Sudden death during sports activities is a relatively rare, yet tragic event, with significant economic and social consequences. According to the existing studies, the annual incidence ranges from 3/100,000 to 1/250,000. The victims are mainly males with ratios varying from 18/1 to 9/1. The colored athletes are proportionally more susceptible and have a particular sensitivity to hypertrophic cardiomyopathy. Athletes have a 2.8/1 higher risk to have sudden death than non-athletes. But this is usually due to the undetected underlying cardiovascular disease. The main cause of sudden cardiac death in individuals older than 35 years by far is the coronary heart disease. With regard to people less than 35 years old, opinions differ, with studies on the U.S. having the first cause the hypertrophic cardiomyopathy, while studies in Italy, the arrhythmogenic right ventricular heart disease. Other common causes of sudden death during exercise are congenital anomalies of the coronary arteries, Commotio Cordis, myocarditis, diastolic cardiomyopathy, prolapse of the mitral valve, aortic stenosis, aortic rupture, etc. Regarding the non-cardiac sudden deaths, blunt trauma is the most frequent.

**Key Words:** Sudden death, Physical activities, Coronary artery disease, Heart
cause, and the parts of the body that are more frequently battered are the head and neck. Other, less common causes are stroke, the use of illicit drugs and pulmonary diseases. The sports in which most frequently occur sudden deaths are football, basketball, running, cycling and swimming. The death rate in each sport is due to the combination of the cardiovascular requirement and the total number of people involved with it. Screening of athletes for their participation in the athletic procedure saves lives and everyone agrees that it is necessary. But there is a serious disagreement between the U.S. and Europe, whether the screening procedure should include the electrocardiograms. The main reason cited by the U.S. for the non-use of the ECG is the high cost it requires. Chronic exercise induces the remodeling of the heart in order to respond to the cardiovascular demands. This phenomenon is called “the athlete’s heart”. This depends on the intensity and type of the exercise. In endurance athletes, it causes mainly the increasing of the internal diameter of the left ventricle and in power athletes, the increasing of the wall thickness of the left ventricle. There is often confusion between the «athlete’s» and the abnormal heart since in mild forms of the disease, some features such as the thickness of the wall of the left ventricle, can have similar sizes. The mechanisms leading to these sudden deaths are either of mechanical nature or electrical with the latter constituting for over 90% of the cases.

**GENERAL INFORMATION**

*Definition*

Sudden death in sports is defined as one that occurs during or within one hour of stopping this sports activity (65).

*Frequency of sudden death in sports*

The annual incidence of sudden death during exercise by healthy young people is one case per 200,000-250,000. In competitive sports, sudden cardiac death is very rare (47, 48). This frequency in young athletes aged 12-35 years amounts to 0.5-2/100,000 per year (11) or 1/50,000 in young athletes generally (43). In a study conducted in Italy concerning sudden death, a frequency of 3 cases per 100,000 was found in young competitive athletes that were under 35 years old and who trained regularly and participated in official sporting events (22). Concerning the general population, in a study conducted in Germany for people aged 10-79 years old, the incidence of Sports Related Sudden Cardiac Deaths (SrSCD) was found to be 1.2–1.5/million/year (12) and in France for people aged 10-75 years old concerning Sports Related Sudden Death, 4.6/million/year (65).
Gender

A study in the USA found that sudden cardiac death during exercise is uncommon in young athletes, regardless of race and the ratio of women to men is about 1/9 (69). In Denmark, in a non-athletic population, the ratio was 1/13.67 (86). In France, 1/18 (66), while in Finland, 1/16.2 (102).

