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**REVIEWS**
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# Athletes and the cardiovascular system
## Afectarea cardiovasculară la sportivi

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**Abstract**

The cardiac changes of athletes in response to systematic conditioning are somewhat variable, with some degree of cardiac remodeling in approximately one-half of trained athletes. A variety of cardiovascular diseases represent the most common causes of sudden death in young athletes. The vast majority of these deaths in athletes younger than 35 years of age are due to several congenital or acquired cardiac malformations. Hypertrophic cardiomyopathy is the single most common cause of athlete deaths (responsible for approximately one third of the cases), followed by congenital coronary artery anomalies. The vast majority of deaths in middle-aged athletes are due to unsuspected atherosclerotic coronary artery disease.

Recently, recommendations of the European Society of Cardiology (ESC) and the International Olympic Committee (IOC) have triggered a new debate regarding the most appropriate strategy for screening trained athletes and other sports participants.

**Key words:** cardiovascular system, athletes, sudden death

**Rezumat**

Modificările cardiace la sportivi, ca răspuns la condiţionarea sistemică, sunt variabile, iar la aproximativ o jumătate dintre aceştia, este întâlnit un anumit grad de remodelare cardiacă. Cele mai frecvente cauze de moarte subită la tinerii sportivi sunt reprezentate de o varietate de boli cardiovasculare. Majoritatea acestor decese la sportivii sub 35 de ani sunt cauzate de anomalii cardiace congenitale sau dobândite. Cardiomiopatia hipertrofică este cauza cea mai frecventă a deceselor la sportivi (responsabilă pentru aproximativ o treime din cazuri), urmată de anomalii coronariene congenitale. Majoritatea deceselor la sportivii de vârstă medie se datorează bolii aterosclerotice coronariene nediagnosticate.

Recent, Societatea Europeană de Cardiologie (ESC) şi Comitetul Olimpic Internaţional (CIO) au dezbătut cea mai potrivită strategie privind screeningul cardiovascular al sportivilor.

**Cuvinte cheie:** sistem cardiovascular, sportivi, moarte subită.

## Introduction

Physical exercise plays an important role in the primary and secondary prevention of cardiovascular diseases, reducing the effect of risk factors (arterial hypertension, diabetes mellitus, dyslipidemia, obesity) (Chandra et al., 2013; Cordero et al., 2014; Brinker et al., 2014). However, paradoxically, high performance athletes have a higher risk of exercise-induced sudden cardiac death (Lawless et al., 2014). The term athlete refers to persons who train for more than six months, more than six hours a week (Carre, 2012). The first who noted that the heart of trained athletes differed from that of the general population was Henschen in 1899 (Weiner & Baggish, 2012). Based on auscultation and percussion, he found that skiers had increased cardiac cavity dimensions. The clinical observations made in 1899 were proved only in 1950 using chest radiography (Pelliccia et al., 2012). With the development of ultrasound, cardiac remodeling in athletes could be better studied.

The constant, intense practice of a sport for a prolonged time period may induce adaptive clinical, electrical and morphological changes in the cardiovascular system, known as "athlete's heart" (Carre, 2012). Cardiac changes in athletes in response to systemic conditioning are variable, and a certain degree of cardiac remodeling is found in approximately half of these (Pelliccia et al., 2012). Dynamic, endurance aerobic isotonic exercise leads to a chronic volume overload, which results in an increase of

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left ventricular mass in parallel to left ventricular diameter, and cardiac remodeling through eccentric hypertrophy develops. In contrast, strength anaerobic isometric exercise increases pressure, resulting in concentric hypertrophy, through the increase of parietal thickness, without the dilation of cardiac cavities (Cordero et al., 2014; Lawless et al., 2014; Weiner & Baggish, 2012; Pelliccia et al., 2012; Ginghină, 2010). Cardiovascular changes associated with athlete's heart include: the increase of parietal thickness and biventricular cavity dimensions, the increase of the left atrial cavity size (and volume), associated with normal or supranormal systolic and diastolic ventricular function (Cabanolas, 2013; Bonow et al., 2012; Apor et al., 2013; Griffet et al., 2013); however, a mild systolic dysfunction was found to occur in 11% of cyclists (Lawless et al., 2014). These changes are most frequently reversible after short deconditioning periods (4-6 weeks up to 3 months), but there is evidence that cardiac remodeling is not completely reversible even after many years of deconditioning, which raises the suspicion that prolonged training may induce permanent myocardial impairment (Ginghină, 2010; Bonow et al., 2012; Luthi et al., 2008; Baldesberger et al., 2008).

