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# Causes of sudden cardiac death in professional athletes

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# Causes of sudden cardiac death in professional athletes

## Abstract

### Introduction

Extreme physical exertion, as experienced by professional athletes, tends to be a significant risk factor for adverse cardiovascular events, including sudden cardiac death (SCD). Despite advances in sports medicine, the precise impact of training intensity, duration, and functional adaptations on the development of cardiovascular disorders remains not sufficient. Moreover, individual predispositions play a crucial role in the manifestation of these conditions.

### Purpose

This review aims to analyze the relationship between intense sports training and the risk of SCD with a focus on how different sports influence cardiac vulnerability.

### State of Knowledge

To the most frequent causes of cardiac arrest in young athletes (≤ 34 years of age) belongs hypertrophic cardiomyopathy, and in the case of older sportsmen (> 35 years of age) it is ischemic heart disease. Pathological abnormalities of the cardiovascular system, inducing sudden cardiac death in professional competitors, are based on the mechanism of severe ventricular aberrations of cardiac rhythm, i.e. ventricular fibrillation and tachycardia.

### Conclusions

Sudden cardiac death in athletes is a complex issue influenced by multiple factors, including genetic predisposition, training intensity, and the type of sport performed. A better understanding of the mechanisms leading to SCD are essential for developing effective preventive strategies.

**Key words**: sudden cardiac death; athletes

## Background

Sudden cardiac death (SCD) is described by cardiologists as *unavoidable rarity* and draws specific attention due to its incidence in a group of young highly physically active people who are commonly considered an example of health. It is defined as sudden death with a possible cardiac cause, which occurred within one hour from the onset of its symptoms in the cases documented by its witnesses, or within 24 hours from the last contact with the person who suffered it, if nobody witnessed to that incident [1]. SCD is often the first and last manifestation of the cardiovascular system disease. Its other worrying indications may include: faints, weaknesses, weariness, decreased physical function, dyspnoea, palpitations, dizziness or the thoracic cage pain. It has been proved that SCD happens almost three to five times more often in male athletes [1, 4, 5, 6, 7] and presumably it is caused in them by more frequent incidence of myocardial fibrosis, being a base for life threatening arrhythmias, greater prevalence of coronary atherosclerosis and its hormonal aspects (oestrogens play a protective role for the cardiovascular system in females) [1]. It has also been discovered that the probability of SCD incidence is three times higher in black athletes, as compared to the white race [1, 6, 7]. SCD is a predominantly registered medical cause of death in sportsmen, occurring with the frequency from 1 to 40,000 to 1 to 80,000 athletes annually [1, 3]. Astonishingly enough, those potentially fatal cardiac incidents usually befall when sportsmen perform exercises, particularly when we recall the general truth saying that active lifestyle decreases a long-term risk of cardiovascular diseases. Paradoxically, intensive training enhances the risk of sudden cardiac attack (SCA). The above-mentioned illustrates how physical exercises may ensure long-term protection against adverse cardiovascular accidents and also predispose a vulnerable individual to the incidence of sudden cardiac failure [1, 8]. On the one hand, physical activity is expected to improve cardiac electrical stability and at the same time protect the heart against ventricular arrhythmias and SCA, but on the other hand, strenuous exercises may activate the sympathetic nervous system and cause severe ventricular rhythm disorders in a person susceptible to such incidents [1]. SCD generally concerns athletes playing football (usually in Europe), basketball (most often in the USA), American football, rugby and those training swimming or long distance running [1, 5, 8, 9]. In persons ≤ 34 years of age, SCD is activated in the mechanism of ventricular disorders of cardiac rhythm (atrial fibrillation and ventricular tachycardia) which may often be generated by asymptomatic