Race

The increasing participation, but also the great performance of black athletes in recent years has made the researchers to deal increasingly with this group. In a study in the USA, the rate of sudden death in young athletes due to cardiovascular diseases was 64% among blacks, compared to 51% for whites (67). Of the overall incidences of sudden deaths on the playing field due to hypertrophic cardiomyopathy, in school and college athletes, more than 50% regards blacks (69). Finally, the black athletes exhibit noticeable repolarization changes in the electrocardiogram and hypertrophy of the wall of the left ventricle in the echocardiography. These features can cause confusion about whether this refers to the “athlete’s heart” or mild hypertrophic cardiomyopathy, which may result in a misdiagnosis and a possible disqualification from competitive sports (17). The repolarization changes refer to the rise of the ST interval and deep reversals of the T wave (76).

Athletes over 35 years old

Specifically for this category, the frequency for sudden cardiac death to occur ranges from 1/15,000 to 1/50,000. The leading cause of S.C.D. is coronary heart disease; the gender most commonly affected is male. Most of the cases had presented previous symptoms and were involved in individual competitions. On the contrary, concerning the athletes under 35 years old, only 30% had reported symptoms and were involved in team sports (80). There is a reduction of 40-60% of the mortality due to cardiovascular or general causes, with moderate intense training three or more times a week and the benefits of regular exercise outweigh the risk SCD in the elderly, especially those who train properly (19).

Athletes – non athletes

Regular participation in training and competitive sporting activities is associated with an increased risk of SCD, with the average relative risk for athletes to be 2.8 times higher compared to non-athletes. However, it is worth noting that sports are not by their own the cause for the higher incidence of SCD. It is the combination of intense physical activity in athletes with underlying cardiovascular disease, which can cause arrhythmias that lead to cardiac arrest. The relative risk of participation
in sports is different depending on the underlying disease and is greater in the case of cardiomyopathy (such as hypertrophic cardiomyopathy or arrhythmogenic right ventricular cardiomyopathy) or congenital abnormalities of the coronary arteries (62). Regarding the size of the “negative dose of sport”, this is unknown, but an everyday expenditure of 490 kcal in physical activity in the aerobic zone and adequate pre participation cardiovascular screening are recommended to avoid SCD and other negative effects of athletic activity (60).

Relative time of sports related sudden death

In a study conducted in France from 2005 to 2010, it was found that 92% of the Sports – Related Sudden Deaths occurred during the sports activity, 7.4% within 30 min of the cessation of the sports activity and rarely between 30-60 min of the cessation (65).

STUDIES

In the Italian region of Veneto over a span of 1979-1999, the main causes of sudden death in athletes between the ages of 12-35 years old, were: Arrhythmogenic right ventricular cardiomyopathy (ARCV) (23.5%), Atherosclerotic coronary artery disease (19.6%), Anomalous origin of CAD (13.7%), Mitral valve prolapse (MVP) (11.1%), Myocarditis (9.8%), diseases of the conduction system (7.8%) etc (21).

In a study in the USA during the years 1980-2006 and concerning 1866 sudden death events in young competitive athletes, the leading causes of death were found to be: hypertrophic cardiomyopathy (251 cases), followed by Coronary artery anomalies of wrong sinus origin (119), Myocarditis (41), Arrhythmogenic right ventricular cardiomyopathy, etc (67).

In 2006, Bille et al. essentially made a summary of the available articles up until 2006. They selected studies related to SCD in athletes of up to 35 years old, were related to sports and in the absence of proven abuse of drugs. This resulted in 47 articles and 1101 cases of death, which were analyzed. The results were: Congenital heart diseases (coronary artery anomalies, 262, 23.8%; mitral valve prolapse: 27, 2.5%; aortic valve stenosis: 20, 1.8%, unspecificed, 3, 0.3%). Cardiomyopathies (hypertrophic cardiomyopathy, 261, 23.7%; dilated cardiomyopathy, 24, 2.2%; various, 37, 3.4%). Arrhythmia (arrhythmogenic right ventricular cardiomyopathy/ dysplasia, 97, 8.8%; long QT syndrome, 5, 0.5%; conduction system pathology, 20, 1.8%; wolff-parkinson-white syndrome, 1, 0.1%; various, 3, 0.3%). Atherosclerotic (atherosclerotic artery disease, 112, 10.2%). Trauma (commotio cordis 103, 9.4%). Infectious (myocarditis 78, 7.1%). Degenerative (cardiac sarcoidosis, 4, 0.4%).
Marfan syndrome (4, 0.4%). Ruptured Aorta (aneurism, 23, 2.1%). Undetermined (10, 0.9%). Acquired (kawasaki 3, 0.3%). “Normal Heart, 3, 0.3% (11).

