilizing SCORE2/SCORE2-OP models,[105] the maximal EST acts as the "gatekeeper", as previously described.[106] While CCTA remains the gold standard in the diagnostic flow chart for "ACS- REV-" athletes with a positive EST, identifying subjects who might benefit from CCTA in athletes with a negative EST is crucial. In this scenario, successful management of modifiable risk factors could

also influence the decision to use CCTA for some categories.

It is crucial to know whether the subject has performed a previous coronary imaging examination (Table XVI) or not (Table XVII).

ACS- REV+

For athletes with no history of acute coronary syndrome (ACS-) and who have undergone previous coronary revascularization (REV+), a comprehensive assessment is necessary. This assessment should include a maximal EST conducted under optimal medical therapy, an echocardiogram, and ultrasonography of the supra-aortic trunks. While CCTA is considered the gold standard in the diagnostic flow chart for "ACS-REV+" athletes with a positive EST, those with a negative maximal EST may be considered for competitive eligibility in Group A sports, provided all suggested diagnostic tests return negative results and there is vigilant monitoring of disease progression and risk factors. Eligibility for Group A sports can only be considered 12 months after any revascularization procedure and following "double therapy" with acetylsalicylic acid and P2Y12 receptor inhibitors (clopidogrel, prasugrel, ticagrelor). For individuals with a history of stenting in a single coronary vessel, the presence of previous coronary artery imaging can help establish different risk categories (Table XVIII).

ACS+

Athletes with a history of acute coronary syndrome (ACS+) include individuals who have experienced ACS, regardless of whether they have undergone myocardial revascularization (either bypass or coronary angioplasty). Key indicators of an adverse prognosis for these subjects are:

- the extent of coronary disease;
- the presence of ventricular arrhythmias;
- the presence of inducible ischemia;
- the degree of potential LV dysfunction.

Cardiac diagnostic investigations, such as electrocardiograms, echocardiograms, ultrasonography of the supra-aortic trunks, and ambulatory ECG monitoring, along with a maximal EST, should be conducted under optimal medical therapy. However, ACS+ athletes are considered a high-risk population, irrespective of prior coronary imaging examinations or if cardiac diagnostic tests remain substantially unchanged from previous assessments. In selected cases with a lower risk profile, eligibility for Group A sports may be considered (Table XIX), with stringent

TABLE XVI.—*Recommendations for eligibility to competitive sports in athletes with a negative history of acute coronary syndrome (ACS-) and no previous coronary revascularization (REV-) with previous imaging for coronary arteries (CCTA<24 months).*

Recommendation	LOE	COE
Sports eligibility must be denied in: • Athletes documenting 1) fibro-lipid (fibro-fatty) plaque, or 2) plaque progression with arterial expansion (positive remodeling), or 3) micro-calcifications leading to stenosis of >30% in the left main trunk or left anterior descending coronary artery; • Athletes documenting stenosis of >50% in any coronary artery; • Athletes with cardiac stress testing of unequivocally ischemic significance.	III	C
Athletes documenting stenosis between 30-50% in ≥1 coronary artery, in the absence of plaques showing at least 2 of the high-risk characteristics above described (fibro-lipid plaque, positive remodeling and micro-calcifications) may be considered for group-A sports.	II	C
Subjects documenting stenosis <30% in ≥1 coronary artery or documenting negative CCTA can be considered eligible for all competitive sports.	I	C

TABLE XVII.—*Recommendations for eligibility to competitive sports in athletes with a negative history of acute coronary syndrome (ACS-) and no previous coronary revascularization (REV-).*

Recommendation	LOE	COE
Male subjects older than 40 years or female subjects older than 50 years with a negative maximal EST and low-moderate CV risk can be considered eligible for sports activity.	I	C
Subjects with negative maximal EST and very high CV risk should consider CCTA according to Figure 4 recommendation.	II	C

TABLE XVIII.—*Recommendations for eligibility to competitive sports in athletes with a negative history of acute coronary syndrome (ACS-) and previous coronary revascularization (REV+).*

Recommendation	LOE	COE
Sports eligibility must be denied in: • Subjects with previous stenting involving the left main trunk or coronary bifurcation; • Subjects with a previous stenting in >1 coronary artery; • Subjects documenting other stenosis of >50% in any coronary artery; • Subjects documenting any other fibro-lipid plaque >30%, with positive remodeling and micro-calcifications.	III	C
Subjects with a previous single vessel stenting (not involving left main trunk or coronary bifurcation) and documenting other stenoses of 30-50% in one or more coronary arteries in the absence of plaques showing at least 2 of the high-risk characteristics above described (fibro-lipid plaque, positive remodeling and micro-calcifications) may be considered for group-A sports, after performing echocardiogram and ultrasonography of supra-aortic trunks.	II	C
Subjects with a previous single vessel stenting (not involving left main trunk or coronary bifurcation) and documenting stenosis of 30% in the presence of plaques showing at least 2 of the high-risk characteristics above described (fibro-lipid plaque, positive remodeling and micro-calcifications) may be considered as case by case according to the type of sport, after performing echocardiogram and ultrasonography of supra-aortic trunks.	II	C

TABLE XIX.—*Recommendations for eligibility to competitive sports in athletes with a history of acute coronary syndrome (ACS+).*

Recommendation	LOE	COE
Sports eligibility must be denied in: • Subjects with chronotropic incompetence or abnormal BP response during EST; • Subjects with angina, early detection of myocardial ischemia in resting electrocardiogram, or silent myocardial ischemia at low threshold; • Subjects with documented ventricular arrhythmias; • Subjects with EF <50%; • Subjects with a history of cardiac arrest; • Subjects with a diagnosis of heart failure.	III	C
Subjects with none of the characteristics listed above may be considered for group-A sports based on a case-by-case assessment.	II	C

disease progression and risk factors monitoring. Such eligibility can only be granted 12 months after any revascularization procedure and following "double therapy" with acetylsalicylic acid and currently available P2Y12 receptor inhibitors (clopidogrel, prasugrel, ticagrelor).

