REVIEW ARTICLE

Sports cardiology field as a key player in optimizing cardiovascular health during sports

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ABSTRACT

Background: Sudden cardiac death (SCD) in athletes, though rare, continues to make headlines, raising questions about its underlying causes. Athletes are generally healthy, yet life-threatening cardiac events can occur unexpectedly in those without prior symptoms. Early detection of high-risk cardiac conditions is critical for prevention. Main text: Sports cardiology is an emerging specialty focused on the cardiovascular health of competitive athletes and highly active individuals, including those with known or undiagnosed heart disease. It encompasses structural, functional, and electrical cardiac assessment, exercise physiology, and preventive strategies. Screening and preparticipation evaluation are vital to identify hidden cardiovascular risks and differentiate physiological adaptations from pathology. This review highlights the importance of sports cardiology in optimizing cardiovascular performance, preventing SCD, and managing cardiovascular conditions such as arrhythmias, cardiomyopathies, coronary artery disease, and hypertension in physically active populations. Conclusions: Sports cardiology is rapidly evolving and plays a crucial role in safeguarding the health of athletes and active individuals. Optimal care requires an integrated approach, specialized knowledge, and targeted research initiatives to ensure safe participation in competitive and recreational exercise while minimizing cardiovascular risks.
Keywords: Sport medicine, Athletic injuries, Cardiovascular diseases, Sudden cardiac death, Exercise.

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INTRODUCTION

An increasing body of evidence substantiates the notion that consistent physical activity (PA) and exercise are detrimental to health. Premature mortality and morbidity associated with metabolic syndrome, stroke, coronary artery disease, osteoporosis, and various cancers (including colon, breast, bladder, kidney, rectal, head and neck, endometrial, gastric cardia, lung, and oesophageal adenocarcinoma), depression, falls, functional decline, and constipation have been shown to have a strong dose–response relationship with PA and exercise (Moore et al., 2016). This relationship has been confirmed in both men and women across different ethnic groups (Pelliccia et al., 2019). Several studies have demonstrated that aerobic activity significantly reduces the risk of premature all-cause mortality and cardiovascular disease (CVD) mortality. Higher cardiorespiratory fitness can be achieved with increased intensity of regular PA, leading to multiple health benefits, and exercise is widely recognized as a key lifestyle modification for improving health (Paquette et al., 2020; Baggish et al., 2017).

There is no doubt that regular PA significantly reduces CVD risk; however, complete protection is not achieved (Pelliccia et al., 2021). Athletes and highly active individuals remain vulnerable to musculoskeletal injury and cardiovascular (CV) events. Musculoskeletal injuries are related to exercise type and intensity, underlying musculoskeletal abnormalities, and preexisting conditions (Teyhen et al., 2020). The most serious CV events associated with high-intensity PA include acute myocardial infarction and sudden cardiac arrest (SCA), which may result in mortality and morbidity (Han et al., 2023).

Sudden cardiac death (SCD) can occur in symptomatic athletes or physically active individuals engaging in strenuous or competitive exercise. The reported incidence of SCD among athletes is variable, ranging from 1 in 40,000 to 1 in 300,000 (Weizman et al., 2023). Physical activity may paradoxically increase the incidence of acute CV events, even in well-trained individuals. In rare cases, exercise may unexpectedly precipitate SCA in individuals with underlying cardiovascular disease, particularly in previously sedentary individuals or those with advanced disease (Pelliccia et al., 2019).

Figure 1. Core curriculum of sports cardiology.

The principal aim of sports cardiology is to ensure safe and optimal cardiovascular system performance during exercise (Baggish et al., 2017). The core curriculum of sports cardiology includes four fundamental domains of knowledge essential for the care of competitive athletes and highly active individuals (CAHAP) (Baggish et al., 2017). Sports cardiology is a novel and emerging subspecialty focused on the cardiovascular care of athletes and active individuals with known or undiagnosed cardiac conditions. It integrates cardiovascular imaging, exercise physiology, cardiac electrophysiology, and structural heart disease, reflecting the increasing population of CAHAP (Figure 1) (Karam et al., 2022).

PHYSIOLOGICAL ADAPTATION TO EXERCISE

Sustained moderate- to high-intensity exercise is associated with adaptive structural and functional changes in the cardiovascular system (Dawes et al., 2016; Baggish & Wood, 2011). These changes include enlargement of cardiac chambers and increased ability to augment cardiac output, a phenomenon known as exercise-induced cardiac remodelling (Scharhag et al., 2002; Gleason & Kim, 2017). Adaptation may involve enlargement of both ventricles and atria, along with enhanced early diastolic filling (Finch & Baggish, 2016). The extent of this remodelling varies among individuals depending on sex, ethnicity, genetic predisposition, type of sport, and duration and intensity of training (Baggish & Wood, 2011).

Isometric (strength) exercises, such as weightlifting, are characterized by transient increases in peripheral vascular resistance and systolic blood pressure, leading to left ventricular pressure overload. In contrast, isotonic (endurance) exercise, such as swimming or running, results in sustained increases in cardiac output with volume overload affecting all cardiac chambers (Ito, 2022; Barker & Eickmeyer, 2020).

A "*grey zone*" exists between physiological adaptation and early pathological cardiac remodelling (Gleason & Kim, 2017). This includes increased left ventricular wall thickness, left ventricular dilatation, and right ventricular dilatation. Differentiation between physiological and pathological changes requires integration of clinical history, including sport type, intensity, duration, sex, and ethnicity, along with imaging findings (Pelliccia et al., 2019). Physiological remodelling typically involves balanced enlargement of all chambers with preserved or enhanced systolic function during exercise, whereas isolated chamber enlargement or functional impairment suggests pathology. Left ventricular wall thickness exceeding 15 mm should prompt evaluation for cardiomyopathy (Gleason & Kim, 2017).

Exercise detraining may be used in ambiguous cases where differentiation between physiological adaptation and cardiomyopathy is uncertain (Corrado et al., 2010). The concept is based on the reversibility of physiological remodelling following cessation of exercise; however, data remain limited regarding the duration and extent of reversibility (McDiarmid et al., 2016). Moreover, the effects of detraining in patients with established cardiomyopathies remain unclear, warranting cautious application (Teyhen et al., 2020).

