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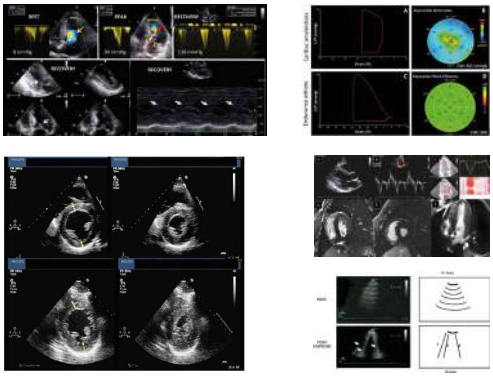

Decoding the Athlete's Heart : The Power of Stress Echocardiography in Cardiac Assessment

Left ventricular hypertrophy and hypertrophic cardiomyopathy in athletes

Amateur athlete, professional athlete : What cardiological evaluation ?

Sport and congenital heart disease : a dangerous connection ?

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Editorial

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Sports cardiology focuses on patients with cardiovascular conditions who wish to return to physical activity, as well as elite athletes who require regular annual monitoring—such as football players who are assigned a PCMA (Pre-Competition Medical Assessment) file by FIFA.

Sport is beneficial to health and should be encouraged for both healthy individuals and those with medical conditions. However, the cardiovascular system, which plays a crucial role in the body's adaptation to physical exertion, can sometimes become the "weak link" in an athlete's life, leading to potentially serious incidents.

The physical stresses and adaptations induced by sport vary across disciplines and activities. Understanding these variations is key to helping each athlete maximize the benefits of their training while minimizing risks to their health.

This issue's articles are dedicated to athletes, both amateur and professional, and offer in-depth coverage of all aspects of their monitoring and the management of various conditions, particularly those related to heart rhythm. Special attention is given to distinguishing between a "sports heart" and hypertrophic cardiomyopathy, as well as preventing sudden cardiac death in sport, which remains a tragic and preventable event.

Pr Nabil Berrada

Sudden death in athletes : How to avoid

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Summary

Sudden death of athletes is generally a public concern, a tragic event for families, sports communities, and the public. It often receives significant media coverage. Statistics are classified by gender, age, ethnicity, and the type of sport practiced. The causes are mainly structural heart diseases and genetics for athletes under 35 year's old, and ischemic origins for the elder ones.
Although silent, scientific societies have come together to agree that screening, including at least a personal and family medical history, an appropriate physical examination, a resting 12-lead ECG, and an echocardiogram, can help detect early warning signs that allow athletes to be classified and monitored according to their risk level. While the number of sudden death incidents remains stable, the implementation of protocols for cardiac arrest management, continuous training of sports medical staff, and proper education on first aid, cardiopulmonary resuscitation (CPR), and the presence of automated defibrillators on-site have significantly improved survival rates

Keywords :
Sudden Cardiac Death (SCD), Athlete Screening, Cardiovascular Risk, Electrocardiogram (ECG), Emergency Action Plan (EAP), Automated External Defibrillators (AEDs), Cardiopulmonary Resuscitation (CPR), Prevention Strategies

Résumé

La mort subite chez les athlètes représente généralement une préoccupation d'ordre public, un événement tragique pour la famille, le public et la communauté sportive, souvent largement médiatisé. Les statistiques varient selon le sexe, l'âge, l'origine ethnique et le type de sport pratiqué. Les causes sont principalement des maladies cardiaques génétiques et structurelles chez les athlètes de moins de 35 ans, et d'origine ischémique chez les athlètes plus âgés.
Bien que silencieuses, les sociétés scientifiques se sont réunies pour convenir qu'un dépistage, incluant au minimum des antécédents médicaux personnels et familiaux, un examen physique approprié, un ECG de repos à 12 dérivations, et une échocardiographie, peut aider à détecter des signes d'alerte précoces permettant de classer et de suivre les athlètes en fonction de leur niveau de risque. Bien que le nombre d'incidents de mort subite reste stable, la mise en place de protocoles de gestion des arrêts cardiaques, la formation continue du personnel médical sportif, ainsi que l'éducation aux gestes de premiers secours, à la réanimation cardio-pulmonaire (RCP) et la présence de défibrillateurs automatiques sur place ont permis d'améliorer considérablement les taux de survie.

Mots clés :
Mort cardiaque subite (MSC), dépistage chez les sportifs, risque cardiovasculaire, électrocardiogramme (ECG), plan d'action d'urgence (PAE), défibrillateurs externes automatisés (DEA), réanimation cardio-pulmonaire (RCP), stratégies de prévention

Introduction

Sudden death in athletes is a rare but alarming event, often raising concerns about the cardiovascular risks of intense physical activity. Although athletes have a lower overall risk of SCD compared to the general population, those with undiagnosed heart conditions, such as coronary artery disease or cardiomyopathies, are at higher risk. In these predisposed individuals, exercise can trigger fatal arrhythmias. (1) (2)

Regular physical exercise significantly reduces the risk of cardiovascular death, but sudden cardiac death (SCD) can still occur, especially during high-intensity sports. To mitigate this risk, pre-participation screening is crucial. Screening typically includes medical history, physical exams, ECGs, and echocardiograms, which help identify athletes with hidden cardiovascular conditions. Countries like Italy have seen reductions in SCD through mandatory ECG screenings.
For older athletes, particularly marathoners, coronary artery disease becomes a key risk factor. These individuals should be

evaluated for symptoms and risk factors, with high-risk participants guided toward supervised exercise programs. Additionally, having resuscitation measures, such as automated external defibrillators (AEDs) and CPR-trained staff at sporting events, is essential for quick response in emergencies.

There is also a growing call for national registries and data collection on SCD in athletes. Such data would help establish guidelines for preventing SCD and improve the safety of both competitive and recreational athletes. (3)

I- Definitions

A 'competitive athlete' is defined as an individual….

- Who participates in an organized team or individual sport,
- Who engages in regular competition against others,
- Who places a high priority or premium on athletic excellence and achievement,
- Who engages in vigorous training in a systematic fashion to achieve all the above goals (5)

Sudden cardiac death in sports and exercise – is cardiac arrest occurring during or within 1h of exercise or sports related activity. (4)

II- Epidemiology and causes of Sudden Cardiac Death (SCD) in Athletes

Sudden cardiac death (SCD) in athletes remains a rare but significant concern, particularly during high-intensity sports.

American College of Cardiology Sports and Exercise Cardiology defines athlete as any individual who engages in routine vigorous physical exercise in the settings of competition, recreation, or occupation. (4)

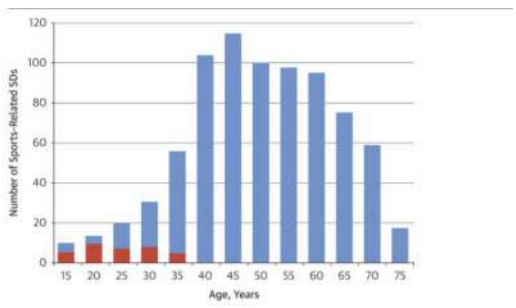
1- Epidemiology

The annual incidence of sports-related SCDs was reported to be 4.6 per 1 million population in a five-year prospective study as compared to 50–100 per million in the general population. (6)

The incidence of SCD in young athletes, particularly those competing in organized sports, is estimated at 1 in 50,000 to 1 in 80,000 athletes annually. These cases are often caused by undiagnosed structural or electrical heart abnormalities, such as hypertrophic cardiomyopathy (HCM) or arrhythmogenic right ventricular cardiomyopathy (ARVC).

In athletes over 35 years old, the leading cause of SCD shifts to coronary artery disease (CAD). A study found that CAD accounts for the majority of SCD cases in this age group, especially in endurance athletes such as marathon runners. Despite being less common in younger athletes, CAD becomes a significant risk as athletes age. (7) (8)

Figure 1 :
Relationship between Age and Sports-Related Sudden Deaths (6)



SCD is more common in males than females, and the risk varies across different sports. High-risk sports include:

Basketball and Football (Soccer): These sports have some of the highest rates of SCD.

Endurance Sports: Long-duration endurance events, such as marathons, pose a higher risk of SCD, especially for athletes over 35 years old with underlying coronary artery disease. Studies indicate that older marathoners face an increased likelihood of cardiac events during or shortly after races.

Post-COVID-19 Impact on SCD Trends

During and after the COVID-19 pandemic, newest studies suggest changes in SCD rates, athletes recovering from COVID-19 seems to be potentially at a higher risk of cardiac complications. There is ongoing research to better understand the long-term effects of the pandemic on cardiovascular health in athletes. (9)

2- Causes of SCD in athletes

Sudden death in athletes typically occurs due to undetected cardiovascular abnormalities. Other causes can include heat stroke, head trauma, and in rare cases, genetic disorders. Sudden cardiac death (SCD) is the most common medical cause of mortality in athletes, particularly in those under the age of 35, It accounts for approximately 75% of all sudden deaths in athletes. For male athletes, the risk is significantly higher, with around 90% of SCD cases occurring in men.In professional sports leagues, such as the NCAA, SCD remains a prominent concern, despite the relatively low overall incidence. (10)

The primary causes of sudden cardiac death (SCD) in athletes are outlined in table 1.

For athletes under the age of 35, inherited cardiac conditions are the most common, with hypertrophic cardiomyopathy (HCM) and anomalous coronary artery origins being the leading causes in the United States. In athletes over 35, the majority of SCD cases are attributed to acquired atherosclerotic coronary artery disease (CAD). (5) (11) Many of these conditions may not show clinical signs prior to the event and may only manifest with sudden death. Although it is challenging to estimate the proportion of athletes experiencing warning symptoms due to the lack of direct accounts, studies suggest that up to 30% of athletes with SCD may have experienced symptoms such as chest pain, shortness of breath, performance decline, palpitations, or episodes of fainting prior to the event. Proper evaluation of these symptoms by sports medicine and cardiology specialists plays a critical role in the medical management of athletes and in preventing SCD. (4)

Table 1 :
Common cardiovascular conditions associated with sudden cardiac death (SCD) in athletes. (12)

Congenital/Genetic	
Structurally Abnormal Heart	**Structurally Normal Heart**
Hypertrophic cardiomyopathy	Congenital long QT syndrome
Arrhythmogenic right ventricular cardiomyopathy	Catecholaminergic polymorphic ventricular tachycardia
Dilated cardiomyopathy	Wolff-Parkinson-White syndrome or other accessory pathway
Other cardiomyopathy (i.e., left ventricular noncompaction)	Brugada syndrome
Congenital anomalies of coronary origin & course	Other ion channelopathies
Aortopathy (i.e., Marfan syndrome & ascending aortic aneurysm/ dissection)	
Valvular heart disease (i.e., congenital aortic stenosis, mitral valve prolapse)	
Acquired	
Structurally Abnormal Heart	**Structurally Normal Heart**
Atherosclerotic coronary artery disease	Commotio cordis
Kawasaki's disease	Acquired long QT (i.e., drug-induced)
Myocarditis	Other substance ingestion or environmental factors (i.e., hypo- or hyperthermia)

III- How to avoid SCD in athletes

1- Pre participation screening (PPS)

Pre-participation screening (PPS) has an important role in prevention of SCD in competitive athletes. The main goal of the cardiovascular PPS is to detect a cardiac disease that can be worsened by intensive sports practice or result in a SCD.

Scientific committees such as the American College of Cardiology, the American Heart Association (AHA), and the European Society of Cardiology (ESC), and sports associations such as the International Olympic Committee (IOC) Medical Commission, Fédération Internationale de Football Association (FIFA) and others recommend a PPS program to provide medical clearance for participation in competitive sports.

All recommendations include questionnaires relating to family and personal history, and a physical examination.

The necessity of a systematic resting 12-lead ECG is no more a matter debate between, the ESC recommends that the first PPS for competitive sports in subjects aged 12–35 includes a resting ECG. This ECG must be repeated every 2 years . In 2014, at the AHA session, 60% of the audience members believed that screening programs should include an ECG. (13) (14) For exemple, FIFA includes a systematic 12-lead ECG and an echocardiogram in the Pre-Competition Medical Assessment.

i- Personal and family history

The primary aim of screening is to detect conditions that increase the risk of sudden cardiac arrest (SCA) or sudden cardiac death (SCD). Key elements of the screening process include personal history, family history, and physical examination findings. Four essential screening questions are suggested : (15)

- History of fainting, or having an unexplained seizure suddenly, especially during exercise or in response to loud noises like doorbells or alarms
- history of chest pain or shortness of breath during exercise
- History of a sydden death or heart problems in the immediate or extended family before age 50, including drownings, unexplained car accidents, or sudden infant death
- Being related to anyone with conditions like hypertrophic cardiomyopathy, Marfan syndrome, long/short QT syndrome, Brugada syndrome, or someone who needed a pacemaker or ICD before age 50

Intensive questioning allows for the early detection of a number of anomalies but may not be sufficient to prevent sudden death. Most symptoms are not pathognomonic of cardiovascular diseases, and the terminology can also vary. For instance, feeling out of breath and experiencing dyspnea, feeling dizzy and having a lipothymia , being hurt in the chest or having a chest pain .. —different presentations of symptoms—can lead to diagnostic uncertainty.

A questionnaire consisting of 14 items has been developed by the AHA, a positive response to one or more items may be deemed sufficient to warrant a referral for a cardiovascular evaluation.(Table 2)

Table 2 :
The 14-Element American Heart Association Recommendations for Preparticipation Screening of competitive athletes (15)

Personal history:
1. Chest pain/discomfort/tightness/pressure related to exertion
2. Unexplained syncope/near-syncope[a]
3. Excessive exertional and unexplained dyspnea/fatigue or palpitations, associated with exercise
4. Previous recognition of a heart murmur
5. Elevated systemic blood pressure
6. Previous restriction from participation in sports
7. Previous testing for the heart, ordered by a physician
Family history:
8. Premature death (sudden and unexpected, or otherwise) before age 50 y attributable to heart disease in ≥1 relative
9. Disability from heart disease in a close relative aged <50 y
10. Hypertrophic or dilated cardiomyopathy, long QT syndrome or other ion channelopathies, Marfan syndrome, or clinically significant arrhythmias; specific knowledge of certain cardiac conditions in family members
Physical examination:
11. Heart murmur[b]
12. Femoral pulses to exclude aortic coarctation
13. Physical stigmata of Marfan syndrome
14. Brachial artery blood pressure (sitting position)[c]
[a]Judged not to be of neurocardiogenic (vasovagal) origin; of particular concern when occurring during or after physical exertion.
[b]Refers to heart murmurs judged likely to be organic and unlikely to be innocent; auscultation should be performed with the patient in both the supine and standing positions (or with the Valsalva maneuver), specifically to identify murmurs of dynamic left ventricular outflow tract obstruction.
[c]Preferably taken in both arms.

ii- Physical examination

The physical examination places a strong emphasis on identifying abnormal findings during cardiac auscultation. These abnormalities may include heart murmurs, particularly those that are diastolic or systolic and have an intensity greater than 2/6. Murmurs that are fixed by respiration or become more pronounced after exercise are of particular concern. Additionally, a systolic click, which can indicate a valvular abnormality, or an irregular heart rhythm are also key findings that may suggest underlying cardiac issues.

Beyond auscultation, the examiner should assess for asymmetrical arterial pulses, especially between the arms and legs, which could indicate aortic coarctation. Checking for differences in blood pressure between both arms, is another important aspect of the examination.

In addition to the cardiovascular focus, the physical exam also includes a detailed evaluation of musculoskeletal and ocular features that may indicate the presence of systemic conditions like Marfan syndrome. Musculoskeletal signs, such as long, hyper-flexible joints or a tall, thin body frame, combined with ocular abnormalities, particularly lens dislocation or other visual disturbances, are characteristic of this genetic disorder, which is associated with an increased risk of cardiovascular complications like aortic aneurysm. Thus, the physical examination aims to comprehensively assess both direct cardiac symptoms and systemic features that may predispose an individual to sudden cardiac events.

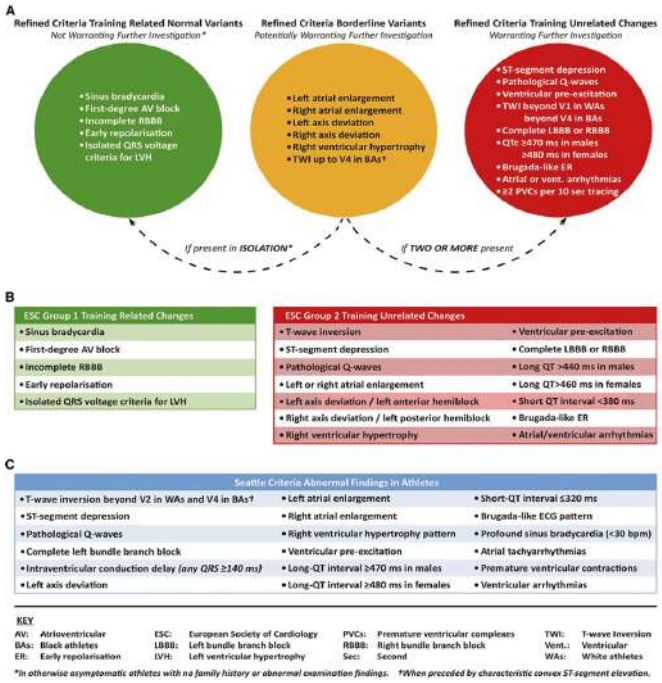
iii- Resting ECG

The athlete's heart is different; vagotonia can cause sinus bradycardia, first-degree AV block, and tall T waves. Increased chamber size can lead to left ventricular hypertrophy and incomplete right bundle branch block. When an athlete's ECG is interpreted using outdated criteria, it can result in many false positives, leading to costly and stressful tests that may not be necessary.

The 2010 European Society of Cardiology (ESC) guidelines for interpreting ECGs in athletes aimed to help distinguish between normal physiological ECG patterns (group 1) and those that may signal cardiac disease (group 2).

While this categorization has enhanced specificity, false-positive rates ranging from 10% to 20% have led to ongoing requests for further refinement. Recently, a collaboration among international experts resulted in the Seattle criteria, which have improved specificity in certain populations.

Figure 2 :
The definition of an abnormal ECG using the (A) refined criteria, (B) European Society of Cardiology (ESC) recommendations, 9 and (C) Seattle criteria. (16)



iv- Echocardiography

Overall, resting transthoracic echocardiography (TTE) remains, for the vast majority of athletes, a secondary diagnostic examination. Its indication should therefore be targeted. However, it is important to note that, legally, TTE has been mandatory since 2004 (decree and government order No. 2004-120 of February 2004, amended in 2006) for high-level athletes (HLA) registered by their federation on an annually updated list. This examination must be performed within 3 months prior to registration on the high-level or promising lists. It should be conducted at least once during an athlete's career after the age of 18 and must be renewed if previously done. Some federations, such as rugby, are even more stringent and require a cardiac ultrasound every 4 years. Professional leagues have their own regulations, with annual TTE for football and every 2 years for cycling, for example.

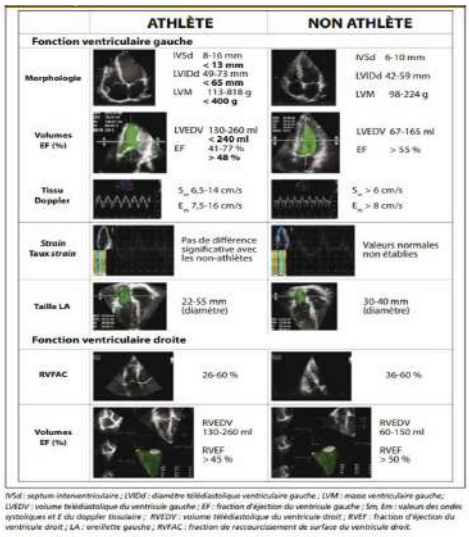
The use of echocardiography clarified the diagnosis in the evaluation of athletes, regardless of ECG findings, Among the conditions assessed, cardiomyopathies ranked highest (average score: 2.79), followed by aortic aneurysm/dilatation (average score: 2.6), bicuspid aortic valve (average score: 2.53), and other valvular heart diseases (average score: 2.08). Additionally, echocardiography can detect conditions such as coronary artery anomalies, myocarditis, ischemic heart disease, and congenital heart diseases when the ECG appears normal.

Table 3 :
List of conditions of which echocardiography may mak the diagnosis in the context of a normal electrocardiogram (17)

Classification of conditions pre-identified by the survey
1. Cardiomyopathies
2. Aortic aneurysm/dilatation
3. Bicuspid aortic valve
4. Other valvular heart disease
Conditions identified by the respondents
Coronary artery abnormalities
Myocarditis
Asymptomatic/silent ischaemic heart disease
Congenital heart disease (including mitral valve prolapse, patent foramen ovale, small patent ductus arteriosus, interatrial and interventricular defects, and subaortic membrane)
Hypertensive cardiomyopathy
Arrhythmias
Pulmonary embolism
Heart failure with preserved left ventricular ejection fraction
Marfan syndrome
Pericardial disease (pericardial effusion and pericarditis)
Atrial myxoma
Endocarditis

Table 4 presents the echocardiographic norms proposed for the athlete's heart compared to that of a sedentary individual. Here are the main measurements and their values: (18)

Table 4 :
Normes échocardiographiques proposées pour le cœur d'athlète en comparaison avec le sédentaire (18)



These steps will allow athletes to be classified according to their risk, identify those who will need further investigation, and those whose follow-up will need to be specific.

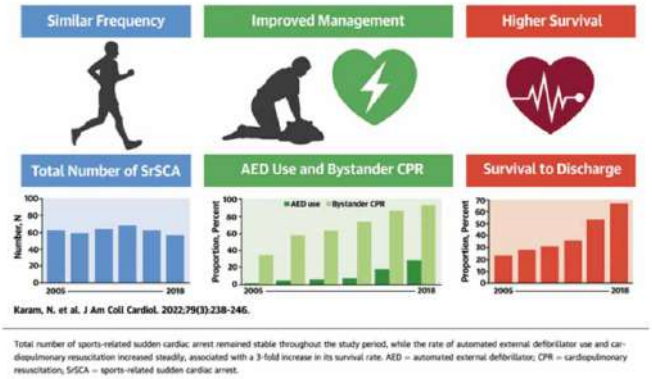
2- Comprehensive Field Strategy for Preventing Sudden Cardiac Death

To effectively prevent sudden cardiac death (SCD) on the field, it's crucial to develop a detailed Emergency Action Plan (EAP) outlining the steps to take during a cardiac arrest. This plan should designate a trained emergency team, including coaches, medical staff, and trainers certified in CPR and the use of Automated External Defibrillators (AEDs).

AEDs must be easily accessible at all training sessions and competitions, with staff regularly trained in their operation and AEDs routinely inspected for functionality. Additionally, mandatory CPR certification for all key personnel and frequent emergency response drills will ensure swift and effective reactions in real-life scenarios. Educating athletes, coaches, and staff to recognize early warning signs of cardiac distress, such as dizziness, chest pain, or palpitations, will further enhance preparedness.

Immediate response protocols are essential when an athlete shows signs of cardiac arrest. Emergency services should be called right away, with CPR initiated immediately and AEDs used as soon as possible. Having medical personnel on-site, easy ambulance access, and practicing regular emergency drills are critical to saving lives. Additionally, managing athlete exertion by monitoring physical activity levels, maintaining hydration, and offering personalized medical plans for those with pre-existing heart conditions will help minimize the risk of SCD. By following these strategies and maintaining ongoing education on best practices, the risk of SCD on the field can be reduced, and survival rates improved.

The cardiology team from Georges-Pompidou European Hospital AP-HP, in collaboration with the Paris Sudden Death Expertise Center, Université Paris Cité, and Inserm, analyzed the survival rates of athletes who experienced cardiac arrest between 2005 and 2018. This study followed the introduction of prevention and training strategies, alongside the provision of essential equipment. Led by Dr. Nicole Karam and Prof. Eloi Marijon, the research revealed that while the incidence of cardiac arrests remained unchanged, the survival rate of affected athletes tripled . (19)



Total number of sports-related sudden cardiac arrest remained stable throughout the study period, while the rate of automated external defibrillator use and cardiopulmonary resuscitation increased steadily, associated with a 3-fold increase in its survival rate. AED = automated external defibrillator; CPR = cardiopulmonary resuscitation; SrSCA = sports-related sudden cardiac arrest.

Conclusion

In conclusion, preventing sudden cardiac death (SCD) in athletes requires a multi-faceted approach combining thorough preparticipation screenings, access to life-saving equipment like Automated External Defibrillators (AEDs), and effective emergency preparedness. Early detection of underlying cardiovascular conditions through regular medical evaluations is crucial in identifying at-risk athletes. Education on recognizing early warning signs, mandatory CPR and AED training for staff and athletes, and the implementation of well-coordinated Emergency Action Plans (EAPs) are key to improving response times and survival rates in case of cardiac events. By integrating these strategies, the risk of SCD on the field can be significantly minimized, ensuring a safer environment for athletes to perform.

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Decoding the Athlete's Heart : The Power of Stress Echocardiography in Cardiac Assessment

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Summary

This review offers an in-depth examination of the role of stress echocardiography (SE) in the cardiovascular assessment of athletes. Due to the intense physical demands, athletes often exhibit significant cardiac adaptations, which can complicate the distinction between normal physiological changes and underlying pathological conditions.ESE serves as a vital noninvasive tool, providing comprehensive insights into the cardiac performance, reserve capacity, and the potential for exercise-induced arrhythmias. SE is also crucial in differentiating between the benign phenomenon of the "athlete's heart" and serious cardiac diseases. Emerging modalities such as left ventricular global longitudinal strain (GLS) and myocardial work (MW) are discussed as innovative approaches for detecting subtle myocardial dysfunction. However, despite its numerous advantages, SE is not without limitations, including challenges related to interpretation and the risk of false-positive and false-negative findings, which are explored throughout this review.

Keywords :
Exercise stress echocardiography, sports cardiology, athlete's heart, cardiovascular adaptation.

Résumé

Cette revue propose une analyse approfondie du rôle de l'échocardiographie de stress dans l'évaluation cardiovasculaire en cardiologie de sport. En raison des exigences physiques intenses auxquelles les athlètes sont soumis, ils présentent souvent des adaptations cardiaques importantes, rendant parfois difficile la distinction entre les adaptations physiologiques des affections cardiovasculaires pathologiques. L'échocardiographie de stress constitue un outil non invasif essentiel, fournissant des informations complètes sur la performance cardiaque, la capacité de réserve et le risque potentiel d'arythmies induites par l'exercice. Les nouvelles techniques, telles que la déformation longitudinale globale du ventricule gauche et le travail myocardique, sont évoquées comme des approches innovantes pour détecter des atteintes myocardiques subtiles. Cependant, malgré ses nombreux avantages, l'échocardiographie de stress présente parfois des limites, liées aux difficultés d'interprétation et aux risques de faux positifs ou faux négatifs, qui seront abordés tout au long de cette revue.