In a study conducted in France from 2005 to 2010, it was found that in the general population (excluding young competitive athletes), 78% of the instances of sudden death during sports activities were unexplained. 19% were due to Coronary heart disease, followed by HCM, possible HCM, ARVD, Dilated cardiomyopathy, Myocarditis, etc (65).

In a study carried out in Spain during the years 1995-2010, and concerning the sudden death during sports of 168 people, 81 up to 35 years old and 87 over 35 years old, it was found that 50.5% of the cases concerned Coronary atherosclerotic disease, 7.7% arrhythmogenic cardiomyopathy, 7.1% hypertrophic cardiomyopathy, etc (98).

In Germany during the period from May 2012 to October 2014, involving 144 cases of sudden cardiac death associated with sports, for individuals under 35 years old, the main cause was Myocarditis and for those over 35, Coronary artery disease (12). CAD was the main cause of SrSCD in a study conducted in Switzerland concerning individuals of 10-39 years old (39).

Regarding the non-cardiac sudden deaths, blunt trauma (22%) was found to be the most frequent cause of sudden death. The head and neck were most frequently struck. Other causes were stroke (2.5%), the use of illegal drugs (1.8%) and pulmonary diseases (1.5%) (67). Other non-cardiac causes were found to be hyperthermia, sickle cell disease, being hit by lightning and bleeding (104). Few sudden deaths can be attributed to cerebral aneurysm (21), drowning (103).

CAUSES

Hypertrophic cardiomyopathy (HCM)

Hypertrophic Cardiomyopathy (HCM) is characterized by the hypertrophy of the left or the right ventricle of the heart, which is often asymmetrical (85). This hypertrophy may range from the mild 13-15mm thickness and may reach 50mm. It is a commonly inherited disease affecting approximately 2% of the population. It is the most common cause of sudden death for people under 30 years and is not influenced by gender (71). However, it has been observed that people that have been diagnosed with this disease to eventually reach and surpass the age of 90 years (68). Symptoms may include shortness of breath, angina, dizziness, muscular weakness, fainting, sudden death (31). The diagnosis of the disease can be made by an echocardiography. However, if the results are not clear, then the cardiac MRI may be used (37). Treatments vary depending on the circumstance.
These may be medication (such as beta blockers, Verapamil, Disopyramide, Amiodarone and a combination of other drugs), surgical myectomy, alcohol septal ablation, pacemaker, transplant (this is the last option and involves people that the disease has reached its final stage) (20).

**Coronary artery disease (CAD)**

The progression of coronary artery disease is caused either by the development of atherosclerotic plaque, or the concentration of thrombotic material in a coronary lesion (106). It is the most important cause of death for people aged over 40 years in the developed Western countries and the most common cause of myocardial infarction (112). The main symptom is chest pain (angina). Also, patients may complain of shortness of breath or fatigue. Still, symptoms can occur in other parts of the body such as the shoulder, hand, chin and elsewhere. The degree of pain does not reflect the extent of coronary artery disease. Mild chest pains do not mean necessarily a mild disease (1). Due to the significance of the disease, there is an ongoing research to improve diagnostic procedures. The usual diagnostic procedure of coronary artery disease consists of the following four steps: 1. Evaluation of the signs and symptoms of the disease and an Electrocardiography (ECG) while resting. 2. ECG during controlled exercise. 3. Scintigraphy of the myocardium. 4. Coronary angiography (55). The treatments are: 1) Pharmaceuticals: Aspirin, Cholesterol regulators, Beta Blockers, Nitroglycerin, ACE and ARB, Calcium channel blockers. 2) Procedures that restore and improve blood flow such as Angioplasty and stent placement, Bypass. 3) Change of lifestyle: quitting smoking, healthy diet, exercise, weight loss, reduction of stress (48).