In addition to structural and functional changes, athletes exhibit electrical remodelling manifested as distinct electrocardiographic (ECG) patterns. Sports cardiologists must differentiate benign exercise-induced ECG changes from pathological findings. Athletes commonly exhibit benign bradyarrhythmias due to increased vagal tone (Sharma et al., 2018; U.S. Department of Health and Human Services, 2008).

PREVENTION AND SCREENING FOR SUDDEN CARDIAC DEATH

Comprehensive recommendations regarding athletics participation are revised periodically. However, evidence is scarce about the normal course of disease progression or the risk of mortality during vigorous exercise and competitive sports among patients with CVD (Petek & Baggish, 2020).

As there is still debate about the true incidence and causes of SCD in athletes, the optimal modalities of preparticipation cardiovascular screening (PPCS) are still controversial. It is important to highlight that there is a paucity of data supporting the effectiveness of PPCS as a strategy for lowering the occurrence of sport-related cardiac arrest and SCD. However, PPCS is compulsory for competitive athletes, Controversies exist on the value of several screening modalities such as ECG and cardiac imaging.

Preparticipation physical evaluation (PPE) is recommended to detect concealed CVD, especially those linked to the risk of SCD and to provide a chance to mitigate negative outcomes by implementing targeted treatments or imposing limitations on PA. Existing evidence supports the value of standardized comprehensive medical history and thorough physical examination protocols such as PPCS. PPCS encompasses several aspects such as education, screening for substance addiction, identification of other medical conditions, and facilitation of access to healthcare providers for young athletes involved in competitive sports (MacLachlan & Drezner, 2020; Maron et al., 1996).

Clinical evaluation is of limited sensitivity and specificity for CVD. However, it is a cost-effective tool in limited resource settings for utilization of the limited care provider skills and implementation with minimal resource commitment. Hence, incorporating additional diagnostic testing such as ECG can enhance the overall precision of PPCS diagnosis. Meanwhile, standardization of preparticipation protocols such as PPE-monograph entailing high-quality primary care preventive services. Additionally, cardiac imaging may help identify congenital, structural, and functional cardiac abnormalities. However, this is opposed by high cost and the required high-skill and financial infrastructure. ECG interpretation has a special approach for athletes which could decrease the false positive diagnosis and is usually best achieved by sports cardiologists (MacLachlan & Drezner, 2020; American Heart Association, 2021; Pescatello et al., 2015).

EVALUATION OF SYMPTOMS IN ATHLETES

Concerns regarding SCD in athletes are the top hot topics during sports seasons. Evaluating different cardiac symptoms in an athlete or highly active individual is a challenging process that requires an integrated approach with the implementation of the basics of cardiology, exercise physiology, and specific issues related to this subgroup of population. Also, careful evaluation of symptoms requires awareness of the atypical presentation. Solving this puzzle is one of the important skills of sports cardiologists. The main symptoms among CAHAP include chest pain, palpitations, syncope, and impaired exercise tolerance (Weizman et al., 2023).

Chest pain

The approach for the diagnosis and management of chest pain in athletes is different from that in the general population. It is crucial to keep in mind the different incidence and presentation as well as the differences that exist according to the type and level of exercise and preexisting cardiac condition.

Non-cardiac causes of chest pain such as gastrointestinal, chest, and musculoskeletal problems are common among athletes of all age groups (Baggish et al., 2017). Cardiac causes of chest pain are age specific. The incidence of chest pain due to CVD in young athletes ≤35 years old is much lower than that in athletes>35 years and is usually due to life-threatening conditions such as hypertrophic cardiomyopathy, coronary artery anomalies, and congenital valvular heart diseases (Maron et al., 1996).

Diagnosis of chest pain due to coronary anomalies in young athletes requires a high index of clinical suspicion due to atypical presentation in most cases. The most common presenting symptom of coronary anomalies is exertional dyspnoea with no chest pain which is usually misdiagnosed. Sometimes it is described as chest discomfort at violent and intense exercise (Williams et al., 2014). The main cause of exertional chest pain in senior athletes > 35 years old is atherosclerotic CVD which usually presents as a warm-up angina (Williams et al., 2014).

Generally, exertional chest discomfort that is associated with newly developed decreased exercise tolerance, dizziness, or syncope in all athletes is an alarming sign of possible and high suspicion of CVD (Weizman et al., 2023).

Exercise-induced asthma can be one of the causes of exercise-induced chest discomfort and can be diagnosed by including spirometry in the work-up before and after the cardiac stress test (Ora et al., 2024). Pleuritic and musculoskeletal chest pain can be associated with strenuous exercise and usually occur in less-trained individuals (Maron et al., 1996).

Thorough and careful assessment of chest pain should be done including history taking with a special focus on the family history of any cardiovascular risk factors, sudden cardiac arrest, familial cardiomyopathies, premature coronary artery diseases (CAD), medications, and illicit or energetic drug use besides a comprehensive clinical examination. The sports cardiologist should postulate the possible aetiology and subsequently request supporting investigations including 12 leads resting ECG, echocardiography, exercise testing, and any further investigations required to discover cardiac aetiology (Gulati et al., 2021).

Echocardiography is the initial investigation of choice to diagnose suspected coronary anomalies being specific and non-invasive, if echocardiography is inconclusive then cardiac magnetic resonance or computed tomography is indicated (Barker & Eickmeyer, 2020).

Syncope

Sudden loss of consciousness and collapse during or shortly after exercise is considered an emergency that needs immediate evaluation (Colivicchi et al., 2004).

More than 6% of athletes experience syncope during their athletic life, preparticipation syncope is usually of neurally mediated type and is of benign outcome (Troiano, 2023). Causes of collapse in athletes, as in the general population, include cardiac and neurally mediated syncope, and other non-cardiac causes like seizures, hyponatremia, and heat stroke. In the case of non-cardiac causes, the collapsed person remains conscious to some extent during the event while in cardiac and neurally mediated syncope the collapsed person develops typical complete loss of consciousness. Cardiac and neurally mediated syncope may mimic the seizures due to acute brain anoxia. The clinical clues of differentiation are that in neurological causes, seizures occur at any time including exercise and the convulsions usually happen before the loss of consciousness. Syncope may be caused by potentially life-threatening circumstances like LV outflow tract obstruction, tachyarrhythmias, bradyarrhythmia (Hosenpud et al., 1987). It may be also related to post-exercise hypotension (Halliwill et al., 2013).