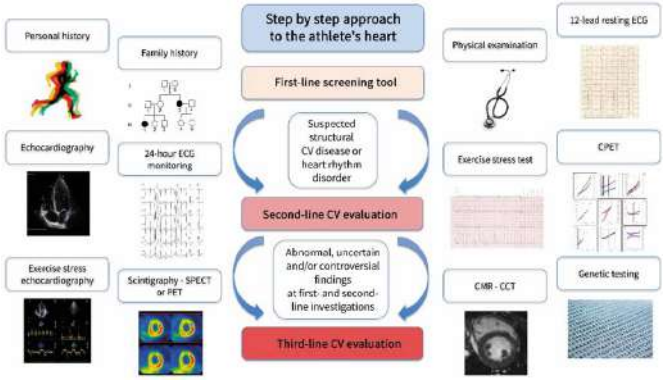
Mots clés :
Échocardiographie de stress, cardiologie du sport, cœur d'athlète, adaptation cardiovasculaire.

Introduction

Athletes expose their cardiovascular systems to intense and repeated physical strain, leading to both short-term and long-term physiological adaptations, as well as an elevated risk of cardiovascular pathologies over time [1]. These adaptations manifest as morphological, functional, and regulatory changes, often including increased cardiac mass, enlarged cavity dimensions, and augmented myocardial wall thickness, all while maintaining normal systolic and diastolic functions [1].

These changes are typically benign, but can resemble certain pathological conditions, thereby complicating the differentiation between normal physiological remodeling and potential disease states [2]. This overlap, commonly referred to as the "grey zone," presents a significant diagnostic challenge, necessitating a thorough and precise assessment to ensure appropriate clinical decision-making and management, as shown in Figure 1 [2].

Figure 1 :
The step-by-step approach to diagnosing athlete's heart [2]



ECG: electrocardiogram; CV: cardiovascular; CPET: cardiopulmonary exercise test;
CMR: cardiac magnetic resonance; CCT: computed coronary tomography;
SPECT: single photon emission computed tomography; PET: positron emission tomography.

When initial diagnostic evaluations yield inconclusive findings, advanced cardiovascular imaging techniques become essential for distinguishing between physiological adaptations and pathological abnormalities. However, the high costs and limited availability of some of these advanced imaging modalities restrict their routine use, and their application is generally reserved for cases with clear clinical indications [3].
Among the various imaging options, SE has proven invaluable in unmasking latent cardiac dysfunctions or concealed pathologies that may not be apparent at rest. This is particularly relevant in athletes who are suspected of harboring arrhythmias or incipient forms of cardiomyopathy [4].
In athletes, SE holds particular value, as it allows for the detection of abnormalities that only manifest during physical exertion. By correlating symptoms with induced cardiovascular stress, SE enhances diagnostic accuracy, especially when assessing exercise-related symptoms [5].
This review aims to explore the specific benefits and applications of SE within the field of sports cardiology, highlighting its critical role in distinguishing between physiological adaptations in athletes and underlying cardiovascular pathologies.

Methodology of Stress Echocardiography :
SE is ideally conducted using supine bicycle ergometer, which allows for continuous echocardiographic imaging during exercise. In situations where this setup is unavailable, standard treadmills or upright cycle ergometers may serve as alternatives. Echocardiographic assessments should be performed at baseline (rest), during various stages of exercise (early and peak), and in the recovery phase, as shown in Figure 2 [1].
Whenever possible, physical exercise should be prioritized over pharmacological stress, as this ensures a more physiologically accurate representation of the cardiovascular system's electromechanical responses and provides critical insights into functional cardiac performance, particularly in athletes [6].

While exercice stress echocardiography (ESE) remains a robust tool for dynamic cardiac assessment under physical stress, pharmacological stress testing may be indicated in individuals with limited exercise tolerance or in those with specific health conditions precluding exercise [6].
Pharmacological stress with agents such as dobutamine or dipyridamole, is typically reserved for assessing myocardial ischemia, especially when there is significant left ventricular dysfunction or when evaluating myocardial viability in patients who are unable to complete physical exertion [7].
A recent advancement in echocardiographic imaging is the use of myocardial contrast stress echocardiography, which can significantly enhance the quality of stress echocardiography by improving the delineation of the endocardial borders. This is particularly valuable in athletes with suboptimal imaging windows, offering superior accuracy in assessing myocardial perfusion and function during stress conditions [8].
Throughout the ESE procedure, the athlete's heart rate, blood pressure, and electrocardiogram should be continuously monitored to ensure safety and provide comprehensive data regarding the cardiovascular response to exercise. The supine bicycle ergometer is generally favored over the treadmill or upright bicycle for exercise testing, especially due to its compatibility with echocardiographic imaging throughout the exercise phases. This setup is particularly advantageous in athletes, who tend to exhibit rapid heart rate and blood pressure normalization during recovery, making continuous imaging essential.
Compared to treadmill or upright cycling, the semisupine position simplifies the acquisition of echocardiographic images during peak exercise, allowing for more reliable real-time imaging. It enables clinicians to capture cardiac images at different stages of the exercise protocol rather than solely relying on post-exercise imaging, which may miss critical dynamic changes.
In the semisupine position, imaging from various anatomical angles can be easily performed as exercise intensity escalates. For upright cycling, adjustments such as forward leaning over the handlebars can facilitate the acquisition of apical views, ensuring adequate image quality during peak stress [9]. However, successful ESE also relies on the athlete's ability to maintain a steady pedaling cadence and proper coordination throughout the test [9].
The exercise protocol typically involves a gradual increase in workload, either in 25-watt increments every two minutes or in higher increments, such as 50 watts, to shorten the duration of the test in well-conditioned athletes. The goal is to achieve maximal exertion or a point where symptoms limit further exercise.
Test termination criteria include significant symptoms, muscular fatigue, elevated blood pressure beyond 220/120 mmHg, symptomatic hypotension with a drop greater than 40 mmHg, abnormal ventricular repolarization, or the development of arrhythmias such as supraventricular tachycardia, atrial fibrillation, or frequent ventricular ectopy [10].

Several cardiac parameters are evaluated during ESE, including biventricular function, transvalvular gradients, and regurgitant flows. Hemodynamic assessments of both the left and right heart, such as systolic pulmonary artery pressure and ventricular volumes, are also critical [11]. While it is not feasible to assess every conceivable parameter under stress conditions, priority should be given to those that are diagnostically relevant to the clinical context.

The specific ESE protocol used should be clearly documented in the final report. A typical physiological response to both exercise and inotropic stress includes an improvement in function across all left ventricular (LV) segments, accompanied by an increase in LV ejection fraction (EF) and cardiac output. The appearance or worsening of regional wall motion abnormalities in two or more adjacent LV segments suggests ischemia, while a one-grade improvement in previously dysfunctional segments points to viable myocardium [12].

For individuals without regional wall motion abnormalities at rest, global contractile reserve is often defined as 5% or greater increase in LV ejection fraction during stress. Flow reserve is identified by a rise in forward stroke volume of at least 20%.

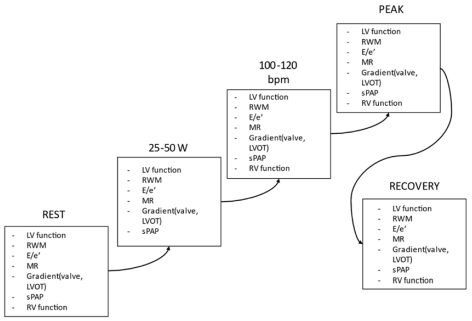
Changes in key cardiac parameters such as wall motion, EF, or global longitudinal strain must be meticulously recorded. Hemodynamic variables like stroke volume, SPAP, the E/e' ratio, and LV outflow tract gradients should also be detailed [12].

Similarly, any variation in the severity of valvular dysfunction, including mitral regurgitation or aortic valve area and associated gradients, must be described with respect to the specific diagnostic query. Blood pressure and heart rate data are essential to contextualize the contractile and hemodynamic responses during the test [12].

ESE is also valuable for the detection of myocardial viability and ischemia, often through assessments of coronary flow reserve, particularly in the left anterior descending artery. This can be measured using pulsed Doppler sampling of the proximal segment [13].

The results of ESE must always be interpreted with consideration of the athlete's overall health and risk factors. Given the heightened cardiovascular conditioning in athletes, false-positive and false-negative results can occur. As such, accurate interpretation of ESE findings requires specialized expertise in both echocardiography and sports cardiology.

Figure 2 :
ESE protocol and echocardiographic parameters assessed at each stage [1]



LV: left ventricle; RWM: regional wall motion; E/e': ratio of early transmitral diastolic velocity to early TDI velocity of the mitral annulus; MR: mitral regurgitation; LVOT: LV outflow tract; sPAP: systolic pulmonary artery pressure; RV: right ventricle; W: watt; BPM: beats per minute.

Indications for Stress Echocardiography in Athletes :

Despite ongoing discussions in the scientific community regarding the most effective strategies for mass preparticipation screening in athletes, SE is not typically endorsed as a screening tool in this context. The rationale behind this recommendation lies in the relatively low prevalence of atherosclerotic coronary artery disease among young athletic populations, coupled with the limited diagnostic utility of SE for detecting anomalies in coronary anatomy, such as aberrant coronary artery origins or trajectories [14].

However, SE remains a cornerstone diagnostic modality in the cardiovascular evaluation of athletes, offering comprehensive insights into cardiac function, functional reserve, exercise tolerance, and potential arrhythmias [15]. Its wide accessibility, affordability, and absence of ionizing radiation make it particularly advantageous as a third-line diagnostic option in sports cardiology. According to the latest European guidelines in sports cardiology, SE is highly recommended in cases where exercise stress testing yields equivocal or uninterpretable results, particularly when evaluating for the presence of coronary artery disease. Furthermore, it is employed to assess the hemodynamic response to exercise in individuals with valvular heart disease, as well as in cases of LV hypertrabeculation, where complications related to the condition are suspected [16].

A profound understanding of the normal physiological echocardiographic adaptations in athletes is essential to determine when SE should be indicated. Common echocardiographic changes resulting from regular physical training include symmetrical enlargement of both left and right heart chambers, increased LV wall thickness and mass, and enhanced indices of systolic and diastolic function beyond normal resting values [2].

Exercise-Induced Ischemia :

One of the main indications for SE in athletes is the detection of exercise-induced ischemia, particularly in those presenting with chest pain or abnormal electrocardiogram findings.

In such clinical scenarios, ruling out CAD or congenital anomalies of the coronary arteries, especially anomalies related to the origin or course of the vessels, is paramount [17].

SE demonstrates moderate sensitivity and specificity for the diagnosis of CAD, approximately 76% and 88%, respectively, which compares favorably with other available stress-testing modalities [17]. However, the decision to indicate SE versus alternative third-line diagnostic techniques, such as CMR or CT angiography, is still a topic of active debate in the literature, particularly when assessing athletes for CAD [17].

While coronary artery disease is rare in younger athletes, especially those without cardiovascular risk factors, it remains a concern in older athletes or in those with exercice-induced symptoms. SE serves as a valuable tool for evaluating coronary perfusion abnormalities under stress, allowing for the dynamic assessment of ischemic thresholds that may not be apparent at rest. Furthermore, in athletes with inconclusive ECG or ambiguous symptoms, SE can offer a nuanced view of myocardial perfusion, wall motion, and functional reserve, aiding in the differentiation between ischemic heart disease and benign athletic heart adaptations [2].

Athlete's Heart Grey Zone :

In endurance athletes presenting with left ventricular (LV) and/or right ventricular (RV) dilation accompanied by a mildly reduced ejection fraction (EF) at rest, exercise stress echocardiography (ESE) serves as a valuable tool for assessing the heart's contractile reserve under physical stress, as shown in Figure 3 [3]. A significant enhancement in contractility during exercise, such as an increase in LVEF greater than 5%, reflects physiological adaptations associated with athletic training. Conversely, a lack of sufficient contractile augmentation may indicate underlying pathological conditions, such as hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy (DCM), left ventricular non-compaction (LVNC), or arrhythmogenic cardiomyopathy (AC) [18]. A pronounced EF improvement during exertion suggests that a reduced resting EF is likely attributable to a decreased preload rather than intrinsic LV systolic dysfunction [15].

In a notable study by Abernethy et al., an increase in LVEF during ESE was observed across all participants, irrespective of their baseline resting values. This underscores the importance of tissue velocity imaging as a key technique when evaluating cardiac performance during physical activity [19].

RV strain, as assessed through speckle-tracking echocardiography (STE), along with RV ejection fraction and RV annular peak systolic velocity, has proven moderately to highly accurate in distinguishing patients with AC from healthy individuals [20].

In the context of isometric exercise, highly trained resistance athletes show greater increases in stroke volume and enhanced diastolic function compared to sedentary controls, as several studies have corroborated [21].

The potential of ESE in differentiating between athlete's heart and DCM has also been demonstrated by Millar et al., who revealed that during ESE, 96% of athletes in the "grey zone" displayed an increase in LVEF exceeding 11% from baseline to peak exercise, contrasting sharply with only 23% of patients with DCM ($p < 0.0001$). Moreover, a reduction in LV end-systolic volume was observed during exercise in both athletes and healthy controls, but not in individuals with DCM or HCM [22]. These findings suggest that monitoring LV function during exercise may serve as a robust means of distinguishing physiological adaptations of athlete's heart from pathological cardiomyopathies [22].

In terms of systolic function, young endurance-trained athletes generally exhibit a normal diastolic response to ESE. For instance, marathon runners demonstrate an increase in mitral inflow velocity (E) and lateral mitral annular velocity (e') during exercise, with a modest rise in both E/e' septal and E/e' lateral ratios, though these remain within normal physiological limits [23].

However, strenuous endurance exercise may induce elevated systolic pulmonary artery pressure (sPAP). Mirea et al. found that 12.9% of the athletes developed elevated sPAP, further exacerbated by exercise on a bicycle ergometer, which correlated with significant RV dilation. Despite these findings, conventional echocardiographic techniques and STE consistently demonstrated preserved RV function in these athletes [24].

In fact, recent research has underscored the utility of ESE in evaluating both the pulmonary circulation and right ventricular function under stress. This approach has revealed prognostically significant differences not only among healthy individuals and athletes but also among high-altitude dwellers and patients with various cardio-respiratory disorders [25].

Such insights further highlight the importance of ESE in the comprehensive assessment of athlete's heart and in distinguishing it from potentially life-threatening conditions.

Figure 3 :
The use of stress echocardiography in the differential diagnosis of grey zones in athlete's heart.

Parameters during Effort	Findings Suggestive of Normal Heart	Finding Suggestive of CV Pathologies
Contractile reserve	Significant improvement	Absent or subnormal improvement
Dynamic obstruction	No dynamic intraventricular obstruction	LVOTO or mid-cavity obstruction
Diastolic function	Normal/supranormal diastolic function indexes	Diastolic dysfunction
Heart valve diseases	Absent	Dynamic/functional new onset/worsening valve diseases
Ischemia	Absent	Inducible ischemia
Lung echocardiography	Normal	Pulmonary congestion

CV : cardiovascular; LVOT : left ventricular outflow obstruction [3].

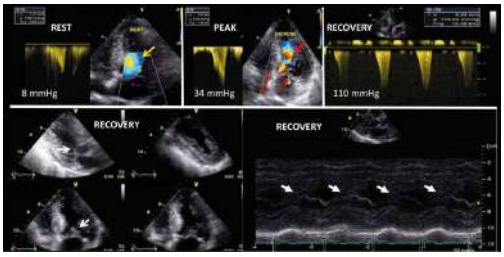
Hypertrophic Cardiomyopathy (HCM) :

ESE plays a pivotal role in the diagnosis of HCM, a main cause of sudden cardiac death among athletes, as shown in Figure 4 [26].

For patients who do not present with outflow gradients at rest, ESE emerges as the preferred technique for inducing obstruction. This dynamic assessment not only facilitates the prediction of future progressive heart failure symptoms, but also aids in differentiating between individuals with inducible obstruction and those without. Such distinctions bear critical implications for determining optimal treatment strategies [27].

An LV outflow tract gradient exceeding 50 mmHg during or immediately post-exercise in athletes exhibiting LV hypertrophy, coupled with symptoms such as syncope or exertional dyspnea, may strongly indicate HCM [28]. Gaitonde et al. demonstrated that athletes diagnosed with HCM exhibited significantly higher peak gradients at rest and during ESE compared to their non-HCM counterparts. Typically, individuals with a structurally normal heart do not display dynamic intraventricular obstruction in response to aerobic exertion [29].

Figure 4 :
Example of dynamic changements during ESE in a dyspneic athlete with HCM [26].



Top: Increase in left ventricular outflow tract velocity and gradient associated with a marked flow acceleration. Bottom: Systolic anterior motion of the mitral valve identified on 2D and M-mode echocardiography.

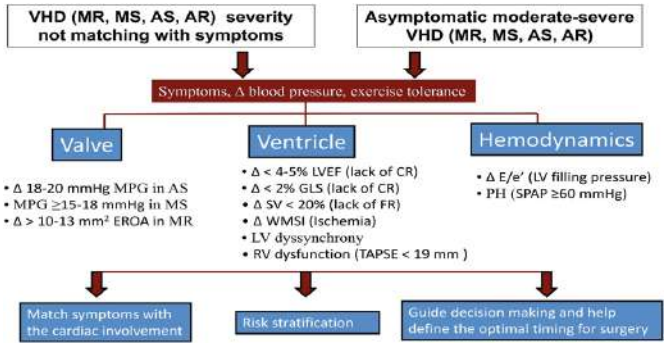
Valvular Heart Disease (VHD) :

The assessment of athletes with valvular heart disease warrants particular attention, as ESE can provide complementary insights into functional status, exercise tolerance, biventricular contractile reserve, and alterations in hemodynamic and valvular parameters.

This includes detailed evaluations of transvalvular gradients, quantification of regurgitation, estimation of sPAP, and assessment of diastolic function, as shown in Figure 5 [30].

Consequently, athletes diagnosed with mild to moderate VHD should be subjected to ESE protocols that closely replicate the physical exertion expected in their respective sports.

Figure 5 :
Usefulness of exercise stress echocardiography in evaluation of athletes with VHD [30]



MR : mitral regurgitation; MS : mitral stenosis; AS : aortic stenosis; AR : aortic regurgitation; Δ : changes from rest to peak exercise; MPG : mean pressure gradient; EROA : Effective regurgitant orifice area; LVEF : left ventricular ejection fraction; FR : flow reserve; GLS : global longitudinal strain; RV : right ventricle; PH : pulmonary hypertension; SPAP : systolic pulmonary artery pressure; SV : stroke volume; TAPSE : tricuspid annulus plane systolic excursion; WMSI : wall motion score index.

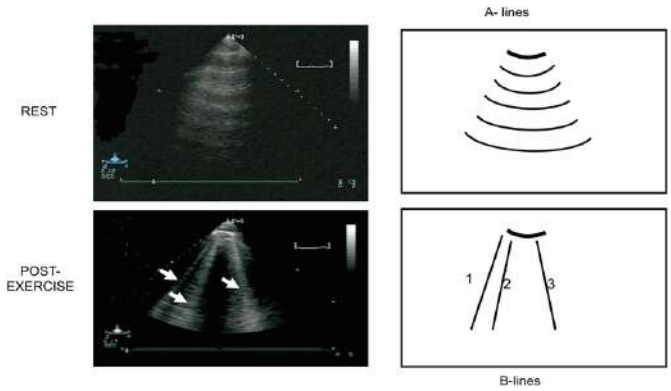
Stress Lung Ultrasound :

The assessment of B-lines utilizing lung ultrasound, often referred to as "ultrasound lung comets", provides a straightforward and effective approach to visualize extravascular lung congestion. An accurate evaluation involves thorough scanning of both the anterior and posterior chest, as well as quantifying the number of B-line artifacts across each intercostal space [31].

Stress lung ultrasound, which entails the detection of B-lines during or immediately following exercise, proves particularly valuable in two distinct clinical scenarios: heart failure and extreme physiological states. In contexts such as high-altitude trekking or among healthy elite athletes (apnea divers, scuba divers, underwater fishermen, and those participating in triathlons or marathons), B-lines may be detected even in the absence of overt pulmonary edema symptoms [32]. Thus, B-line assessment should be routinely incorporated into the ESE protocol for athletes [32].

Moreover, recent studies, including one by D'Andrea and colleagues, have highlighted the use of B-line evaluation during ESE as a means to differentiate between athletes and individuals using anabolic androgenic steroids. This finding suggests a potential application of B-line assessment in anti-doping evaluations, thereby underscoring its importance in the comprehensive assessment of athlete health [33].

Figure 6 :
Lung ultrasound at rest and immediately after exercise [31].



Emerging Techniques and Innovations in Stress Echocardiography :

Left Ventricular Global Longitudinal Strain (LV GLS) and Mechanical Dispersion :

In the last decade, the assessment of LV GLS through speckle-tracking echocardiography has gained considerable recognition as a robust tool for analyzing myocardial mechanics. This advanced imaging technique provides nuanced insights into cardiac performance that extend beyond traditional parameters of LV systolic function, such as EF [34].

Endurance athletes often exhibit lower LV GLS at rest compared to their sedentary counterparts, a phenomenon likely attributable to factors such as increased afterload, cardiac hypertrophy, and sinus bradycardia [35].

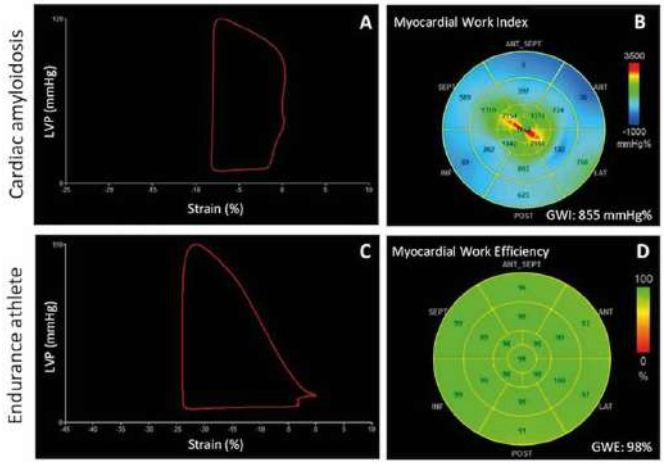
Gruca et al. demonstrated significant increase in GLS following a stress test in a cohort of 111 basketball athletes. This finding holds potential diagnostic implications for differentiating athlete's heart from other cardiovascular conditions [36]. Indeed, several investigations have revealed that a reduction in LV GLS is infrequently seen in athlete's heart, suggesting that GLS alterations cannot be dismissed as mere physiological adaptations to training. Such findings may be instrumental in elucidating the nature of cardiovascular adaptations in particular circumstances [36].

Additionally, the measurement of mechanical dispersion of GLS, defined as the standard deviation of the time to peak longitudinal strain across all LV segments, has emerged as a promising diagnostic tool for identifying HCM in athletes [36].

Myocardial Work (MW):

The concept of MW has emerged as an innovative noninvasive index for evaluating LV myocardial deformation through LV pressure-strain loop analysis. This advancement over GLS allows for deeper insights into LV performance under varying levels of exertion by incorporating afterload and assessing myocardial efficiency [37]. MW provides additional context beyond EF and strain measurements, particularly under different LV loading conditions prevalent in athletic populations. This capability renders MW particularly valuable for evaluating LV myocardial deformation and contractile reserve in a manner that is less susceptible to loading variations, as shown in Figure 7 [38].

Figure 7 :
Pressure-strain loop (A) and 17-segment bull's-eye representation of Global Work Index (B) in a patient with cardiac amyloidosis, showing a reduced loop area and significant impairment of Myocardial Work Index in the basal segments. In endurance athletes, Myocardial Work Efficicency is an early predictor of physiologic remodeling in baseline examination (C and D) [38].



GWI : global work index; GWE : global work efficiency; LVP : left ventricular pressure;
GWI : global work index; GWE : global work efficiency; LVP : left ventricular pressure.

Innovative Combinations with Other Techniques :
Recent investigations have begun to explore the synergistic potential of combining SE with other diagnostic modalities, such as cardiopulmonary exercise testing (CPET) [40]. Furthermore, the advent of artificial intelligence (AI) applications related to SE is paving the way for enhanced diagnostic precision.
In a recent study, Upton et al. highlighted the significant contributions of AI-driven methodologies in improving the accuracy, confidence, and reproducibility of stress echocardiography interpretations [41].
These innovations not only broaden the diagnostic toolkit available for the assessment of cardiovascular health in athletes, but also hold the potential to refine clinical decision-making processes, ultimately improving athlete management and outcomes.

Limitations of Stress Echocardiography in Athletes :
While SE serves as a valuable diagnostic tool for evaluating cardiovascular health in athletes, several limitations merit consideration [5]:
Accessibility and Resource Intensity:
SE necessitates specialized equipment and expertise, which may not be universally available or affordable across all healthcare settings. The requirement for trained personnel to conduct and interpret the results can further limit its accessibility, particularly in resource-limited environments.
Time Commitment:
The procedure entails a substantial time commitment, as it typically involves an extensive imaging protocol alongside continuous monitoring of the athlete throughout the testing process. This time demand may be a constraint for both athletes and clinical staff, potentially impacting scheduling and logistical planning.
Influence of Athlete's Physiological State:
The variability in athletes' physiological states can complicate SE evaluations. Factors such as hydration status, fatigue level, and acute physiological responses to exercise may influence echocardiographic measurements, leading to variability in results that complicate consistent interpretation.
Risk of False Positives:
A significant limitation of SE in the athletic population is the propensity for false positive results. Exercise-induced alterations in LV function, such as transient wall motion abnormalities, can occur in otherwise healthy athletes.
These physiological changes may be misinterpreted as indicators of underlying cardiovascular disease, leading to unnecessary additional testing, unwarranted treatments, and unjustified restrictions on athletic participation.
Risk of False Negatives:
Conversely, SE also has the potential for false negative results, particularly in athletes with early or mild manifestations of cardiovascular disease.
The inability to detect subtle abnormalities may result in missed diagnoses, delayed therapeutic interventions, and an elevated risk of serious complications, including sudden cardiac events during physical exertion.
Interpretation Challenges:
The interpretation of SE findings can be particularly challenging in highly conditioned athletes. These individuals may exhibit physiological adaptations such as LV hypertrophy, LV cavity enlargement, or altered wall motion that are not indicative of pathological conditions. Differentiating between normal physiological adaptations and true pathological changes often requires a nuanced understanding and may necessitate supplementary imaging techniques, such as CMR, for a comprehensive assessment.