The positive impact of sports on CAD is increasingly recognized. Nowadays, a carefully tailored and highly personalized exercise prescription holds a Class I indication with a level of evidence A for the secondary prevention of CAD.[107] Additionally, such an exercise regimen can be instrumental in preventing comorbidities and enhancing personal well-being.[108-110] This evaluation should be conducted in experienced centers. Individuals with CAD may participate in structured physical exercise if risk factors and symptoms are properly managed and regular clinical monitoring is maintained.

Cardiological competitive sports eligibility in the paralympic athlete

Pre-participation screening in paralympic athletes: the added value of the Italian experience

Since 1993, Italian regulations have required pre-participation screening for Paralympic athletes (PAs), classifying sports into Group A and Group B based on the level of muscular and cardiorespiratory engagement, each demanding specific medical examinations. To date, pre-participation screening has uncovered a higher prevalence of CV abnormalities in PAs compared to their able-bodied counterparts (12% *vs.* 3.9%, with AH accounting for one-third of these cases), and a significant (2%) prevalence of conditions associated with the risk of SCD.[111]

Motor impairments, especially those resulting from spinal cord injury (SCI), often lead to a sed-

entary lifestyle, contributing to reduced exercise capacity, sarcopenia, osteoporosis, chronic inflammation, dyslipidemia, and insulin resistance, all of which elevate CV risk.[112] However, engaging in sports with medium to high CV demands has been shown to offer protective benefits against CV events and is particularly encouraged for individuals with SCI and lower limb amputations.[113]

A thorough clinical evaluation, including 12-lead rest and EST with protocols specifically adapted to each athlete's impairment, is crucial for identifying conditions that could lead to SCD and determining the need for further examination. The goal of the EST is to reach the highest possible HR, considering the athlete's specific impairment. In the presence of abnormalities or heart disease, PAs are evaluated based on the same criteria as able-bodied athletes.

There is a consensus to adopt a more permissive approach toward eligibility for Group A sports, recognizing the important role of sports in social integration and psycho-physical well-being. For Group B sports, eligibility decisions for PAs with heart disease must adhere to the guidelines for able-bodied athletes, with the final judgment in individual cases left to sports medicine facilities that are highly experienced in the medical assessment of PAs.

Pathophysiology and exercise-induced CV adaptations in paralympic athletes

CV changes in athletes with SCI include alterations in CV reflexes and hemodynamics, leading to decreased venous return, preload, and afterload.[114] These athletes may experience cardiac hypotrophy, characterized by reduced stroke volume, smaller mass, and size of the cardiac chambers – similar to changes observed in able-bodied individuals after prolonged bed rest – which results in a hypokinetic circulation marked by reductions in cardiac output, mean arterial pressure, and VO_{2max}.[115] Additionally, SCI athletes may encounter alterations in sweating and vasodilation, impairing thermoregulation and increasing the risk of hyperthermia during exercise.

Exercise is known to counteract the reduction in cardiopulmonary capacity caused by SCI,[116] but the attainable VO2max is influenced by several factors, such as the level and severity of the injury and the type of sport practiced.[117] While non-SCI athletes may exhibit cardiac adaptations similar to those of able-bodied individuals, the extent of training-induced CV adaptations in SCI athletes remains uncertain.[111] Distinguishing between physiological and pathological conditions presents challenges and necessitates specialized expertise in the pathophysiology of these athletes. Consequently, CPET is recommended for all high-level PAs, particularly when differentiating between physiological and pathological conditions during pre-participation screening.

Disease-specific cardiological issues

The loss of sympathetic control in quadriplegic individuals leads to sinus bradycardia at rest and orthostatic hypotension during postural changes. During physical activity, these athletes exhibit a reduced increase in HR, vasodilation in the muscles being exercised, and an absence of vasoconstriction in areas not engaged in the exercise, potentially leading to exertional hypotension, characterized by general malaise, cold sweats, and episodes of pre-syncope or syncope. These symptoms may diminish over time with regular training.

Additionally, individuals with SCI may experience autonomic dysreflexia (AD), a condition arising from uncontrolled sympathetic nervous system discharge triggered by stimuli below the lesion level in injuries at or above T6. AD is marked by a sudden and significant increase in BP, manifesting symptoms such as headache, arrhythmias, flushing, and sweating, and can escalate to a hypertensive crisis, AF, or cerebral hemorrhage. Athletes might intentionally induce AD, using it as a form of "boosting" to enhance cardiac output and performance, a practice that needs immediate recognition and treatment (Table XX).

For a comprehensive assessment, an echocardiogram is advised at the initial evaluation for athletes in specific categories:

1. individuals with Down Syndrome, due to a high prevalence of congenital heart disease;[118]

2. those with tuberous sclerosis, given the association with multiple myocardial rhabdomyomas;[119]

3. athletes with motor impairments from neuromuscular diseases, like muscular dystrophies

TABLE XX.—*Recommendations for pre-participation screening in Paralympic athletes.*

Pre-participation screening in Paralympic athletes is even more important than in able-bodied athletes due to the underlying pathology, the CV risk and the high mean age of the athletes involved.
Pre-participation screening should consist of a clinical examination, a 12-lead resting ECG and an EST with protocols and modalities tailored to the athlete's specific impairment. A stress test should be performed to achieve the highest possible HR, considering the underlying pathology.
In case of suspicion, second-level tests must be performed according to the disease-specific indications provided for able-bodied athletes.
CPET should be performed in all high-level athletes and every case of doubt between physiological and pathological conditions during pre-participation screening.
The execution of a color-Doppler echocardiography in Paralympic athletes treated with antiblastic therapy (in particular with anthracyclines) is required for competitive sport clearance.
Athletes with Down Syndrome and tuberous sclerosis need to undergo an echocardiogram at least at the first medical assessment.

or metabolic myopathies, which often involve myocardial dysfunction;

4. amputees due to bone tumors, who may develop anthracycline cardiomyopathy, often clinically silent, warranting annual echocardiographic monitoring.