Neurally mediated syncope happens shortly after exercise as well as in special circumstances that are not related to exercise. It is usually preceded by alarming symptoms like flushing and warmth forcing the affected person to lie down or fall slowly. Syncope due to arrhythmia is typically associated with the abrupt loss of consciousness with dramatic fall and possible harm (Colivicchi et al., 2004).

The clues for the diagnosis of syncope include careful history taking, review of circumstances preceding, or if available video capture of the event, time and duration of the event, and potential risks for recurrence (Barker & Eickmeyer, 2020). Adjunct tests like 12 leads ECG and cardiac imaging can be done to exclude underlying structural, electrical, or myocardial disease in case of inconclusive history. Exercise testing may be required to uncover hidden ECG features of electrical disorders like arrhythmia and congenital long QT syndrome (LQTS). Ambulatory rhythm monitoring may be required to diagnose arrhythmias. Prolonged rhythm monitoring, like implantable loop recorders, should be done in some cases of arrhythmia when ordinary tests are non-diagnostic. It should be noted that relative orthostatic intolerance is common in CAHAP as one mechanism of cardiovascular adaptation, positive tilt-table testing is not specific and should not be used to confirm the diagnosis of neurally mediated syncope (García-Sánchez et al., 2020).

Palpitations

There is no doubt that palpitation is common in the athletic population due to the presence of times of excitement, fear, psychological stress, and increased awareness. Benign conditions are the most common causes of palpitation in athletes with subsequent reassurance, and only in rare cases medications to suppress the arrhythmia are required. Palpitations not associated with triggers, those of sudden onset and offset pattern (Abdelfattah & Froelicher, 2015), and palpitations associated with syncope are mostly due to pathological arrhythmia (Abdelfattah & Froelicher, 2015). Alcohol consumption and high caffeine intake including soda and energy drinks are non-cardiac causes of palpitations (Reissig et al., 2009). A family history of SCD, syncope, and palpitations may be an alarm to high risk of Wolf Parkinson White (WPW) syndrome and LQTS (Moore et al., 2016). Clues for detection of life-threatening causes include but are not limited to resting ECG for detection of preexcitation, structural heart disease, and evidence of repolarization abnormalities like the case in LQTS and Brugada syndrome. When the initial diagnostic approach fails to diagnose the nature of the palpitations, ambulatory rhythm monitoring should be done to detect abnormal rhythm correlated to the symptoms. Cardiac stress testing and cardiac imaging may be required to uncover underlying structural or electrical pathology (Abdelfattah & Froelicher, 2015; Reissig et al., 2009).

Shortness of breath and impaired ability to exercise

CAHAP with limited exercise capacity offer subjective accounts of their exercise tolerance by comparing it to that of their more proficient counterparts. Exercise intolerance can manifest either independently or in combination with many symptoms, including chest pain, shortness of breath, dizziness, fainting, palpitations resembling fainting, and overall weariness (Baggish et al., 2017).

Dyspnoea is a major concern in CAHAP. Exertional dyspnoea may be due to respiratory causes such as exercise-induced asthma (EIA) or exercise-induced bronchospasm (EIB), decreased oxygen transport, or may be due to cardiac or musculoskeletal abnormalities. Sometimes it is subjective and does not indicate a true pathology (Carlsen, 2012).

An integrated strategy combining several medical professionals is necessary for a full evaluation of exercise intolerance in CAHAP, depending on the clinical context (Stöhr & Pugh, 2021). The evaluation involves a comprehensive medical history, with a specific focus on exercise training techniques, prospective modifications to lifestyle, previous performance records, and data obtained from wearable monitors. Physical examination involves evaluation of CVD alongside the identification of potential indicators of alternative pathologies such as overtraining, autoimmune diseases, iron deficiencies, viral illnesses, asthma, laryngeal dysfunction, and endocrine problems. Further diagnostic testing should be focused on ruling out CVD, including conditions such as chronotropic incompetence, reduced LV function, coronary artery disease, atrial fibrillation (AF), aortic stenosis, and hypertrophic cardiomyopathy. This should be followed by a more comprehensive evaluation aimed at excluding dysfunction in other organ systems, systemic diseases, and psychiatric disorders, such as depression and anxiety. According to Rajendran et al. (2023), it is recommended that athletes who experience exercise intolerance should regularly check their peak oxygen consumption alongside a relevant measure of external effort, such as treadmill speed/incline or cycle ergometer power. This approach allows for an objective evaluation of their exercise capacity. The differential diagnosis of athletes aged ≥35 years should include an assessment of normal aging. It is important to acknowledge, however, that the process of aging does not lead to sudden and significant decreases in exercise capacity (Maron et al., 2009).

MANAGEMENT OF CARDIOVASCULAR DISEASES IN ATHLETES

One of the most essential duties of a sports cardiologist is to provide athletes with confirmed CVD with continuous and optimal care. Health and safety should always take precedence over athletic objectives, and in most cases, simultaneous attention to both outcomes based on risk-benefit facilitates optimal care (Borjesson et al., 2019).

Myocardial diseases

Hypertrophic cardiomyopathy was the initial genetic cardiac disorder to be associated with sudden death occurring during physical exertion (Zelenović et al., 2021). The diagnostic criteria and clinical management procedures for CAHAP with cardiomyopathy should be consistent with those utilized for less physically active individuals within the broader community. PA and exercise may provoke cardiac dysrhythmias. CAHAP with coexisting cardiomyopathy face a heightened susceptibility to the development of malignant tachyarrhythmias, as well as a greater likelihood of experiencing the advancement of the underlying cardiac condition (Dickie & Terblanche, 2020). Recent results indicate that regular intense PA may potentially elevate the likelihood of developing congestive heart failure and subsequent arrhythmias in individuals diagnosed with arrhythmogenic RV cardiomyopathy (Ahmadi et al., 2022). It is recommended that those diagnosed with or potentially having arrhythmogenic RV cardiomyopathy are discouraged from engaging in strenuous PA. The absolute risk of arrhythmia and disease development in CAHAP with other cardiomyopathies is currently not well understood and is challenging to measure. As a result, there is a lack of consensus advice on involvement in sports, leading to a conservative approach. Conversely, imposing limitations on PA within this demographic could potentially result in noteworthy negative consequences, such as increased body weight and diminished psychological welfare (Corrado et al., 2010). Sports cardiologists have a significant role in the management of CAHAP with cardiomyopathy. They are responsible for developing and improving treatment strategies, which include providing exercise recommendations that aim to minimize risk and maximize the health benefits of regular low- to moderate-intensity exercise according to the study conducted by Ahmadi and colleagues (Ahmadi et al., 2022).