Conclusion

SE is an essential instrument in the cardiovascular assessment of athletes, offering a sophisticated integration of exercise testing and echocardiographic imaging. This method yields critical insights into cardiac function, reserve capacity, exercise tolerance, and arrhythmias. It stands out as a dependable, safe, and non-invasive diagnostic modality, capable of revealing hidden pathological conditions that may not be apparent in resting evaluations. The growing adoption of SE within sports cardiology echocardiography laboratories underscores its acknowledged diagnostic and prognostic significance. While there remains a necessity for further research to develop comprehensive guidelines governing its application and execution, SE's distinctive capacity to distinguish between the athlete's heart and other cardiovascular disorders establishes it as a vital element in the diagnostic landscape.

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Bradycardia in athletes: When to worry ?

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Summary

Bradyarrhythmias are a common feature of the athlete's ECG. Although these arrhythmias are generally well tolerated in young athletes, there is evidence that they can progress to symptomatic pathological bradycardia, possibly requiring pacemaker implantation. The high vagal tone has always been described as the cause. However, recent research has challenged this hypothesis, revealing a significant role in intrinsic electrophysiological remodelling of the sinus and atrioventricular nodes. What mechanism is involved and when should we worry?

Keywords :
Bradycardia, Athletes, Atrioventricular block, Exercise, Eligibility

Résumé

Les bradyarythmies sont une caractéristique commune de l'ECG de l'athlète. Bien que ces arythmies soient généralement bien tolérées chez les jeunes athlètes, il est prouvé qu'elles peuvent évoluer vers une bradycardie pathologique symptomatique, nécessitant éventuellement l'implantation d'un stimulateur cardiaque. Le tonus vagal élevé a toujours été décrit comme la cause de ces arythmies. Cependant, des recherches récentes ont remis en question cette hypothèse, révélant un rôle important dans le remodelage électrophysiologique intrinsèque du sinus et des nœuds auriculo-ventriculaires. Quel est le mécanisme en cause et quand faut-il s'inquiéter ?

Mots clés :
Bradycardie, athlètes, bloc auriculo-ventriculaire, exercice, éligibilité

Introduction

Bradycardia is the hallmark of the athlete's heart. While the general population widely recognizes this phenomenon, the underlying mechanisms and clinical implications remain complex and somewhat elusive [1]. Clinicians should determine when slow becomes too slow, that is when physiological adaptation transitions into pathophysiology. The overarching goal of this document is to provide evidence-based and expert consensus recommendations on the diagnosis and management of bradycardia in athletes of all ages, emphasizing shared decision-making.

II. General concepts and principles

1. Definition of athletes :

Although there is no universal definition of "athlete" in the medical literature [2], since 2020, the European Society of Cardiology (ESC) defines an athlete as an individual of young or adult age, either amateur or professional, who is engaged in regular exercise training and participates in official sports competition [3]. Similarly, the Heart Rhythm Society defines athletes as individuals who are engaged in habitual and vigorous training for the purpose of obtaining a high level of fitness. This includes competitive athletes, high-level recreational exercise enthusiasts, and occupational athletes [2].

This document applies to both adult and pediatric athletes. Given the complexity of the interaction between age and different arrhythmic diseases, rather than using arbitrary age cut points, athletes are considered in different age domains based on the stage of development [2].

- Young= prepubertal and adolescent
  - o Prepubertal: <12 years old
  - o Adolescent = 13-17 years old
- Young adult = 18-24 years old
- Adult > 25 years old
- Master > 35 years old

2. Physiology of exercise :

The cardiovascular system undergoes several modifications as a consequence of exercise training, with acute responses, which occur within a few seconds of intensive exercise, and chronic adaptations that include more profound structural remodelling resulting from long-term conditioning. The normal resting cardiac output is approximately 5L/min. The liver, kidneys, muscles, and brain account for the largest blood volume consumption at approximately 27%, 22%, 20%, and 14%, respectively. The situation changes dramatically during strenuous exercise when the cardiac output increases to 25–35L/min. Skeletal muscle demands approximately 84% of this blood volume when exerting strenuously,and this is critical to satisfy the oxygen demand for ATP generation and carbon dioxide removal necessary for acid-base homeostasis.

Chronic cardiovascular adaptations to exercise training determine structural remodelling of the cardiac chambers and vessels, facilitating an increased capacity to deliver oxygen to the working muscles during prolonged bouts of exercise. Expression of cardiac remodeling consists of an increase in left and right ventricular and left atrial cavity size associated with normal diastolic function [4].

3. Athletes with bradycardia :

Sinus and Atrio-ventricular (AV) nodal slowing are expected adaptations to athletic training, particularly in high vagal states such as sleep or rest. More profound bradycardias should first be evaluated with response to light exercise and, if they persist, with ambulatory monitoring and/or exercise stress testing. If they remain persistent, further testing may be indicated to exclude the possibility of cardiomyopathy. Findings should be correlated with history to ensure that the patient is asymptomatic and has no family history of cardiomyopathy or conduction system disease. In the absence of symptoms of structural heart disease, current consensus guidelines consider sinus bradycardia > 30 beats/min, PR interval < 400 ms, Mobitz type I second-degree AV block, and junctional escape rhythms as benign physiological adaptations accompanying a long training history [5,6].

III. Mechanisms underlying sinus bradycardia and AV bloc in athletes

1. OUT goes the OLD :

It is well known that athletes have a low resting heart rate. Bradycardia can be moderate or severe: reports of heart rates of 40-60 beats min in athletes are common, and heart rate <30 beats min in elite athletes at night. This bradycardia is generally the result of high vagal tone, which is a natural assumption as high vagal tone reduces heart rate. However, despite this widespread belief, vagal nerve activity efferent to the sinus node has never been recorded. Measuring it is not straightforward, as the vagal nerve carries afferent and efferent nerve fibres [1].

For this reason, the scientific community uses what is assumed to be a surrogate for vagal nerve activity at the sinus node, namely heart rate variability. Heart rate variability is a beat-to-beat variability of heart rate. It is assumed to result from stochastic fluctuations in autonomic nerve activity at the sinus node, and changes in heart rate variability are assumed to represent changes in this activity. Heart rate variability is higher in athletes, which is considered evidence of elevated vagal tone in athletes, which is then assumed to be responsible for bradycardia. However, a causal link between autonomic nerve activity and heart rate variability has never been demonstrated. Furthermore, we have recently analyzed the biophysics underlying heart rate variability [7].

2. And IN comes the NEW :

The electrical activity in the heart is influenced by ion channel activity, and studies have shown that plasticity in the expression of rhythmic ion channels plays a key role in regulating heart rate and AV node conduction in response to chronic physiological and pathological stimuli. Research in humans and rodents has demonstrated that after exercise training, there is a change in the intrinsic electrophysiology of the AV node, including a prolonged Wenckebach cycle length, prolonged AV node refractory period, PR interval extension, or reduced heart rate, even in the absence of autonomic tone. A widespread transcriptional remodelling of key ion channels in both the sinus and AV nodes has been observed. Exercise leads to the downregulation of hyperpolarization-activated and cyclic nucleotide-gated (HCN) Ca2+ channels as well as L- and T-type Ca2+ channels at these nodes' protein and mRNA levels. A corresponding reduction in ion currents, such as the funny current (If), L-type Ca2+ current (ICa, L), and T-type Ca2+ current (ICa, T), has been shown in the myocytes of the sinus and AV nodes. The ICa, L and ICa, T currents are crucial in sinus node pacing and AV node conduction. While If's role in heart rate regulation is well established, its influence on AV node conduction is only now being recognized. Studies involving If blockers, HCN4 loss-of-function mutations, and HCN4 knockouts have shown that a reduction in If not only slows heart rate but also AV node conduction. This suggests that in human athletes, downregulation of HCN channels and If contributes to sinus bradycardia. In the case of the AV node, biophysical modeling indicates that downregulation of If and ICa-L contributes to the slowed AV node conduction following exercise training [1,5].

IV. How to manage bradycardia in athlete

1. Sinus bradycardia :

Sinus bradycardia is a heart rate <60bpm in up to 80% of highly trained athletes. Resting sinus bradycardia is particularly prevalent in endurance athletes. It is mainly mediated by intrinsic electrophysiological adaptations in the sinus node (decrease in sinus automaticity) and increased parasympathetic and reduced sympathetic input during the resting state. Resting bradycardia is related to training level and may predispose to increased atrial or ventricular ectopic activity and rarely atrial fibrillation. However, sinus bradycardia is considered a normal finding in the athlete unless symptoms such as fatigue, dizziness, or syncope are present. Even heart rates ≥ 30 bpm are considered normal in the asymptomatic athlete. Interestingly, in some cases of severe sinus bradycardia, the sinus rhythm competes with the nodal rhythm, and an isorhythmic atrioventricular dissociation can be seen. However, ventricular escape rhythm is a rare finding. The effects of increasing age may modify some of the physiological adaptations at the sinus node level. In this context, resting bradycardia is more pronounced in older athletes whose maximum heart rate is also less. This may account in part for the drop-off in performance that occurs with age [4,6].

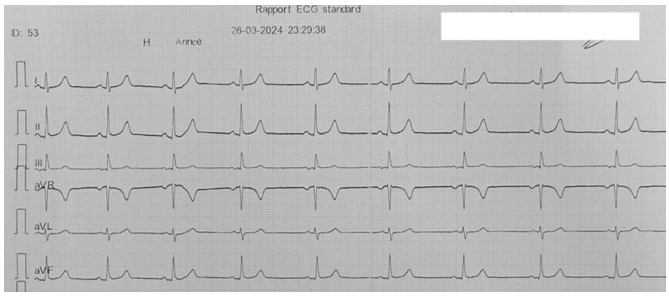
Sinus pauses are very frequent among athletes, with almost a third of subjects presenting pauses greater than 2s. Holter monitoring captures sinus pauses in up to 25% of highly trained endurance athletes during rest. These typically occur during sleep and can result in prolonged sinus node arrest (>6s) in some cases. These phenomena may occur in athletically trained individuals with slow resting heart rates. Invasive electrophysiological studies typically demonstrate normal sinus node and AV node function and normal sinus rhythm appears during exercise [4].

Recommendations

1. Athletes with sinus bradycardia, sinus exit block, sinus pauses, and sinus arrhythmia without symptoms can participate in all competitive athletic activities unless otherwise excluded by underlying structural heart disease or other arrhythmias (Class I; Level of Evidence C).

2. Athletes with symptomatic bradycardia should be evaluated for structural heart disease and be treated for the bradycardia, generally by an implanted pace-maker. They should be restricted from training and athletic competition while being evaluated. If treatment of the bradycardia eliminates symptoms, they can participate in athletic training and competition unless otherwise excluded by structural heart disease or other arrhythmias (Class I; Level of Evidence C).

Figure 1 :
6-lead resting electrocardiogram: sinus bradycardia



2. AV Bloc :

The prevalence of first-degree and Mobitz type I second-degree atrioventricular block (Luciani–Wenckebach) is very high in endurance athletes. In contrast, Mobitz type II second-degree and third-degree atrioventricular blocks are rare. Pathological causes of atrioventricular block in the young athlete include specific forms of infective myocarditis (e.g. Lyme disease and Chagas disease), immune diseases (e.g. cardiac sarcoidosis), and genetic disorders (e.g. Lénègre disease, PRKAG2 syndrome, myotonic dystrophy, and Laminin mutation-related dilated cardiomyopathy). In healthy athletes, first-degree and Luciani–Wenckebach atrioventricular blocks reflect the effect of increased vagal tone on the atrioventricular node and typically disappear with exercise and hyperpnea. Persistent forms of atrioventricular block and those unrelated to hyper-vagotonia contraindicate sports activity. Second-degree atrioventricular Mobitz type 2 advanced or complete atrioventricular block requires careful investigation of the conduction system [4].

2.1 First-degree AV block :

In the first-degree AV block, the PR interval is prolonged (>200ms), but it presents the same duration on every beat. Mild to moderate first-degree AV block (PR interval up to 399ms) may be present in athletes due to intrinsic adaptations and/or increased parasympathetic drive. In competitive endurance athletes, the frequency of this finding can be as high as a third. This represents a delay in AV nodal conduction in athletes due to increased vagal activity and/or intrinsic AV node changes and typically resolves with the onset of exercise [4,6].

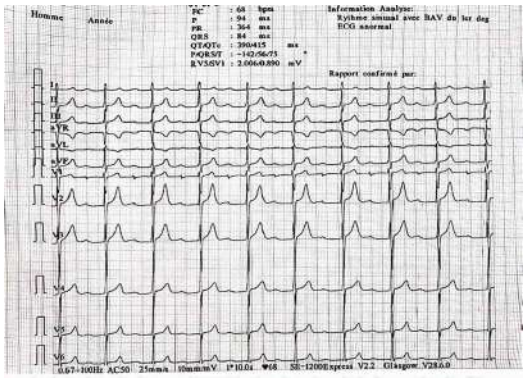
Recommendations :

1. Asymptomatic athletes with no structural heart dis- ease and first-degree AV block (PR interval <0.3 ms) can participate in all competitive sports unless there are findings that indicate that the person is at risk for progression to higher-degree block that would symptoms (Class I; Level of Evidence C).

2. Asymptomatic athletes with first-degree AV block, in whom type I second-degree AV block appears with exercise, should be evaluated further for possible intra-His or infra-His block with EPS (Class I; Level of Evidence C).

3. If structural heart disease is present, athletic restrictions should be recommended as appropriate for the type of structural heart disease (Class I; Level of Evidence C).

Figure 2 :
12-lead resting electrocardiogram: first-degree AV block
(PR interval up to 380ms)



2.2 Type I second-degree (Wenckebach) AV block :

In Mobitz type I second-degree AV block the PR interval progressively lengthens from beat to beat until there is a non-conducted P wave with no QRS complex. The first PR interval after the dropped beat is shorter than the last conducted. Even if the PR interval can be augmented by increased vagal tone, the occurrence of AV blocks depends mostly on individual susceptibility [4,6].

Recommendations :

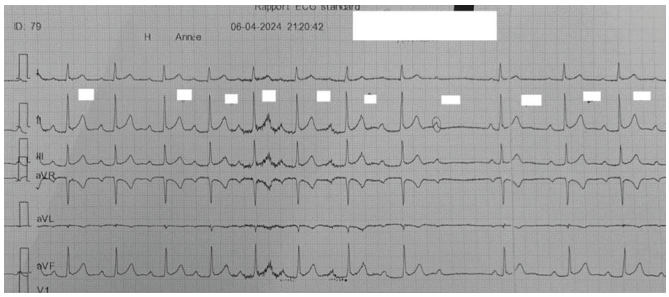
1. Asymptomatic athletes with structurally normal hearts and Wenckebach AV block (type I second-degree AV block) with improvement in conduction with exercise or recovery can participate in all competitive sports (Class I; Level of Evidence C).

2. Asymptomatic athletes with structurally abnormal hearts with improvement in Wenckebach AV block with exercise can participate in all competitive sports, unless there are restrictions based on heart disease (Class I; Level of Evidence C).

3. Athletes with Wenckebach AV block that does not improve with exercise should be evaluated with an EPS for intra-His or infra-His block that may require pacemaker therapy (Class I; Level of Evidence C).

4. In athletes with Wenckebach AV block and coexisting bundle-branch block or with any indication that they are at risk for progression to higher-degree AV block, EPS should be performed to identify the presence of intra–His-Purkinje or infra–His-Purkinje block that may require pacemaker therapy (Class I; Level of Evidence C).

Figure 3 :
6-lead resting electrocardiogram: Wenckebach AV block



2.3 Type II second-degree (Mobitz II) AV block :

Recommendations :

1. Athletes with Mobitz type II second-degree AV block with a wide QRS, including isolated right bundle-branch block (RBBB) should receive a permanent pacemaker (Class I; Level of Evidence C). Restrictions for athletic participation for those with pacemakers are in the section on "Athletes With Permanent Pacemakers."

2. Permanent pacemaker implantation is reasonable for athletes with asymptomatic Mobitz type II second- degree AV block with a narrow QRS (Class IIa; Level of Evidence C).

Figure 4 :
6-lead resting electrocardiogram: 2:1

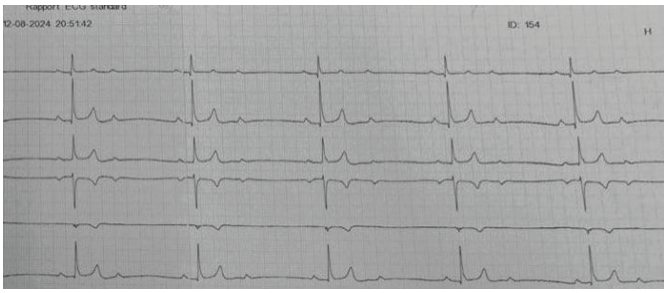
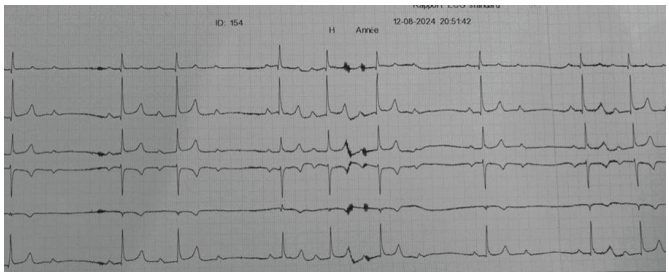


Figure 5 :
6-lead resting electrocardiogram: Type II second-degree (Mobitz II) AV block



2.4 Congenital High-grade or Complete heart block :

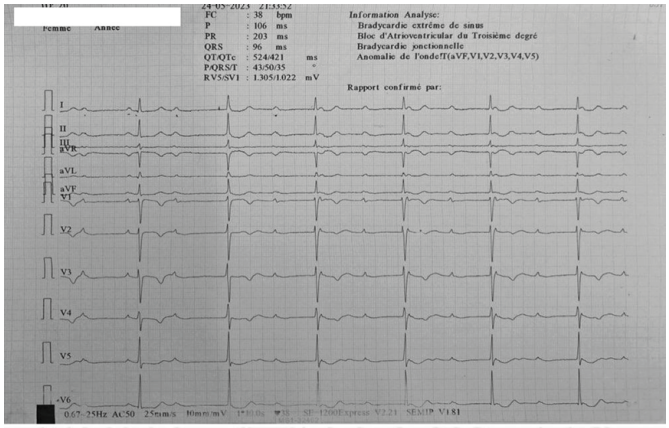
Recommendations :

1. Asymptomatic athletes without heart disease who have a junctional escape rhythm that has a QRS duration <120 ms, resting ventricular rates >40 bpm that increase appropriately with exertion, and exercise capacity that approximates that of the relevant sport can participate in athletic activity without re- striction (Class I; Level of Evidence C).

2. Athletes with symptomatic heart block, resting ventricular rates <40 bpm, or ventricular escape rhythm with a QRS width >120 ms should have a pacemaker implanted before they participate in competitive sports. Before athletes are allowed to resume sports, an exercise test should be conducted to ensure patient safety and that the exercise capacity of the athlete is similar to that required for the relevant sport (Class I; Level of Evidence C).

3. Athletes with structural heart disease and congenital complete heart block should be restricted from, or allowed to participate in, competitive athletics based on the recommendations for the type of structural heart disease with or without a permanent pacemaker (Class I; Level of Evidence C).

Figure 6 :
12-lead resting electrocardiogram: resting heart rate of 38 beats/min, complete atrio ventricular bloc



2.5 Acquired Complete heart block :

Recommendations :

1. Athletes with acquired complete heart block should have a permanent pacemaker placed regardless of symptoms, type of structural heart disease, and exercise capacity unless the heart block is attributable to completely reversible causes and resolves completely (Class I; Level of Evidence C).

2. Athletes with structural heart disease and acquired complete heart block should be restricted from, or allowed to participate in, athletic activities based on the recommendations for the type of structural heart disease (Class I; Level of Evidence C).

3. Before athletes with a permanent pacemaker are allowed to engage in athletic activities, an exercise test should be conducted to ensure that the exercise capacity of the athlete is similar to that required by the relevant sport (Class I; Level of Evidence C).

Table 1 :
Recommendations for athletes with bradycardia

Class of recommendation	Level of evidence	Recommendations
Class 1	B-NR	For athletes with significant distal conduction disease, including left bundle branch block, bifascicular block, or complete heart block at any level, evaluation prior to return to play is recommended.
Class 2a	C-EO	In athletes with significant sinus and/or AV node disease that does not correct with light exercise, further evaluation during return to play is reasonable.
Class 3	B-NR	In athletes with asymptomatic sinus node slowing or first-degree heart block/second- degree Mobitz type I AV block (Wenckebach) at rest, further evaluation is not recommended because these are expected adaptation to training.

V. How to follow an athlete with bradycardia

European and North American scientific organizations have provided guidelines for evaluating and managing athletes with bradycardia. In asymptomatic athletes with expected physiological bradycardia, an annual general sports medical check-up is sufficient, and they can continue participating in all sports. However, if symptomatic sinus bradycardia or pauses occur, temporarily stopping sports activity for 1-2 months may lead to symptom resolution and rhythm improvement. After the symptoms resolve, athletes can resume sports with a follow-up evaluation after six months, though they should be advised to consult earlier if symptoms reappear. It may also be helpful to shift the focus toward technical or power-based sports rather than endurance training. In rare cases where symptoms persist despite stopping sports, pacemaker implantation might be necessary [4,8,9,10].

Table 2 :
Recommendations for competitive sport participation in athletes with bradycardia

Lesion	Evaluation	Criteria for eligibility	Recommendations	Follow-up
Marked sinus bradycardia (<30 bpm) and/or sinus pauses > 3s with symptoms	History, ECG, ET, 24-h Holter, Echo	If no symptoms, no cardiac disease, with resolution during exercise	All competitive sports	Not required
		If symptoms are present	Temporary interruption of sport and re-evaluation	Yearly
		After symptom resolution and off therapy	All sports	Yearly
AV block first and second degree, type 1	History, ECG, ET, 24-h Holter, Echo, (CMR, EP study selectively)	If no symptoms, no cardiac disease, with resolution during exercise.	All competitive sports	Not required
AV block second degree, type 2 or advanced		In the absence of symptoms, cardiac disease, ventricular arrhythmias during exercise, and of resting heart rate is > 40 bpm	All sports (endurance discouraged)	Yearly

Conclusion

Participation in sports has innumerable benefits, both physical and psychological. However, restriction from sports can significantly negatively impact psychological well-being and overall quality of life. Deciding whether to maintain, stop or facilitate an athlete's return to sport, especially after experiencing any incidents, a comprehensive approach is essential.

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Left ventricular hypertrophy and hypertrophic cardiomyopathy in athletes

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Summary

The changes in an athlete's heart are influenced by factors such as age, gender, ethnicity, and the type of cardiovascular training. It is crucial for clinicians to consider these variables when assessing athletes to guide appropriate investigations and differentiate between physiological and pathological conditions. This article addresses the challenges in distinguishing physiological left ventricular hypertrophy from pathological hypertrophic cardiomyopathy (HCM) in athletes, discussing the mechanisms behind both types of hypertrophy, along with clinical histories and investigative findings that aid in identifying pathology from electrocardiogram to echocardiography and cardiac magnetic resonance.

Keywords :
Athlete's heart, hypertrophic cardiomyopathy, suddencardiac death,Diastolic and systolic function, echocardiography,strain, cardiac magnetic resonance

Résumé

Les changements survenant dans le cœur d'un athlète sont influencés par des facteurs tels que l'âge, le sexe, l'ethnicité et le type d'entraînement cardiovasculaire. Il est crucial que les cliniciens prennent en compte ces variables lors de l'évaluation des athlètes afin de guider des investigations appropriées et de faire la distinction entre les conditions physiologiques et pathologiques. Cet article aborde les défis liés à la distinction entre l'hypertrophie ventriculaire gauche physiologique et la cardiomyopathie hypertrophique (CMH) pathologique chez les athlètes, en discutant des mécanismes derrière les deux types d'hypertrophie, ainsi que des antécédents cliniques et des résultats d'investigation qui aident à identifier la pathologie, allant de l'électrocardiogramme à l'échocardiographie jusqu' à l'IRM cardiaque.

Mots clés :
Cœur d'athlète, cardiomyopathie hypertrophique, mort subite, fonction diastolique et systolique, échocardiographie, déformation, résonance magnétique cardiaque.

Introduction

Intensive athletic training is associated with a spectrum of morphologic and functional cardiac changes (athlete's heart), considered to be physiologic adaptations to increased hemodynamic load and neurohormonal changes (1). In most athletes, morphologic cardiac changes are mild and do not raise clinical concern, but in some highly trained athletes, left ventricular (LV) remodeling may be substantial, prompting differential diagnosis with structural heart disease, most commonly hypertrophic cardiomyopathy(HCM). Indeed, accurate identification of HCM in athletes has relevant clinical implications, because this disease is one of the most common causes of athletic field deaths (2) and usually represents the basis for disqualification from competitive or professional sports(3,4). In this overlap between athlete's heart (AH) and disease states, the so called 'grey zone', the ability to differentiate physiological adaptation from pathological changes, is critical for the safety of athletes. False reassurance may have devastating consequences, whilst inappropriate investigations or false diagnosis and disqualification from sport have considerable financial and emotional implications (5).
Therefore, the aim of this article is to explore the various facets of the difference between athlete's heart and hypertrophic cardiomyopathy.

Why is it important to distinguish between Athlete's heart and Hypertrophic cardiomyopathy?

The main risk is the sudden cardiac death among athlete's, in fact hypertrophic cardiomyopathy is one of sudden death causes among athletes, that's why it is necessary to study the tools to differentiate between physiology from pathology.
Many etiologies can be responsible for sudden cardiac death among athletes (Table 1).

Table 1 Most common aetiologies of sudden cardiac death in athletes

Heritability	Structurally abnormal heart	Structurally normal heart
Inherited	Cardiomyopathies	Channelopathies
	HCM	Long QT syndrome
	ARVC	Brugada syndrome
	IDCM	Cathecol-aminergic polimorphic ventricular tachycardia
	Congenital coronary anomalies	
	Bridged coronary arteries	Wolff–Parkinson–White
	Aortic diseases	
Acquired	Myocardial ischaemia	Commotio cordis
	Myocarditis	Drugs and stimulants
	Cardiac sarcoidosis	Electrolyte imbalance
	Valvular heart disease	

Definitions

Athlete :

The ESC defines an athlete as 'an individual of young or adult age, either amateur or professional, who is engaged in regular exercise training and participates in official sports competition'. Similarly, the American Heart Association (AHA) and others define a competitive athlete as an individual involved in regular (usually intense) training in organized individual or team sports, with an emphasis on competition and performance (6,7)

Hypertrophic cardiomyopathy :

Hypertrophic cardiomyopathy (HCM) is defined as the presence of increased LV wall thickness (with or without Right ventricule hypertrophy) or mass that is not solely explained by abnormal loading conditions. (8)
Hypertrophic cardiomyopathy affects around 1:500 of the population, and the majority of the cases are caused by genetic mutations. In up to 60%, the disease follows an autosomal dominant inheritance due to mutations in cardiac sarcomere protein genes (9).