**Dilated cardiomyopathy (DCM)**

Dilated Cardiomyopathy is the presence of a dilatation of the left ventricle and left ventricular systolic dysfunction without abnormal loading conditions (hypertension, valve disease), or coronary artery disease, sufficient to cause global systolic impairment. Dilation and dysfunction of the right ventricle may be present, but are not necessary for the diagnosis (28). During the disease, the mass of myocardial muscle increases and the wall of the ventricle becomes thinner. The myocardium becomes thin and weak and cannot pump blood efficiently. The myocardium expands so that it can hold more blood, but in time becomes even weaker and will lead to heart failure symptoms (24). It is the most common form of cardiomyopathy and has many causes. In the adult population, its prevalence is 1 case in 2500. The causes are: 1. Hereditary, genetic. 2. Infection by viruses, bacteria, fungi, parasites, etc. 3. Toxic, from the use of drugs, medication, alcohol (50). The therapy may be either pharmaceutical or involve the implanting of a cardiac defibrillator (29).

**Idiopathic concentric left ventricular hypertrophy**
This condition shows some important differences compared with hypertrophic cardiomyopathy and for that reason, differs from this. 1. The hypertrophy of the left ventricle is symmetric (concentric). 2. There is no disorganization of the cardiac cells in the left ventricular myocardium. 3. There is no evidence that it is hereditary in first degree relatives (70).

**Congenital anomalies of the coronary arteries**

This condition affects around 1% of the general population (with studies ranging from 0.3% to 5.6%). Most are discovered randomly, while others are after lethal or sublethal events such as arrhythmias, myocardial infarction, sudden death, during adolescence or adulthood. They constitute about 5-10% of the cases of people suffering from symptomatic congenital heart disease (63). It affects both men and women and has not been found to be a hereditary disease (64). The main anomalies regard: 1. The anomalous origin of the left coronary artery from the right atrium of Valsalva, which has been implicated in sudden cardiac deaths in young adults, especially during and immediately after intense exercise. 2. Less frequently occurring is the abnormal origin of the right coronary artery from the left atrium of Valsalva, which is also associated with sudden death and ischemia. 3. Rarely occurring, is the left coronary artery having started from the right atrium of Valsalva, enters directly into the myocardium. 4. Finally, an even more rare case is in which the origin of the coronary arteries are not even from the aorta (109).

The symptoms are not always the same and depend on the kind of anomalies and the age of patients. They include atypical angina, shortness of breath, cardiomyopathy, atrial fibrillation, syncope, acute myocardial infarction (72). The evaluation of coronary artery anomalies is done by conventional angiography, cardiac CT, and cardiac MRI, with the second method being superior to the first and the third growing its role (94). The detection methods include ECG, radiography, ultrasound, angiography, the stress test and echocardiogram. Methods of treatment are angioplasty, surgery, and medication (2).

**Aortic rupture**

Young athletes may die suddenly because of rupture of the aorta. Such individuals may have a decreased number of elastic fibers in the aortic media, an abnormality that presumably causes the intrinsic weakening of the wall. This is commonly known as cystic medial necrosis. People with this condition may successfully participate in strenuous competitive sports for many years without experiencing a disastrous event (70).
Myocarditis

Myocarditis involves the inflammation of the myocardial tissue. It may be responsible for up to 12% of the sudden deaths of young adults and is a major underlying cause of other diseases such as myocardial dilated cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy. Most cases involve infectious disease in previously healthy patients typically either because of a direct viral infection or post-viral immune-mediated reaction. It can, however, be caused by reversible and/or irreversible toxic, ischemic, mechanical trauma, inflammation due to drugs, transplant rejection, or other immune reactions (35). Symptoms that can occur are chest pains, dyspnea, paroxysmal nocturnal dyspnoea and orthopnoea, fatigue, exercise intolerance, palpitations, presyncope or syncope, heart failure (88).