pathologies of the cardiovascular system, i.e. hypertrophic cardiomyopathy (the most frequent cause of SCD in young athletes in the USA, accounting for 36% of cases), congenital anomalies of coronary arteries, commotio cordis, post heart inflammation complications, arrhythmogenic right ventricular cardiomyopathy (the most usual cause of SCD in young athletes in Italy, accounting for 23% of cases), thoracic aortic aneurism in Marfan Syndrome, mitral valve prolapse, dilated cardiomyopathy, channelopathies (Long QT Syndrome, Brugada Syndrome), Wolf-Parkinson-White (WPW) Syndrome and injuries to the region of the heart and aorta. Whereas in the group of persons > 35 years of age, SCD is provoked generally by ischemic heart disease (approx. 73% of demises), originated by coronary atherosclerosis [1, 2, 3, 4, 6, 8, 10, 11]. Factors participating in the mechanism of inducing SCD, apart from asymptomatic cardiovascular disease, may include a considerable physical effort, excessive secretion of catecholamines connected with a stressful situation, generated by e.g. competitions, dehydration, electrolyte or acid and alkaline disorders [1, 10].

**Material and methods**

The survey is based on the content of 33 articles written between 2010 - 2023. The selection of References was conditioned by its compliance with the Evidence Based Medicine and reliability of the medical data included in them. The entries presented in References come from the Pubmed data base.

**Results**

Hypertrophic cardiomyopathy (HCM) is a genetically conditioned autosomal dominant disorder induced by mutations within the gene encoding the albumen of the cardiac sarcomere. It occurs with the frequency from 1:200 to 1:500 in the general population of adults, whereas in young people, especially in professional athletes, it is responsible for the most frequent cause of SCD. The left ventricular hypertrophy may be mild (>15 mm) or severe (>30 mm), and its histopathologic changes include enlargement and disorders of the system, as well as cardiomyocytes with fibrotic tissues. The above-mentioned result in impairing diastolic heart function, obstruction of the left ventricular outflow tract and disorders to cardiac rhythms. Its many sufferers are asymptomatic or produce slight symptoms which may include: dyspnoea provoked by physical exertion, fatigue, palpitations, fainting, atypical pain in the thoracic cage, dizziness and even sudden cardiac death. Considerable attention should be paid to positive family medical history on SCD. Professional athletes with diagnosed hypertrophic cardiomyopathy are recommended to resign of practising sport since a strenuous physical activity in connection with the above cardiac condition considerably enhances the risk of

ventricular arrhythmias leading to sudden cardiac failure. It especially concerns those sports requiring isometric weight workout or accelerated running which result in a sudden increase in cardiac action or a greater left ventricle outflow of the blood. However, moderate aerobic exercises are recommended since they improve human circulatory and respiratory condition and belong to a part of healthy lifestyle habits [12, 13, 14, 15].

Arrhythmogenic right ventricle cardiomyopathy (ARVC) is a genetically conditioned autosomal dominant disease mainly caused by mutations in desmosomal genes, resulting in development of improper structure of albumins building cell-cell connectivity called desmosomes. Their mechanical injury (e.g. during strenuous physical exercises) enhances the activity of calcium channels, overloads cells with calcium ions and leads to the onset of apoptosis inducing repair mechanisms, i.e. fibrillation and adiposity, whose tissues replace cardiomyocytes, particularly in the wall of the right ventricle (however, according to the latest research findings, more frequently that condition is identified with both cardiac ventricles). Disorders originate in the epicardium and progress towards the inside, sparing the interventricular septum. This condition favours development of inclinations to ventricular arrhythmias since the number of metabolic and electrical connections between cardiomyocytes decreases and in turn disorders concerning conduction of electrical signals are generated. The advancement of the above condition leads to the right ventricular enlargement, thinning of its wall, disorders in its contractibility (regions of dyskinesis and akinesis) and to enlarging the right ventricle outflow tract of the blood. This disease occurs with the frequency from 1:2,000 to 1:5,000 and it is more common in men (1.6 – 3.2 times greater risk in males, as compared to females). The following belong to its first symptoms: a syncopal episode, fainting, idiopathic consolidated ventricular tachycardia (VT) or death caused by ventricular tachycardia. Especially young people between their second and fourth decade of life are afflicted by sudden deaths, and in Italy that disease is the most usual cause of SCD in sportsmen. Undertaking physical activity by individuals with arrhythmogenic right ventricular cardiomyopathy is absolutely not recommended since emotional stress and physical effort may become factors provoking ventricular tachycardia, which is one of the mechanisms of SCD, and may also accelerate the progress and development of the disease [16, 17, 18].