Exercise and even PA can trigger and/or exaggerate myocardial damage in CAHAP. Myocarditis may be due to infectious and non-infectious causes (Dickie & Terblanche, 2020). It is of note that moderate-level PA has a positive effect on the immune system while extreme exertion may impair immune response (Pyne, 1994). The clinical presentation is variable and usually presented as flu-like symptoms for a few days followed by chest pain which may be atypical, a prominent effort intolerance and fatigue. CAHAP are more sensitive to the changes in physical effort. Magnetic resonance imaging of the heart is the most sensitive diagnostic method in athletes. It is important to avoid any form of exertion during the early phase of myocarditis and perform a comprehensive assessment of cardiac function before returning to physical activity and/or competitive exercise (Hurwitz & Issa, 2020).

There is a correlation between myocarditis and a heightened likelihood of experiencing arrhythmia while engaging in athletic activities. Additionally, existing animal studies indicate that exercising during the acute stages of myocarditis may worsen cardiac dysfunction. Therefore, it is advisable to impose limitations on athletic involvement when there is suspicion or confirmation of myocarditis in the individual. Typically, myocarditis is a temporary and restricted condition. After confirming the remission of the disease using a combination of biochemical tests, exercise testing, non-invasive imaging, and ambulatory rhythm monitoring, it is generally suitable to resume exercise fully. The thorough characterization of the time course for the resolution of myocarditis and the prognostic implication of persistent myocardial scarring is still lacking. Most CAHAP will experience full resolution within a period of 3 to 6 months. However, it is worth noting that there are cases when symptoms persist or return, and there may be evidence of persistent inflammation through laboratory tests or imaging. In such instances, it may take longer for individuals to safely resume their training and participation in competitive activities. In addition, it is important to note that exercise alone does not solely account for morbidity and mortality rates. Various other risk variables, like stress and competitive environments, as previously indicated, also play a significant role in contributing to these outcomes (Dickie & Terblanche, 2020; Bonhorst, 2018).

Arrhythmias

In recent decades there has been an ongoing discussion about the relationship between physical activity and arrythmias. Several factors interact with the effect of PA on cardiac rhythm including demographic criteria and level and type of exercise (Newman et al., 2021).

AF is well recognized as the predominant arrhythmia within the general population as well as in athletes. There is a substantial body of published evidence indicating that younger athletes and those who participate in endurance and mixed-type sports have a higher susceptibility to AF (Wilhelm, 2014). The mechanisms and risk factors responsible for the development of atrial arrhythmia in CAHAP are mixed, complex, and not fully comprehended. Causal factors encompass exercise-related atrial remodelling and/or elevated atrial pressure, imbalance in autonomic control including exaggerated vagal tone, inflammation, and genetic predisposition (Hindricks et al., 2021). Diagnosis involves a thorough history targeting to identify the factors that precipitate arrhythmia, physical examination, resting ECG, ergometry, and possibly signal-average analysis of P-wave (Rao & Shipon, 2019).

Due to limited data, the management of atrial arrhythmia in CAHAP is like non-athletes. It includes the elimination of risk factors, rate control or rhythm control strategies, and thromboembolic prophylaxis. Catheter radiofrequency ablation is also successful management. Class IC antiarrhythmic drugs are preferred due to the low risk of adverse effects. Controversies exist about whether it is better rate control or rhythm control, and shared decision-making is advisable. Administration of the first dose of anti-arrhythmogenic therapy should be done under medical observation to guard against unexpected responses (Siegel, 2023).

Several patients with chronic AF or those with modest thromboembolic risk profiles may not require anticoagulant therapy. The selection of either warfarin or a novel oral anticoagulant should be determined by employing algorithms that have been suggested for utilization in the broader community. It is advisable to provide athletes who are using these substances with appropriate counselling regarding the potential hazards of experiencing bleeding incidents during contact sports or sports activities that include a significant risk of physical harm, such as cycling or martial arts. After undergoing catheter ablation, it is recommended to maintain anticoagulation for a certain duration, despite the absence of sufficient data to establish the ideal length of treatment (Figure 2) (Tanasescu, 2002).