The diagnosis comprises of the following: 1. Endomyocardial biopsy. It is considered the most accurate method of disease diagnosis. 2. MRI. Its use has significantly increased over the last decade. 3. Echocardiogram 4. Blood tests. 5. Computed cardiac Tomography – Angiography (40).

Mitral valve prolapse (MVP)

Mitral valve prolapse (MVP) is defined as anomalous swelling of the leaflets of the mitral valve in the left atrium during ventricular contraction (41). The prevalence of MVP is estimated at 2-3% of the population (44) and although it is genetically determined, clinical manifestations do not appear before adulthood (51). It is the most prevalent abnormality of the heart valves (49). Contrary to what was believed, it is now generally accepted that the disease occurs at the same rate in both genders. Symptoms range from mild to severe. The most common are heart palpitations, chest discomfort, and dyspnea. Other symptoms include paroxysmal supraventricular tachycardia, nocturnal dyspnea, and fatigue. The diagnosis is based on clinical picture, physical examination, and ultrasound (95). MVP can be diagnosed with a coronary CT angiography with high accuracy (90%–95%) compared with the accuracy of the diagnosis using an echocardiography (30). The management of the disease regards either medication or surgery (93).

Arrhythmogenic right ventricular cardiomyopathy (ARVC)

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a primary disease of the cardiac muscle, characterized by a progressive myocardial atrophy of the right ventricle, with transmural fatty or fibrofatty replacement, either segmental or diffuse, accounting for electrical instability which results in the risk of life-threatening ventricular arrhythmias (99). The disease occurs more in males than in women at a ratio of 1.6/1 (14). Symptoms may include arrhythmias, blackouts,
atypical chest pain, cardiac arrest (18) or even dizziness (36). The diagnosis can be accomplished by angiography of the right ventricle, endomyocardial biopsy, echocardiogram, computed tomography (disadvantage, the emitted radiation), MRI (78). The treatments include implanting a defibrillator, catheter ablation, pharmacological treatment, limiting exercise, and heart transplant (it is a solution that is not used regularly) (13).

Aortic stenosis

It is the most frequent valvular disease in the elderly with about 2-3% of people over 65 years being affected. The most frequent cause of acquiring the disease is calcareous degeneration, characterized by a progressive asymptomatic period that may last decades. It causes the prevention of blood outflow from the left ventricle (34). The symptoms of the disease are angina, dyspnea, and syncope (6). A diagnosis can be accomplished by blood tests, ECG, echocardiogram, chest X-rays, cardiac catheterization, coronary angiography, exercise stress test (47).

Sarcoidosis

Sarcoidosis is a disease where epithelial granulomas form in various organs of the body such as the skin, lungs, lymph nodes, eyes, and heart (92). Patients with cardiac sarcoidosis present a wide variety of symptoms, ranging from asymptomatic electrocardiographic abnormality to sudden death. The initial symptoms depend on the underlying pathology and may include shortness of breath, irregular heartbeat, fainting, dizziness, chest pain, orthopnea or peripheral edema. The clinical manifestations of the disease are associated with the location and the extent of the epithelial granuloma, which seems to depend on several factors, including ethnicity, gender and the duration of the illness (7). The diagnosis of the disease can be done by endomyocardial biopsy, electrocardiogram, echocardiogram, MRI, positron emission tomography (PET), and cardiac catheter (91). The therapy may include pharmacological treatment, the use of a catheter and the destruction of the harmful tissue with radio waves (catheter ablation), placement of a cardiac defibrillator, and heart transplant (74).