The Grey Zone :

A small proportion (2 %) of athletes who are white, male and compete in endurance sports have been reported to demonstrate LVH between 13 and 15 mm, which can also mimic morphologically mild HCM. Further studies revealed that ethnicity affects the degree of LVH, with up to 18 % of African American adult athletes exhibiting an LV wall thickness of >12 mm but never beyond 16 mm.
The 'grey zone' is therefore larger in African American athletes.

History and physical examination

While doing the investigations to differentiate between pathological and physiological left ventricular hypertrophy, a number of factors can be elucidated from initial history and examination, which may favour pathology or physiology.

A history of palpitations, dizziness or syncope, exertional chest discomfort, breathlessness out of proportion to the degree of physical effort or palpitations raises the suspicion of a pathological process. Likewise, a detailed family history is important and may be the main feature which then leads to subsequent investigations. Physical examination may often be normal, but sometimes in case of HCM we can find an ejection systolic murmur at the left sternal edge due to left ventricular outflow tract obstruction or signs of mitral regurgitation due to systolic anterior motion of the mitral valve. A family history of HCM in a first-degree relative in an athlete with LVH should raise the suspicion of HCM, because the disorder is inherited as an autosomal dominant trait.

Electrocardiogram

The importance of the electrocardiogram (ECG) 12-leads is crutial, it could give us early orientation about the difference between pathology and physiology. A variety of ECG changes are commonly observed in trained athletes with physiology LV remodeling; particular suspicion for HCM is raised by certain abnormalities such as marked left-axis deviation, left atrial enlargement pattern, deep Q waves, ST-segment depression, and diffuse T-wave inversion. Occurrence of such abnormal patterns in an athlete with LV hypertrophy should be viewed with suspicion and mandate careful diagnostic investigation and periodical follow-up. On the other hand, about 5% of trained athletes without LV hypertrophy or overt heart disease show distinctly abnormal ECG patterns consistent with HCM(11).
Main changes in the electrocardiogram:
• Physiological T wave inversion (TWI) in white athletes can be normal in leads V1–V2 and is more common in females.
• Physiological TWI in black athletes can be normal in leads V1–V4. The TWI is commonly associated with J point ST elevation.
• T wave inversion in lateral leads, ST segment depression and pathological Q waves warrant further investigation and are more likely to be associated with pathology.
In that sense, the most common, accessible, and cost-effective exams as a second-line examination are echocardiography, exercise stress test (EST), 24 h Holter ECG monitoring, and cardiopulmonary exercise testing (CPET). If the results of one or more of these second-line evaluations are highly suspicious or fall in the grey zone, a third-line evaluation is needed, which is represented by less accessible or more costly diagnostic techniques such as exercise stress echocardiography (ESE), CMR, coronary computer tomography (CCTA), genetic testing, single-photon emission computed tomography (SPECT), and positron emission, tomography (PET)(12) .(figure 1)

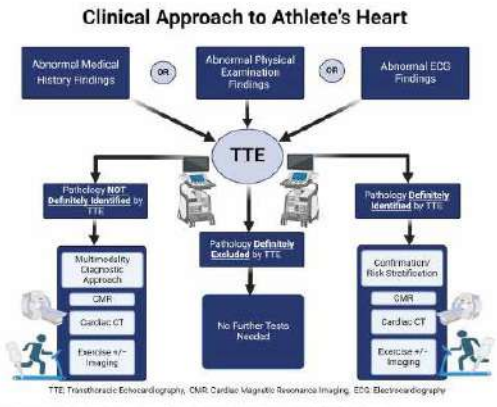


Figure 1. Clinical approach to the evaluation of athlete's heart.

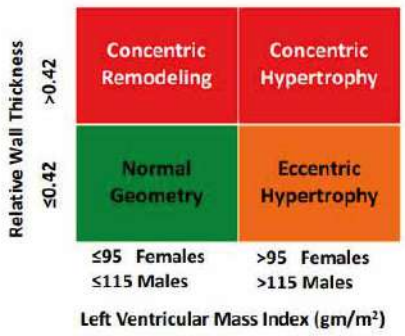
Echocardiographic data

Echocardiographic diagnosis of HCM in young competitive athletes may be challenging when the extent of LV hypertrophy is mild, falling into the so-called gray zone of uncertainty between physiologic LV remodeling and mild phenotypic expression of the disease. This differential diagnosis is a not uncommon clinical dilemma, and it is relevant because of the therapeutic and social implications that HCM diagnosis conveys, with the potential for disqualification from organized sports activities.

Left ventricular geometric changes in athletes

Left ventricular geometry and the pattern of hypertrophy can be determined using a combination of LV mass indexed (LVMI) to body surface area (BSA) and the relative wall thickness (RWT). RWT is calculated by summating septal and posterior wall thickness in diastole and dividing into the LV diastolic cavity dimension. LV geometry can be reported as 'normal', 'eccentric hypertrophy', 'concentric hypertrophy' or 'concentric remodelling' depending on the subjects RWT and LVMI (Figure 2).

Figure 2 :
Relative wall thickness (RWT)

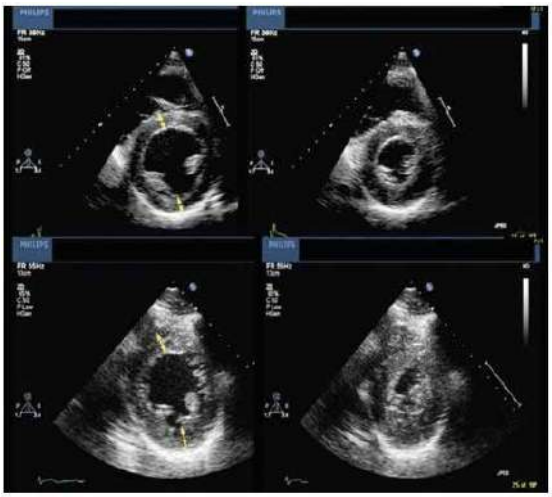


Many studies had shown that most athletes exhibit a normal LV geometry; Up to 10% of white athletes may exhibit concentric hypertrophy or concentric remodelling, which may be more prevalent in black athletes. Furthermore, most conclusions said that It is rare for athletes to have a RWT more than 0,48.

Left ventricular wall thickness

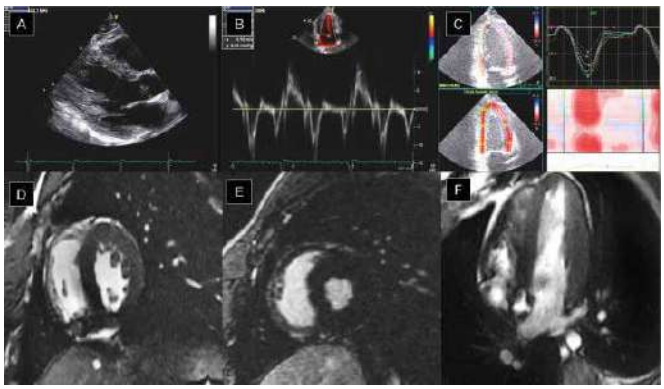
In athletes, left ventricular (LV) wall thickness is usually normal, but measurements between 13 and 16 mm, especially if asymmetric, may indicate mild cases of hypertrophic cardiomyopathy (HCM). Diagnosing left ventricular hypertrophy (LVH) involves calculating the LV mass index through wall and septum thickness measurements. Research by Sharma et al. found that elite adolescent athletes typically had greater LV wall thickness than sedentary individuals, with few exceeding 12 mm. In male athletes, LV wall thickness over 12 mm without ventricular dilation raises concerns for HCM. Assessment of LVEDD(left ventricule end diastolic diameter), systolic/diastolic function, and atrial size can help determine if hypertrophy is reversible and exercise-induced. (12).
LVEDD was found in many studies as an interesting index to use , in fact, among athletes it was found that the LV cavity was larger compared to HCM patients (Example Figure 3)

Figure 3 :
Mid-cavity para-sternal short axis views (diastole and systole) in an international cyclist (top) and a patient with morphologically mild hypertrophic cardiomyopathy (bottom). Showing a left ventricular wall thickness of 13 mm (arrows) in both individuals. However, note the athlete has an enlarged left ventricular cavity (60 mm) when compared with the patient with HCM (44 mm)



Advanced imaging techniques like speckle-tracking echocardiography (Global longitudinal strain : GLS) and cardiac magnetic resonance (CMR) aid in this differentiation. Speckle tracking examines LV twisting patterns, while CMR can reveal myocardial fibrosis, a characteristic of HCM but typically absent in AH.(12).
Echocardiography can sometimes be not enough to distinguish between Athlete's heart and HCM for that we can rely on the Cardiac magnetic resonance with the study of LV thickness and Late gadolinium enhancement to look for the fibrosis that represents a sign of a pathological case taking as an example(figure 4)

Figure 4 :
Transthoracic echocardiography (TTE) and cardiac magnetic resonance (CMR) from a patient with athlete's heart. TTE showed left ventricular hypertrophy and borderline left ventricle diameters (A), maintaining normal diastolic (B) and systolic function with normal GLS (C). CMR confirmed the diagnosis of athletic adaptation, showing concentric left ventricular hypertrophy most marked in the mid-region of the lateral wall (19 mm), without LGE (D–F). Left ventricle ejection fraction EF is 69%.



To sum up this point :

• The majority of athletes exhibit normal LVWT thickness (≤ 12 mm).
• Athletes can exhibit physiological hypertrophy but this is symmetrical and does not exceed 16 mm.
• It is unusual for white female athletes to have a LVWT 9-11 mm.
• Physiological hypertrophy is more commonly seen in black athletes.
• Athletes with HCM have a lower degree of hypertrophy than sedentary patients with HCM but still more than 85% have asymmetric hypertrophy more than 16 mm.

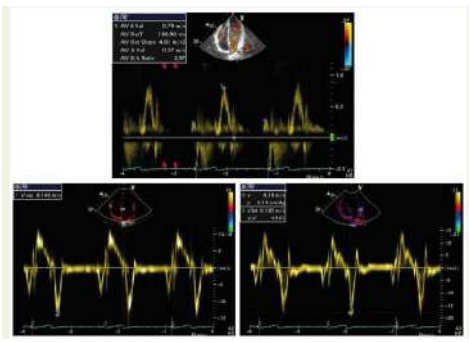
Systolic and diastolic function

In athletes, an increase in left ventricular (LV) mass is typically associated with a normal ejection fraction (EF) at rest, while systolic velocity (SV) is normal or even elevated due to increased preload (LV end-diastolic volume). Pulsed tissue Doppler provides further insights for a thorough evaluation of the functional characteristics of an athlete's heart, showing either normal or supranormal myocardial systolic performance at rest. A cutoff value of less than 9 cm/s for systolic peak velocity (s , averaged from four mitral annular sites) has demonstrated 87% sensitivity and 97% specificity in differentiating pathological left ventricular hypertrophy (LVH) due to arterial hypertension or hypertrophic cardiomyopathy (HCM) from athletic LVH. (13).

Additionally, LV diastolic function at rest may appear normal but is often supranormal compared to untrained individuals, particularly in endurance athletes (figure 5) . Typically, athletes exhibit a transmitral E/A ratio greater than 2, which helps distinguish physiological LVH from pathological LVH, the latter being marked by an E/A ratio less than 1 and a prolonged deceleration time of E velocity. Measurements of early diastolic myocardial velocity (e') and the e/a ratio for the basal septal and basal lateral walls are also found to be elevated in athletes. (13).

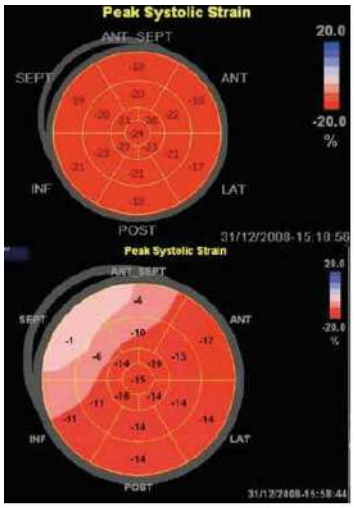
In contrast, patients with HCM show reduced e' velocity in both the hypertrophied septum and the normally thick lateral wall. Regional diastolic dysfunction (e'/a' < 1) was identified in 25% of myocardial segments in HCM patients, including those with non-hypertrophic walls, while no such dysfunction was observed in any segments of athletes. (13).

Figure 5 :
Supranormal LV diastolic function in a competitive runner. In the upper panel, standard Doppler-derived transmitral patterns shows an E/A ratio ¼ 2.97. In the lower panel, pulsed tissue Doppler-derived e' velocity is very high at both septal (left) and lateral mitral annulus (right) and E/e' ratio (¼ 4.9) is consistent with a normal diastolic function.



Measurement of myocardial deformation (strain imaging), by either color coded tissue Doppler or more recently two dimensional speckle tracking, has improved our ability to quantify regional myocardial function. A recent study demonstrated that in patients with HCM strain and strain rate are abnormal even in the absence of myocardial fibrosis on cardiac MRI. Conversely, studies in athletes with LVH reveal normal circumferential, radial, and longitudinal profiles raising the possibility that myocardial strain imaging is yet another echocardiographic modality that may facilitate the differentiation between athlete's heart and HCM(14)(figure 6)

Figure 6 :
'Bulls eye' plots of speckle tracking derived longitudinal strain in an athlete (top) and a patient with morphologically mild hypertrophic cardiomyopathy (bottom). Paler shades represent lower peak systolic strains. Mean global strain of the athlete was -20%, compared with -14% in the HCM patient. Specifically note the reduced peak strain values in the septum/antero-septum represented by paler shades of red.

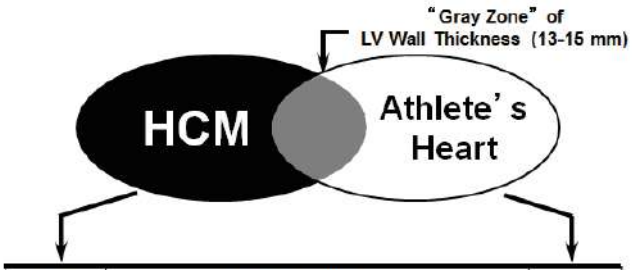


Cardiac magnetic resonance

Cardiac MRI is a valuable tool for differentiating athlete's heart from hypertrophic cardiomyopathy (HCM), it can offer improved image quality in subjects with poor echocardiographic windows. It is recognized as the gold standard imaging method for evaluating hypertrophic cardiomyopathy (HCM) in both athletes and non-athletes. Its capability to clearly identify left ventricular hypertrophy, detect underlying fibrosis, and employ advanced techniques such as T1 mapping places cardiac MRI in a unique position among imaging modalities. In trained athletes whose left ventricular wall thickness falls within a "grey zone," where physiological non-pathological left ventricular hypertrophy due to systemic training overlaps with mild phenotypic expression of hypertrophic cardiomyopathy (HCM), several factors must be considered to distinguish between these two conditions (Figure 7). In this context, cardiac magnetic resonance imaging (CMR) is becoming increasingly vital for differentiating HCM from athlete's heart for several reasons. First, when echocardiographic images are inconclusive, CMR offers the advantage of providing high-resolution measurements of LVWT. Additionally, CMR has proven beneficial in detecting areas of LVH that may not be visible on echocardiography,

especially when the increased wall thickness is confined to specific regions of the left ventricle, such as the anterior free wall, posterior septum, and apex.(15,16,17)

Figure 7 :
Clinical criteria used to distinguish HCM from athlete's heart. ECG: electrocardiogram; LA: left atrium; LV left ventricle; LVH: left ventricular hypertrophy; Modified from Maron BJ, Pelliccia A. The heart of trained athletes: cardiac remodeling and the risks of sports, including sudden death. Circulation 2006;114:1633-44



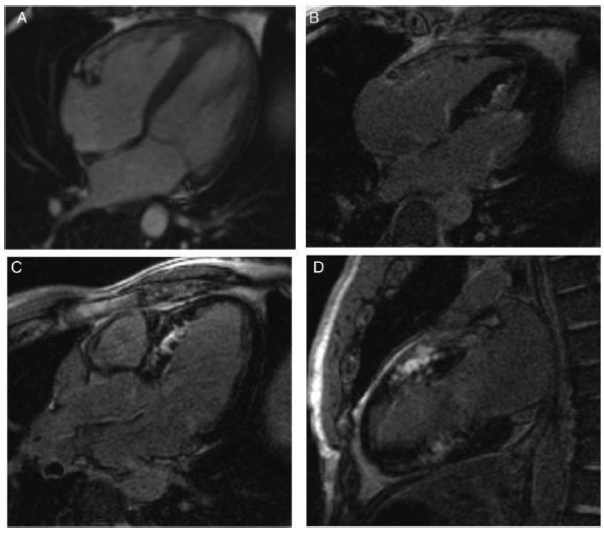
+	Focal Pattern of LVH	-
+	LV Cavity < 45mm	-
+	LV Cavity > 55mm	-
-	LA Enlargement	+
+	Bizarre ECG Patterns	-
+	Abnormal LV Filling	-
+	Female Gender	-
+	Family History of HCM	-
-	↓ Thickness with Deconditioning	+
-	Peak VO2 > 110% predicted	+
+	Late Gadolinium Enhancement	-

The late gadolinium enhancement (LGE) can be instrumental in distinguishing hypertrophic cardiomyopathy (HCM) from athlete's heart. LGE is observed in approximately half of individuals with HCM. In contrast, left ventricular remodeling associated with athlete's heart typically does not lead to focal areas of myocardial scarring or fibrosis, particularly in younger individuals. Several small studies based on CMR have shown that young competitive athletes often do not exhibit LGE. in an athlete under suspicion of having HCM, the presence of LGE on contrast-enhanced CMR supports a diagnosis of HCM. However, it is important to note that the absence of LGE cannot definitively rule out HCM, as it is present in half of patients diagnosed clinically with the condition (18,19)

A novel and promising technique in CMR, T1 mapping, assesses the overall extent of expanded extracellular space rather than just identifying regional areas of myocardial fibrosis through traditional LGE imaging. Thus, T1 mapping may become a valuable diagnostic tool for differentiating between pathological cardiovascular diseases like HCM and physiological remodeling associated with athlete's heart. (18,19)

Here's an example of diagnosed HCM in an athlete using CMR (figure 8)

Figure 8 :
Diagnosis of HCM in a 41-year old male endurance athlete by CMR. (A) The left ventricle is enlarged (LV end-diastolic diameter =58 mm), septal thickness is 15 mm. (B–D). LGE revealed myocardial fibrosis of anterior and inferior septum. Genetic analysis confirmed the diagnosis of HCM.



Guidelines ESC

Based on the latest European society of cardiology guidelines of practicing sports among athletes with HCM; the available data indicate that participation in vigorous exercise and competitive sport may be considered in a select group of predominantly adult patients who have mild morphology and a low-risk profile based on emerging evidence, the Task Force agreed to adopt a comparatively liberal approach, advocating that, after appropriate selection, some individuals with a low-risk profile may participate in high-intensity exercise and competitive sport after comprehensive expert evaluation and shared discussion, which highlights the unpredictable nature of exercise-related SCD in HCM. Sporting disciplines in which syncope may result in fatal accidental injury or danger to others are not recommended. (figure 9)

Figure 9 :
risk assessment in HCM

HCM		
High-intensity exercise and competitive sport should be considered in genotype-positive/ phenotype-negative individuals who seek to do so.[1124]	IIa	C
High-intensity exercise and competitive sport may be considered in asymptomatic low-risk[c] individuals with morphologically mild hypertrophic cardiomyopathy in the absence of resting or inducible left ventricular outflow obstruction and exercise-induced complex ventricular arrhythmias.[1107,1113,1125,1126]	IIb	B
High-intensity exercise, including competitive sport, is not recommended in high-risk individuals and in individuals with left ventricular outflow tract obstruction and exercise-induced complex ventricular arrhythmias.	III	C

Conclusion

Cardiac imaging is essential for detecting cardiovascular disease in athletes, but final diagnoses must consider factors like medical and sports history, training load, symptoms, age, gender, ECG results, and genetic analysis. The ongoing debate over the use of echocardiography in pre-participation screening for competitive athletes stems from its high cost-effectiveness ratio and the risk of false-positive results affecting athletes and their families. Limited research suggests that pocket-sized imaging devices for cardiac ultrasound may serve as an affordable initial assessment tool. When needed, advanced imaging techniques such as cardiac computed tomography (CCT), cardiac magnetic resonance (CMR), and nuclear imaging can provide further diagnostic and prognostic information, with CMR LGE, CMR T1 mapping, low-dose CCT, and new PET tracers showing the greatest potential.

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Atrial fibrillation and extrasystoles in athletes

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Summary

Moderate regular physical exercise improves cardiovascular health. However, athletes, despite their generally optimal health, are at risk for arrhythmias, notably extrasystoles and atrial fibrillation (AF), which can lead to higher morbidity and mortality. The physiological adaptations of the cardiovascular system in athletes, known as "athlete's heart" create favorable conditions for arrhythmias, particularly AF. AF is the most common arrhythmia in athletes and can increase with high-intensity endurance sports, especially in middle-aged male athletes. Extrasystoles can occur in athletes with healthy hearts as part of the athlete's heart syndrome and are generally benign. However, they may also indicate underlying heart disease carrying the risk of serious ventricular arrhythmias and SCD. Pre-participation screening for amateur athletes is vital to preventing SCD.

Keywords :
Atrial fibrillation, Extrasystoles, Athletes, Sudden cardiac arrest, The athlete's heart.

Résumé

Un exercice physique modéré et régulier améliore la santé cardiovasculaire. Cependant, les athlètes, malgré leur santé généralement optimale, sont exposés au risque d'arythmies, notamment d'extrasystoles et de fibrillation auriculaire (FA), qui peuvent entraîner une morbidité et une mortalité accrues. Les adaptations physiologiques du système cardiovasculaire des athlètes, connues sous le nom de « cœur d'athlète », créent des conditions favorables aux arythmies, en particulier à la FA. La FA est l'arythmie la plus fréquente chez les athlètes et peut augmenter avec les sports d'endurance de haute intensité, en particulier chez les athlètes masculins d'âge moyen. Les extrasystoles peuvent survenir chez des athlètes au cœur sain dans le cadre du syndrome du cœur d'athlète et sont généralement bénignes. Cependant, elles peuvent également indiquer une maladie cardiaque sous-jacente entraînant un risque d'arythmie ventriculaire grave et de mort subite du nourrisson. Le dépistage des sportifs amateurs avant leur participation est essentiel pour prévenir les troubles cardiaques graves.

Mots clés :
Atrial fibrillation, Extrasystoles, Athletes, Sudden cardiac arrest, The athlete's heart.

Introduction

Regular physical exercise offers numerous benefits to individuals of all ages. It significantly reduces the risk of metabolic disease and enhances cardiovascular health depending on its low, moderate, or intense level. It's linked to improved mental well-being and healthy aging. Athletes have long been symbols of optimal health, which makes the connection between physical activity and the occurrence of arrhythmia seem paradoxical. Extrasystoles and atrial fibrillation (AF) are the most common rhythm disorders and can affect both the general population and athletes. The study of these 2 conditions in athletes remains significant, due to their high morbidity and mortality. AF with its potential for thrombo-embolic complications, and certain types of extrasystoles that carry a risk of sudden cardiac arrest (SCA) which is the leading cause of sports and exercise-related mortality in athletes, underscores the importance of understanding and addressing these issues in athletic populations.

Pathophysiological pathways : "The Athlete's HEART"

The cardiovascular system undergoes several modifications as a consequence of exercise training, with acute responses, which occur within a few seconds of intensive exercise, and chronic adaptations that include more profound structural remodeling and are a result of long-term endurance training, described by scientists since 1899, as "The athlete's heart".[1]
The acute response to exercise training includes increases in heart rate, stroke volume, cardiac output, systolic blood pressure, and maximal oxygen consumption. On the other hand, the chronic cardiovascular adaptations to exercise result in structural remodeling of the heart chambers and vessels which enables an improved ability to deliver oxygen to working muscles over extended periods of exercise. It is commonly known that the primary factors influencing remodeling include age, sex, ethnic origin, body size, type of sport, and training volume, exhibiting a dose-response

relationship. A multivariate analysis of data from large populations of athletes reveals that non-genetic factors account for 75% of the variability in left ventricular cavity size and the remaining 25% remains unexplained and may be partially attributed to genetic factors. As a result, an athlete's structural adaptability may differ significantly from another's athletes. [2], [3]

The expression of cardiac remodeling is an increase in the size of the ventricles and atriums, associated with normal, or even improved, diastolic function in response to the state of volume and pressure overload, which can increase during exercise by five to sixfold, to all cardiac chambers with atrial stretch. The right ventricle is subject to a lower pressure overload than the left because pulmonary resistance is lower than systemic resistance. The cut-off level of physical activity at which changes appear is poorly defined, but it is known that even the course of 2 to 4 months, 4 hours or more per week of severe endurance exercise can lead to cardiac changes.[4] The structural changes observed in the athlete's heart are mainly atrial and ventricular dilatation, left ventricular hypertrophy, and increased cardiac mass with fibrosis and inflammatory changes. They create a favorable substrate for arrhythmia, particularly AF, and induce changes in conduction velocity. Knowing, the LV hypertrophy is partially reversible upon sport cessation. The most extreme increases in LV cavity dimension and/or wall thickness are observed in male athletes engaged in endurance sports such as rowing, cross-country skiing, cycling, and swimming. Conversely, female athletes typically experience a milder degree of hypertrophy.[5], [6]

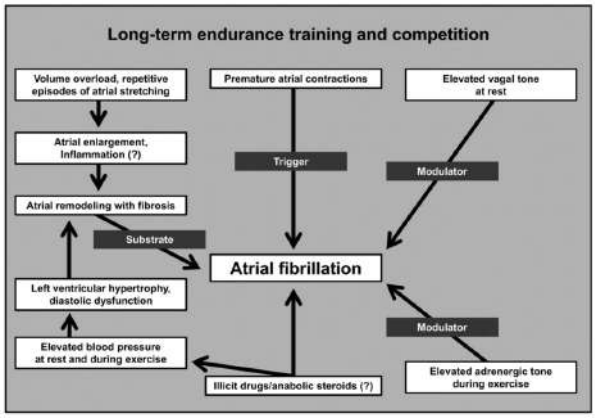
During exercise, the initial increase in heart rate is due to the suppression of parasympathetic vagal tone and activation of the sympathetic nervous system, which releases catecholamines such as epinephrine and norepinephrine. These hormones stimulate cardiac contractility and rate, increasing cardiac output.[4] Endurance athletes, on the other hand, exhibit an increase in vagal tone, resulting in resting sinus bradycardia, first-degree and Mobitz type 1 second-degree atrioventricular block, partial right bundle branch block, and the early repolarization pattern, which can act as a modulator, predisposing normal hearts to the onset of vagal AF by shortening the atrial refractory period and increasing AF dispersion, thus creating the conditions for re-entry. The increase in cardiac output in endurance athletes is not due to an increase in heart rate, but rather to an increase in systolic ejection volume as a result of higher blood volume, dilated heart chambers, and improved myocardial relaxation and contraction capacity.[5]

More physiological pathways of arrhythmias in athletes' hearts are described with less supporting evidence. For example, At the molecular level, training is associated with an over-expression of the isoform α of the myosin-heavy chain, which enhances cardiac contractility. The expression of microRNAs (miRNAs) for the α myosin heavy chain is an early event in the adaptation to chronic exercise and occurs before cardiac growth is evident. The miRNAs are significant mediators of pro-arrhythmogenic remodeling and could be proposed as potential biomarkers of AF. Following a marathon, their serum levels differ, with trained elite runners showing higher levels than less-trained non-elite runners. This could account for the difference between possibly dangerous severe endurance activity and good moderate exercise.[6], [7]

There is a difference in heart remodeling between male and female athletes, regardless of age and sports discipline. In general, compared to male, female athletes have less significant alterations. The exact mechanisms behind these differences are still unclear. Several hypotheses have been suggested, including variations in body size, blood pressure response to exercise, and circulating androgen concentration.[8]

It should be noted that physical exercise may simply contribute to uncovering genetic structural heart diseases such as hypertrophic cardiomyopathy (HMC), congenital coronary anomalies, arrhythmogenic right ventricular cardiomyopathy, Wolf-Parkinson-White syndrome, channelopathies, etc.