## Competition preparticipation screening

Screening recommendations for athletes differ depending on the country, sport, and competition level, and are mainly aimed at detecting cardiac diseases with a risk for sudden death (La Gerche et al., 2013). In 1982, the first screening program for athletes, compulsory from 12 to 35 years, with re-evaluation every 2 years, including anamnesis, objective examination and ECG, was implemented in Italy. This program proved to be highly effective, decreasing mortality among athletes by 90% (Dougherthy et al., 2013; Myerson et al., 2012). In USA, the screening program became compulsory only starting with 2007 (according to AHA), and it only comprises anamnesis and objective examination, without ECG (Myerson et al., 2012; Grazioli et al., 2014). In Japan, a screening program was implemented in 1973, with the identification of athletes at risk, these measures proving to be effective for the decrease of morbidity/mortality in time (Nistiuchi et al., 2014). In 2004, ESC (European Society of Cardiology) and the International Olympic Committee decided to include ECG in the screening of athletes (Myerson et al., 2012).

## Causes of sudden death in athletes

Sudden cardiac death (SCD) is the main cause of death in athletes and occurs within an hour from the onset of symptomatology (Dougherthy et al., 2013; Leikin et al., 2013). The incidence of sudden death in young athletes aged less than 35 years is 0.4 per 100,000/year (Italy), between 2.3-4.4/100,000/year (USA), and is 10 times more frequent in men (Chandra et al., 2013; Cordero et al., 2014; La Gerche et al., 2013; Dougherthy et al., 2013; Demorest, 2013). Only 30% of athletes with SCD have a positive family history (Leikin et al., 2013). The most frequent causes of sudden death in young athletes in USA are: hypertrophic cardiomyopathy, coronary anomalies, RV arrhythmogenic dysplasia, myocarditis, channelopathies, frequently through the ventricular arrhythmias that they induce. In Italy, the most common cause of sudden cardiac death is RV arrhythmogenic dysplasia (Grubb et al., 2012). In USA, these deaths are more frequently found in basketball and football, sports with the highest participation levels, which involve intense physical activity. Athletes aged over 35 years have a different demographic profile, with a more frequent participation in individual sports such as the marathon, and in 80% of them, sudden death is secondary to undiagnosed coronary ischemic disease (Heidbuchel et al., 2012; Massoure et al., 2014). This is why a preparticipation screening program is extremely important, in order to detect potential cardiovascular diseases with an increased risk of sudden death, to provide treatment, and sometimes to recommend the cessation of high performance sports activity.

## ECG changes in athletes

Electrocardiographic changes are present in 60% of athletes, being more frequent in men (10 times more frequent) and in athletes who practice endurance sports (Williams et al., 2012; Brosnan et al., 2014; Drezner & Ackerman, 2013). The majority of cardiovascular diseases can be suspected based on ECG changes. In February 2012, a group of experts in sports cardiology set up the ECG criteria allowing for a differential diagnosis between normal ECG in athletes and ECG changes requiring additional investigations for the detection of underlying cardiovascular disorders. These are found in the literature as the Seattle ECG criteria (Drezner & Ackerman, 2013). Due to regular training > 4 hours/week, ECG changes occur, which reflect the benign structural and electrical remodeling of the heart, secondary to the increase of vagal tone and cardiac cavity dimensions (Drezner & Fischbach, 2013). According to the Seattle criteria, these *physiological* changes are: *sinus bradycardia* (a ventricular rate between 30 and 60 beats per minute; it occurs in 80% of athletes), *sinus arrhythmia* (in 55% of athletes, more frequently in young athletes), *atrial or junctional rhythm*, *first degree AVB* (PR > 200 ms) and *Mobitz 1 AVB II* (benign if disappearing during exercise), *incomplete RBBB* (in 40% of athletes), *early repolarization syndrome; left ventricular hypertrophy* only based on increased QRS voltage criteria (Sokolow Lyon index) (Drezner & Ackerman, 2013; Drezner & Fischbach, 2013). In athletes of Afro-American origin, early repolarization changes are frequently found, with convex ST segment elevation (in two thirds of athletes), with J point elevation, followed by negative T waves in the anterior territory (25%) (Drezner & Fischbach, 2013; Noseworthy & Baggish, 2013; Muramoto et al., 2013; Pagourelios et al., 2011).