Special attention is required for bilateral congenital sensorineural deafness to exclude Jervell and Lange-Nielsen Syndrome, a form of autosomal recessive LQT Syndrome.

Recreational physical activity in patients with at-risk CV diseases

Although diagnosing certain CV diseases might deem competitive sports participation incompatible, this does not mean that patients should become sedentary. Indeed, there is no clear evidence that low-to-moderate intensity physical activity increases the risk of arrhythmias or worsens the disease phenotype of CV diseases, while there are undoubted benefits for physical and mental health and social inclusion. Patients should be evaluated in a center of proven experience to receive an individualized exercise prescription based on the disease phenotype, the clinical condition, including the possible presence of arrhythmias and/or an ICD, the current drug therapy and the individual definition of the work capacity through EST. Further diagnostic and functional tests to provide a tailored exercise prescription should be performed, including CPET,[120, 121] particularly in patients with specific conditions such as cardiomyopathies,[122] where an appropriate definition of the exercise-intensity domains is essential.[123, 124] For these patients, excluding those with severely limiting symptoms, regular physical activity (at least 150 minutes per week of light-moderate/moderate-intensity aerobic exercise or at least 6000 steps per day) is recommended in any case. In addition, a cardiological follow-up at least once a year is recommended for all subjects with at-risk CV diseases and for redosing and remodulating exercise prescriptions for patients who are engaged in an exercise program.

References

1. Biffi A, Delise P, Zeppilli P, Giada F, Pelliccia A, Penco M, *et al.*; Italian Society of Sports Cardiology and Italian Sports Medicine Federation. Italian cardiological guidelines for sports eligibility in athletes with heart disease: part 1. J Cardiovasc Med (Hagerstown) 2013;14:477–99.

2. Biffi A, Delise P, Zeppilli P, Giada F, Pelliccia A, Penco M, *et al.*; Italian Society of Sports Cardiology and Italian Sports Medicine Federation. Italian cardiological guidelines for sports eligibility in athletes with heart disease: part 2. J Cardiovasc Med (Hagerstown) 2013;14:500–15.

3. Delise P, Mos L, Sciarra L, Basso C, Biffi A, Cecchi F, *et al.* Italian Cardiological Guidelines (COCIS) for Competitive Sport Eligibility in athletes with heart disease: update 2020. J Cardiovasc Med (Hagerstown) 2021;22:874–91.

4. Pelliccia A, Sharma S, Gati S, Bäck M, Börjesson M, Caselli S, *et al.*; ESC Scientific Document Group. 2020 ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease. Eur Heart J 2021;42:17–96.

5. Palermi S, Vecchiato M, Saglietto A, Niederseer D, Oxborough D, Ortega-Martorell S, *et al.* Unlocking the potential of artificial intelligence in sports cardiology: does it have a role in evaluating athlete's heart? Eur J Prev Cardiol 2024;31:470–82.

6. Graziano F, Schiavon M, Cipriani A, Savalla F, De Gaspari M, Bauce B, *et al.* Causes of sudden cardiac arrest and death and the diagnostic yield of sport preparticipation screening in children. Br J Sports Med 2024;58:255–60.

7. Sarto P, Zorzi A, Merlo L, Vessella T, Pegoraro C, Giorgiano F, *et al.* Value of screening for the risk of sudden cardiac death in young competitive athletes. Eur Heart J 2023;44:1084–92.

8. Corrado D, Basso C, Schiavon M, Thiene G. Screening for hypertrophic cardiomyopathy in young athletes. N Engl J Med 1998;339:364–9.

9. Palermi S, Cavarretta E, D'Ascenzi F, Castelletti S, Ricci F, Vecchiato M, *et al.* Athlete's Heart: A Cardiovascular Step-By-Step Multimodality Approach. Rev Cardiovasc Med 2023;24:151.

10. D'Andrea A, Sperlongano S, Russo V, D'Ascenzi F, Benfari G, Renon F, *et al.* The Role of Multimodality Imaging in Athlete's Heart Diagnosis: Current Status and Future Directions. J Clin Med 2021;10:10.

11. Sharma S, Drezner JA, Baggish A, Papadakis M, Wilson MG, Prutkin JM, *et al.* International recommendations for electrocardiographic interpretation in athletes. Eur Heart J 2018;39:1466–80.

12. Keen EN, Sloan AW. Observations on the Harvard step test. J Appl Physiol 1958;13:241–3.

13. Sirico F, Fernando F, Di Paolo F, Adami PE, Signorello MG, Sannino G, *et al.* Exercise stress test in apparently healthy individuals - where to place the finish line? The Ferrari corporate wellness programme experience. Eur J Prev Cardiol 2019;26:731–8.

14. SCORE2 working group and ESC Cardiovascular risk collaboration. SCORE2 risk prediction algorithms: new models to estimate 10-year risk of cardiovascular disease in Europe. Eur Heart J 2021;42:2439–54.

15. Palermi S, Sperlongano S, Mandoli GE, Pastore MC, Lisi M, Benfari G, *et al.*; Echocardiography Working Group of the Italian Society of Cardiology (SIC). Exercise Stress Echocardiography in Athletes: Applications, Methodology, and Challenges. J Clin Med 2023;12:7678.

16. Andreini D, Magnoni M, Conte E, Masson S, Mushtaq S, Berti S, *et al.*; CAPIRE Investigators. Coronary Plaque Features on CTA Can Identify Patients at Increased Risk of Cardiovascular Events. JACC Cardiovasc Imaging 2020;13:1704–17.

17. Motoyama S, Ito H, Sarai M, Kondo T, Kawai H, Nagahara Y, *et al.* Plaque Characterization by Coronary Computed Tomography Angiography and the Likelihood of Acute Coronary Events in Mid-Term Follow-Up. J Am Coll Cardiol 2015;66:337–46.