Factors influencing the development of atrial fibrillation in athletes[9]



Atrial fibrillation in athletes :

AF is the most common sustained cardiac arrhythmia in adults with a prevalence between 2 and 4%, and its incidence gradually increases. It is also notably the most prevalent arrhythmia observed in athletes.[10] AF is a supraventricular tachyarrhythmia with uncoordinated atrial electrical activation and ineffective atrial contraction. Diagnosing AF requires documentation through an electrocardiogram (ECG). Its asymptomatic and paroxysmal nature makes diagnosis difficult. Holter monitoring should be considered for its early detection. To link symptoms to the existence of an arrhythmia during Holter monitoring, athletes must exercise at their typical volume and maintain a detailed activity journal. New screening tools and event recorders are used for AF screening and have made it easier, which benefits the general population and athletes. AF may result in complications such as stroke, peripheral arterial embolism, and heart failure.[11]

Several risk factors contribute to the onset of AF, including age and co-morbidities such as hypertension, diabetes, heart failure (HF), and coronary artery disease (CAD).[11] The relationship between exercise and AF is complex and continues to be a topic of significant debate, particularly regarding the intensity of physical activity. A non-linear relationship between physical activity and AF, often depicted as a U-shaped curve, suggests that moderate exercise may offer protective benefits against the onset of AF. Moderate, regular physical activity is recommended to prevent AF by modifying many of its predisposing factors. In contrast, both a sedentary lifestyle and intense physical activity have been linked to an increased risk of developing AF. The association between AF and high-intensity endurance sports has not been confirmed in women.[10], [12], [13]

Age, volume, intensity, type, and level of sport may contribute to AF risk in athletes. The incidence of AF appears to be higher among elite athletes, particularly those with long-term or endurance sports, such as running and cycling. For athletes who have accumulated more than 4500 hours of training in their lifetime, the risk of developing AF can be as high as 82%.[6] High-intensity endurance athletes have been found to experience AF 2 to 10 times more frequently than sedentary individuals.[14] Furthermore, the frequency of AF in athletes tends to increase with age, which is more common in middle-aged male athletes (45-65 years) compared to younger athletes (18-30 years); it doubles with each advancing decade.[6], [15], [16] The risk of AF was 8.8 times higher in marathon runners (mean age 39±9 years) and 1.7 times higher in vigorous joggers (less than 50 years old) as compared to controls. [17], [18]

Although AF in the absence of structural heart disease or other identifiable causes such as hyperthyroidism or alcohol use that should be corrected, is considered a benign condition, but can be associated with severe and disturbing symptoms.[17] Two strategies, rate control, and rhythm control have to be discussed with the athlete. Also, considering the link between practicing endurance sports and AF, it is important to talk about lowering training volume and intensity. Rate control is equally complicated. Physical activity should be stopped and rate control optimized as soon as symptoms appear. Class III antiarrhythmic drugs are often ineffective (sotalol) or not suitable for younger patients (amiodarone). Beta-blockers may not be well tolerated (even prohibited in the special case of competitive athletes). Class I antiarrhythmic drugs can prevent AF recurrences but should not be used alone due to the risk of causing atrial flutter with potentially dangerous 1:1 atrioventricular conduction, leading to high ventricular rates, slowed intraventricular conduction, and hemodynamic instability. Thus, they may be considered only for acute cardioversion, as a 'pill-in-the-pocket' approach. These patients should refrain from sports as long as AF persists, and until two half-lives of the antiarrhythmic drug have passed. In the rhythm control strategy, before catheter ablation, at least one round of AAD therapy should be attempted. Catheter ablation, however, may be used as a first-line treatment in certain athletes whose physical performance is significantly compromised and who intend to continue competing in sports. All sports participation at any level is possible in asymptomatic persons if there is evidence of appropriate rate control while in AF, determined by an exercise stress test or ECG monitoring during training or competition. Anticoagulation prescription depends on overall risk, as assessed by the recently updated CHA2DS2-VA Score.[13], [19]

Extrasystoles in athletes :

Atrial and ventricular premature beats may occur in an athlete with a healthy heart as part of athlete's heart syndrome and are most often benign resulting from the activity of an automatic and idiopathic focus. Still, they could also be a sign of an underlying heart disease, for which there is a poor prognosis, even in those who are asymptomatic, with a risk of serious ventricular arrhythmias and SCD.[20]

On the 24-hour ambulatory ECG, in the healthy general population, we find atrial premature beats (APBs) with a prevalence of up to 60.8% and ventricular premature beats (VPBs) up to 43.3%, and their incidence increases with age.[21] Only a few athletes have more than 100 APBs in 24 hours. Some studies have established a correlation between long-term endurance exercise and the APB load. They could act as a trigger for AF in particular or other supraventricular tachyarrhythmias. However, APBs pass unnoticed in most cases, even though they might cause subjective palpitations without corresponding hemodynamic deterioration Other than a physical examination, thyroid function tests, and 12-lead ECG, echocardiography is useful in determining the functional status of the heart and identifying potential underlying structural abnormalities. There is no need for therapy if structural heart disease is not present. All sports are permitted, whether they are competitive or recreational.[9], [22], [23]

PVBs are not related to the volume or duration of physical activity, and their prevalence, which also increases with age, is similar to that of non-athletes. On 24-hour Holter tracings, the prevalence of PVBs varies from 0.05% to 1.1%. Frequent or complex PVBs are rare, with rates similar to those of sedentary people: 10% in athletes under 35 years of age (versus 11% in sedentary people) and 26% in athletes over 30 years of age (versus 23% in sedentary people).[13]

The morphology of VPBs provides clues to the presence of a possible etiology. PVBs with specific features morphology (origin from the apex or left ventricular free wall or left ventricle), high burden, complexity (couplets, triplets, or non-sustained runs), multifocal origin, and/or increased rate during exercise should raise the possibility of electrical, ischemic, or structural heart disease. There is no absolute threshold for the number of PVBs that can be used as a cutoff to detect underlying disease. VPBs with LBBB morphology and an inferior axis indicate a right ventricular outflow tract origin consistent with idiopathic right ventricular outflow tract arrhythmia, which is a benign condition. On the other hand, VPBs with an LBBB morphology and superior axis indicate a right ventricular free wall or apex origin and are more suggestive of Arrhythmogenic right ventricular cardiomyopathy. Athletes with more than one VPB on a resting 12-lead ECG must get additional testing. This evaluation includes 24-hour Holter monitoring and cardiac structure and function assessment using echocardiography. Ambulatory ECG registration is an invaluable diagnostic tool for differentiating benign arrhythmias commonly seen in the athlete's heart syndrome and life-threatening arrhythmias caused by cardiac pathologies. Intense exercise may paradoxically act as a trigger for life-threatening ventricular arrhythmias in the presence of underlying CVD.[13] Compared to single PVCs, NSVT is more likely to be indicative of an underlying disease. When NSVTs found during rest disappear with exercise, this suggests a likely benign condition.[24], [25]

A study has shown that among asymptomatic competitive athletes with more than 2000 PVBs per day, there is a 30% possibility of discovering an underlying structural or cardio-genetic disease. The athletes with complex and frequent ventricular arrhythmia who do not have structural heart

disease represent the most challenging grouping when it comes to clinical management guidelines. These people provide a clinical challenge regarding their eligibility to participate in sports. [20]
Complex forms of ventricular ectopy are more common in athletes. A significant correlation was found between the grade of arrhythmia and the total number of ventricular extrasystoles recorded during the 24-hour monitoring period in athletes. However, no significant correlation was observed between the grade of arrhythmia or the number of ectopic beats and the dimensional or functional echocardiographic parameters.[26]

Recommendations for exercise in individuals with PVCs or non-sustained ventricular tachycardia[13]

Recommendations	Class[a]	Level[b]
In exercising individuals with ≥2 PVCs on a baseline ECG (or ≥1 PVC in the case of high-endurance athletes) thorough evaluation (including a detailed family history) to exclude underlying structural or arrhythmogenic conditions is recommended.[503,522]	I	C
Among individuals with frequent PVCs and non-sustained VT a thorough investigation with Holter monitoring, 12-lead ECG, exercise test, and suitable imaging is recommmended.[503]	I	C
It is recommended that all competitive and leisure-time sports activities are permitted, with periodic re-evaluation in individuals without familial or structural underlying disease.[503]	I	C

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Doping and arrhythmia :

Doping in sports is the administration of active substances or their metabolites or the use of methods prohibited by the World Anti-Doping Agency (WADA), which updates its list of prohibitions at least once a year. To combat doping and to protect the rights of athletes, WADA was founded in 1999. Unfortunately, athletes continue to use doping to enhance their performance, reduce anxiety, increase muscle mass, reduce weight, or mask the use of other drugs during testing. The substances and methods vary from sport to sport. For example, in precision sports such as archery and shooting, beta-blockers, which reduce heart rate and muscle tremors, are considered doping because they enhance performance. WADA bans a substance or method if it meets at least two criteria: enhancement of sporting performance, danger to the athlete's health, or contradiction with the spirit of sport. When treating athletes, physicians must be aware of the possibility of improved performance.[27], [28]
The abuse of doping substances and methods has been associated with the occurrence of numerous severe side effects that vary based on the type of drug, dosage, duration of use, and individual sensitivity. In doping, doses are typically much higher than therapeutic levels, and combining multiple drugs is common, increasing the risk of side effects. Cardiovascular side effects are the most deleterious, with the occurrence of myocardial infarction, thrombosis, heart failure, arrhythmias, and sudden cardiac death. Many substances are considered to be pro-arrhythmogenic. [29]
Androgenic-anabolic steroids (AAS), for example, are pro-arrhythmic due to the direct structural changes to the myocardium, exacerbated by hypertension from mineralocorticoid-like effects. The resulting cardiac hypertrophy is persistent, unlike the temporary hypertrophy from exercise. AAS also has electrophysiological effects, including prolonged depression of the cardiac stimulation threshold and altered electrolyte levels, which can lead to atrial and ventricular fibrillation. They affect the cardiac sympathetic nervous system, increasing vulnerability to severe arrhythmias. Another hormone known to have a pro-arrhythmic effect is human growth hormone (HGH), secondary to the myocardial hypertrophy associated with interstitial fibrosis it causes. The most frequently observed arrhythmia, often occurring during physical activity, are AF, supraventricular and ventricular ectopic beats, sustained and non-sustained ventricular tachycardia, and ventricular fibrillation.[28], [29], [30] Athletes tend to use diuretics mainly to mask the presence of drugs in the urine, which in turn leads to electrolyte imbalance that can cause QT interval prolongation and arrhythmias. Other substances that have been implicated in the development of rhythm disorders include cocaine and ephedrine-containing preparations such as mahuang, the "herbal ecstasy", as well as cannabis preparations such as marijuana and hashish.[29]
Awareness has to be raised, even among physicians, about which effects are due to training and which are side effects of prohibited substances.

Pre-participation screening (PPS) for amateur athletes :

Regular physical exercise lowers the risk of atherosclerotic cardiovascular disease and enhances cardiorespiratory fitness, body composition, and psychological well-being. However, sports, especially high-intensity sports, have been linked to an increased risk of SCD in those who are already at risk. Thus, athletes must benefit from a minimum of cardiovascular (CV) safety during their sports activities, to avoid catastrophic SCD, which can often be prevented by PPS for amateur athletes. Such screening should now be a challenge for all leading sports organizations. Early detection of life-threatening disorders in athletes can reduce CV morbidity and mortality by offering athletes at high risk a medical or surgical treatment, or an implantable defibrillator as a primary prevention. [13], [31]
PPS is widely used for athletes of all skill levels in various nations, although it differs according to local regulations. The screening methods proposed by the ESC guidelines vary according to the athlete's age and are limited. CV screening in young athletes by history physical examination and a 12-lead electrocardiogram presents difficulties and limitations. Several studies have demonstrated that ECG is still more effective than just history and physical examination when performed by experienced clinicians, mostly due to the capability to identify arrhythmogenic conditions at risk (cardiomyopathies and channelopathies). Echocardiography can detect other structural abnormalities, but to date, there is insufficient data to justify the addition of echocardiography in the regular screening of children and young athletes. For athletes aged over 35, screening should focus on atherosclerotic coronary disease, which is more prevalent in this age group. They should understand the nature of cardiac prodromal symptoms and the need for prompt medical attention. Risk factor assessment for CV

disease may identify higher-risk individuals who warrant additional testing. Routine screening for ischemia with exercise testing in asymptomatic adults is not recommended. Thus, exercise testing should be reserved for symptomatic athletes or those deemed at high risk of CAD based on the ESC Systematic Coronary Risk Evaluation system. Exercise testing may also be useful to evaluate the blood pressure (BP) response to exercise, the occurrence of exercise-induced arrhythmias, and to assess symptoms or physical performance and its relation to exercise training. A screening ECG may still discover undiagnosed cardiomyopathies and primary electrical disorders in older athletes.[13], [32]

It is important to point out that certain sports associations such as the Fédération International de Football Association (FIFA) and the Union of European Football Associations (UEFA), while the International Olympic Committee (IOC) have their protocols.

Conclusion

The incidence of AF is increased by intense and endurance sports activity, while moderate exercise improves cardiovascular health and reduces co-morbidities and the risk of developing AF. The overlap between physiological adaptation to exercise and cardiomyopathies remains significant, hence the need for further studies with long-term follow-up data to investigate the long-term rhythmic consequences of the athlete's heart as a means of better prevention to avoid the tragic deaths of high-level athletes.

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Amateur athlete, professional athlete : What cardiological evaluation ?

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Summary

Sudden cardiac death (SCD) is a devastating event that can affect seemingly healthy individuals, including athletes. Cardiac conditions may increase the risk of SCD, especially when abnormal electrical or structural issues combine with the physical stress of intense exercise, potentially leading to fatal arrhythmias. The risk of SCD is higher in men and older athletes. In younger athletes, common causes include cardiomyopathies, channelopathies, and congenital coronary artery anomalies, while atherosclerotic coronary artery disease is more prevalent in older individuals.
Pre-participation cardiac screening offers a potential method to detect conditions that may lead to SCD in athletes. European guidelines recommend the inclusion of a 12-lead ECG in these screenings, whereas American guidelines emphasize personal and family history alongside a physical examination. Cardiac screening protocols should be tailored to different age groups, as the causes of SCD vary between younger and older athletes.
Although early detection of silent cardiac diseases is key to prevention, the widespread availability of Automated external defibrillator (AEDs) and timely Cardiopulmonary resuscitation (CPR) remain essential measures in preventing SCD tragedies.

Keywords :
Sudden cardiac death (SCD) , Automated external defibrillator (AEDs), Cardiopulmonary resuscitation (CPR)

Résumé

La mort subite d'origine cardiaque (MSC) est un événement tragique qui peut toucher des personnes apparemment en bonne santé, y compris des athlètes. Certaines pathologies cardiaques peuvent augmenter le risque de MSC, particulièrement lorsque des anomalies électriques ou structurelles sont associées aux exigences physiques de l'exercice intense, ce qui peut entraîner des arythmies fatales. Le risque de MSC est plus élevé chez les hommes et les athlètes plus âgés. Chez les jeunes athlètes, les cardiomyopathies, les canalopathies et les anomalies congénitales des artères coronaires sont des causes fréquentes de MSC, tandis que la maladie coronarienne athérosclérotique est plus courante chez les individus plus âgés.
Le dépistage cardiaque avant la participation sportive constitue une méthode possible pour identifier les affections cardiaques pouvant entraîner une MSC chez les athlètes. Les directives européennes recommandent l'utilisation d'un ECG à 12 dérivations dans ces dépistages, tandis que les directives américaines se concentrent sur les antécédents personnels et familiaux ainsi que sur l'examen physique. Le contenu du dépistage cardiaque doit être adapté selon les groupes d'âge, car les causes de MSC varient entre les jeunes et les athlètes plus âgés.
Bien que l'identification précoce de maladies cardiaques silencieuses puisse aider à prévenir ces tragédies, la mise en place de politiques favorisant l'accès généralisé aux défibrillateurs externes automatisés (DEA) et la réanimation cardiopulmonaire (RCP) rapide reste cruciale.

Mots clés :
mort subite d'origine cardiaque (MSC), défibrillateurs externes automatisés (DEA), réanimation cardiopulmonaire (RCP)

Introduction

Regular physical activity is well-known for its beneficial effects on overall health. Research consistently shows that individuals who exercise regularly face a lower risk of mortality from various causes, as well as reduced incidence of cardiovascular diseases, cancer, and metabolic disorders. However, sudden cardiac death (SCD) can still occur in otherwise healthy individuals, including athletes.

Sudden cardiac death (SCD) is defined as an unexpected death resulting from cardiac causes that occurs within one hour of the onset of an acute change in cardiovascular status, or within 24 hours in unwitnessed cases, in the absence of external contributing factors. [1]
In athletes, a variety of conditions can lead to SCD. While atherosclerotic coronary artery disease is more common in those over 35, primary cardiomyopathies and ion channelopathies are frequently seen in younger individuals. [2]

To potentially prevent sudden cardiac death in athletes, it's crucial to implement policies that focus on two main areas: screening for cardiac conditions in asymptomatic individuals and improving the likelihood of successful resuscitation during cardiac emergencies [3].
Cardiac screening for athletes is commonly practiced in many countries and is endorsed by various international organizations. The screening typically includes a personal and family medical history along with a physical examination (H&P), often accompanied by a resting 12-lead electrocardiogram (ECG). Some protocols also recommend additional tests such as echocardiograms or stress ECGs.[4]

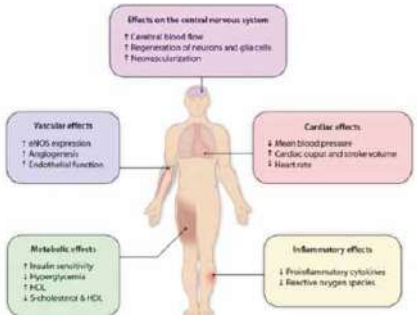
2. Amateur vs professional athletes (2)[5]:

The European Society of Cardiology (ESC) defines an athlete as anyone who regularly engages in exercise training and participates in official sports competitions, whether amateur or professional. Similarly, the American Heart Association (AHA) describes competitive athletes as those involved in rigorous, organized training aimed at competition. Athletes can compete at various levels, from youth leagues to the Olympics.In contrast, recreational athletes participate in sports primarily for enjoyment, while competitive athletes are highly trained and focused on performance.
Athletes are often categorized by their training volume:
• 'elite' athletes train for over 10 hours a week
• 'competitive' athletes for more than 6 hours,
• and 'recreational' athletes for over 4 hours.
• However, these categories can overlap, as some recreational athletes may train more than certain professional athletes.

3. The 'paradox of sport' :

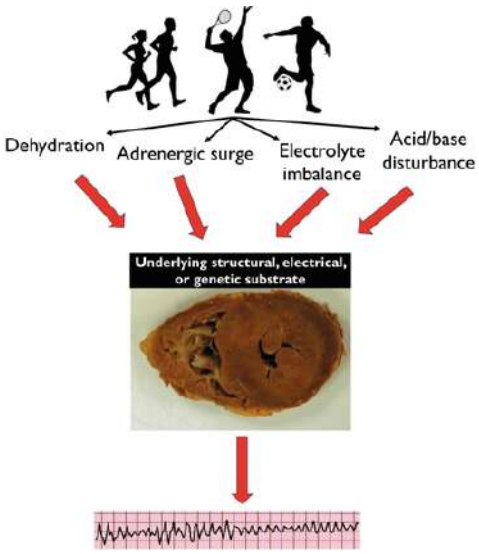
Regular physical exercise provides numerous health benefits, including reduced mortality, lower risks of metabolic disorders (like diabetes and dyslipidemia), and certain cancers. Aerobic activity can also decrease the risk of acute myocardial infarction and sudden cardiac death (figure 1) However, the "paradox of sport" highlights that, while exercise is beneficial, vigorous physical exertion temporarily raises the risk of acute cardiac events, with the chance of SCD doubling during and shortly after intense activity. Despite this, the health benefits far outweigh the risks. Pre-participation screening can help detect individuals with underlying cardiac conditions, thereby reducing their risk of SCD in sports[6].

Figure 1 :
The general effects of exercise. [7]



Exercise can act as a trigger for lethal ventricular tachyarrhythmias, particularly in individuals with underlying, often unsuspected,
cardiac conditions [8]. Most sudden cardiac events in athletes are caused by malignant tachyarrhythmias, typically ventricular fibrillation (VF) or ventricular tachycardia (VT) that progresses to VF, particularly in those with arrhythmogenic disorders such as hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, and channelopathies. Intensive exercise and participation in competitive sports can precipitate these dangerous ventricular arrhythmias in predisposed individuals [9].
The development of these arrhythmias is influenced by three interconnected factors [Coumel's triangle of arrhythmogenesis] (figure2): [10]
1. Arrhythmogenic substrates: Congenital or acquired cardiovascular diseases.
2. Triggers: For endurance athletes, these include extreme physical exertion or trauma to the chest during sports events.
3. Pathophysiological conditions: Factors like autonomic nervous system dysregulation, excessive adrenergic stimulation, dehydration, heat stress, and electrolyte imbalances, which are common during intense physical activity.

Figure 2 :
Common physiological effects of intense exercise [2]



4. Incidance of scd in athletes : The significance of the challenge

A prospective Italian study determined that athletes had a relative risk of SCD of 2.5 compared to non-athletes [9]. Estimates of sudden cardiac death (SCD) incidence among competitive athletes vary significantly, ranging from 1 in 1 million to 1 in 5,000 per year, according to the European Society of Cardiology (ESC)[5].

A comprehensive study evaluating sports-related sudden cardiac death (SrSCD) in the young adult population (ages 18–35) revealed a higher frequency of events in recreational athletes compared to elite athletes, with a notable predominance among males[11]. However, research by Risgaard et al. [12]found no significant difference in SrSCD rates between non-competitive and competitive athletes.

Notably, the study also observed an increase in SrSCD incidence among individuals aged 36–49 in both competitive and non-competitive categories compared to those aged 12–35. This suggests that age, rather than competitive status, may be a significant factor in the increased riskIt's important to emphasize that sport itself is not considered the primary cause of increased mortality in athletes. Instead, physical activity may act as a trigger for life-threatening ventricular arrhythmias and sudden cardiac arrest in individuals who are predisposed to these conditions due to underlying cardiovascular abnormalities. Identifying and managing these risks is crucial for prevention. [12].

A large regional registry in the UK found that out of 748 SCD cases among individuals participating in sports (more than 3 hours of organized training per week), only 98 (13%) were women [13]. Possible explanations for the higher incidence of SCD in male athletes include differences in physiological cardiac adaptation to exercise, chamber remodeling, and a higher prevalence of myocardial fibrosis, which can create a substrate for dangerous arrhythmias [9].

An Italian study indicated that 45% of all SCD events in athletes occurred during football, with a similar percentage (39%) reported in occupied Palestine [14][15].

5. Aetiology of sudden cardiac death during exercise : The Hidden Depths of the Iceberg

In young athletes under the age of 35, a wide range of cardiovascular abnormalities contribute to sudden cardiac death (SCD). The most common are cardiomyopathies, such as hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), and dilated cardiomyopathy (DCM). Other frequent causes include congenital anomalies of the coronary arteries. Less commonly, other conditions are responsible, such as aortic rupture related to Marfan syndrome, myocarditis, valvular diseases (like aortic stenosis and mitral valve prolapse), ion channel disorders (such as long or short QT syndrome, Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia), and blunt chest trauma leading to malignant arrhythmias (commotio cordis)[16]. Table 1

In athletes > 35 years the primary cause of death, as in the general population, is coronary artery disease (CAD) figure 3

FIGURE 3 :
Aetiologies of SCD in athletes and age. The size of the circles relate to the relative frequency of SCD caused by the respective pathology. Age 35 has been used as a threshold as this is the most frequently used age in the literature; however, a clear line in terms of age is difficult to draw [2]

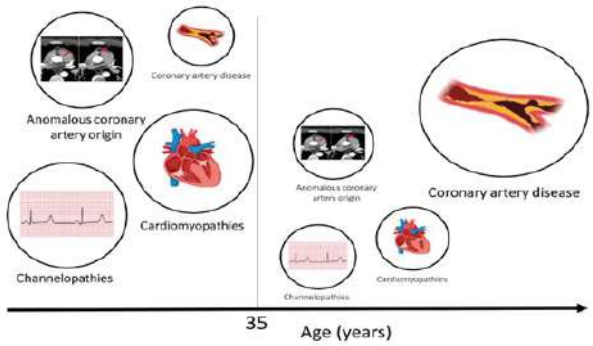


Table 1 :
causes of sudden cardiac death(SCD) in young athletes [17]

Congenital/genetic pathology	
Disease of the myocardium	Hypertrophic cardiomyopathy Arrhythmogenic ventricular cardiomyopathy Dilated cardiomyopathy
Coronary artery disease/anomalies	Congenital coronary artery anomalies Premature atheromateus coronary artery disease
Cardiac conduction tissue abnormalities	Wolff-Parkinson-White syndrome Right ventricular outflow tachycardia
Valvular heart disease and disorders of the aorta	Mitral valve prolapse Congenital aortic stenosis Marfan syndrome
Ion channelopathies	Congenital long QT syndrome Catecholaminergic polymorphic ventricular tachycardi Brugada syndrome
Acquired causes	
Infections (myocarditis)	
Drugs (cocaine, amphetamine)	
Electrolyte disturbances (hypokalemia or hyperkalemia)	
Hypothermia	
Hyperthermia	
Trauma (commotio cordis)	

6. Screening modalities for cardiovascular disease in young athletes :

Both the American Heart Association/American College of Cardiology (AHA/ACC) and the European Society of Cardiology (ESC) advocate for pre-participation cardiac screening to identify cardiac conditions that could lead to sudden cardiac death (SCD) [5] [18].