ECG changes considered to be *pathological* in athletes according to the Seattle criteria are: T wave inversion, ST segment depression, pathological Q waves, bundle branch blocks, left or right axial deviation, left atrial or right ventricular hypertrophy, ventricular preexcitation syndrome, long QT syndrome (>470 ms in men, >480 ms in women) or short QT syndrome (<340 ms), Brugada like early repolarization, sinus bradycardia <30/minute or sinus pause >3 seconds, ventricular extrasystoles >2 in 10 seconds, supraventricular or ventricular tachyarrhythmia

(Drezner & Ackerman, 2013; Drezner & Fischbach, 2013; Erz et al., 2013).

## Structural cardiac diseases associated with SCD

*Hypertrophic cardiomyopathy* (HCM) is the most frequent cause of sudden cardiac death in USA, occurring in approximately one third of athletes, and in UK in 11% of athletes. In current practice, this pathology frequently poses problems of differential diagnosis with changes specific for athlete's heart, which occurs as benign cardiac remodeling during exercise. HCM is an asymmetrical ventricular hypertrophy (with a parietal thickness > 15 mm), with a small LV (frequently less than 45 mm), dilated left atrium, with diastolic dysfunction (due to microvascular dysfunction that frequently leads to ischemia), with altered LV geometry (affected mitral valve), with left ventricular ejection obstruction, being found in approximately 25% of athletes (Pelliccia et al., 2012; La Gerche et al., 2013; Leikin et al., 2013; Drezner & Ashley, 2013). Contrast MRI can detect myocardial fibrosis areas, which underlie subsequent ventricular arrhythmias (La Gerche et al., 2013). ECG is pathological in approximately 90% of athletes with HCM. Anomalies include LVH accompanied by T wave inversion in the infero-lateral territory, ST segment depression, pathological Q, LBBB, left axial deviation, left atrial dilation (Drezner & Ashley, 2013). Risk factors for SCD are: syncope, decrease of blood pressure during exercise, a positive family history, LV parietal thickness > 30 mm, sustained or non-sustained ventricular arrhythmia, which requires the implantation of a prophylactic intracardiac defibrillator (Demorest, 2013). A possible variant of HCM is solitary papillary muscle hypertrophy (Panduranga et al., 2013).

*Right ventricular arrhythmogenic dysplasia (RVAD)*. In Italy, a quarter of autopsied sudden deaths were of right ventricular arrhythmogenic dysplasia etiology (Drezner & Ashley, 2013). The presence of this disease increases 5 times the risk of exercise-induced sudden death (Chandra et al., 2013; James et al., 2013). It consists of the replacement of myocardial tissue with steatofibrous deposits, which cause RV dilation and dysfunction, with the formation of aneurysms, with an increased risk of ventricular arrhythmia and SCD (Chandra et al., 2013; Demorest, 2013; Heidbuchel et al., 2012; Williams et al., 2012; Drezner & Ashley, 2013). Only in 50% of the cases, it is associated with specific desmosomal mutations (Drezner & Ashley, 2013). In 10% of the cases, it can manifest only in the left ventricle (Drezner & Ashley, 2013). ECG is abnormal only in 80% of the cases, with the presence of the following: negative V1-V4 T waves with an isoelectric ST segment, epsilon waves, microvoltage in the limb leads, ventricular extrasystoles with a LBBB appearance (Chandra et al., 2013; Drezner & Ashley, 2013).