18. Bergström G, Rosengren A, Bacsovics Brolin E, Brandberg J, Cederlund K, Engström G, *et al.* Body weight at age 20 and in midlife is more important than weight gain for coronary atherosclerosis: results from SCAPIS. Atherosclerosis 2023;373:46–54.

19. Doutreleau S, Pistea C, Lonsdorfer E, Charloux A. Exercise-induced second-degree atrioventricular block in endurance athletes. Med Sci Sports Exerc 2013;45:411–4.

20. Rezazadeh S, Duff HJ. Genetic Determinants of Hereditary Bradyarrhythmias: A Contemporary Review of a Diverse Group of Disorders. Can J Cardiol 2017;33:758–67.

21. Matelot D, Schnell F, Khodor N, Endjah N, Kervio G, Carrault G, *et al.* Does Deep Bradycardia Increase the Risk of Arrhythmias and Syncope in Endurance Athletes? Int J Sports Med 2016;37:792–8.

22. Chiu SN, Lin LY, Wang JK, Lu CW, Chang CW, Lin MT, *et al.* Long-term outcomes of pediatric sinus bradycardia. J Pediatr 2013;163:885–9.e1.

23. Kim JH, Baggish AL. Electrocardiographic right and left bundle branch block patterns in athletes: prevalence, pathology, and clinical significance. J Electrocardiol 2015;48:380–4.

24. Calò L, Della Bona R, Martino A, Crescenzi C, Panattoni G, d'Amati G, *et al.* Left Posterior Fascicular Block and Increased Risk of Sudden Cardiac Death in Young People. J Am Coll Cardiol 2021;77:1143–5.

25. Brugada J, Katritsis DG, Arbelo E, Arribas F, Bax JJ, Blomström-Lundqvist C, *et al.*; ESC Scientific Document Group. 2019 ESC Guidelines for the management of patients with supraventricular tachycardiaThe Task Force for the management of patients with supraventricular tachycardia of the European Society of Cardiology (ESC). Eur Heart J 2020;41:655–720.

26. Ofman P, Khawaja O, Rahilly-Tierney CR, Peralta A, Hoffmeister P, Reynolds MR, *et al.* Regular physical activity and risk of atrial fibrillation: a systematic review and meta-analysis. Circ Arrhythm Electrophysiol 2013;6:252–6.

27. Goudis CA, Ntalas IV, Ketikoglou DG. Atrial Fibrillation in Athletes. Cardiol Rev 2015;23:247–51.

28. Turagam MK, Flaker GC, Velagapudi P, Vadali S, Alpert MA. Atrial Fibrillation In Athletes: Pathophysiology, Clinical Presentation, Evaluation and Management. J Atr Fibrillation 2015;8:1309.

29. Palamà Z, Robles AG, Paoletti M, Nesti M, De Ruvo E, Scarà A, *et al.* Long-Term Follow-Up In Paroxysmal Atrial Fibrillation Patients With Documented Isolated Trigger. Front Cardiovasc Med 2023;10:1115328.

30. Obeyesekere MN, Leong-Sit P, Massel D, Manlucu J, Modi S, Krahn AD, *et al.* Risk of arrhythmia and sudden death in patients with asymptomatic preexcitation: a meta-analysis. Circulation 2012;125:2308–15.

31. Robles AG, Palamà Z, Pernat A, Gianfrancesco D, Bartolomucci F, Scarà A, *et al.* Intermittent ventricular pre-excitation in symptomatic adults: always a marker of low risk? Pacing Clin Electrophysiol 2023;46:1049–55.

32. Sawyer TJ, Cianci M. Wolff-Parkinson-White in a College Athlete: Why Didn't We Pursue an EP Study? JACC Case Rep 2022;9:101531–101531.

33. D'Ascenzi F, Zorzi A, Alvino F, Bonifazi M, Corrado D, Mondillo S. The prevalence and clinical significance of premature ventricular beats in the athlete. Scand J Med Sci Sports 2017;27:140–51.

34. Delise P, Sitta N, Lanari E, Berton G, Centa M, Allocca G, *et al.* Long-term effect of continuing sports activity in competitive athletes with frequent ventricular premature complexes and apparently normal heart. Am J Cardiol 2013;112:1396–402.

35. Haruta D, Akahoshi M, Hida A, Sera N, Imaizumi M, Ichimaru S, *et al.* Prognostic Significance of Premature Ventricular Contractions without Obvious Heart Diseases Determined by Standard 12-Lead Electrocardiography Considering their Morphology. Ann Noninvasive Electrocardiol 2016;21:142–51.

36. Di Florio A, Fusi C, Anselmi F, Cavigli L, Focardi M, Cameli M, *et al.* Clinical management of young competitive athletes with premature ventricular beats: A prospective cohort study. Int J Cardiol 2021;330:59–64.

37. Marine JE. Nonsustained Ventricular Tachycardia in the Normal Heart: Risk Stratification and Management. Card Electrophysiol Clin 2016;8:525–43.

38. Zorzi A, D'Ascenzi F, Andreini D, Castelletti S, Casella M, Cavarretta E, *et al.* Interpretation and management of premature ventricular beats in athletes: an expert opinion document of the Italian Society of Sports Cardiology (SICS-PORT). Int J Cardiol 2023;391:131220.

39. Zorzi A, Perazzolo Marra M, Rigato I, De Lazzari M, Susana A, Niero A, *et al.* Nonischemic Left Ventricular Scar as a Substrate of Life-Threatening Ventricular Arrhythmias and Sudden Cardiac Death in Competitive Athletes. Circ Arrhythm Electrophysiol 2016;9:e004229.

40. Biffi A, Palermi S, D'Ascenzi F, Bonifazi M, Zorzi A, Corrado D. Premature ventricular beats in athletes: to detrain or not to detrain? Br J Sports Med 2024;58:407–8.