Anomalous origin of the left coronary artery from the opposite sinus, anomalous origin of the left coronary artery from the pulmonary artery and congenital arteriovenous fistulas constitute three main types of congenital coronary artery anomalies. In the first case mentioned, which is at the same time the most frequent type of this anomaly, the artery running between the aorta

and the pulmonary artery is bent, hypoplastic or may be pressed by other greater arteries. During physical exercises, cardiac musculature requires a greater inflow of oxygenated blood indispensable for aerobic respiration of the cells to acquire energy. It is very frequently not possible to supply an adequate amount of oxygenated blood to the intensely working heart because of abnormally developed coronary arteries, which results in its temporary ischaemia manifested by angina pectoris, ventricular arrhythmia or exercise-induced SCD. The second type of anomalous coronary arteries is manifested by segmental impairment of the left ventricular contractibility, disfunction of the papillary muscle and mitral regurgitation. In the third case, arteries are joined with cardiac ventricles and/or large arteries which produces congestive heart failure (volume overload), cardiac ischaemia (coronary steal syndrome) or atrial fibrillation (when arteries extend to atria) [19].

Dilated cardiomyopathy (DCM) is a condition where the heart becomes abnormally enlarged (its ventricles become larger and its walls thinner), its systolic activity is impaired and the left ventricular ejection fraction - due to structural changes in myocardium, i.e. increased fibrillation, necrosis and scarring, as well as changes in junctions between sarcomeres - affects the development of zones of decreased conduction. The condition produces no changes in arterial hypertension, gives no symptoms of coronary artery disease or mitral insufficiency and is caused by non-genetic factors (e.g. myocarditis, alcohol and drug abuse or self-violence) or genetic factors (e.g. mutations of genes encoding proteins of the cytoskeleton, sarcomere or the nuclear envelope). Symptoms of DCM occur in its onset (dyspnoea, orthopnoea, decreased functional efficiency or swelling of ankles) and in its advanced stage palpitations, diastolic heart failure, cardiogenic shock or blood clot and thrombosis complications are typical. SCD is usually caused by bradyarrhythmia or ventricular tachycardia. In the general population, DCM occurs with the frequency of 1:2,500, and five to seven cases are recorded per 100,000 persons within a year. It is more typical of males (1:1.3) than females (1:1.5) and commonly develops between 20 and 60 year of age. This condition also concerns children (DCM is responsible for 60% of paediatric cardiomyopathies). Distinguishing mild, subclinical DCM from a healthy athlete's heart (physiologically enlarged left cardiac ventricle) in the case of athletes undergoing intensive endurance training presents a challenge since both those conditions have a similar cardiac phenotype. Professional athletes with diagnosed DCM should be excluded from training most competitive sports and qualifications for practising a sport should be based on recognizing the athletes' genotypes [20,21,22].

Long QT Syndrome (LQTS) is a channelopathy generated by a mutation of the ion channel genes, usually of potassium or sodium cardiomyocytes, whose abnormal functioning affects differentiation of duration time of functional potentials in different layers of myocardium, which in turn may induce disorders of cardiac rhythm in the re-entry or early secondary potentials, i.e. arrhythmias linked with the elongation of repolarization cardiomyocytes. This may produce polymorphic ventricular tachycardia of the torsade de pointes type and sudden cardiac arrest. In LQTS, the ECG record shows characteristic long QT interval (≥ 500 ms) and alterations in the morphology of the T wave. Recurrent incidents of fainting induced by physical effort (especially swimming), sudden audio stimuli (e.g. morning alarm-clock set off) or emotional stress belong to the manifestations of this disease. Other symptoms include, e.g. tremors, atrial arrhythmias and even SCD, and can be activated by such factors as: physical effort, strong emotions or noise. LQTS occurs at young age (5-15 years of age), and it is extremely significant to have a positive medical history record concerning sudden cardiac deaths in a family. LQTS occurs with the frequency of 1:200, it is usually diagnosed in females and constitutes an absolute contraindication to perform physical activities [23, 24].