Long QT syndrome

The Long QT syndrome, characterized by the prolongation of the QT interval on the electrocardiogram can lead to polymorphic tachycardia, which can cause sudden death (52). It can be obtained either hereditary or subsequently by the use of certain drugs such as quinidine sotalol and dofetilide but also from additional drugs that are not directly related to the cardiovascular (108). The cases of sudden death in young athletes due to long QT syndrome is about 2% (67) and occurs
at a frequency ranging from 1/2000 to 1/5000 in the general population (13). The diagnosis of the disease is based on clinical features, electrocardiographic findings and family history (81). Treatments that could be followed are pharmaceutical, surgery, cardiac pacing and the placement of a defibrillator (53).

**Brugada syndrome**

The Brugada syndrome is characterized by an elevation of the ST segment on the electrocardiogram and a high incidence of sudden deaths in patients with a structurally normal heart. The syndrome is estimated to be responsible for at least 4% of the overall sudden deaths and at least 20% of the sudden deaths in patients with structurally normal hearts. The disease prevalence is estimated at 5 per 10,000 inhabitants and besides accidents is the leading cause of death in men younger than 40 years (3). The arrhythmia usually occurs during sleep, rest and after large meals. The symptoms are fainting, polymorphic ventricular tachycardia, ventricular fibrillation, and sudden death. Diagnosis of the disease can be done with medications, and with an electrocardiogram considering the family history and the above-mentioned symptoms (10). As for how to tackle the disease, the implantable cardiac defibrillator should be used in patients with a history of cardiac arrests or aborted sudden death. The use of drugs is another solution and the effectiveness of catheter ablation has been observed (56).

**Wolff-parkinson-white syndrome (WPW)**

The Wolff-Parkinson-White syndrome (WPW) is estimated to appear at a rate ranging from about 0.1% to 3% in the general population. It is a form of ventricular preexcitation and includes an abnormal conduction pathway. The accessory pathway bypasses the AV node, creating a direct electrical connection between the atria and ventricles. The majority of patients with preexcitation syndromes remain asymptomatic throughout their lives. It is important to recognize this syndrome because the patients may be at risk for the development of various types of supraventricular tachyarrhythmias causing disabling symptoms and, in extreme cases, sudden cardiac death (87). Symptoms include syncope, presyncope, arrhythmias, supraventricular tachycardia, ventricular fibrillation and SCD (46). The diagnosis is made by an ECG and therapy usually involves the use of drugs or catheter ablation (89). For asymptomatic patients, it is suggested a more aggressive approach with invasive testing and therapies, such as therapeutic ablation (15).

**Catecholaminergic polymorphic ventricular tachycardia (CPVT)**

Catecholaminergic polymorphic ventricular tachycardia (CPVT) is a rare but highly malignant inherited arrhythmia disorder. It is characterized by ventricular
tachycardia (VT) which is polymorphic or bidirectional. Physical exercise or emotional stress trigger catecholamines which generate (CPVT), typically in the absence of structural heart disease. The symptoms usually begin in childhood and include palpitations, dizziness, convulsions and syncopal spell (111). The mortality rate in patients that will not seek treatment is 30% - 50%, up to forty years old. The diagnosis may be made by the exercise stress test, electrocardiogram (Holder), genetic testing (61) and an exercise ECG (111). The treatment may be pharmaceutical (b-blockers) for controlling arrhythmias. Nevertheless, 30% of patients continue to have arrhythmias resulting implanted cardiac defibrillator (61).

**Commotio cordis**

Commotio Cordis is a fatal cardiac arrhythmia, caused by a blunt hit to the chest during the phase of ventricular repolarization, leading to sudden cardiac death. The transfer of the kinetic energy from the hockey ball or baseball to the myocardium is considered to lead to the arrhythmia (59). The above factors have to do with the exact time of impact relative to the cardiac cycle, the exact impact position relative to the heart, the toughness and the speed of the object and the ineffectiveness of the chest protections (75).