41. Sung R, Scheinman M. Spectrum of Fascicular Arrhythmias. Card Electrophysiol Clin 2016;8:567–80.

42. Kean KA, Fitzgerald DJ, Ayers MD, Kean AC. The Ef-

fects of Cardiac Implantable Electronic Device Personal Protective Equipment Use in Patients with Pediatric and Adult Congenital Heart Disease. Pediatr Cardiol 2023;44:1721–5.

43. Reynolds D, Duray GZ, Omar R, Soejima K, Neuzil P, Zhang S, *et al.*; Micra Transcatheter Pacing Study Group. A Leadless Intracardiac Transcatheter Pacing System. N Engl J Med 2016;374:533–41.

44. Sciarra L, Salustri E, Petroni R, Calò L, Delise P, Penco M, *et al.* Sport activity in patients with cardiac implantable electronic devices: evidence and perspectives. J Cardiovasc Med (Hagerstown) 2021;22:335–43.

45. Nesti M, Russo V, Palamà Z, Panchetti L, Garibaldi S, Startari U, *et al.* The Subcutaneous Implantable Cardioverter-Defibrillator: A Patient Perspective. J Clin Med 2023;12:12.

46. Russo V, Ciabatti M, Brunacci M, Dendramis G, Santobuono V, Tola G, *et al.* Opportunities and drawbacks of the subcutaneous defibrillator across different clinical settings. Expert Rev Cardiovasc Ther 2023;21:151–64.

47. Mazzanti A, Kanthan A, Monteforte N, Memmi M, Bloise R, Novelli V, *et al.* Novel insight into the natural history of short QT syndrome. J Am Coll Cardiol 2014;63:1300–8.

48. Anselm DD, Evans JM, Baranchuk A; DD. Brugada phenocopy: A new electrocardiogram phenomenon. World J Cardiol 2014;6:81–6.

49. Sciarra L, Moriya M, Robles AG, Sarubbi B. Type 2 Brugada pattern: more doubts than certainties. Minerva Cardiol Angiol 2021;69:426–8.

50. Sieira J, Ciconte G, Conte G, Chierchia GB, de Asmundis C, Baltogiannis G, *et al.* Asymptomatic Brugada Syndrome: Clinical Characterization and Long-Term Prognosis. Circ Arrhythm Electrophysiol 2015;8:1144–50.

51. Russo V, Caturano A, Guerra F, Migliore F, Mascia G, Rossi A, *et al.*; IBRYD Study Group. Subcutaneous versus transvenous implantable cardioverter-defibrillator among drug-induced type-1 ECG pattern Brugada syndrome: a propensity score matching analysis from IBRYD study. Heart Vessels 2023;38:680–8.

52. Delise P, Allocca G, Sitta N. Risk of sudden death in subjects with Brugada type 1 electrocardiographic pattern and no previous cardiac arrest: is it high enough to justify an extensive use of prophylactic ICD? J Cardiovasc Med (Hagerstown) 2016;17:408–10.

53. Sroubek J, Probst V, Mazzanti A, Delise P, Hevia JC, Ohkubo K, *et al.* Programmed Ventricular Stimulation for Risk Stratification in the Brugada Syndrome: A Pooled Analysis. Circulation 2016;133:622–30.

54. Delise P, Probst V, Allocca G, Sitta N, Sciarra L, Brugada J, *et al.* Clinical outcome of patients with the Brugada type 1 electrocardiogram without prophylactic implantable cardioverter defibrillator in primary prevention: a cumulative analysis of seven large prospective studies. Europace 2018;20(FI1):f77–85.

55. Sieira J, Conte G, Ciconte G, Chierchia GB, Casado-Arroyo R, Baltogiannis G, *et al.* A score model to predict risk of events in patients with Brugada Syndrome. Eur Heart J 2017;38:1756–63.

56. Kawada S, Morita H, Antzelevitch C, Morimoto Y, Nakagawa K, Watanabe A, *et al.* Shanghai Score System for Diagnosis of Brugada Syndrome: Validation of the Score System and System and Reclassification of the Patients. JACC Clin Electrophysiol 2018;4:724–30.

57. Sieira J, Conte G, Ciconte G, de Asmundis C, Chierchia GB, Baltogiannis G, *et al.* Prognostic value of programmed electrical stimulation in Brugada syndrome: 20 years experience. Circ Arrhythm Electrophysiol 2015;8:777–84.

58. Mascia G, Brugada J, Arbelo E, Porto I. Athletes and suspected catecholaminergic polymorphic ventricular tachycardia: awareness and current knowledge. J Cardiovasc Electrophysiol 2023;34:2095–101.

59. Bay A, Dellborg M, Berghammer M, Sandberg C, Engström G, Moons P, *et al.* Patient reported outcomes are associated with physical activity level in adults with congenital heart disease. Int J Cardiol 2017;243:174–9.

60. Budts W, Pieles GE, Roos-Hesselink JW, Sanz de la Garza M, D'Ascenzi F, Giannakoulas G, *et al.* Recommendations for participation in competitive sport in adolescent and adult athletes with Congenital Heart Disease (CHD): position statement of the Sports Cardiology & Exercise Section of the European Association of Preventive Cardiology (EAPC), the European Society of Cardiology (ESC) Working Group on Adult Congenital Heart Disease and the Sports Cardiology, Physical Activity and Prevention Working Group of the Association for European Paediatric and Congenital Cardiology (AEPC). Eur Heart J 2020;41:4191–9.

61. Palermi S, Serio A, Vecchiato M, Sirico F, Gambardella F, Ricci F, *et al.* Potential role of an athlete-focused echocardiogram in sports eligibility. World J Cardiol 2021;13:271–97.

62. Di Salvo G, Miller O, Babu Narayan S, Li W, Budts W, Valsangiacomo Buechel ER, *et al.*; 2016–2018 EACVI Scientific Documents Committee. Imaging the adult with congenital heart disease: a multimodality imaging approach-position paper from the EACVI. Eur Heart J Cardiovasc Imaging 2018;19:1077–98.