Wolff-Parkinson-White Syndrome is another congenital cardiologic condition which may lead to SCD in the mechanism of tachyarrhythmia (tachycardia with narrow or broad QRS symptoms, atrial fibrillation, flutter or arrythmia). It consists in developing accessory pathway of conducting an impulse to cardiac ventricle and stimulating it outside the cardiac conduction system – most often it is the bundle of Kent joining atrium with ventricle, the so-called atrio-ventricular furrow, situated usually on the left side of the heart. WPW occurs in the general population with the frequency of 1-3/1,000 persons (0.1 to 0.3%). Those suffering from it usually do not produce any changes in bodily function but can experience palpitations, dizziness, light-headedness and fainting, and the first manifestation can even be a sudden heart arrest. Sports cardiologists categorically advise against practising sports with moderate or high intensity in the case of persons with pre-excitation syndrome [25, 26].

Commotio cordis with subsequent atrial fibrillation and SCD occurs as a result of a nonpenetrating dull high energy blow in the pre-heart region of the thoracic cage. It can be a blow caused by a fast moving object (e.g. a rugby, baseball, softball, lacrosse ball or a hockey puck) or by a physical contact with another player (e.g. a high impact collision with another player). The incident is not accompanied by the rib or sternum fracture or heart injury and no structural or functional heart disease coexists with it. The following need to be fulfilled to cause commotio cordis: a blow on a precise spot in a thoracic cage, its overlapping

with a specific moment of the heart cycle, composite energy of the blow, density, size and arrangement of the object blowing (hard, small and ball-shaped objects are of greatest risk) and the shape of the thoracic cage (younger persons are more susceptible due to thinner, weaker ribs and worse developed muscles). In most cases, it leads to death and effective circulatory and respiratory resuscitation or defibrillation concerns only 35% of cases. Commotio cordis occurs also in children, teenagers and young male adults who do sports as amateurs or competitively. The following sports are mainly exposed to the incidence of commotio cordis (mentioned in descending order in respect of frequency of commotio cordis occurrence in a given type of activity): baseball, softball, hockey, American football, football, lacrosse, boxing, cricket, rugby, karate and basketball. It seems groundless to forbid competitors to practise sports due to experienced commotio cordis; nevertheless, athletes should be instructed on how to protect themselves against injuries or should be recommended to wear protective vests on their thoracic cages during competitions (however, it turns out that they are not fully effective against SCD) [27, 28, 29].

Burgada Syndrome is a genetically conditioned, congenital autosomal dominant arrhythmogenic channelopathy caused by a mutation of the gene encoding the sodium channel of cardiomyocytes and leading to decreased expression in cardiac muscle. It reduces the time of functional potential and generates disorders of repolarisation and the re-entry phenomenon. This condition predisposes to ventricular tachycardia, may transform into ventricular fibrillation and cause sudden cardiac arrest. A total of 1/3 of Burgada Syndrome sufferers experienced faints and the remaining 2/3 did not produce any symptoms. The following can induce its indications: fever, a lavish meal and physical activity, and that is why Burgada Syndrome is a condition excluding from practising competitive sports [30].

Marfan Syndrome is a genetic autosomal dominant disorder with a mutation of the fibrilline-1 gene encoding protein, which is one of the main structural elements of the connective tissue fibres, and it occurs in approximately 1:3,000 – 5,000 individuals worldwide. Its clinical symptoms are heterogenous and concern mostly the musculoskeletal system, organs of vision, heart and blood vessels. A considerable height (often above 95th centile), long arms and legs, and arachnodactyly (long and slender fingers) are typical physiognomic features of those with Marfan Syndrome with a defective structure of elastin and excessive stretchability of the connective tissue. That phenotype makes such persons be willingly recruited to sports favouring tall players, e.g. basketball or volleyball. Apart from possessing advantageous body built, they also have increased probability of developing dissecting aortic aneurysm of the

ascending aorta due to the weakening of the medial layer of the aortic wall causing enlargement of the aortic bulbus, aortic regurgitation, dilations and aneurysms which have a tendency to rupture, producing severe internal haemorrhage during events affecting increased arterial blood pressure, e.g. emotionally stressful and physically demanding situations such as competitions or matches. Developmental age Marfan Syndrome competitors are often diagnosed too late because distinguishing its nature poses considerable problems due to diversities of clinical signs and symptoms, and their changeability at the developmental age [31].