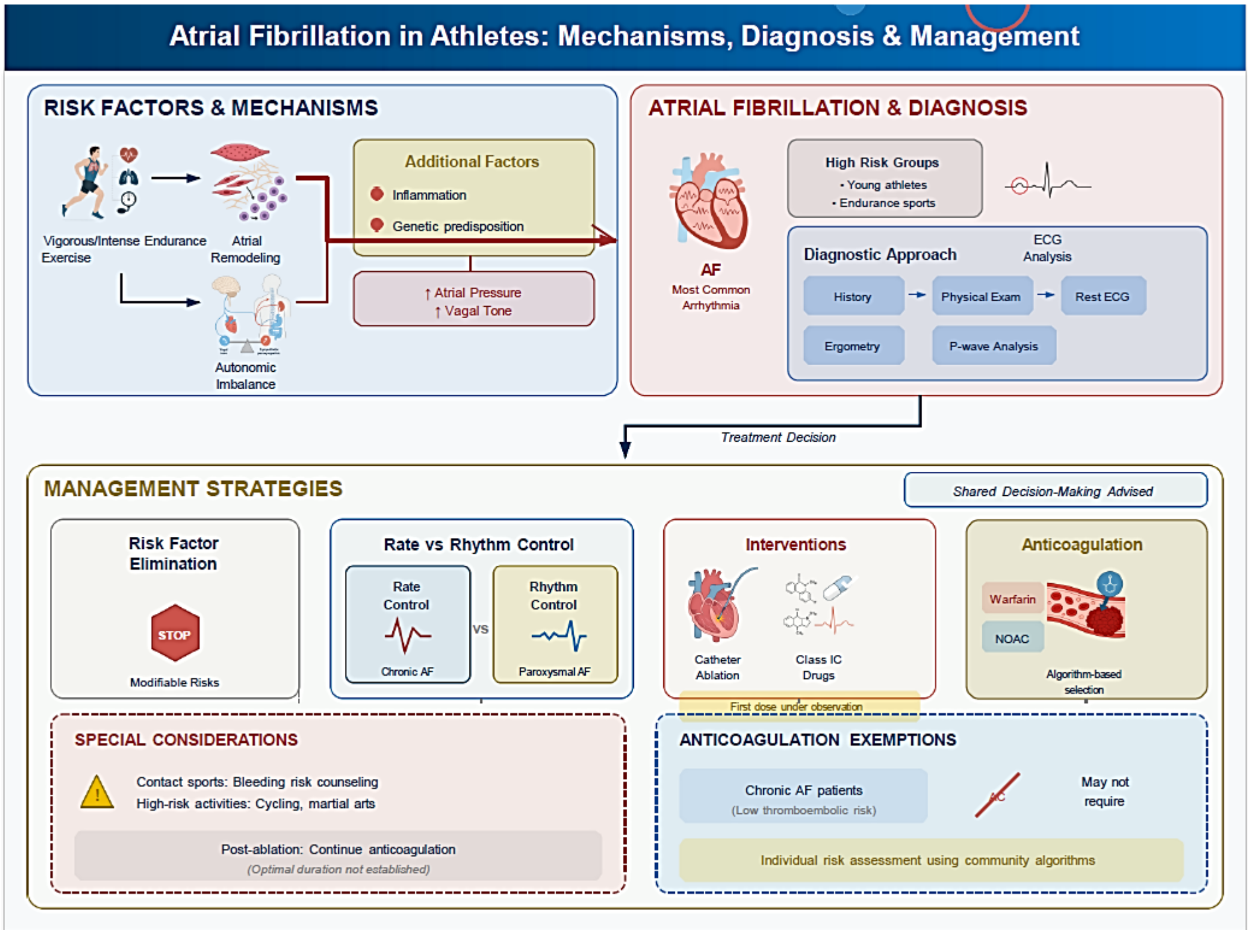

Figure 2. Atrial fibrillation in athletes: mechanisms, diagnosis and management.

Channelopathies

The presence of ion channel abnormalities can potentially elevate susceptibility to malignant arrhythmias, which may lead to catastrophic outcomes, particularly in the context of engaging in athletic activities. Patients with suspected or confirmed channelopathy should undergo evaluation at a specialist centre that demonstrates proficiency in the fields of cardiac electrophysiology and sports cardiology. The utilization of a multidisciplinary approach enables precise assessment of the risk faced by each patient, the tailoring of treatment plans that align with current guidelines, and the provision of informative guidance on long-term strategies for engaging in competitive sports and recreational physical activities.

Preventive measures for CAHAP with channelopathy encompass the avoidance of specific prescription and over-the-counter drugs, as well as the prevention of dehydration, fever, and heat stroke. It is crucial to construct an emergency action plan that prioritizes accessibility to external defibrillators. The engagement in physical activity by individuals diagnosed with LQTS, namely those with type-1, has been linked to a heightened susceptibility to experiencing abnormal heart rhythms (MacLachlan & Drezner, 2020). Nevertheless, the most recent observational data indicates that there have been no reported fatalities among athletes with LQTS who choose to continue participating in sports while receiving adequate treatment for their condition (Lampert et al., 2023).

A comprehensive collaborative decision-making process is required to engage the sports cardiologist in the decision-making to permit unrestricted sports participation among athletes diagnosed with LQTS or other comparable conditions carrying equivalent risks. CAHAP often exhibits WPW syndrome, as well as benign ventricular and supraventricular arrhythmias. The definitive diagnosis, risk classification, and therapy are the joint responsibilities of the sports cardiologist and the cardiovascular electrophysiologist. The assessment may encompass exercise testing that is restricted by maximal exertion and an invasive electrophysiological study, which can be employed to ascertain the most effective approaches for managing the condition. These approaches may involve observation, medication therapy, or catheter ablation. Catheter-based ablation provides a long-lasting remedy. Usually, the implementation of an efficacious treatment regimen will adequately diminish or eradicate potential risks, enabling individuals to engage in competitive athletic activities without any limitations (Mora et al., 2007).

Coronary artery disease

Regular PA has been shown to have positive impacts on conventional risk factors associated with CVD (Borjesson et al., 2019). Despite this fact, atherosclerotic coronary artery disease is a common cause of sudden death in veteran athletes. Usually CAD is due to rupture of/or haemorrhage inside superficial plaques. Exercise-related vascular effects are blunted with increased exercise intensity. High-intensity exercise is associated with increased inflammatory mediators and oxygen-free radicals. Mismatched myocardial needs and supply is the main mechanism for CAD. Other factors contributing to CAD in athletes are age, genetic factors, infections, and structural, functional, and physiologic changes associated with exercise. Endurance-type is also associated with increased calcification of coronary arteries. The relationship between exercise and CAD has several grey areas and unclear mechanisms that are still under discussion (Dores et al., 2018; Katsi et al., 2021; Borjesson et al., 2019).

The presentation of CAD in athletes may be silent or with symptoms of angina. The management is based on the recent guidelines. Exercise tests and stress imaging are important to select those with suspected CAD for further coronary artery evaluation. Those with established CAD are to be treated according to guidelines and restriction of high-intensity PA or complete prevention of competitive sports is recommended based on the risk of cardiac events category (Borjesson et al., 2019).

Statins or cholesterol-lowering agents and antiplatelet drugs are prescribed according to the results of imaging of coronary arteries and risk for cardiac events (Aengevaeren & Eijsvogels, 2020). It is worth mentioning that musculoskeletal adverse events resulting from statins can be exaggerated by PA. Nevertheless, it is imperative to emphasize statin therapy after an acute coronary syndrome, even if it necessitates reducing exercise training and competitiveness (Deichmann et al., n.d.).

However, the potential utilization of these agents could be contemplated. It is strongly recommended that individuals with coronary artery disease who are experiencing acute coronary syndrome should actively engage in a structured cardiac rehabilitation program under the supervision of a sports cardiologist. This professional can customize an exercise plan that may involve higher intensity levels than those typically prescribed for individuals with a more sedentary lifestyle (van Hattum et al., 2022).

Hypertension

There is an inverse relationship between PA and blood pressure (Bp). However, athletes are not fully protected. Hypertension is reported to be prevalent among CAHAP, though underdiagnosed and undertreated. Preparticipation measurement of Bp as well as evaluation of those at risk of hypertension. Evaluation of Bp response to exercise can be evaluated during stress tests (Schweiger et al., 2021).

Office BP measurements are recommended, and ambulatory measurement is to be carried out for those with suspected white-coat or masked hypertension. Besides, evaluation of organ dysfunction and associated comorbidities. It is recommended to evaluate the blood pressure in both brachial arteries during each clinical contact in CAHAP. Additionally, it is advisable to monitor blood pressure at least once in the leg (Mancia et al., 2023).