63. Vecchiato M, Neunhaeuserer D, Fabris M, Aghi A, Quinto G, Battista F, *et al.* The impact of the COVID-19 pandemic on functional capacity in a population of young athletes: should we expect long-time consequences? J Sports Med Phys Fitness 2023;63:828–34.

64. Vecchiato M, Ermolao A, Zanardo E, Battista F, Ruvoletto G, Palermi S, *et al.* Overshoot of the Respiratory Exchange Ratio during Recovery from Maximal Exercise Testing in Young Patients with Congenital Heart Disease. Children (Basel) 2023;10:10.

65. Dallaire F, Wald RM, Marelli A. The Role of Cardiopulmonary Exercise Testing for Decision Making in Patients with Repaired Tetralogy of Fallot. Pediatr Cardiol 2017;38:1097–105.

66. Sillesen AS, Vøgg O, Pihl C, Raja AA, Sundberg K, Vedel C, *et al.* Prevalence of Bicuspid Aortic Valve and Associated Aortopathy in Newborns in Copenhagen, Denmark. JAMA 2021;325:561–7.

67. Michelena HI, Corte AD, Evangelista A, Maleszewski JJ, Edwards WD, Roman MJ, *et al.* International Consensus Statement on Nomenclature and Classification of the Congenital Bicuspid Aortic Valve and Its Aortopathy, for Clinical, Surgical, Interventional and Research Purposes. Radiol Cardiothorac Imaging 2021;3:e200496.

68. Balistreri CR, Forte M, Greco E, Paneni F, Cavarretta E, Frati G, *et al.* An overview of the molecular mechanisms underlying development and progression of bicuspid aortic valve disease. J Mol Cell Cardiol 2019;132:146–53.

69. D'Ascenzi F, Valentini F, Anselmi F, Cavigli L, Bandera F, Benfari G, *et al.*; Working Group of Echocardiography of the Italian Society of Cardiology (SIC). Bicuspid aortic valve and sports: from the echocardiographic evaluation to the eligibility for sports competition. Scand J Med Sci Sports 2021;31:510–20.

70. Zeppilli P, Bianco M, Gervasi SF, Cammarano M, Monti R, Sollazzo F, *et al.* Congenital coronary artery anomalies in sports medicine. Why to know them. Clin Cardiol 2023;46:1038–48.

71. Donati F, Guicciardi C, Lodi E, Fernando F, Palermi S, Modena MG, *et al.* Echocardiography in the preparticipation

screening: an old topic revisited. J Cardiovasc Med (Hagerstown) 2023;24:297–301.

72. Chatrath N, Papadakis M. Physical activity and exercise recommendations for patients with valvular heart disease. Heart 2022;108:1938–44.

73. Frommelt P, Lopez L, Dimas VV, Eidem B, Han BK, Ko HH, *et al.* Recommendations for Multimodality Assessment of Congenital Coronary Anomalies: A Guide from the American Society of Echocardiography: Developed in Collaboration with the Society for Cardiovascular Angiography and Interventions, Japanese Society of Echocardiography, and Society for Cardiovascular Magnetic Resonance. J Am Soc Echocardiogr 2020;33:259–94.

74. Figliozzi S, Georgiopoulos G, Lopes PM, Bauer KB, Moura-Ferreira S, Tondi L, *et al.* Myocardial Fibrosis at Cardiac MRI Helps Predict Adverse Clinical Outcome in Patients with Mitral Valve Prolapse. Radiology 2023;306:112–21.

75. Cavarretta E, Peruzzi M, Versaci F, Frati G, Sciarra L. How to manage an athlete with mitral valve prolapse. Eur J Prev Cardiol 2021;28:1110–7.

76. Arbelo E, Protonotarios A, Gimeno JR, Arbustini E, Barriales-Villa R, Basso C, *et al.*; ESC Scientific Document Group. 2023 ESC Guidelines for the management of cardiomyopathies. Eur Heart J 2023;44:3503–626.

77. Bryde R, Martinez MW, Emery MS. Exercise recommendations for patients with hypertrophic cardiomyopathy. Prog Cardiovasc Dis 2023;80:53–9.

78. Pelliccia A, Caselli S, Pelliccia M, Musumeci MB, Lemme E, Di Paolo FM, *et al.* Clinical outcomes in adult athletes with hypertrophic cardiomyopathy: a 7-year follow-up study. Br J Sports Med 2020;54:1008–12.

79. Basu J, Finocchiaro G, Jayakumar S, Schönfeld J, MacLachlan H, Miles C, *et al.* Impact of Exercise on Outcomes and Phenotypic Expression in Athletes With Nonobstructive Hypertrophic Cardiomyopathy. J Am Coll Cardiol 2022;80:1498–500.

80. Lampert R, Olshansky B, Heidbuchel H, Lawless C, Saarel E, Ackerman M, *et al.* Safety of Sports for Athletes With Implantable Cardioverter-Defibrillators: Long-Term Results of a Prospective Multinational Registry. Circulation 2017;135:2310–2.

81. Lampert R, Ackerman MJ, Marino BS, Burg M, Ainsworth B, Salberg L, *et al.*; LIVE Consortium. Vigorous Exercise in Patients With Hypertrophic Cardiomyopathy. JAMA Cardiol 2023;8:595–605.

82. Corrado D, Link MS, Calkins H. Arrhythmogenic Right Ventricular Cardiomyopathy. N Engl J Med 2017;376:61–72.

83. Corrado D, Perazzolo Marra M, Zorzi A, Beffagna G, Cipriani A, Lazzari M, *et al.* Diagnosis of arrhythmogenic cardiomyopathy: the Padua criteria. Int J Cardiol 2020;319:106–14.

84. Graziano F, Cipriani A, Balla D, Bondarev S, Marra MP, Bauce B, *et al.* Evolving spectrum of arrhythmogenic cardiomyopathy: Implications for Sports Cardiology. Clin Cardiol 2023;46:1072–81.