Mitral valve prolapse (MVP) known as Barlow Syndrome is a valvar disease characterized by displacement of an abnormally thickened mitral valve leaflet into the left atrium during systole, which causes incomplete mitral valve prolapse of different degree. Mitral leaflets are thickened and elongated by myxomatous degeneration which consists in depositing mucopolysaccharides in the mitral connective tissue generating fragmentation of the collagen fibres and elongation of the leaflets and chordae tendineae. MVP concerns a total of 3% of the world population. This disorder may be asymptomatic or may produce symptoms resulting from mitral valve prolapse (decrease of physical function, dyspnoea, cardiac arrhythmia), displacement of mitral valve leaflet (untypical pains in the thoracic cage, palpitations, weaknesses, dyspnoeic attacks not related to physical effort, dizziness, faints) or increased adrenergic stress (overexcitability, anxiety attacks). It can be related to higher incidence of SCD, especially in persons with anatomically deformed valvar leaflet and syncopal episodes or faints [32, 33].

Coronary artery disease is responsible for the most frequent cause of SCD in professional athletes above 35 years of age. The atherosclerotic plaque built up in the coronary artery may tear off and its piece may clog the artery causing inadequate blood supply to a part of cardiac muscle and result in acute coronary syndrome (ACS). It happens when coronary arteries are tensed and contracted under the influence of factors related to increased physical activity, i.e. tachycardia, higher arterial blood pressure, greater contractibility of cardiac muscle and emotional stress during competitions. Apart from the above mentioned, practising sport is accompanied by dehydration, making blood thicker, and release of catecholamines in relation to emotionally stressful situations which induces catecholamine-dependent activation of platelets and increases thrombogenic blood potential. These factors also favour the incidence of ACS. Coronary artery obstruction responsible for inadequate blood supply to the heart

results in electric instability, producing danger of incidence of ventricular fibrillation which may be the cause of SCD [1].

**Discussion**

The research findings suggest that excessive physical activity may negatively affect the general health of an individual, among others in the context of the cardiovascular system. However, specialist literature cannot precisely tell within what time span the athlete's heart gets deformed and how long should the time without physical training be indispensable for regression of those disorders. According to accessible sources, one may conclude that actually both those issues to a considerable degree depend on the athlete's age, intensity of their training or their individual genetic predispositions. Moreover, the results of the research mentioned may also be influenced by general lifestyle of the athlete and their possible use of endurance enhancing substances, which was not taken into account in most of the research works to date. Omitting those significant issues could to a considerable degree affect the credibility of the research works mentioned in the manuscript and contribute to drawing adverse conclusions from the publications oscillating within identical subject matters.

It is, no doubt, alarming that despite extensive knowledge on cardio-vascular diseases, cases of sudden cardiac deaths in professional athletes still occur. Taking into account the present knowledge on the subject, it is significant to determine optimum time of physical activity enabling proper planning of the physical effort reducing the risk of possible deterioration in the athletes' state of health. It is also of great importance to improve social awareness of the cardiovascular diseases occurrence which contribute to the incidence of sudden cardiac death.

**Summary**

- To the most frequent acute cardiac arrhythmias (ventricular tachycardia and ventricular fibrillation), which may lead to SCD in young athletes (below 35 years of age), belong hypertrophic cardiomyopathy and channelopathies, whereas in older athletes (above 35 years of age) it is coronary atherosclerosis.
- SCD more often occurs in male athletes and in the black race.
- SCD most frequently concerns athletes playing football, basketball, American football, rugby or soccer, those training swimming or long distance running.

**Disclosure**

**Authors contribution:**

Conceptualisation: ZK, TA

Methodology: DJ, TA, AJ

Formal analysis: ZK, DJ, AJ

Investigation: ZK, DJ, RMu, AJ

Writing - Rough Preparation: ZK, DJ, RMa

Writing - Review and Editing: RMa, RMu
Visualisation: RMa, RMu, AJ
All authors have reviewed and approved the final version of the manuscript for publication.

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