A. History and Physical Examination

A comprehensive pre-participation evaluation should include a focused medical history and physical examination, as recommended by various organizations, including the AHA/ACC, ESC, International Olympic Committee (IOC), and FIFA [18] [19] [20] [21].Most healthcare providers utilize standardized questionnaires, such as the Preparticipation Examination Monograph (PPE-4) or the AHA's 14-point screening questions. These questionnaires aim to identify congenital and genetic cardiovascular diseases that heighten the risk of adverse outcomes during exercise.

However, they can yield a significant false-positive rate, with poor sensitivity and low positive predictive value, leading to unnecessary secondary testing [23]. The physical examination focuses on diagnosing specific conditions, such as valvular diseases and hypertrophic cardiomyopathy, using techniques like cardiac auscultation in various positions and assessing for signs of Marfan syndrome and hypertension [22].

Table 2 :
Comparison of AHA-14 Questionnaire and PPE-4 Monograph [22]

AHA-14 questionnaire	PPE-4 monograph
Personal History	Heart Health Questions About You
1. Chest pain/discomfort/tightness/pressure related to exertion	6. Have you ever had discomfort, pain, tightness, or pressure in your chest during exercise?
2. Unexplained syncope/near syncope	5. Have you ever passed out or nearly passed out during or after exercise?
3. Excessive and unexplained dyspnea/ fatigue or palpitations, associated with exercise	12. Do you get more tired or short of breath more quickly than your friends during exercise? 10. Do you get lightheaded or feel more short of breath than expected during exercise? 7. Does your heart ever race or skip beats (irregular beats) during exercise?
4. Prior recognition of a heart murmur 5. Elevated systemic blood pressure	8. Has a doctor ever told you that you have any heart problems? If so, check all that apply: ▫ High blood pressure ▫ A heart murmur ▫ High cholesterol ▫ A heart infection ▫ Kawasaki disease Other: _____
6. Prior restriction from sports	1. Has a doctor ever denied or restricted your participation in sports for any reason?
7. Prior testing for heart disease, ordered by a physician	9. Has a doctor ever ordered a test for your heart? (For example, ECG/EKG, echocardiogram) *11. Have you ever had an unexplained seizure?*
Family History	Heart Health Questions About Your family
8. Premature death (sudden and unexpected or otherwise) before 50 yrs. of age attributable to heart disease in >1 relative	13. Has any family member or relative died of heart problems or had an unexpected death before age 50 yrs. (including drowning, unexplained car accident, or sudden infant death syndrome)?
9. Disability from heart disease in a close relative < 50 yrs of age	
10. Hypertrophic or dilated cardiomyopathy, long QT syndrome or other ion channelopathies, Marfan syndrome, or clinically significant arrhythmias; specific knowledge of genetic cardiac condition in family member	14. Does anyone in your family have hypertrophic cardiomyopathy, Marfan syndrome, arrhythmogenic right ventricular cardiomyopathy, long QT syndrome, short QT syndrome, Brugada syndrome, or catecholaminergic polymorphic ventricular tachycardia? *15. Does anyone in your family have a heart problem, pacemaker, or implanted defibrillator?* *16. Has anyone in your family had unexplained fainting, unexplained seizures, or near drowning?*
Physical Examination	Physical Examination
11. Heart murmur	a. Heart • Murmurs (auscultation standing, supine, ± Valsalva) • Location of point of maximal impulse
12. Femoral pulses to exclude coarctation	b. Pulses • Simultaneous femoral and radial pulses
13. Physical stigmata of Marfan syndrome	c. Appearance • Marfan stigmata (kyphoscoliosis, high-arched palate, pectus excavatum, arachnodactyly, arm span > height, hyperlaxity, myopia, MVP, aortic insufficiency)
14. Brachial artery blood pressure (sitting position)	d. Blood pressure

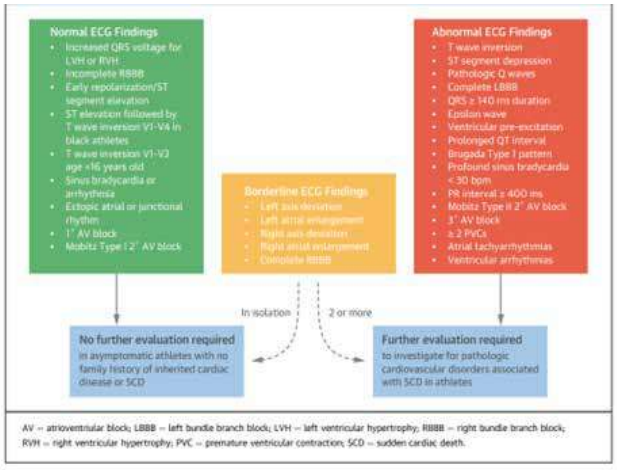
B. Electrocardiogram (ECG)

The inclusion of an ECG in screening remains debated, yet many sports and cardiology associations, including the ESC, IOC, and FIFA, recommend it as part of a comprehensive cardiac evaluation. A 12-lead ECG significantly improves the detection of cardiomyopathies and channelopathies, which often present with ECG abnormalities [14]; [24].

The sensitivity of screening improves dramatically with ECG inclusion—from under 25% for history and physical examination to around 90% for ECG [25]. However, ECG changes in athletes can result from normal adaptations to exercise, making correct interpretation crucial to distinguish physiological changes from pathological ones.

International standards for ECG interpretation have improved the quality of cardiovascular care for athletes, reducing false positives and associated screening costs [26]. which should be applied only to those exercising vigorously for at least 4– 8 hours per week. ECG findings in athletes are classified as normal, abnormal and borderline

Figure 4 :
international consensus standards for electrocardiographic interpretation in athletes [26]



• Normal ECG Findings do not require any further evaluation
• Some ECG findings previously characterized as abnormal have since been reclassified as a borderline or possibly normal finding in an athlete . Specifically, the ECG findings below are considered normal if present in isolation, but if two or more borderline findings are present then further evaluation with an echocardiogram to exclude cardiomyopathy is recommended [27]
• Abnormal ECG findings require further evaluation to exclude a pathologic cardiac disorder. The ECG abnormality alone does not necessarily imply a disease process, but rather indicates that more evaluation is needed. Temporary restriction from sport should be considered while the secondary evaluation is completed. Some ECG abnormalities may be the first manifestation of a cardiomyopathy before morphologic changes are present. Thus, serial evaluation is recommended on an annual basis for athletes with abnormal T wave inversion, ST segment depression, and/or pathologic Q waves [28]
Despite its benefits, the clinical limitations of ECG screening include imperfect sensitivity and specificity. For instance, while the sensitivity of ECG for detecting hypertrophic cardiomyopathy is about 90%, congenital coronary artery anomalies—another common cause of SCD—are often silent on ECG [29] [30].

C. Multimodality Imaging

At present, no major sporting or professional organizations, aside from FIFA, advocate for the routine use of multimodality imaging in pre-participation cardiovascular screening (PPCS). However, various screening programs, universities, national teams, and professional organizations have incorporated imaging into their standard screening protocols. The efficacy of a multimodality imaging strategy has not been thoroughly tested or validated, leaving its potential impact on PPCS uncertain. The transthoracic echocardiogram (TTE) is the most frequently considered imaging method, with proponents arguing that a focused TTE approach could enhance screening sensitivity and possibly decrease the number of days athletes miss from sports due to the need for further cardiovascular evaluations. [22]

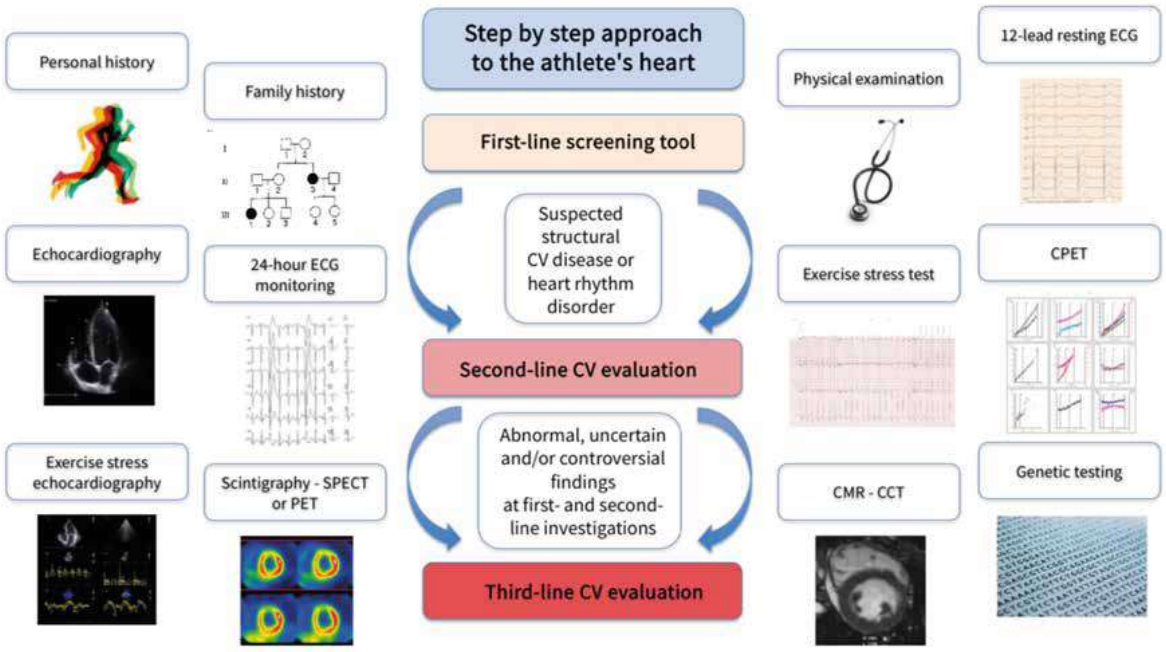
Echocardiography can detect the origin of the coronary arteries in the majority of cases . Furthermore, congenital abnomalities such as bicuspid aortic valve, aortopathies and mitral valve prolapse can be detected, which can pose an increased risk depending on the sporting discipline. Due to its advantages, some major sporting bodies have implemented echocardiography in their mandated CV evaluation protocols. The Fédération Internationale de Football Association (FIFA) and the Union of European Football association (UEFA), as well as the Union Cycliste Internationale (UCI) mandate an echocardiography in the screening of their athletes . Concerns against performing an echocardiography are mostly additional cost, and an increased rate of false-positive findings due to training-induced changes. [31] (29)
While there is ongoing debate regarding the necessity of multimodality imaging in routine athlete screenings, such as exercise ECG testing, ambulatory ECG monitoring, cardiac magnetic resonance imaging (MRI), or computed tomography coronary angiography (CTCA), it is recognized as a crucial element of secondary testing when initial exams reveal abnormalities. Common reasons for pursuing secondary imaging include unexplained prior syncope, a family history of sudden cardiac death among first-degree relatives, exertional chest pain or excessive shortness of breath, and certain abnormal ECG findings, as outlined in the recent international guidelines[22] [26]

7. Recommandations for cardiovascular evaluation :

A. Step by step approach to the athlete's heart [32]

The optimal way to begin the PPS should include family and personal history collection, physical examination and 12-lead resting ECG, as proposed by several scientific societies and as shown as a first-line evaluation in our step-by-step approach (Figure 5). Only if in the presence of clinical suspicion or ECG abnormalities, it may be necessary to request other examinations, as indicated in the International Recommendations for Electrocardiographic Interpretation in athletes . In that sense, the most common, accessible and cost-effective exams as a second-line examination are echocardiography, Exercise-Stress Test (EST), 24-hours Holter ECG monitoring and cardiopulmonary exercise testing (CPET). If the results of one or more of these second-line evaluations are highly suspicious or fall in the grey zone, a third-line evaluation is needed, which is represented by less accessible or more costly diagnostic techniques such as exercise stress echocardiography (ESE), cardiovascular magnetic resonance (CMR), coronary computer tomography (CCT), genetic testing, single photon emission computed tomography (SPECT) and positron emission tomography (PET).
• First-Line Evaluation : family / personal history and physical examination , Electrocardiogram
• Second-Line Evaluation : Echocardiogram, Exercise-Stress Test, 24-Hours ECG Holter Monitoring, Cardiopulmonary Exercise Test
• Third-Line Evaluation : Exercise-Stress Echocardiography, Cardiovascular Magnetic Resonance, Coronary Computed Tomography, Nuclear Imaging Techniques, Genetic Testing in Athletes

Figure 5 :
The step-by-step approach in the management of athlete's heart [32].
CV, cardiovascular; ECG, electrocardiogram; CPET, cardiopulmonary exercise test; CMR, cardiac magnetic resonance; CCT, cardiac computer tomography; SPECT, single photon emission computer tomography; PET, positron emission tomography.



B. Recommendations Based on Age

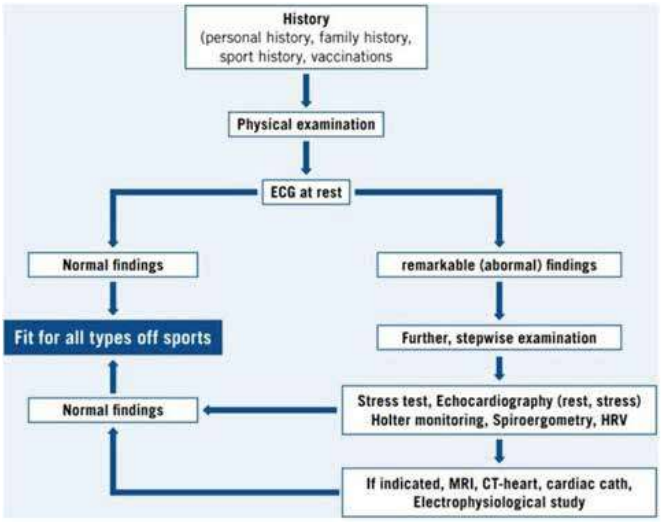
a) Recommendations for cardiovascular evaluation in healthy athletes under the age of 35

Under the aegis of the French Society of Cardiology (SFC), a group of French experts met and proposed a text specifying the content of the systematic cardiovascular examination for any subject aged between 12 and 35 wishing to take part in competitive sport. This text validates the usefulness of systematic resting ECG screening in this population. However, its content is more nuanced than the European recommendations. The French group recommends systematic renewal of the ECG every 3 years between the ages of 12 and 20 (the age at which genetic cardiomyopathies most frequently emerge) and then every 5 years between the ages of 20 and 35[33] .

b. Recommendations for cardiovascular evaluation of athletes over the age of 35

The ESC guidelines (2) recommend the following Four Steps for Cardiovascular Evaluation and Exercise Recommendations (figure 8) (table 4):

figure 6 :
Flow chart of preparticipation examination as recommended by EFSMA . HRV, heart rate variability.[34]



• Step 1 : Assessment of risk of CVD :

To evaluate an individual's risk of subclinical cardiovascular disease (CVD), accumulated risk can be calculated using established tools like the SCORE risk charts (figure7) [35][36], taking into account key factors such as very high total cholesterol, low-density lipoprotein (LDL) levels, diabetes mellitus, and a strong family history of CVD. This assessment allows for categorizing cardiovascular risk from low to very high (table 3) [5].

Figure 7 :
SCORE charts for European populations
at high cardiovascular disease (CVD) risk[35][36]

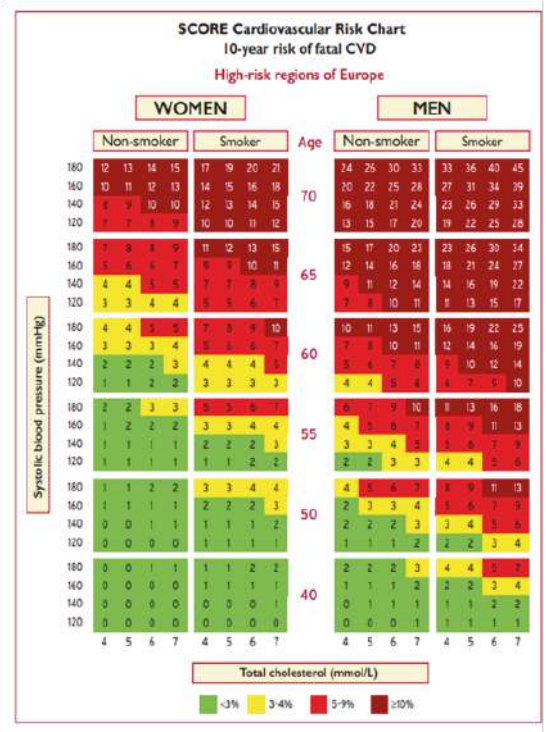


Table 3 :
Cardiovascular risk categories [5]

Very high-risk	People with any of the following: • Documented ASCVD, either clinical or unequivocal on imaging. Documented ASCVD includes previous ACS (MI or unstable angina), stable angina, coronary revascularization (PCI, CABG, and other arterial revascularization procedures), stroke and TIA, and peripheral arterial disease. Unequivocally documented ASCVD on imaging includes those findings that are known to be predictive of clinical events, such as significant plaque on coronary angiography or CT scan (multivessel coronary disease with two major epicardial arteries having >50% stenosis), or on carotid ultrasound. • DM with target organ damage,* or at least three major risk factors, or early onset of T1DM of long duration (>20 years). • Severe CKD (eGFR <30 mL/min/1.73 m²). • A calculated SCORE ≥10% for 10-year risk of fatal CVD. • FH with ASCVD or with another major risk factor.
High-risk	People with: • Markedly elevated single risk factors, in particular TC >8 mmol/L (>310 mg/dL), LDL-C >4.9 mmol/L (>190 mg/dL), or BP ≥180/110 mmHg. • Patients with FH without other major risk factors. • Patients with DM without target organ damage,* with DM duration ≥10 years or another additional risk factor. • Moderate CKD (eGFR 30–59 mL/min/1.73m²). • A calculated SCORE ≥5% and <10% for 10-year risk of fatal CVD.
Moderate-risk	Young patients (T1DM <35 years; T2DM <50 years) with DM duration <10 years, without other risk factors. Calculated SCORE ≥1% and <5% for 10-year risk of fatal CVD.
Low-risk	Calculated SCORE <1% for 10-year risk of fatal CVD.

• step 2 : Consideration of intensity of physical activity (figure8)

Habitually active individuals at low or moderate risk face no restrictions regarding exercise, including competitive sports. Sedentary individuals or those at high or very high risk may participate in low-intensity exercise without additional evaluation. However, those in these groups planning to engage in high-intensity exercise, as well as certain individuals considering moderate-intensity exercise, should undergo a physical examination, a 12-lead ECG, and an exercise stress test. The purpose of the exercise test is to identify significant coronary artery disease (CAD) and detect any exercise-induced arrhythmias.[5]

figure 8 :
intensity of physical activity [5]

• step 3 :Investigation including maximal exercise test , functional imaging or CTCA

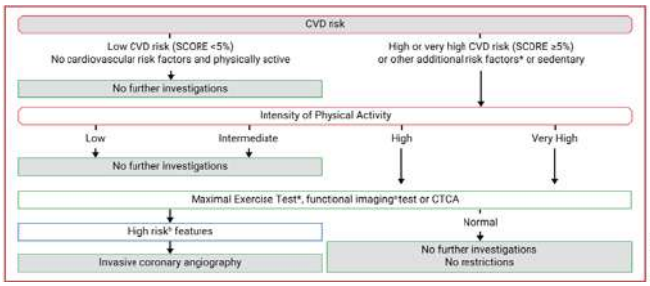
Currently there is no evidence for incorporating routine cardiac imaging in preparticipation screening among asymptomatic individuals aged >35 years old with a normal exercise stress test. However, in asymptomatic adults considered to be at high risk or very high risk (diabetes, strong family history of CAD, previous risk assessment suggesting high risk for CAD) a functional imaging test or coronary computed tomography angiography (CCTA) should be considered in the risk assessment (Figure 8). Identification of atherosclerotic CAD should prompt aggressive management of risk factors and preventive medical treatment. Among individuals with proven obstructive CAD, further assessment and treatment is indicated[5].

• step 4 : Further diagnostic testing and treatement in selected individuals : invasive coronary angiography in the presence of high risk features [5]:

- Single-photon emission computed tomography: area of ischaemia >_10% of the left ventricular myocardium

- stress echocardiography: >_3 of 16 segments with stress-induced hypokinesia or akinesia
- stress cardiovascular magnetic resonance: >_2 of 16 segments with stress perfusion defects or >_3 dobutamine-induced dysfunctional segments
- coronary computed tomography angiography (CCTA): three-vessel disease with proximal stenoses; left main disease

Figure 8 :
Proposed algorithm for cardiovascular assessment in asymptomatic individuals aged >35-years-old with risk factors for cardiovascular disease and possible subclinical chronic coronary syndrome before engaging in sports.[5]



*Consider functional test or CCTA if exercise stress test is equivocal or the ECG is uninterpretable.

Table 4 :
Recommendations for cardiovascular evaluation and regular exercise in healthy individuals aged >35 years[5]

Recommendations	Class[a]	Level[b]
Among individuals with low to moderate CVD risk, the participation in all recreational sports should be considered without further CV evaluation.	IIa	C
Cardiac screening with family history, symptoms, physical examination, and 12-lead resting ECG should be considered for competitive athletes.	IIa	C
Clinical evaluation, including maximal exercise testing, should be considered for prognostic purposes in sedentary people and individuals with high or very high CV risk who intend to engage in intensive exercise programmes or competitive sports.	IIa	C
In selected individuals without known CAD who have very high CVD risk (e.g. SCORE>10%, strong family history, or familial hypercholesterolaemia) and want to engage in high- or very high-intensity exercise, risk assessment with a functional imaging test, coronary CCTA, or carotid or femoral artery ultrasound imaging may be considered.	IIb	B

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CCTA = coronary computed tomography angiography; CV = cardiovascular; CVD = cardiovascular disease; SCORE = Systematic Coronary Risk Evaluation.
[a]Class of recommendation.
[b]Level of evidence.

8. Summary of Recommendations According to Scientific Societies:

Currently, a pre participation screening (PPS) is also enforced in the United States (history andphysical examination only) and Israel and is advised by theEuropean Society of Cardiology (ESC)and the Association ofEuropean Paediatric Cardiology (AEPC). Furthermore, although a PPS is not mandatory in most countries, several major sportsorganizations (e.g., FIFA, UEFA, and NBA) either recommend orenforce a cardiovascular evaluation that includes at least history,physical examination, and 12-lead resting ECG [37]

Table 5 :
Existing published recommendations for cardiac screening of athletes [37]

Guidelines/position statements from expert groups				
US	American Medical Society for Sports Medicine (AMSSM)[7]	All	Young **competitive** athletes, aged 12 +	H&P, ECG optional (interpreted by expert)
US	National Collegiate Athletic Association (NCAA)[14]	All NCAA sports	College athletes, usually aged 17 +	H&P, noting that some institutions include ECG
US	American Academy of Family Physicians, American Academy of Pediatrics, American College of Sports Medicine (ACSM), AMSSM, American Orthopaedic Society for Sports Medicine and the American Osteopathic Academy of Sports Medicine[39]	All	PPE recommended for athletes (broadly defined) from grade school and up, from age 6 +	H&P
US	American Heart Association, American College of Cardiology[40]	All	Competitive athletes	H&P
Australia & New Zealand	Australasian College of Sport and Exercise Physicians (ACSEP)[13]	All	Young **elite (professional, semi-professional)** athletes, aged 16 +	H&P, ECG
Brazil	Brazilian Society of Cardiology and Brazilian Society of Exercise and Sports Medicine[24]	All	Young **professional** athletes aged 12 + (also amateur athletes)	H&P, ECG
Canada	Canadian Cardiovascular Society and Canadian Heart Rhythm Society[41]	All	Young **competitive** athletes (engaged in regular training with high CV demands, likely ≥10 h/week), no age specified	History; consider physical examination as an adjunct component ECG only if indicated from H&P
Europe	European Heart Rhythm Association (EHRA) and European Association of Preventive Cardiology (EAPC), endorsed by APHRS, HRS, and SOLAECE[5]	All	All athletic participants of any age (performing regular intense exercise), no age specified	H&P, ECG
Europe	Association of European Paediatric Cardiology[42]	All	Young competitive athletes when they start competitive sport	H&P, ECG
World sporting organizations				
World	Federation Internationale de Football Association (FIFA)[15]	Football (soccer)	PCMA mandatory for any player in a FIFA tournament, commencing with under 17 teams	H&P, ECG and echocardiogram
Europe	Union of European Football Associations (UEFA)[43]	Football (soccer)	Highly recommended for players in UEFA competitions (mandatory for some) (i.e. elite level), no age specified	H&P, ECG Echocardiogram and stress ECG (every 2 years)
World	International Olympic Committee (IOC)[44]	Olympic sports	PHE recommended for Olympic athletes. Minimum age depends on the country,[45] generally aged 13 +	H&P, ECG
World	World Netball[46]	Netball	Age 16 +	H&P, ECG
World	World Rugby[47]	Rugby: Community level National/international	Recommended from age 14 + Recommended for national level players under age 20	History only H&P Consider ECG if logistically possible
		World Rugby tournaments	Screening mandatory	H&P, ECG recommended (mandatory in some tournaments)
World	Union Cycliste Internationale (UCI)[48]	Cycling	Elite, age not specified	H&P, ECG Echocardiogram and stress ECG for men's world teams and pro-teams

Conclusion

The primary goal of the Pre-Participation Evaluation (PPE) is to detect clinically silent cardiac conditions that may lead to cardiac arrest or sudden cardiac death, particularly in relation to exercise training and competitive sports participation.
The cardiological evaluation, which includes clinical history, physical examination, and a 12-lead ECG, has been shown to provide superior diagnostic accuracy compared to history and physical examination alone. Strong scientific evidence supports that the 12-lead ECG significantly enhances the diagnostic effectiveness of the PPE, primarily by identifying arrhythmogenic conditions at risk, such as cardiomyopathies and channelopathies. Current data suggest that routine use of echocardiography or other imaging techniques does not substantially improve the diagnostic capability of PPE as a mass screening method and is not considered cost-effective [21].