85. Petersen SE, Jensen B, Aung N, Friedrich MG, McMahon CJ, Mohiddin SA, *et al.* Excessive Trabeculation of the Left Ventricle: JACC: Cardiovascular Imaging Expert Panel Paper. JACC Cardiovasc Imaging 2023;16:408–25.

86. Gati S, Chandra N, Bennett RL, Reed M, Kervio G, Panoulas VF, *et al.* Increased left ventricular trabeculation in highly trained athletes: do we need more stringent criteria for the diagnosis of left ventricular non-compaction in athletes? Heart 2013;99:401–8.

87. Bryde RE, Cooper LT Jr, Fairweather D, Di Florio DN, Martinez MW. Exercise After Acute Myocarditis: When and How to Return to Sports. Cardiol Clin 2023;41:107–15.

88. Imazio M, Gaita F, LeWinter M. Evaluation and Treatment of Pericarditis: A Systematic Review. JAMA 2015;314:1498–506.

89. Brunetti G, Cipriani A, Perazzolo Marra M, De Lazzari M, Bauce B, Calore C, *et al.* Role of Cardiac Magnetic Resonance Imaging in the Evaluation of Athletes with Premature Ventricular Beats. J Clin Med 2022;11:11.

90. Szabo L, Brunetti G, Cipriani A, Juhasz V, Graziano F. Hirschberg, K, *et al.* Certainties and Uncertainties of Cardiac Magnetic Resonance Imaging in Athletes. J Cardiovasc Dev Dis 2022;9.

91. di Gioia CR, Giordano C, Cerbelli B, Pisano A, Perli E, De Dominicis E, *et al.* Nonischemic left ventricular scar and cardiac sudden death in the young. Hum Pathol 2016;58:78–89.

92. Niebauer J, Börjesson M, Carre F, Caselli S, Palatini P, Quattrini F, *et al.* Recommendations for participation in competitive sports of athletes with arterial hypertension: a position statement from the sports cardiology section of the European Association of Preventive Cardiology (EAPC). Eur Heart J 2018;39:3664–71.

93. Mancia G, Kreutz R, Brunström M, Burnier M, Grassi G, Januszewicz A, *et al.* 2023 ESH Guidelines for the management of arterial hypertension The Task Force for the management of arterial hypertension of the European Society of Hypertension: Endorsed by the International Society of Hypertension (ISH) and the European Renal Association (ERA). J Hypertens 2023;41:1874–2071.

94. Stergiou GS, Palatini P, Parati G, O'Brien E, Januszewicz A, Lurbe E, *et al.*; European Society of Hypertension Council and the European Society of Hypertension Working Group on Blood Pressure Monitoring and Cardiovascular Variability. 2021 European Society of Hypertension practice guidelines for office and out-of-office blood pressure measurement. J Hypertens 2021;39:1293–302.

95. Lurbe E, Agabiti-Rosei E, Cruickshank JK, Dominiczak A, Erdine S, Hirth A, *et al.* 2016 European Society of Hypertension guidelines for the management of high blood pressure in children and adolescents. J Hypertens 2016;34:1887–920.

96. Perrone-Filardi P, Coca A, Galderisi M, Paolillo S, Alpendurada F, de Simone G, *et al.* Noninvasive cardiovascular imaging for evaluating subclinical target organ damage in hypertensive patients: a consensus article from the European Association of Cardiovascular Imaging, the European Society of Cardiology Council on Hypertension and the European Society of Hypertension. J Hypertens 2017;35:1727–41.

97. Schultz MG, Otahal P, Cleland VJ, Blizzard L, Marwick TH, Sharman JE. Exercise-induced hypertension, cardiovascular events, and mortality in patients undergoing exercise stress testing: a systematic review and meta-analysis. Am J Hypertens 2013;26:357–66.

98. Biffi A, Gallo G, Fernando F, Sirico F, Signorello MG, De Martino L, *et al.* Relationship Between Cardiorespiratory Fitness, Baseline Blood Pressure and Hypertensive Response to Exercise in the Ferrari Corporate Population. High Blood Press Cardiovasc Prev 2022;29:81–8.

99. Palatini P, Rosei EA, Avolio A, Bilo G, Casiglia E, Ghiadoni L, *et al.* Isolated systolic hypertension in the young: a position paper endorsed by the European Society of Hypertension. J Hypertens 2018;36:1222–36.

100. Caselli S, Vaquer Sequì A, Lemme E, Quattrini F, Milan A, D'Ascenzi F, *et al.* Prevalence and Management of Systemic Hypertension in Athletes. Am J Cardiol 2017;119:1616–22.

101. Keller K, Stelzer K, Ostad MA, Post F. Impact of exag-

gerated blood pressure response in normotensive individuals on future hypertension and prognosis: systematic review according to PRISMA guideline. Adv Med Sci 2017;62:317–29.

102. Knuuti J, Wijns W, Saraste A, Capodanno D, Barbato E, Funck-Brentano C, *et al.*; ESC Scientific Document Group. 2019 ESC Guidelines for the diagnosis and management of chronic coronary syndromes. Eur Heart J 2020;41:407–77.

103. Kim JH, Malhotra R, Chiampas G, d'Hemecourt P, Troyanos C, Cianca J, *et al.*; Race Associated Cardiac Arrest Event Registry (RACER) Study Group. Cardiac arrest during long-distance running races. N Engl J Med 2012;366:130–40.

104. De Bosscher R, Dausin C, Claus P, Bogaert J, Dymarkowski S, Goetschalckx K, *et al.* Endurance exercise and the risk of cardiovascular pathology in men: a comparison between lifelong and late-onset endurance training and a non-athletic lifestyle - rationale and design of the Master@Heart study, a prospective cohort trial. BMJ Open Sport Exerc Med 2021;7:e001048.

105. Visseren FL, Mach F, Smulders YM, Carballo D, Koskinas KC, Bäck M, *et al.*; ESC National Cardiac Societies; ESC Scientific Document Group. 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice. Eur Heart J 2021;42:3227–337.