**Marfan syndrome (MFS)**

Marfan syndrome (MFS) is a multisystem connective tissue disorder, which involves primarily the cardiovascular, ocular and skeletal systems. It is an autosomal heritable disease, mainly attributable to a defect in the FBN1 gene and affects about 1 in 5000 people. The vast majority of the patients with MFS present aortic dilatation which is the main cause of morbidity and mortality among them. Fewer cases regarding ectopia lentis, dural ectasia, and skeletal features may occur (33). Treatments used to prevent or delay an aortic aneurysm are pharmaceuticals such as b-blockers, and surgery when the maximum diameter of the aorta reaches 5 cm (45).

**Use of anabolics and drugs**

Athletes use anabolic-androgenic steroids to increase strength, fat-free body mass and in some cases, improving their physical appearance. The use of anabolic-androgenic steroids by adolescent boys in the Western countries range from 1% to 5%. Moreover, in southeastern Spain, a systematic toxicology study showed that 3.1% of sudden deaths were associated with cocaine and due to cardiovascular causes (9). Despite the widespread perception of the role of doping in SCA/SCD in sports, and a large number of circumstantial reports and indirect evidence, there is no clear scientific or categorical proof to support it or not. The
clarification of its influence on SCA/SCD may happen only after the registration of toxicological agents (97).

**SPORTS IN WHICH SUDDEN DEATHS OCCUR**

In a study summarizing the findings of many previous studies, they ranked 388 sudden deaths in young athletes regarding sports as follows: Football 132 (34%), Basketball 98 (25%), Running 64 (16%), Swimming 19 (5%) etc (11). On the contrary, in a study involving 770 cases of sudden cardiac deaths during exercise of the general population in France, the results showed: Cycling 251 (31%), Jogging, 175 (21%), Football 107 (13%), Hiking 41 (5%), Swimming 31 (4%), basketball 29 (3.5%) etc (65). In the USA, in 136 cases of sudden death of people aged 13-24 years old, the results were: American Football 67, Basketball 22, Athletics 12, Football 9, Baseball 7 etc (104).

Involving studies that have to do with sudden death during recreational sports activity: In the USA, in 81 cases of sudden death of people aged 16-91 years old, the results showed: Golf 19, Jogging 16, Swimming 9, Bowling 6, Tennis 5 etc (83). In Ireland, in 51 cases of sudden death of people aged 15-78 years old, the results showed: Golf 16, Gaelic football 11, Jogging 5, Tennis 5 etc (82). In France, in 31 cases of sudden death of people aged 7-60 years old, the results showed: Running 13, Cycling 4, Soccer 3 etc (32).

From the above results, one may conclude that the frequency of SCD in sports does not depend exclusively on whether it is demanding cardiovascular, but also has to do with how many people are involved in the sport.

**PREPARTICIPATION CARDIOVASCULAR SCREENING OF ATHLETES (PPS)**

Preparticipation cardiovascular screening (PPS) is the systematic practice of evaluating athletes before their participation in sports in order to identify or raise suspicion of abnormalities that could lead to sudden death (5). There is the consensus of most sports organizations that cardiovascular prescreening should be carried out, considering the fact that there is an increase of 250% of the risk of sudden death during athletic activities by vulnerable people. Also, both the American Heart Association (AHA) and the European Society of Cardiology (ESC), agree on cardiac screening and that the history and physical examination should be included, but the interpretation of the results or the lack of them, has caused
a dispute over whether an ECG should be included in the process. Using the AHA guidelines, the US continues to recommend testing only with the history and physical examination, without the ECG. The above argument has been the subject of many articles and discussions on what is the best method of cardiovascular prescreening (105). Regarding the addition of an echo along with the 12 Lead ECG and the history and physical examination, in PPS, it was found that the echo did not add a significant number of findings (25).