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Sport and congenital heart disease: a dangerous connection?

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Summary

Congenital heart disease (CHD) encompasses a variety of structural heart defects that hinder effective blood circulation. Advances in surgical and medical management have significantly improved survival rates, allowing many individuals with CHD to lead active lives. Alarmingly, only about 19% of these patients receive guidance on recommended physical activity, leading to a predominantly sedentary lifestyle. Traditional exercise recommendations for this group tend to be overly restrictive, lacking robust evidence, even though physical activity has been shown to provide significant cardiovascular benefits. Recent studies indicate that, following appropriate screening, physical activity and exercise can be both safe and beneficial for most individuals with CHD. The American Heart Association (AHA) and the European Society of Cardiology (ESC) provides updated guidelines advocating for individualized exercise plans tailored to the specific types of heart defects and the overall functional capacity of patients. The purpose of this contemporary article is to elucidate the benefits and risks of sports participation in patients with CHD and provide up-to-date guidelines for safe engagement.

Keywords :
Congenital heart disease; fitness; sports; physical activity; aerobic training, resistance training, Fontan circulation, tetralogy of Fallot, transposition of the great arteries, pulmonary hypertension; quality of life; sudden cardiac death.

Résumé

Les cardiopathies congénitales (CHD) englobent une variété de défauts structurels du cœur qui entravent l'efficacité de la circulation sanguine. Les progrès de la prise en charge chirurgicale et médicale ont considérablement amélioré les taux de survie, permettant à de nombreuses personnes atteintes d'une cardiopathie congénitale de mener une vie active. Il est alarmant de constater qu'environ 19 % seulement de ces patients reçoivent des conseils sur l'activité physique recommandée, ce qui se traduit par un mode de vie essentiellement sédentaire. Les recommandations traditionnelles en matière d'exercice physique pour ce groupe ont tendance à être trop restrictives et manquent de preuves solides, même s'il a été démontré que l'activité physique apporte des bénéfices cardiovasculaires significatifs. Des études récentes indiquent qu'après un dépistage approprié, l'activité physique et l'exercice peuvent être à la fois sûrs et bénéfiques pour la plupart des personnes atteintes de coronaropathie. L'American Heart Association (AHA) et la Société européenne de cardiologie (ESC) ont publié des lignes directrices actualisées préconisant des plans d'exercice individualisés adaptés aux types spécifiques de malformations cardiaques et à la capacité fonctionnelle globale des patients. L'objectif de cet article contemporain est d'élucider les avantages et les risques de la pratique sportive chez les patients atteints de coronaropathie et de fournir des directives actualisées pour un engagement en toute sécurité.

Mots clés :
Cardiopathie congénitale ; forme physique ; sports ; activité physique ; entraînement aérobique, entraînement à la résistance, circulation de Fontan, tétralogie de Fallot, transposition des grandes artères, hypertension pulmonaire ; qualité de vie ; mort cardiaque subite.

Introduction

Congenital heart disease (CHD) includes various structural abnormalities of the heart that can affect its ability to pump blood efficiently. It is estimated to affect around 1% of newborns [1]. Advances in surgical and medical management have significantly improved survival rates, allowing many individuals with CHD to lead active lives. Exercise training (ET) is a planned program of physical activity (PA) designed to boost or preserve fitness, performance, or health.

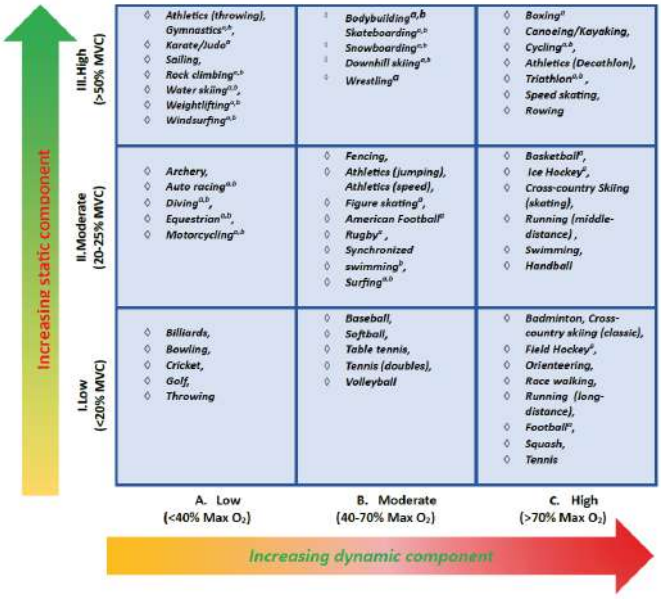
Nonetheless, participation in sports for this subgroup of patients presents potential risks that necessitate careful evaluation. Fewer than 20% of congenital heart disease patients receive advice on recommended PA. As a result, many adopt a sedentary lifestyle, often due to being overly protected and unclear on the type and intensity of exercise they should engage in [2]. Recommendations on exercise in this subgroup of patients are usually pretty restrictive without having clear evidence for this. This is especially relevant considering that sports participation has been shown to provide considerable cardiovascular benefits for both the general population and individuals with heart issues [3].

However, a small but growing body of literature has demonstrated that PA and exercise are safe and beneficial for the vast majority of people with CHD following appropriate screening [4]. American Heart Association (AHA) and European Society of Cardiology (ESC) provide a classification of the static and dynamic components of the currently most practised sports based on the type and severity of CHD, functional status, and overall health; Table I illustrates this classification with adaptations for the sports carried out in Morocco.

Studies carried out on patients with CHD indicate that the majority of people participating in training programs and receiving appropriate recommendations reveal a significant improvement in their exercise capacity and psychological state [3]. Varied and complex forms of CHD, along with the limited availability of evidence-based recommendations, make it difficult to apply a uniform approach to prescribing exercise. The current challenge is to ensure safe participation in regular physical activity in order to avoid the detrimental effects associated with a sedentary lifestyle.

The purpose of this contemporary article is to elucidate the benefits and risks of sports participation in patients with CHD, focusing on the most common congenital heart diseases, and provide up-to-date guidelines for safe engagement.

Table I.
Classification of sports depending on cardiovascular needs (based on combined static and dynamic components) [3], based on American Heart Association recommendations, adapted to sports carried out in Morocco.



This classification is based on peak static and dynamic components achieved during competition. It should be noted, however, that higher values may be reached during training. The increasing dynamic component is defined in terms of the estimated percent of maximal oxygen uptake (MaxO2) achieved and results in an increasing cardiac output. The increasing static component is related to the estimated percent of maximal voluntary contraction (MVC) reached and results in an increasing blood pressure load.

a: danger of corporal collision; b: increased risk in the event of a syncope.

Assessing Congenital Heart Disease: Types, Repairs, and Exercise Considerations

Most individuals with CHD have undergone surgical repair of their lesions; however, many still have significant cardiac residuals that must be considered when prescribing exercise training (ET). The type of CHD lesion, the method of repair, and the overall function of the heart and valves can all affect how a patient responds to ET. This section outlines common causes of CHD and their implications for ET, as well as typical surgical repair techniques and their effects on hemodynamic status. Table II outlines a classification of various types of CHD and their associated physiological stages, derived from the guidelines set forth by AHA and ESC [5] [6].

Table II.
Adult congenital heart disease anatomic and physiological Classification [7].

Anatomic classification*		
I. Simple	II. Moderate Complexity	III. Great Complexity (or Complex)
◊ Native disease • Isolated small ASD • Isolated small VSD • Mild isolated pulmonic stenosis ◊ Repaired conditions • Previously ligated or occluded ductus arteriosus • Repaired secundum ASD or sinus venosus defect without significant residual shunt or chamber enlargement • Repaired VSD without significant residual shunt or chamber enlargement	◊ Repaired or unrepaired conditions • Aorto-left ventricular fistula • Anomalous pulmonary venous connection, partial or Total • Anomalous coronary artery arising from the pulmonary artery • Anomalous aortic origin of a coronary artery from the opposite sinus • AVSD (partial or complete, including primum ASD) • Congenital aortic valve disease • Congenital mitral valve disease • Coarctation of the aorta • Ebstein anomaly (disease spectrum includes mild, moderate, and severe variations) • Infundibular right ventricular outflow obstruction • Ostium primum ASD • Moderate and large unrepaired secundum ASD • Moderate and large persistently patent ductus arteriosus • Pulmonary valve regurgitation (moderate or greater) • Pulmonary valve stenosis (moderate or greater) • Peripheral pulmonary stenosis • Sinus of Valsalva fistula/aneurysm • Sinus venosus defect • Subvalvar aortic stenosis (excluding HCM; HCM not addressed in these guidelines) • Supravalvar aortic stenosis • Straddling atrioventricular valve • Repaired tetralogy of Fallot • VSD with associated abnormality and/or moderate or greater shunt	• Cyanotic congenital heart defect (unrepaired or palliated, all forms) • Double-outlet ventricle • Fontan procedure • Interrupted aortic arch • Mitral atresia • Single ventricle (including double inlet left ventricle, tricuspid atresia, hypoplastic left heart, any other anatomic abnormality with a functionally single ventricle) • Pulmonary atresia (all forms) • TGA (classic or d-TGA, CC-TGA, or l-TGA) • Truncus arteriosus • Other abnormalities of atrioventricular and ventriculoarterial connection (i.e., crisscross heart, isomerism, heterotaxy syndromes, ventricular inversion)
Physiological Stage		
A	B	C	D
• NYHA FC I symptoms • No hemodynamic or anatomic sequelae • No arrhythmias • Normal exercise capacity • Normal renal/hepatic/pulmonary function	• NYHA FC II symptoms • Mild hemodynamic sequelae (mild aortic enlargement, mild ventricular enlargement, mild ventricular dysfunction) • Mild valvular disease • Trivial or small shunt (not hemodynamically significant) • Arrhythmias not requiring treatment • Abnormal objective cardiac limitation to exercise	• NYHA FC III symptoms • Significant (moderate or greater) valvular disease, moderate or greater ventricular dysfunction (systemic, pulmonic, or both) • Moderate aortic enlargement • Venous or arterial stenosis • Mild or moderate hypoxemia/cyanosis • Hemodynamically significant shunt • Arrhythmias controlled with treatment • Pulmonary hypertension (less than severe) • End-organ dysfunction responsive to therapy	• NYHA FC IV symptoms • Severe aortic enlargement • Arrhythmias refractory to treatment • Severe hypoxemia (almost always associated with cyanosis) • Severe pulmonary hypertension • Eisenmenger syndrome • Refractory end-organ dysfunction

*This list is not exhaustive; other conditions may be important in individual patients. ACHD indicates adult congenital heart disease; AP, anatomic and physiological; ASD, atrial septal defect; AVSD, atrioventricular septal defect; CC-TGA, congenitally corrected transposition of the great arteries; CHD, congenital heart disease; d- TGA, dextro-transposition of the great arteries; FC, functional class; HCM, hypertrophic cardiomyopathy; l-TGA, levo-transposition of the great arteries; NYHA, New York Heart Association; TGA, transposition of the great arteries; and VSD, ventricular septal defect. Reprinted from Journal of the American College of Cardiology, Vol 73, Stout et al, 2018 AHA/ACC Guideline for the Management of Adults With Congenital Heart Disease: Executive Summary A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines, page 1504, © 2019 [6].

A. Atrial Septal Defects and Ventricular Septal Defects

Atrial septal defects (ASDs) involve an abnormal opening in the atrial septum, which typically results in a left-to-right blood flow shunt. This shunt pattern can shift under certain conditions, such as when additional congenital heart anomalies are present or when the defect is associated with complications like pulmonary vascular disease [7]. Ventricular septal defects (VSDs) are characterized by a defect in the ventricular septum, resulting in a left-to-right shunt unless conditions like Eisenmenger syndrome develop or right ventricular outflow obstruction is present [7].

Patients with an isolated small or repaired ASD or VSD with normal ventricular and valve function usually have no exercise limitations, as they have a very low risk of adverse responses to physical activity. However, those with pulmonary hypertension may experience long-term complications such as valve dysfunction and ventricular overload, which must be carefully evaluated when prescribing exercise training based on their severity [7].

B. Bicuspid Aortic Valve

Bicuspid aortic valve (BAV) is the most prevalent congenital heart defect, affecting about 1-2% of the population [8]. Characterized by having only two cusps instead of the normal three, BAV often leads to complications like aortic stenosis and regurgitation. Before initiating an ET program, it's crucial to screen for aortic enlargement and valve dysfunction, including the quantification of associated aortic stenosis or insufficiency, while also assessing ventricular size and function. Patients with no significant aortic dilation (aortic diameter < 40 mm and z-score < 2) or notable valve issues can typically engage in physical activities. However, those with larger aortic diameters or higher z-scores should avoid high-load exercises, and regular monitoring is essential for all patients to track potential progression of their condition [8]. Having a bicuspid aortic valve generally does not preclude individuals from participating in sports. However, the long-term effects of high-intensity training on athletes with this condition remain unclear. Intense and sustained exercise might theoretically lead to early deterioration of the abnormal valve and dilation of the ascending aorta. Therefore, early detection in these athletes is crucial for monitoring and preventing potential negative outcomes related to rigorous training [8].

C. Ebstein's Anomaly

Ebstein's anomaly is a rare congenital heart condition defined by the abnormal downward displacement of the tricuspid valve towards the apex of the right ventricle. This displacement leads to the fusion of the septal and posterior valve leaflets with the underlying myocardium, resulting in "atrialization" of the inlet portion of the right ventricle [9]. As a consequence, this structural abnormality can significantly impair right ventricular function, which may persist even after surgical interventions, such as tricuspid valve repair or replacement. These patients are at increased risk for atrial arrhythmias due to this dysfunction [9]. Furthermore, it is common for patients with Ebstein's anomaly to have coexisting atrial septal defects or patent foramen ovale, which can cause right-to-left shunting due to elevated diastolic pressures in the right atrium [10]. This shunting often leads to oxygen desaturation during exercise, posing additional challenges for physical activity.

D. Coarctation of the Aorta and Aortic Dilatation

Patients who have undergone repair for isolated aortic coarctation frequently exhibit reduced exercise capacity, which may persist even after successful correction of the defect. Hypertension remains common, with some individuals showing normal resting blood pressure but significant increases during minimal activity. Therefore, monitoring blood pressure and heart rate during exercise is vital. An exercise test to evaluate blood pressure response is advisable, and if there is no anatomical obstruction requiring intervention, antihypertensive medications may be considered to prevent severe hypertension during physical activity [6]. Discrepancies in blood pressure readings between arms can occur due to subclavian artery anomalies or prior ligation during surgery, making right arm measurements more reliable. Coarctation is also associated with aortic wall abnormalities and bicuspid aortic valve, increasing the risk of complications like aneurysm or dissection. Regular imaging of the aorta through computed tomography or magnetic resonance imaging is essential before starting an exercise program, as echocardiography alone is inadequate for screening [6]. For patients with severe aortic dilation (z-score >3) or obstruction, exercise should be limited to low-intensity activities until surgical intervention and medical clearance are obtained. Moderate to high-intensity training could exacerbate aortic dilation or lead to dissection, especially in those with significant dilation, aneurysms, or false aneurysms. Various factors, such as patient size, congenital defects, and the type of surgical repair, influence the risk of aortic complications, highlighting the importance of collaboration with the medical team for accurate assessment and guidance [7].

E. Tetralogy of Fallot

Tetralogy of Fallot (ToF) is a congenital heart condition defined by four key abnormalities: right ventricular (RV) outflow obstruction, RV hypertrophy, a ventricular septal defect (VSD), and an overriding aorta. It is the most common form of cyanotic congenital heart disease, typically requiring surgical correction in childhood. However, even after repair, many patients continue to experience residual complications, including pulmonary valve regurgitation, persistent RV outflow tract obstruction, and mild RV dysfunction, all of which may reduce exercise capacity [11]. Congenital coronary anomalies are also common in this population but rarely lead to ischemia. However, there is a heightened risk of malignant ventricular arrhythmias, particularly in patients with risk factors such as right bundle branch block (QRS duration >180 ms), pulmonary regurgitation, ventricular dysfunction, documented arrhythmia, syncope, and a history of ventriculotomy incision. While the likelihood of ventricular tachycardia remains low when RV size and function are preserved, it is advisable to conduct exercise testing to evaluate the arrhythmia burden before starting an exercise training program, especially in the presence of any high- risk indicators [7].

F. Transposition of the Great Arteries

Transposition of the great arteries (TGA) includes congenitally corrected transposition (Cc-TGA), also known as levo-transposition (l-TGA), and dextro-transposition (d-TGA). Cc-TGA results in a condition known as "double discordance," where the right atrium connects to a sub-pulmonary morphological left ventricle, and the left atrium connects to a systemic right ventricle. In d-TGA, the pulmonary artery arises from the left ventricle while the aorta originates from the right ventricle. Many patients with d-TGA have undergone atrial switch procedures (Mustard or Senning procedure) to reroute blood flow, leading to a systemic right ventricle [10]. Patients with a systemic right ventricle face risks such as bradyarrhythmia, various arrhythmias, and potential heart failure. Some may experience outflow obstruction or regurgitation of the systemic atrioventricular valve.

Those who have had an atrial switch operation might also develop obstructions in the atrial baffles, leading to venous blockages or shunting that can cause desaturation during exercise [12]. Therefore, exercise testing is crucial for assessing arrhythmia burden and cardiac function to inform exercise training recommendations [7]. Currently, the preferred treatment for d-TGA is the arterial switch operation (Jatene procedure), where the aorta, pulmonary artery and coronary arteries are translocated to the correct anatomical position re-establishing a systemic LV [10]. These patients are generally less prone to arrhythmias and heart failure, but long-term complications such as pulmonary stenosis, coronary ischemia, neoaortic insufficiency, and arrhythmias can still arise, necessitating regular follow-up and monitoring. In the absence of significant complications, these patients typically achieve near-normal exercise capacity [13].

G. Fontan Circulation

The Fontan procedure, used for children with single ventricle physiology, improves prognosis and exercise capacity by connecting the vena cavae directly to the pulmonary arteries, bypassing the heart and eliminating the need for a sub-pulmonary right ventricle. While effective in reducing cyanosis and cardiac overload, long-term complications such as high venous pressure, arrhythmias, and circulatory failure may arise. Exercise capacity is limited by reduced preload, which decreases stroke volume, while factors like abnormal heart rate dynamics and diminished muscle function also contribute [14]. Resistance and inspiratory muscle training can enhance cardiac output and improve exercise capacity by strengthening the peripheral and respiratory muscle pumps. These interventions improve blood flow and mitigate fatigue, offering potential benefits for long- term outcomes [7].

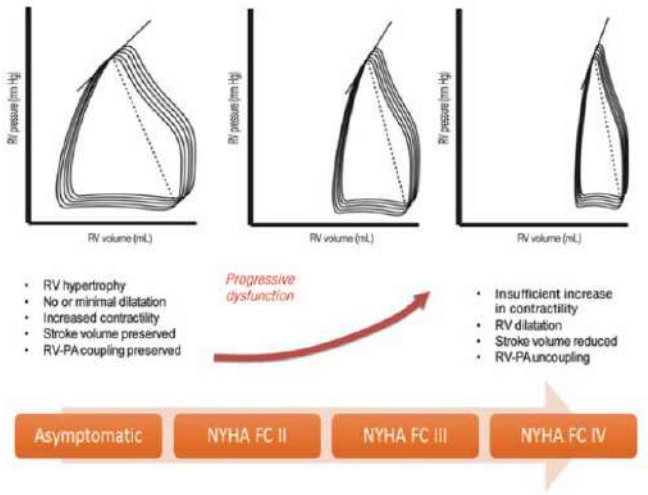
Key Considerations for Tailoring Exercise Training in Patients with Congenital Heart Disease

Most patients with CHD can safely participate in regular moderate-intensity exercise training, as long as no contraindications are present. However, many CHD patients exhibit varying levels of reduced exercise capacity, even if they are asymptomatic, with the severity often related to the specific type of CHD [15]. When crafting individualized exercise prescriptions, including both resistance and aerobic training, several key factors must be carefully considered for each patient.

1. Pulmonary Hypertension (PH)

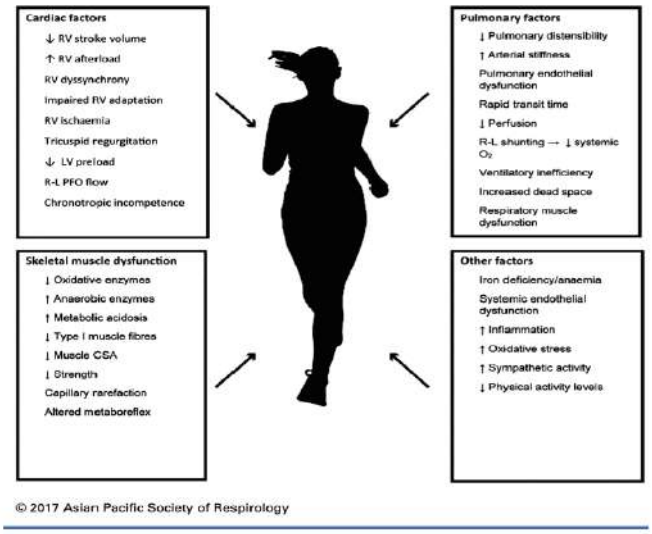
Patients with pulmonary arterial hypertension (PAH) related to CHD experience increased afterload on the right ventricle (RV). Initially, the right heart compensates by developing concentric hypertrophy to preserve systolic function. However, over time, this adaptation can lead to a maladaptive state where systolic function declines and the RV dilates [16]. In individuals with Eisenmenger's Syndrome, the RV may maintain adaptive concentric hypertrophy well into adulthood. For non-cyanotic patients, ventriculoarterial uncoupling is often the main factor limiting exercise performance (Figure1), though additional factors may also influence this outcome (Figure 2) [17]. Those with chronic cyanosis or Eisenmenger physiology may experience significant desaturation during physical activity, which severely impacts their exercise capacity [17]. While light to moderate intensity exercise is generally well tolerated and beneficial for patients with PH, vigorous physical activity carries risks, such as decreased blood pressure due to low cardiac output, syncope, and sudden death [18]. This risk may be lower in patients with Eisenmenger syndrome, as the presence of a shunt can help maintain left ventricular volume. Additionally, some individuals with PAH may experience angina caused by compression of the left main coronary artery by a dilated main pulmonary artery, a condition that may be exacerbated by exercise [19].

Figure 1 :
Right ventricular- pulmonary artery (RV-PA) coupling [17].



The coupling between the right ventricle and the pulmonary artery (RV-PA) is represented by the ratio of end-systolic ventricular elastance (Ees) to arterial elastance (Ea). Ees is determined by the slope of the end-systolic pressure–volume relationship, derived from a series of pressure–volume loops at progressively decreasing venous return, and serves as a load-independent measure of ventricular contractility. Conversely, Ea is calculated by dividing end-systolic pressure by stroke volume (SV), reflecting the afterload that the right ventricle must overcome. RV-PA coupling can be conceptualized as the ratio Ees/Ea, which represents right ventricular contractility adjusted for afterload. In the early stages of certain diseases, this coupling may remain intact, as increases in contractility correspond with rising afterload. However, RV decompensation occurs when the heart's contractility no longer suffices to meet the demands of afterload, leading to a decline in stroke volume.

Figure 2 :
Factors contributing to exercise intolerance in pulmonary arterial hypertension [17].



CSA, cross-sectional area; LV, left ventricle; PFO, patent foramen ovale; R-L, right-to-left; RV, right ventricle.

2. Cardiac Rhythm Issues

Patients with both unrepaired and repaired congenital heart disease face an elevated risk of supraventricular and ventricular arrhythmias. Bradycardia and chronotropic incompetence are frequently observed in these individuals, often resulting from the cardiac lesions themselves, damage to the conduction system during surgery, or the effects of heart rate-limiting medications. Approximately 30% to 60% of patients with CHD experience chronotropic incompetence, defined as achieving less than 80% or 85% of the predicted maximal heart rate [20]. Even those who have undergone surgical repair for simple lesions like atrial septal defects and ventricular septal defects may still exhibit this impairment. The combination of peak oxygen consumption and heart rate reserve serves as a significant predictor of five-year mortality in patients with congenital heart disease. This strong association may be attributed to the predictive capability of peak oxygen consumption for heart failure-related mortality and the role of heart rate reserve in forecasting mortality associated with arrhythmias [20]. The underlying causes may include disrupted cardiac autonomic regulation, injury to the sinoatrial or atrioventricular nodes during surgery, or insufficient preload. For patients with Fontan physiology, maximal HR (HRmax) typically plateaus around 155 to 165 beats per minute. This reduction in peak heart rate may serve as a compensatory mechanism in response to decreased preload [7].

3. Sudden Cardiac Death (SCD) and Uncontrolled Arrhythmias

Although sudden cardiac death (SCD) during exercise training is rare among patients with CHD, appropriate screening can help reduce this risk.

Patients with complex CHD should undergo assessment for potentially harmful arrhythmias through a 24-hour Holter monitor and an exercise stress test before starting an exercise training program. It is crucial to effectively manage any uncontrolled cardiac arrhythmias [6]. However, relying on a single test may not be adequate for detecting all arrhythmias, so patients exhibiting new symptoms indicative of arrhythmias should be promptly referred for further evaluation and treatment [7].

4. Implanted Cardiac Defibrillators (ICD) and Pacemakers

After the implantation of a pacemaker or implantable cardiac defibrillator (ICD), patients should avoid excessive upper limb exercise for at least 3-4 weeks and refrain from high-impact activities to prevent potential dislodgement of the devices.
Activities like long-distance swimming that involve repetitive arm movement may also risk damaging the transvenous leads. Exercise professionals need to be mindful of the rate threshold set on an ICD; exercise prescriptions should typically not exceed a heart rate that is 10-15 beats per minute below the device's discharge threshold to avoid inappropriate shocks unless the ICD has sufficient arrhythmia discrimination, which can be confirmed with the cardiology team [21].
Using heart rate to monitor exercise intensity is not valid for patients with pacemakers that pace via the atrium during exercise. For individuals with complete heart block who have a pacemaker that senses atrial activity and subsequently paces the ventricle, the device may stop tracking atrial activity if the heart rate surpasses the upper limit set on the pacemaker. Since pacing settings can often be optimized, it's advisable to consult the treating cardiologist if any pacing issues arise that could affect the patient's ability to engage in exercise training [7].