Individuals without pre-existing cardiovascular disease or resting hypertension are expected to experience transient maximal systolic blood pressures of 250 mm Hg or greater, which is both normal and expected (Refoyo et al., 2023).

Management of hypertension in CAHAP is the same as in the general population. Non-pharmacologic therapy such as lifestyle Modulation and endurance exercise if not already performed may be beneficial. In the case of grade 3 hypertension and those with associated comorbidities, pharmacotherapy should not be delayed. Diuretics and beta blockers are banned. Vasodilators have a low impact on exercise physiology such as angiotensin-converting enzyme inhibitors, angiotensin-II receptor antagonists, calcium channel blockers or alpha-blockers are the recommended drugs. Exercise intensification or restriction is tailored based on the patient’s clinical condition and other CVD risk factors as well as the results of exercise blood pressure measurement (Mancia et al., 2023).

Coronavirus Disease-2019 (COVID-19) and the athletic heart

It is well-established that COVID-19 has cardiovascular effects (Sobh et al., 2021). These effects are variable in CAHAP. Some studies reported more profound and persistent effects especially myocarditis. However, the exact frequency and significance of cardiac abnormalities associated with COVID-19 in athletes is still unknown. The development of a safe return-to-play algorithm specifically designed for adult athletes engaged in competitive sports is crucial for risk prevention (Gluckman et al., 2022). Finally, shared decision making is important for athletes, especially in areas of controverses or debates.

CONCLUSIONS

Sports cardiology is an emerging field in cardiology with rapid evolution. Optimal care of physically active people requires an integrated approach with enough relevant knowledge and skills. Subsequently, it is of vital importance that high-quality standards of care and target-based research initiatives continue to develop to achieve the main goal of sports cardiology ensuring maximum safety for this group of the population. To advance cardiovascular knowledge and skills among trainees and practitioners, it is imperative to implement educational initiatives in the future. These initiatives should be specifically tailored to meet the needs of both aspiring cardiovascular professionals and those who are already established in the field. Furthermore, the scientific community must assume the responsibility of overseeing outcomes-based research to effectively address the numerous unresolved controversies.

AUTHOR CONTRIBUTIONS

All authors meet the criteria for authorship in accordance with established ethical guidelines. Contributions are specified according to the CRediT (Contributor Roles Taxonomy) as follows:

Conceptualization: Mohammed Mohyeldin, Muhammad Reihan. Methodology: Mohammed Mohyeldin, Mohamed Roshdi. Formal analysis: Eman Sobh. Investigation: Husna Irfan Thalib, Ayesha Jamal. Data curation: Fatma Ahmed, Muhammad Reihan. Writing – original draft: Husna Irfan Thalib, Ayesha Jamal.

Writing – review & editing: Mohammed Mohyeldin, Mohamed Roshdi, Eman Sobh. Supervision: Mohammed Mohyeldin, Eman Sobh All authors have critically reviewed and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this manuscript.

AI USE DISCLOSURE

In accordance with current publishing ethics and transparency recommendations, artificial intelligence (AI) tools were used solely to assist with translation and language editing, with the aim of improving clarity and readability. No AI tools were used in the generation of scientific content, including the study design, data collection, analysis, interpretation of results, or the formulation of conclusions. The authors retain full responsibility for the content of the manuscript and confirm its originality, integrity, and accuracy.

REFERENCES

Abdelfattah, R. S., & Froelicher, V. F. (2015). Palpitations in athletes. Current Sports Medicine Reports, 14(5), 333-336. https://doi.org/10.1097/01.CSMR.0000466789.13643.f1

Aengevaeren, V. L., & Eijsvogels, T. M. H. (2020). Coronary atherosclerosis in middle-aged athletes: Current insights, burning questions, and future perspectives. Clinical Cardiology, 43(8), 863-871. https://doi.org/10.1002/clc.23340

Ahmadi, M. N., Gill, J. M. R., & Stamatakis, E. (2022). Association of changes in physical activity and adiposity with mortality and incidence of cardiovascular disease: Longitudinal findings from the UK Biobank. Mayo Clinic Proceedings, 97(5), 847-861. https://doi.org/10.1016/j.mayocp.2021.11.026

American Heart Association. (2021). Pre-participation cardiovascular screening of young competitive athletes: Policy guidance.

Baggish, A. L., Battle, R. W., Beckerman, J. G., Bove, A. A., Lampert, R. J., Levine, B. D., et al. (2017). The present and future sports cardiology core curriculum for providing cardiovascular care to competitive athletes and highly active people. Journal of the American College of Cardiology, 70(14), 1902-1918. https://doi.org/10.1016/j.jacc.2017.08.055

Baggish, A. L., & Wood, M. J. (2011). Athlete's heart and cardiovascular care of the athlete. Circulation, 123(23), 2723-2735. https://doi.org/10.1161/CIRCULATIONAHA.110.981571

Barker, K., & Eickmeyer, S. (2020). Therapeutic exercise. Medical Clinics of North America, 104(2), 189-198. https://doi.org/10.1016/j.mcna.2019.10.003

Borjesson, M., Dellborg, M., Niebauer, J., LaGerche, A., Schmied, C., Solberg, E. E., et al. (2019). Recommendations for participation in leisure time or competitive sports in athletes-patients with coronary artery disease. European Heart Journal, 40(1), 13-18. https://doi.org/10.1093/eurheartj/ehy408

Burtscher, J., Strasser, B., Burtscher, M., & Millet, G. P. (2022). The impact of training on the loss of cardiorespiratory fitness in aging master's endurance athletes. International Journal of Environmental Research and Public Health, 19, 11050. https://doi.org/10.3390/ijerph191711050

Carlsen, K.-H. (2012). Mechanisms of asthma development in elite athletes. Breathe, 8(4), 278-284. https://doi.org/10.1183/20734735.009512

Colivicchi, F., Ammirati, F., & Santini, M. (2004). Epidemiology and prognostic implications of syncope in young competing athletes. European Heart Journal, 25(20), 1749-1753. https://doi.org/10.1016/j.ehj.2004.07.011

Corrado, D., Pelliccia, A., Heidbuchel, H., Sharma, S., Link, M., Basso, C., et al. (2010). Recommendations for interpretation of 12-lead electrocardiogram in the athlete. European Heart Journal, 31(2), 243-259. https://doi.org/10.1093/eurheartj/ehp473

Dawes, T. J. W., Corden, B., Cotter, S., de Marvao, A., Walsh, R., Ware, J. S., et al. (2016). Moderate physical activity in healthy adults is associated with cardiac remodeling. Circulation: Cardiovascular Imaging, 9(12). https://doi.org/10.1161/CIRCIMAGING.116.004712

Deichmann, R. E., Lavie, C. J., Asher, T., DiNicolantonio, J. J., O'Keefe, J. H., & Thompson, P. D. (n.d.). The interaction between statins and exercise: Mechanisms and strategies to counter musculoskeletal side effects.