106. Löllgen H, Leyk D. Exercise Testing in Sports Medicine. Dtsch Arztebl Int 2018;115:409–16.

107. Zhao W, Yin Y, Cao H, Wang Y. Exercise Improves Endothelial Function via the lncRNA MALAT1/miR-320a Axis in Obese Children and Adolescents. Cardiol Res Pract 2021;2021:8840698.

108. Palermi S, Bragazzi NL, Cular D, Ardigò LP, Padulo J. How Chest Press-Based Exercises Can Alleviate the Burden of Cardiovascular Diseases. Human Mov 2022;23:88–98.

109. Vecchiato M, Quinto G, Palermi S, Foccardi G, Mazzucato B, Battista F, *et al.* Are Gyms a Feasible Setting for Exercise Training Interventions in Patients with Cardiovascular Risk Factors? An Italian 10-Years Cross-Sectional Survey Comparison. Int J Environ Res Public Health 2022;19:19.

110. Compagno S, Palermi S, Pescatore V, Brugin E, Sarto M, Marin R, *et al.* Physical and psychological reconditioning in long COVID syndrome: results of an out-of-hospital exercise and psychological - based rehabilitation program. Int J Cardiol Heart Vasc 2022;41:101080.

111. Pelliccia A, Quattrini FM, Cavarretta E, Squeo MR, Adami PE, Di Paolo FM, *et al.* Physiologic and Clinical Features of the Paralympic Athlete's Heart. JAMA Cardiol 2021;6:30–9.

112. Bauman WA, Spungen AM. Coronary heart disease in individuals with spinal cord injury: assessment of risk factors. Spinal Cord 2008;46:466–76.

113. Bernardi M, Romano S, Squeo MR, Guerra E, Adami PE, Alviti F, *et al.* Aerobic Fitness Is a Potential Crucial Factor in Protecting Paralympic Athletes with Locomotor Impairments from Atherosclerotic Cardiovascular Risk. Sport Sci Health 2021;17:363–74.

114. Hoffman MD. Cardiorespiratory fitness and training in quadriplegics and paraplegics. Sports Med 1986;3:312–30.

115. West CR, Mills P, Krassioukov AV. Influence of the neurological level of spinal cord injury on cardiovascular outcomes in humans: a meta-analysis. Spinal Cord 2012;50:484–92.

116. Huonker M, Schmid A, Sorichter S, Schmidt-Trucksäb A, Mrosek P, Keul J. Cardiovascular differences between sedentary and wheelchair-trained subjects with paraplegia. Med Sci Sports Exerc 1998;30:609–13.

117. Baumgart JK, Brurok B, Sandbakk Ø. Peak oxygen uptake in Paralympic sitting sports: A systematic literature review, meta- and pooled-data analysis. PLoS One 2018;13:e0192903.

118. Bergström S, Carr H, Petersson G, Stephansson O, Bonamy AK, Dahlström A, *et al.* Trends in Congenital Heart Defects in Infants With Down Syndrome. Pediatrics 2016;138:138.

119. Umeoka S, Koyama T, Miki Y, Akai M, Tsutsui K, Togashi K. Pictorial review of tuberous sclerosis in various organs. Radiographics 2008;28:e32.

120. Anselmi F, Cavigli L, Pagliaro A, Valente S, Mondillo S, Focardi M, *et al.* Cardiopulmonary exercise testing: an essential tool for a tailored exercise prescription in patients with cardiac disease. G Ital Cardiol (Rome) 2021;22:716–26.

121. D'Ascenzi F, Cavigli L, Pagliaro A, Focardi M, Valente S, Cameli M, *et al.* Clinician approach to cardiopulmonary exercise testing for exercise prescription in patients at risk of and with cardiovascular disease. Br J Sports Med 2022;56:1180–7.

122. Cavigli L, Olivotto I, Fattirolli F, Mochi N, Favilli S, Mondillo S, *et al.* Prescribing, dosing and titrating exercise in patients with hypertrophic cardiomyopathy for prevention of comorbidities: ready for prime time. Eur J Prev Cardiol 2021;28:1093–9.

123. Hansen D, Bonné K, Alders T, Hermans A, Copermans K, Swinnen H, *et al.* Exercise training intensity determination in cardiovascular rehabilitation: should the guidelines be reconsidered? Eur J Prev Cardiol 2019;26:1921–8.

124. Anselmi F, Cavigli L, Pagliaro A, Valente S, Valentini F, Cameli M, *et al.* The importance of ventilatory thresholds to define aerobic exercise intensity in cardiac patients and healthy subjects. Scand J Med Sci Sports 2021;31:1796–808.

Conflicts of interest

The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

Authors' contributions

Paolo Zeppilli and Alessandro Biffi equally contributed to the manuscript; Maurizio Casasco and Luigi Sciarra contribute to the manuscript as senior authors. All authors read and approved the final version of the manuscript.

Acknowledgments

The authors acknowledge the following authors who served as contributors: Domenico Accettura, Daniele Andreini, Alberto Anedda, Enrico Ammirati, Marco Bernardi, Umberto Berrettini, Massimiliano Bianco, Valentina Bucciarelli, Mattia Canevari, Mario Carminati, Massimo Chessa, Antonio D'Aleo, Domenico D'Amario, Cristina Degano, Giovanni di Salvo, Fredrick Fernando, Alessandro Frigiola, Salvatore Gervasi[§], Antonio Gianfelici, Francesca Girolami, Ferdinando Iellamo, Massimo Imazio, Giuseppe Inama, Loira Leoni, Massimo Lombardi, Mauro Lo Rito, Riccardo Monti, Iacopo Olivotto, Paolo Palatini, Stefania Paolillo, Maria Penco, Fabio Pigozzi, Marco Pozzi, Claudio Rapezzi[§], Silvio Romano, Patrizio Sarto, Berardo Sarubbi, Gianfranco Sinagra, Antonio Spataro.

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History

Manuscript accepted: July 12, 2024 - Manuscript received: June 17, 2024.