Recently, the hypothesis has been advanced that prolonged physical exercise may cause, due to cardiac remodeling that is more important in the right ventricle, RV dilation and dysfunction, with a proarrhythmogenic effect (in one third of athletes). Thus, a new syndrome has been described: *exercise-induced RV arrhythmogenic cardiomyopathy*, which occurs in athletes and is pathogenetically similar to RVAD. This syndrome is more common in cyclists (80%) (Heidbuchel et al., 2012; James et al., 2013; Steriotis et al., 2013; D'Andreea et al., 2013).

*Coronary anomalies* are the second most frequent cause of SCD in USA (12-33%), the first most frequent cause being left coronary artery arising from the right coronary ostium (Chandra et al., 2013; Demorest, 2013; Massoure et al., 2014; Guenarcia et al., 2013; Kaski et al., 2013). Coronary flow decreases during exercise due to: an abnormal ostium, compression of coronary arteries between the great vessels, coronary spasm. Only a third of the affected subjects have preceding symptoms (angina, syncope, dyspnea) (Trahan et al., 2014). Rest ECG is normal, but the exercise test is negative in 20% of them. This pathology favors the development of early ischemia and ventricular arrhythmia (Chandra et al., 2013; Demorest, 2013; Massoure et al., 2014; Trahan et al., 2014; Tan et al., 2014).

*Left ventricular non-compaction* is a rare cause of SCD in athletes < 1%. The myocardium appears trabeculated, with a risk of thrombus and embolism formation. ECG changes that may occur are: repolarization, long QT, ST segment depression, negative T, LVH, LBBB or RBBB (Drezner & Ashley, 2013; Gati et al., 2013).

Another cause of SCD in athletes is *commotio cordis* (20% in USA), more common in children and adolescents, due to increased thoracic compliance, particularly in baseball, lacrosse, hockey, softball players. It is a precordial contusion, which occurs 10-30 ms before the T wave peak (in the vulnerable repolarization period), with the induction of ventricular fibrillation. This syndrome is described with an increasing frequency in the literature, and 60% of the affected subjects successfully respond to resuscitation procedures (Chandra et al., 2013; Leikin et al., 2013; Demorest, 2013; Link, 2014).

## Electrical anomalies associated with SCD

*Long QT syndrome* in athletes has the following diagnostic criteria: QTc >470 ms in men and >480 ms in women; it is a hereditary disease, with a prevalence of 1:2000-5000. Thirteen mutations affecting potassium channels have been described. LQT1 syndrome is most frequently associated with exercise-induced SCD, particularly in swimmers and divers, due to adrenergic discharge induced by immersion in cold water (2-4%). It predisposes to the development of monomorphic or polymorphic ventricular tachycardia (1,20, 21, 44). *Short QT syndrome* (<340 ms) is less common; it also favors the development of ventricular arrhythmia, due to accelerated repolarization (Chandra et al., 2013; Drezner JA et al., 2013).

*Brugada syndrome* is another hereditary channelopathy, through the impairment of natrium channels. ECG shows an atypical ST segment elevation in the right precordial leads (pseudo-RBBB), accompanied by negative or biphasic T waves. It predisposes to ventricular arrhythmia particularly during rest and sleep, but it can also follow exercise, due to exercise-induced hyperthermia; it can be identified using class I anti-arrhythmic drugs (Chandra et al., 2013; Williams E et al, 2012; Drezner JA et al., 2013).

*Wolf-Parkinson-White syndrome* is a ventricular preexcitation syndrome with anterograde conduction,

through an accessory atrioventricular pathway. Associated ECG changes are: short PR (<120 ms), wide QRS (>120 ms) and delta wave. The association of this syndrome with atrial fibrillation has an increased risk of ventricular arrhythmia and SCD, through rapid conduction through the accessory pathway (1:1000 prevalence, occurring in 1% of athletes) (Chandra et al., 2013; Demorest R, 2013; Drezner JA et al., 2013).