Dickie, K. E., & Terblanche, E. (2020). Behavioural lifestyle factors, physical health-related fitness and cardiometabolic disease risk in women from a low socio-economic urban community in Stellenbosch (Western Cape). [Unpublished manuscript].

Dores, H., de Araújo Gonçalves, P., Cardim, N., & Neuparth, N. (2018). Coronary artery disease in athletes: An adverse effect of intense exercise? Revista Portuguesa de Cardiologia, 37(1), 77-85. https://doi.org/10.1016/j.repc.2017.06.006

Finch, J. A. M., & Baggish, A. L. (2016). Cardiovascular evaluation and treatment of the endurance athlete. In Endurance sports medicine (pp. 3-19). Springer. https://doi.org/10.1007/978-3-319-32982-6_1

García-Sánchez, E., Rubio-Arias, J. Á., Ávila-Gandía, V., López-Román, F. J., & Menarguez-Puche, J. F. (2020). Effects of two community-based exercise programs on adherence, cardiometabolic markers, and body composition in older people with cardiovascular risk factors: A prospective observational cohort study. Journal of Personalized Medicine, 10(4), 176. https://doi.org/10.3390/jpm10040176

Gibson, A. L., Wagner, D., & Heyward, V. (2019). Advanced fitness assessment and exercise prescription (8th ed.). Human Kinetics. https://doi.org/10.5040/9781718220966

Gleason, P. T., & Kim, J. H. (2017). Exercise and competitive sport: Physiology, adaptations, and uncertain long-term risks. Current Treatment Options in Cardiovascular Medicine, 19(9), 79. https://doi.org/10.1007/s11936-017-0578-7

Gluckman, T. J., Bhave, N. M., Allen, L. A., Chung, E. H., Spatz, E. S., Ammirati, E., et al. (2022). 2022 ACC expert consensus decision pathway on cardiovascular sequelae of COVID-19 in adults. Journal of the American College of Cardiology, 79(17), 1717-1756. https://doi.org/10.1016/j.jacc.2022.02.003

Gulati, M., Levy, P. D., Mukherjee, D., Amsterdam, E., Bhatt, D. L., Birtcher, K. K., et al. (2021). 2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR guideline for chest pain evaluation. Journal of the American College of Cardiology, 78(22), e187-e285. https://doi.org/10.1016/j.jacc.2021.07.053

Halliwill, J. R., Buck, T. M., Lacewell, A. N., & Romero, S. A. (2013). Postexercise hypotension and sustained postexercise vasodilatation: What happens after we exercise? Experimental Physiology, 98(1), 7-18. https://doi.org/10.1113/expphysiol.2011.058065

Han, J., Lalario, A., Merro, E., Sinagra, G., Sharma, S., Papadakis, M., et al. (2023). Sudden cardiac death in athletes: Facts and fallacies. Journal of Cardiovascular Development and Disease, 10(2), 68. https://doi.org/10.3390/jcdd10020068

Hindricks, G., Potpara, T., Dagres, N., Arbelo, E., Bax, J. J., Blomström-Lundqvist, C., et al. (2021). 2020 ESC guidelines for atrial fibrillation. European Heart Journal, 42(5), 373-498. https://doi.org/10.1093/eurheartj/ehaa612

Hosenpud, J. D., Campbell, S. M., Niles, N. R., Lee, J., Mendelson, D., & Hart, M. V. (1987). Exercise-induced autoimmunity in myocarditis. Cardiovascular Research, 21(3), 217-222. https://doi.org/10.1093/cvr/21.3.217

Hurwitz, B., & Issa, O. (2020). Management and treatment of myocarditis in athletes. Current Treatment Options in Cardiovascular Medicine, 22, 65. https://doi.org/10.1007/s11936-020-00875-1

Ito, S. (2022). High-intensity interval training for older adults: Safety issues. European Journal of Medical and Health Sciences, 4(1), 3-5. https://doi.org/10.24018/ejmed.2022.4.1.1224

Karam, N., Pechmajou, L., Narayanan, K., Bougouin, W., Sharifzadehgan, A., Anys, S., et al. (2022). Sports-related sudden cardiac arrest outcomes. Journal of the American College of Cardiology, 79(3), 238-246. https://doi.org/10.1016/j.jacc.2021.11.011

Katsi, V., Soulaidopoulos, S., Aggeli, C., Latsios, G., Tousoulis, D., & Toutouzas, K. (2021). Extensive coronary artery disease in a long-distance athlete. Journal of Athletic Training, 56(11), 1137-1141. https://doi.org/10.4085/JAT0330-20

Lampert, R., Ackerman, M. J., Marino, B. S., Burg, M., Ainsworth, B., Salberg, L., et al. (2023). Vigorous exercise in hypertrophic cardiomyopathy. JAMA Cardiology, 8(6), 595-603. https://doi.org/10.1001/jamacardio.2023.1042

Livingston, L., Forbes, S. L., Wattie, N., & Cunningham, I. (2020). Sport officiating: Recruitment, development, and retention. Routledge. https://doi.org/10.4324/9780429465291

MacLachlan, H., & Drezner, J. A. (2020). Risk-based cardiac evaluation of young athletes. Clinical Cardiology, 43(9), 906-914. https://doi.org/10.1002/clc.23364

Mancia, G., Kreutz, R., Brunström, M., Burnier, M., Grassi, G., Januszewicz, A., et al. (2023). 2023 ESH hypertension guidelines. Journal of Hypertension, 41(12), 1874-2071. https://doi.org/10.1097/HJH.0000000000003480

Maron, B. J., Doerer, J. J., Haas, T. S., Tierney, D. M., & Mueller, F. O. (2009). Sudden deaths in athletes. Circulation, 119(8), 1085-1092. https://doi.org/10.1161/CIRCULATIONAHA.108.804617