5. Ischemia

Patients who have undergone coronary reimplantation during procedures such as the arterial switch operation, Ross operation, surgical repair of anomalous coronary artery origins, or aortic root replacement face a higher risk of developing occlusive ostial coronary lesions and myocardial ischemia. Those with an anomalous origin of the left coronary artery from the pulmonary artery or an anomalous aortic origin of a coronary artery are particularly susceptible to progressive myocardial ischemia, which can lead to sudden cardiac death. Even in cases where stress testing shows no documented ischemia, high-risk anomalous coronary lesions can still pose significant dangers [22]. Therefore, it is crucial to involve a cardiologist experienced in managing these lesions to guide exercise training prescriptions. Conditions like pulmonary atresia with intact ventricular septum can present complex coronary abnormalities, and patients with severe cyanosis or pulmonary hypertension may experience cardiac ischemia due to insufficient myocardial oxygen delivery. Individuals displaying symptoms of myocardial ischemia should undergo a medically supervised exercise stress test before beginning an exercise training program. For those with confirmed coronary ischemia who have received medical clearance to exercise, the maximum exercise intensity should not exceed 10 beats per minute below the ischemic threshold [21].

6. Cyanosis

Patients with right-to-left shunting or reduced pulmonary blood flow are at risk of desaturation during exercise training. Some individuals may experience slight improvements in oxygen saturation with supplemental oxygen, so in frail patients, this may be trialed during exercise sessions. Those with cyanotic congenital heart disease often have resting oxygen saturations significantly below 90% and can desaturate rapidly, even with minimal exertion. Therefore, for severely cyanotic patients, exercise intensity and duration should be guided by symptom-limited thresholds and ratings of perceived exertion, as measuring oxygen saturation in these cases may be less useful [7].

7. Valvular Heart Disease and Outflow Tract Obstruction

Some patients with valvular atresia or complex congenital heart disease may have an implanted cardiac conduit functioning as a valve. Those with anterior right ventricle-to-pulmonary artery conduits could potentially face risks of damage to the prosthesis, particularly during contact sports. Obstruction of the outflow tract can be dynamic, potentially worsening during exercise.

Patients with mild stenosis, regurgitation, or obstruction can generally engage in most forms of exercise training [23]. However, individuals with more severe valvular stenosis or ventricular outflow tract obstruction are at risk of syncope and hypotension during exercise due to reduced cardiac output. Therefore, these patients should be limited to low to moderate intensity exercise that does not provoke symptoms [24].

8. Musculoskeletal Issues

Individuals with CHD frequently experience diminished muscle function, and the extent of this impairment is typically associated with the complexity of their heart condition. Additionally, there is a relationship between skeletal muscle mass and peak VO2 levels. While the exact causes of muscle dysfunction remain unclear, they are likely multifactorial, including factors such as neurohormonal activation, low cardiac output, cyanosis, endothelial dysfunction, and reduced physical activity [25].

Additionally, structural musculoskeletal abnormalities, such as scoliosis and kyphosis, are frequently observed in CHD patients. Exercise training that causes pain should be avoided or modified, which may involve adjusting the range of motion and incorporating isometric exercises to simplify the regimen [25].

9. Respiratory Function

Pulmonary function can be compromised in some patients with CHD, contributing to exercise limitations. Reduced lung volume is often a consequence of previous cardiothoracic surgeries, such as sternotomies or thoracotomies [26]. In Fontan patients a significant correlation between peak VO2 and pulmonary function has been observed, potentially linked to diminished pulmonary blood flow, which in turn may lead to reduced stroke volume. Inspiratory muscle training could serve as a beneficial intervention to help improve exercise capacity [7].

10. Psychological Considerations

Patients with CHD are at a heightened risk for developing psychological disorders. Issues such as anxiety, body image concerns, and low self-esteem can adversely impact adherence to exercise programs and overall quality of life (QOL) [27]. These psychological factors are also associated with lower participation in sports, and many patients may struggle with feelings of self-efficacy regarding exercise. Commonly, a lack of motivation is observed, necessitating additional strategies to enhance adherence. Depression is often the strongest determinant of a patient's perceived health status but is frequently overlooked.

While exercise limitations have been identified as a major factor affecting QOL in CHD patients, the evidence regarding the impact of exercise training on improving QOL remains inconsistent, highlighting the need for further research in this area [7].

Comprehensive Clinical Approaches to Exercise Testing

Before starting an ET program, patients with complex CHD should undergo cardiopulmonary exercise testing (CPET) to assess peak VO2, a key measure of aerobic capacity and prognosis. Peak VO2 below 15.5 ml.kg-1.min-1 indicates a higher risk of hospitalization or mortality, making it crucial to enhance aerobic fitness. CPET also helps monitor progress and adjust treatments. For patients unable to reach maximal effort during CPET, submaximal indicators such as the first ventilatory threshold (VT), VE/VCO2 slope, and VO2/work rate slope provide valuable insights into cardiopulmonary health [15].

The six-minute walk test (6MWT) is an alternative to CPET for assessing exercise capacity, correlating moderately with peak VO2. However, it offers less insight into the causes of exercise intolerance and may be limited by a "ceiling effect" in less symptomatic patients [28].

Prior to resistance training, strength testing should be done in low-risk patients, with one-repetition maximum (1RM) testing providing essential data for exercise prescriptions. In complex cases, physician evaluation is recommended before exercise. Where 1RM testing isn't feasible, the load-repetition method can estimate strength levels to guide intensity [29]. Patients with complex CHD should undergo CPET prior to initiating an ET program. CPET offers an objective assessment of peak VO2, providing vital information about the patient's aerobic capacity and helping to detect any adverse exercise responses in a controlled environment. Peak VO2 is widely regarded as the gold standard for evaluating aerobic fitness and is a key prognostic indicator for individuals with CHD. In particular, a peak VO2 below 15.5 ml.kg-1.min-1 is linked to an increased risk of hospitalization or mortality, underscoring the need to improve aerobic capacity in this population [15]. Furthermore, periodic CPET evaluations are valuable for monitoring the patient's clinical progress over time and adjusting treatment as needed [30].

Benefits and Considerations in Exercise Training prescription for CHD Patients

Although current research highlights the benefits of ET for patients with CHD and indicates that adverse responses are uncommon, participation in ET remains insufficient. This section outlines the advantages of ET and offers guidance on designing effective exercise programs for adolescents and adults with CHD, based on the latest literature (Table III) [31] delineates the various intensity levels for both aerobic and resistance ET, addressing specific considerations for distinct types of CHD. Additionally, it outlines strategies for safely managing patients who may be at an elevated risk for adverse responses to ET, ensuring a comprehensive approach to exercise intervention.

Research on ET for adolescents and adults with CHD has mainly centered around aerobic (endurance) training. A consistent outcome is the improvement in cardiorespiratory fitness (CRF); a recent systematic review covering various forms of CHD found an average increase in peak VO2 of 8% (2.6 ml.kg-1.-min-1) following ET interventions [32]. This finding corresponds with studies showing a delayed anaerobic threshold (AT) after ET, indicating enhanced oxidative metabolism [33].

Interestingly, there appears to be no significant change in peak heart rate, which aligns with observations in other patient groups. Although there are few studies measuring the direct effects of ET on stroke volume, increases in post-training oxygen pulse during peak exercise (an indirect measure of cardiac output) suggest that ET may positively influence stroke volume in some CHD patients [7]. These results, however, need further investigation in larger, diverse CHD populations. Additionally, adaptations in peripheral systems, including better muscle ergo-receptor function, skeletal muscle hypertrophy, improved vascular endothelial function, enhanced oxygenation in peripheral muscles, and possibly greater skeletal muscle oxidative capacity, seem to play a critical role in the improvements in CRF following ET [7].

Table III.
Exercise intensity categories based on various methods of exercise prescription [31].

Intensity Category	Aerobic Training Measure of Intensity	Aerobic Training Subjective Measures	CPET Ventilatory Threshold points	Resistance Training Measure of Intensity
Low (Light)	20-39% HRR or VO2R 40-54%HRmax 20-39% VO2max	RPE: 8-10	Start of exercise to the 1st VT	30-49% 1RM
Moderate	40-59% HRR or VO2R 55-69% HRmax 40-59% VO2max	RPE:11-13	Start of exercise to the 1st VT	50-69% 1RM
Vigorous	60-84% HRR or VO2R 70-89% HRmax 60-84% VO2max	RPE: 14-16	Between the 1st VT and 2nd VT (RCP)	70-79% 1RM
High	≥85% HRR or VO2R ≥90% HRmax ≥85% VO2max	RPE: ≥17	Above the 2nd VT (RCP)	≥80% 1RM

VO2R, oxygen uptake reserve; 1st VT, first ventilatory threshold (anaerobic threshold); 2nd VT, second ventilatory threshold (RCP: respiratory compensation point); VO2max, maximal oxygen uptake; RM, repetition maximum; HRmax, maximal heart rate; Borg's RPE (ratings of perceived exertion) scale (6- 20).

1. Aerobic Exercise Training Prescription

The FITT-VP principle (frequency, intensity, time, type, volume, and progression) guides ET for patients with cardiovascular conditions, including those with CHD. Intensity is particularly important for improving peak VO2, and higher intensities don't necessarily increase risks, especially in heart failure patients. However, aerobic ET intensity thresholds for CHD patients are less defined, leading to variability based on the individual's condition and baseline cardiorespiratory fitness (CRF) [7].

Typically, aerobic intensity is prescribed based on a percentage of maximum heart rate (HRmax), with most studies recommending a target of 60-80% HRmax. In cases of abnormal heart rate responses, metrics like the Borg Rating of Perceived Exertion (RPE) or the threshold method may be more appropriate. These are independent of HR and take physiological responses into account during incremental work rates. ET sessions commonly last 30-60 minutes and are conducted 2-3 times per week, with studies showing that frequencies above three sessions weekly provide similar benefits [7].

High-intensity interval training (HIIT) offers promising results, particularly in stable CHD patients, improving peak VO2 and vascular function. It has been found safe in conditions like tetralogy of Fallot (ToF) and is recommended with a cautious start, progressing as tolerated. Further research is needed to validate its safety across diverse CHD populations [33].

2. Resistance Exercise Training Prescription

Muscle wasting and dysfunction are common in individuals with CHD, and while aerobic ET improves muscle function, resistance training is more effective for combating muscle loss. It is essential for maintaining muscle mass and boosting exercise capacity, particularly in complex CHD cases. In patients with Fontan circulation, a strong correlation exists between lower limb muscle mass and exercise output ($r = 0.7$, $p = 0.008$) [7]. Despite past concerns over resistance ET's potential impact on cardiac function, research has shown it to be safe and effective. Heavier weights with fewer repetitions can induce a milder hemodynamic response compared to lighter weights with more repetitions. Additionally, resistance ET offers similar benefits to aerobic exercise for individuals with exercise-induced pulmonary arterial hypertension (PAH). Proper technique and avoiding the Valsalva maneuver are crucial to prevent adverse effects like syncope.

Studies have shown that resistance training improves cardiorespiratory fitness, quality of life, and muscle characteristics without negatively affecting left ventricular function during moderate-intensity sessions. A notable study demonstrated significant gains in muscle mass, cardiac output, and exercise capacity after a 20-week high-intensity resistance program in Fontan patients [7]. Training should progress gradually, starting with lower intensities and increasing as tolerated, with rest intervals of over 60 seconds between sets. Sessions should be performed 2-3 times per week, targeting all major muscle groups, especially the lower limbs, to enhance the skeletal muscle pump's effectiveness in Fontan patients [7].

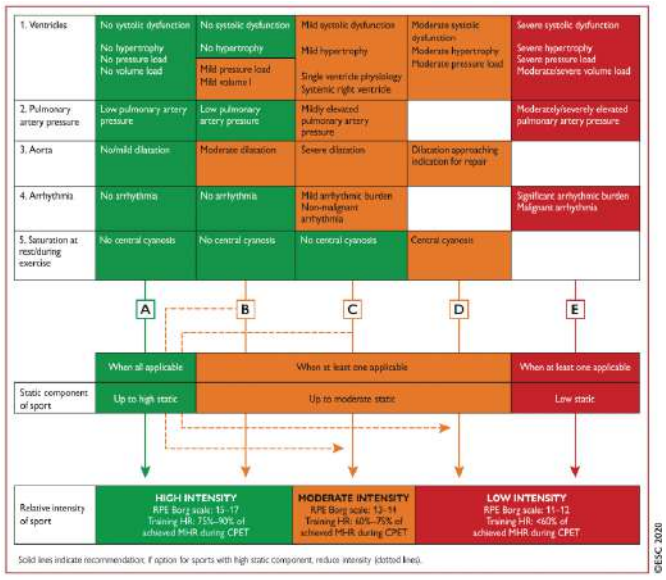
3. General Recommendations

The pre-exercise screening process and ET prescription for patients with CHD are detailed in Figure 3 [6]. Clinically stable CHD patients should receive an individualized ET program as a complement to their ongoing medical care, tailored to the specifics of their condition [7]. Prior to initiating ET, a thorough screening by the patient's CHD specialist is essential, with close collaboration between the specialist and the exercise professionals responsible for prescribing ET to ensure necessary precautions are addressed.

Continued communication between the specialist and exercise professional is crucial to monitor patient progress and manage any adverse symptoms, such as light-headedness, syncope, headaches, or severe dyspnea, which may require further medical evaluation. Supervision, especially during the initial stages, is highly recommended to ensure proper ET techniques, teach self-monitoring, and improve patient adherence and confidence in their exercise regimen [7].

Table IV outlines generalized recommendations for prescribing ET for patients with CHD, serving as a guide based on their risk classification [7]. Nevertheless, certain patients may be cleared for higher intensity exercise following a consultation with their CHD specialist. In addition to structured ET, individuals with CHD should also work toward achieving the minimum physical activity (PA) guidelines set for healthy individuals [34].

Figure 3.
Pre-participation assessment of individuals with congenital heart disease [6].



CPET = cardiopulmonary exercise test; HR = heart rate; MHR = maximum heart rate; RPE = rate of perceived exertion. A-E represent pathways linking static and intensity components for each column. After assessment of CPET and the five variables, an individual recommendation can be given (solid arrow). If a higher static level sport is chosen, then a lower intensity level is advised (dotted arrow).

Table IV.
Aerobic and resistance exercise prescription based on risk classification [7].

Mode	Risk classification	Intensity/relative volume load (resistance training)	Frequency	Duration (aerobic training)/No of sets (resistance training)
Aerobic Training	Low	40–84%HRR or 55 – 89% HR max 11–16 RPE	3 – 5 days/ week	Commence at 5- 10 minutes and increase as tolerated to 30 – 60 minutes or Interval training may be employed to increase tolerance to exercise. Commencing at a work: active rest ratio of 1:2 progressing to 2:1.
	Moderate	20–59%HRR or 40 – 69% HR max 8 – 13 RPE		
	High	20 – 39% HRR or 40 – 54% HR max 8 – 10 RPE		
Resistance training	Low	50 – 79% 1RM 1 – 3 sets, 8 – 10 repetitions ≥1 minute rest between sets	> or = 2 days/week	Commence at 1 set progressing to 3 sets as tolerated. Initial supervision is recommended to provide instruction in correct lifting technique.
	Moderate	30 – 69% 1RM 1 – 3 sets, 10 – 12 repetitions ≥1 minute rest between sets		
	High	30 – 49% 1RM 1 – 3 sets, 12 – 15 repetitions ≥2 minutes rest between sets		

HRR, heart rate reserve; Borg's RPE, rate of perceived exertion; 1RM, one repetition maximum. Exercise prescription using heart rate reserve can be calculated using the Karvonen method (prescribed exercise heart rate = % intensity* (peak HR – resting HR) + resting HR). * % intensity as a decimal.

Latest Guidelines for Exercise in Patients with CHD

The 2018 AHA/ACC and 2020 ESC Guidelines outline a structured methodology for assessing athletes with CHD, designed to optimize their sports participation (Table V) [6] [5]. This assessment begins with a comprehensive evaluation that includes a detailed medical history and physical examination, focusing on the specific CHD diagnosis, previous interventions, medications, symptoms, and exercise history. Understanding the athlete's current training regimen and sports activities is crucial. The second stage involves evaluating key baseline parameters, such as ventricular function through echocardiography, assessing pulmonary hypertension, and identifying potential aortic or arrhythmia issues. Cardiopulmonary exercise testing (CPET) serves as a vital tool in predicting outcomes and measuring various exercise-related metrics. Subsequently, exercise intensity and prescription are customized for each athlete, taking into account unique hemodynamic consequences and lesion-specific considerations. Continuous follow-up and periodic assessments, typically on an annual basis, are essential to monitor age-related changes and the potential onset of cardiovascular diseases. This comprehensive and holistic approach promotes a safe and effective sports participation environment for CHD athletes, ultimately enhancing their overall health and quality of life.

By adhering to these guidelines, healthcare providers can help patients with CHD engage in safe and effective exercise, ultimately improving their overall health and quality of life.

Table V.
Assessment of the athlete with congenital heart disease [6] [5].

Stage 1: Full History and Physical Examination	• Conduct a detailed history and physical exam. • Review the patient's CHD diagnosis and any surgical or catheter interventions. • Assess current medications and cardiovascular symptoms at rest and during exercise. • Take note of any non-cardiac conditions, like pulmonary dysfunction. • Obtain a comprehensive exercise and sports participation history, including details of the training schedule and dietary supplements. • Analyze the static and dynamic components of the planned or ongoing sports activities.
Stage 2: the following five baseline parameters should then be evaluated	• **Ventricular Function**: Evaluate ventricular function using echocardiography to determine if it's mildly, moderately, or severely reduced. Complex cases may need CMR scanning to detect intracardiac scarring and assess arrhythmia risk. • **Pulmonary Hypertension (PH)**: Diagnose PH by assessing mean pulmonary artery pressure (PAP), particularly in athletes with CHD. Consider how increased age, exercise, or conditions like altitude may affect PAP. • **Aortic Assessment**: Assess for aortic dilatation, particularly in CHD patients at risk (e.g., those with Tetralogy of Fallot or 22q11 microdeletion). Avoid contact sports in patients with aortas dilated >5 cm. • **Arrhythmia**: Assess for arrhythmias, which may signal underlying hemodynamic issues. Comprehensive evaluation may include ECG, prolonged monitoring, and possibly electrophysiology testing. • **Saturations/Lung Function**: Evaluate for potential intracardiac shunts or residual cyanosis using pulse oximetry and cardiopulmonary exercise testing, considering pulmonary causes and post-surgical complications.
Stage 3. Cardiopulmonary exercise testing	• **Risk Stratification**: Cardiopulmonary exercise testing (CPET) is critical in assessing risk and predicting outcomes in adults with CHD. • **Comprehensive Evaluation**: CPET helps evaluate pulmonary artery pressure (PAP), respiratory function, cardiac output, exercise-related hemodynamics, and arrhythmias. • **Combined Assessment**: Should be paired with a Borg scale effort evaluation and a 12-lead ECG to monitor for arrhythmias and chronotropic incompetence. • **Key Indicators**: Reduced VO2max and oxygen pulse may suggest decreased stroke volume, especially in complex CHD cases like repaired tetralogy of Fallot. • **Respiratory Efficiency**: A reduced ventilatory anaerobic threshold and elevated VE/VCO2 slope may signal inefficient gas exchange and a limited pulmonary vascular bed. • **Muscle Mass**: Reduced skeletal muscle mass is common in CHD adults and may impair oxygen uptake. Regular resistance exercise can improve muscle mass and long-term outcomes.
Stage 4. Exercise intensity and prescription	• **Exercise Evaluation**: Assess the athlete's participation in sports, focusing on intensity, aerobic, and resistance components, including isometric and dynamic exercises. • **Comprehensive Overview**: Consider both training and competition volumes during the evaluation. • **Hemodynamic Impacts**: Assess the hemodynamic consequences of exercise, factoring in lesion-specific effects and individual athlete-specific adaptations.
Stage 5. Follow-up and repeat assessment	• **Annual Assessments**: Conduct serial assessments annually for CHD athletes involved in sports. • **Monitor Age-related Changes**: Assess for age-related changes and potential onset of degenerative cardiovascular disease (CVD). • **Ongoing Evaluation**: Ensure consistent monitoring to adjust exercise and sports recommendations as needed based on the athlete's evolving condition.

Conclusion

Recent research indicates that ET is safe and provides significant physiological and psychosocial benefits for adolescents and adults with CHD, including those with complex abnormalities. Given the limited availability of effective medical therapies beyond corrective surgical interventions for many CHD defects, ET may represent a critical strategy for enhancing functional capacity and lowering the risk of future cardiovascular events. However, this is still an evolving area of study, and additional research is necessary to refine ET prescriptions across the various types of CHD and to establish ET services that can effectively support this population.

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Instructions aux auteurs Revue Marocaine de Cardiologie

La revue marocaine de cardiologie, est l'organe de presse officiel de la société marocaine de cardiologie à but non lucratif, d'apparition trimestrielle, qui publie en langue française et anglaise des travaux scientifiques originaux.
La revue marocaine de cardiologie assure la création d'un espace de publication d'articles originaux, essai clinique méta-analyse de mises au point et de cas cliniques. Elle permet de communiquer les résultats d'études menées et d'assurer le développement de la recherche scientifique dans le domaine cardiovasculaire.
Les journées du congrès de la SMC bénéficient de numéros exclusifs.

Processus d'évaluation

Avant publication, Tout manuscrit reçu par la revue, doit être soumis à un comité de rédaction qui procède à une évaluation du texte, avec une relecture par des experts associée à d'éventuelles modifications, une vérification de l'originalité de l'article peut être exigée via l'outil de détection de plagiat.
En cas d'approbation, Les articles ne doivent pas être publiés antérieurement ni simultanément dans une autre revue, même électronique.

Déclaration éthique

Recherche comportant des expériences sur des humains ou des animaux ou des prélèvements de spécimens
Les recherches comportant des expériences sur les humains ou des animaux ou des prélèvements de spécimens doivent respecter les principes de la déclaration d'Helsinki « The Code of Ethics of the World Medical Association »:
- pour les expérimentations impliquant l'homme : https://www.wma.net/fr/policies-post/declaration-dhelsinki-de-lamm-principes-ethiques-applicables-a-la-recherche-medicale-impliquant-des-etres-humains/
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Les auteurs doivent obtenir toutes les autorisations de recherche nécessaires avant d'entreprendre les travaux sur le terrain, et les numéros de permis ou de licence de recherche doivent figurer dans le manuscrit.

Protection des patients participants aux études

Un consentement éclairé par écrit est nécessaire pour protéger le droit à la vie privée des patients. Toute information permettant d'identifier l'individu ne doit être publiée, à moins qu'elle ne soit essentielle à des fins scientifiques. Chaque individu qui apparaît en photographie, en vidéo, dans un enregistrement ou simplement nommément dans l'article, doit être préalablement informé.
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Pour assurer la transparence et la crédibilité des articles publiés, la revue se refère aux normes internationales relatives aux conflits d'intérêt. Toute publication soumise doit comporter des documents à l'appui dévoilant les liens d'intérêt et les sources de soutien financier du travail.
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Principes généraux :
le texte des articles répond à la structure « IMRD » divisée en quatre sections : Introduction, Méthodes, Résultats et Discussion, Les formats de fichiers textes utilisables sont MS Word.
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Page de titre :
La page de titre contient :
- le titre de l'article (titre en français et en anglais), avec éventuellement un sous-titre,
- Informations sur les auteurs : Nom et prénom et adresse e-mail dans l'ordre dans lequel ils apparaitront lors de la publication, les affiliations de chacun des auteurs, les départements ou institutions auxquels le travail est attribué, il faut préciser les coordonnées de l'auteur en charge de la publication
- Comptage des mots contenu dans le texte (sans tenir compte du résumé, illustrations références et remerciements).
- Nombre de figures et de tableaux.
- les remerciements éventuels.
- les sources de financements et les liens d'intérêts, s'il y a lieu.
Manuscrit :
La longueur maximale des textes (références comprises) doit être comme suit :
- articles originaux et mises au point : 12 pages ;
- cas cliniques: 4 pages ;
- arrêt sur image: 2 pages.
Les auteurs doivent veiller à ce que les textes soumis soient clairs et facilement compréhensibles, précis et concis.

Abréviations et symboles :
Seules les abréviations normalisées peuvent être utilisées en nombre limité. Éviter de les utiliser dans le titre du manuscrit. Les abréviations doivent être expliquées lors de leur première apparition dans le texte.
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Les documents iconographiques (figures et tableaux) sont obligatoirement appelés dans le texte et conformes aux recommandations suivantes.
- Dans le manuscrit, les légendes des illustrations doivent être présentées sur une page séparée en utilisant les chiffres arabes correspondant aux illustrations (figure 1).
- Les tableaux sont numérotés en chiffres romains, par ordre d'apparition dans le texte : (tableau I).
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Les auteurs doivent fournir les références bibliographiques directes des sources originales, rapportés à la fin de l'article et numérotées consécutivement dans l'ordre de leur première mention dans le texte. Identifier les références dans le texte, les tableaux et les légendes par des chiffres arabes entre crochets les références d'articles parus dans un périodique doivent comporter le nom des six premiers auteurs avec les initiales des prénoms (suivis de « et al. » à partir du 7ème auteur), le titre complet de l'article dans la langue originale, le nom de la revue selon les abréviations de l'Index Medicus, l'année, le numéro du tome, pages (première et dernière).

GUIDE OF AUTHORS

The moroccan journal of cardiology is the official press of the Moroccan Society of Cardiology. It appears quarterly and publishes original French and English scientific works.
The moroccan journal of cardiology ensures the publication of original articles, trials meta-analyses clinical reviews and case reports. It allows to communicate the results of studies and enhance the development of scientific research in the cardiovascular fields.

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Title page :
The title page contains :
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- Number of figures and tables
- Acknowledgments
- Sources of funding and interests
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The maximum length of the texts (including references) must be as follows :
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- Case reports : 4 pages
- Freeze frame : 2 pages.
The submitted text should be clear and easily understandable, Precise and concise. The languae should be simple and correct. Abbreviations should be explained when they first appear in the text and then used consistently and invariably.

Abbraviations and symbols :
Only a limited number of standard abbreviations may be used. Avoid using them in the title of the manuscripts. Abbraviations must be explained when they first appears in the text. Units of measurement must conform to the international nomenclatures.

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Iconographic documents (fugures and tables) must be called up in the text and conform to the following recommendations :
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Authors should provide direct bibliographic references to original sources, reported at the end of the article and numbered consecutively in the order of their first mention in the text. Identify references in the text, tables and legends by Arabic numbers in square brackets.
References to articles in a journal must include the named of the first six authors with first name initials (followed by « and al. » from the 7th author), the full title of the article in the original language, the name of the journal according to the Index Medicus abbreviations, the year, the volume number, pages (first and last).


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