*Catecholaminergic polymorphic ventricular tachycardia (CPVT)* is a hereditary disease, exercise-induced ventricular ectopia, with a 1:10,000 prevalence in the general population. Rest ECG is normal, but the exercise test may evidence multifocal ventricular extrasystoles, ventricular tachycardia or ventricular fibrillation (Chandra et al., 2013; Drezner & Ackerman & Cannon, 2013).

*Ventricular extrasystoles* are frequent in high performance athletes (physiological), but they can be the expression of hidden cardiac disorders and require cardiological evaluation. Ventricular extrasystoles with a LBBB appearance, more than two in 10 seconds (by Holter monitoring > 2000/24 hours), have an increased risk of inducing malignant arrhythmia. In endurance athletes, these have been associated with a higher risk of exercise-induced RV arrhythmogenic cardiopathy (Drezner & Ackerman & Cannon, 2013; Lampert, 2012).

High performance sport is frequently forbidden to athletes with severe cardiovascular disorders. Thus, athletes with *ischemic heart disease* with a negative prognosis, i.e. those with severe ventricular dysfunction, inducible ischemia or electrical instability, hemodynamically significant stenosis over 50%, can practice only low intensity sports activities and require self-training for stopping the effort when symptomatology occurs (Bonow et al., 2012; Thompson et al., 2005). Athletes with recently revascularized myocardial infarction can resume sports activity after 4 weeks (Thompson et al., 2005). Athletes with severe, symptomatic *valvular regurgitation/stenosis*, with important pulmonary hypertension, cannot participate in sports competitions. Athletes under *oral anticoagulant* treatment should avoid contact sports and sports with a high risk of hemorrhage (Bonow et al., 2012; Bonow & Cheitlin, 2005). The presence of *HCM, RVAD, myocarditis, DCM, coronary anomalies* predisposes to an increased risk of SCD, participation in sport competitions being forbidden (Bonow et al., 2012). Athletes with *Marfan syndrome* with an increased risk, i.e. aorta > 40 mm, moderate or severe mitral insufficiency, a positive family history for SCD, have an increased risk of aortic dissection, particularly during weight lifting, and should give up competitive sport (Bonow et al., 2012). *Congenital cardiac malformations* contraindicate high performance sport if uncorrected, cyanogenic, symptomatic, with severe pulmonary hypertension, accompanied by tachy- or bradyarrhythmia, myocardial dysfunction. If these malformations have been surgically corrected, sport can be resumed after 2-4 months, and if they have been solved interventionally, participation in competitions is possible after 3-6 weeks, in the absence of other contraindications (Graham et al., 2005). *Cardiac rhythm and conduction disorders* cause 1% of SCD in athletes and are frequently symptomatic. Chronic tachyarrhythmia may induce cardiac remodeling, and exercise-induced transient tachyarrhythmia can cause injuries to the athlete. Athletes with arrhythmias require re-evaluation every 6-12 months. After interventional treatment through ablation, athletes can resume sports activity in days. Athletes with preexcitation syndrome can resume sports activity 2-4 weeks after the ablation treatment of the accessory pathway (Zipes et al., 2005). Athletes diagnosed with *channelopathies* (long or short QT, Brugada syndrome, CPVT) have an increased risk of exercise-induced SCD, intense physical exercise being prohibited at least until the implantation of an intracardiac defibrillator (Zipes et al., 2005).

## Conclusions

1. Athlete's heart is a structural, morphological and electrical alteration, in response to sustained physical exercise. These changes are physiological and should be known by the treating doctor, in order to be adequately interpreted. Athlete's heart frequently poses problems of electrocardiographic and echocardiographic differential diagnosis with some channelopathies, cardiomyopathies or other cardiovascular disorders.

2. A clear delineation between physiological and pathological changes in athletes is mandatory in order to avoid the prohibition of sport in healthy athletes and also, to prohibit it in cases with a high risk of sudden death. This is why a sports cardiology subspecialty has lately been developed, and ECG, echocardiographic criteria have been established in order to differentiate between pathological and physiological changes.

3. In the majority of the cases, the presence of cardiovascular disorders contraindicates high performance sport, particularly of high intensity, at least until their pharmacological, interventional or surgical treatment.

**Conflicts of interest**

Nothing to declare.

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