Maron, B. J., Thompson, P. D., Puffer, J. C., McGrew, C. A., Strong, W. B., Douglas, P. S., et al. (1996). Preparticipation screening. Circulation, 94(4), 850-856. https://doi.org/10.1161/01.CIR.94.4.850

McDiarmid, A. K., Swoboda, P. P., Erhayiem, B., Lancaster, R. E., Lyall, G. K., Broadbent, D. A., et al. (2016). Athletic cardiac adaptation. Circulation: Cardiovascular Imaging, 9(12), e003579. https://doi.org/10.1161/CIRCIMAGING.115.003579

Mora, S., Cook, N., Buring, J. E., Ridker, P. M., & Lee, I.-M. (2007). Physical activity and cardiovascular risk. Circulation, 116(19), 2110-2118. https://doi.org/10.1161/CIRCULATIONAHA.107.729939

Newman, W., Parry-Williams, G., Wiles, J., Edwards, J., Hulbert, S., Kipourou, K., et al. (2021). Atrial fibrillation in athletes meta-analysis. British Journal of Sports Medicine, 55(21), 1233-1238. https://doi.org/10.1136/bjsports-2021-103994

Ora, J., De Marco, P., Gabriele, M., Cazzola, M., & Rogliani, P. (2024). Exercise-induced asthma. Journal of Functional Morphology and Kinesiology, 9(1), 15. https://doi.org/10.3390/jfmk9010015

Paquette, M. R., Napier, C., Willy, R. W., & Stellingwerff, T. (2020). Training load quantification. Journal of Orthopaedic & Sports Physical Therapy, 50(10), 564-569. https://doi.org/10.2519/jospt.2020.9533

Pescatello, L. S., MacDonald, H. V., Ash, G. I., Lamberti, L. M., Farquhar, W. B., Arena, R., et al. (2015). Hypertension exercise recommendations. Mayo Clinic Proceedings, 90(6), 801-812. https://doi.org/10.1016/j.mayocp.2015.04.008

Petek, B. J., & Baggish, A. L. (2020). Pre-participation screening controversies. Expert Review of Cardiovascular Therapy, 18(7-8), 435-442. https://doi.org/10.1080/14779072.2020.1787154

Pyne, D. B. (1994). Neutrophil function during exercise. Sports Medicine, 17(4), 245-258. https://doi.org/10.2165/00007256-199417040-00005

Rajendran, A. J., George, P., Srichandran, L., & Vijayashree, N. (2023). Cardiovascular rehabilitation. In Cardiovascular and kidney disease within the geriatric population in developing countries (pp. 243-267).

Rao, P., & Shipon, D. M. (2019). Atrial fibrillation in athletes. American College of Cardiology.

Refoyo, E., Troya, J., de la Fuente, A., Beltrán, A., Celada, O. L., Díaz-González, L., et al. (2023). Myocardial work index in football players. Journal of Clinical Medicine, 12(9), 3059. https://doi.org/10.3390/jcm12093059

Reissig, C. J., Strain, E. C., & Griffiths, R. R. (2009). Energy drinks problem. Drug and Alcohol Dependence, 99(1-3), 1-10. https://doi.org/10.1016/j.drugalcdep.2008.08.001

Scharhag, J., Schneider, G., Urhausen, A., Rochette, V., Kramann, B., & Kindermann, W. (2002). Athlete's heart. Journal of the American College of Cardiology, 40(10), 1856-1863. https://doi.org/10.1016/S0735-1097(02)02478-6

Schweiger, V., Niederseer, D., Schmied, C., Attenhofer Jost, C., & Caselli, S. (2021). Athletes and hypertension. Current Cardiology Reports, 23, 176. https://doi.org/10.1007/s11886-021-01608-x

Siegel, A. J. (2023). Marathon aspirin prevention. American Journal of Medicine, 136(5), 613-615. https://doi.org/10.1016/j.amjmed.2023.02.007

Sobh, E., Reihan, M. S., Hifnawy, T. M. S., Abdelsalam, K. G., Awad, S. S., Mahmoud, N. M. H., et al. (2021). COVID-19 cardiovascular effects. The Egyptian Heart Journal, 73, 77. https://doi.org/10.1186/s43044-021-00202-4

Stöhr, E. J., & Pugh, C. J. A. (2021). Endurance athlete circulation. Atherosclerosis, 320, 89-91. https://doi.org/10.1016/j.atherosclerosis.2021.01.019

Tanasescu, M. (2002). Exercise and coronary heart disease. JAMA, 288(16), 1994-2000. https://doi.org/10.1001/jama.288.16.1994

Teyhen, D. S., Shaffer, S. W., Goffar, S. L., Kiesel, K., Butler, R. J., Rhon, D. I., et al. (2020). Injury risk factors in athletes. Sports Health, 12(6), 564-572. https://doi.org/10.1177/1941738120902991

Troiano, R. P. (2023). Physical activity accelerometers. Journal of Measurement in Physical Behaviour, 6(1), 13-18. https://doi.org/10.1123/jmpb.2022-0038

U.S. Department of Health and Human Services. (2008). Physical activity guidelines for Americans.

van Hattum, J. C., Verwijs, S. M., Senden, P. J., Spies, J. L., Boekholdt, S. M., Groenink, M., et al. (2022). Sports cardiology team approach. Mayo Clinic Proceedings: Innovations, Quality & Outcomes, 6(5), 525-535. https://doi.org/10.1016/j.mayocpiqo.2022.08.006

Weizman, O., Empana, J.-P., Blom, M., Tan, H. L., Jonsson, M., Narayanan, K., et al. (2023). Cardiac arrest in women sports. Journal of the American College of Cardiology, 81(10), 1021-1031. https://doi.org/10.1016/j.jacc.2023.01.015

Wilhelm, M. (2014). Atrial fibrillation in endurance athletes. European Journal of Preventive Cardiology, 21(8), 1040-1048. https://doi.org/10.1177/2047487313476414

Williams, R. P., Manou-Stathopoulou, V., Redwood, S. R., & Marber, M. S. (2014). Warm-up angina. Heart, 100(2), 106-114. https://doi.org/10.1136/heartjnl-2013-304187

Zelenović, M., Božić, D., Aksović, N., Iacob, G. S., Alempijević, R., & Bjelica, B. (2021). Physical activity and mortality. International Journal of Sport Culture and Science, 9(3), 255-267.