The Italian model

The application by the Italian Ministry of Health, of a cardiovascular prescreening program for participation in sports since 1982, resulted in a 90% reduction in the sudden deaths of athletes, aged 20-35 years old in the Veneto region during the period 1980 - 2004. This was mainly due to the ECG that could find the cardiomyopathies, but also due to the perception of the existence of these diseases by the sports doctors and cardiologists (23). A physical examination, personal and family history and a 12-lead ECG, represent the first step to evaluate if there is an underlying abnormality. If the results are positive, then begins a second level of exams like stress tests, MRI, CT scans, genetic analysis and a third level such as coronary angiography, endomyocardial biopsy, etc., with the aim of a sure diagnosis (100). The International Olympic Committee (IOC) proposes the use and of the ECG during medical evaluation prior to participation (67), while FIFA introduced the use of it before the finals of the World Cup of 2006 (101).

The American model

The recommendations made by the AHA in 2007 include a 12-point test. It consists of five items related to the personal history, three to the family history, and four points on physical examination (69). However, studies show that the physical examination and history have limited effectiveness in the detection of latent heart disease that predisposes to the sudden death of athletes (5). Despite strong evidence of its usefulness, the AHA does not support the routine use of the ECG. The main arguments include cost-effectiveness, the concerns about false positive results and psychological consequences for the athletes and their families. SCD in sports remains rare and an ECG can not detect all the factors associated with it (16). Also, as an obstacle can be seen the lack of the infrastructure that the doctors have in the USA to evaluate the ECG correctly (26). In a relevant calculation model for the cost of a twenty-year ECG monitoring program for young competitive athletes in the US, the cost would range between 51 and 69 billion US dollars and save 4813 lives. Consequently, the cost per life saved would range from 10.6 to 14.4 million dollars. The application of the Italian strategy screening using ECG in the USA would entail huge costs for each life saved (42).
On the contrary, according to others, screening by history and physical examination alone does not detect the majority of athletes at risk and gives a false complacency in those with a silent underlying heart disease. But adding an ECG enhances the capabilities of detecting the diseases and modern athlete-specific ECG interpretation standards provide low false positive rates and improved cost efficiency (4). Newer screening recommendations are needed from the AHA, that will give fewer false positives and will be based on evidence because the current ones lead to an excess number of athletes that screen positive (27).

Secondary prevention of sudden death in athletes

The most important factor that determines the survival from Sudden Cardiac Arrest is the time that passes until the defibrillation begins. Automated external defibrillators (AEDs) are a basic piece of the “chain of survival” and their right use increases the chances of survival from SCA. Public access defibrillation programs (PAD) train responders in CPR and the use of AED but despite the growing evidence of their usefulness, many obstacles remain for their implementation, mostly concerning the cost (84).

THE ATHLETE’S HEART

Cardiac remodelling induced by exercise enhances the ability of the cardiovascular system to meet the demands of exercising the skeletal muscles (110). In endurance sports, the volume loading of the heart predominates; the cardiac output can increase up to 8 times and sees only modest increases in systemic blood pressure. In power sports, blood pressure may rise dramatically (up to 480/350 mm Hg), while only sees a modest increase in cardiac output. In combined sports (cycling and rowing), there is a significant increase in both volume and pressure loading (57). Regarding cardiac remodeling, the ventricles of endurance athletes feature eccentric hypertrophy, power athletes feature concentric hypertrophy in the ventricles and endurance and strength athletes show a moderate increase in the end-diastolic diameter of the left ventricle (58). Eccentric hypertrophy is a geometric increase of wall thickness and ventricular dilation without causing changes in shape. The term concentric hypertrophy refers to the increasing the volume of the wall, without changing the inner diameter (96).

But generally from the literature concerning remodeling, different opinions can be found. One can find that the right ventricle shows expansion only in endurance athletes and no change in power athletes (110). Elsewhere, concerning marathon runners (endurance athletes), there is a significant expansion of the left ventricle,
without thickening of its walls (90) and according to another study, there was little or no difference in the diameter of the left ventricle (73). In endurance athletes, there was an increase of both the left atrium, and the right, while in power athletes, only the left (110). Finally, another study finds that it is almost impossible to clearly distinguish a heart trained for endurance, from a heart trained for power (8). The remodeling of the heart due to sports may show features that resemble those of a “sick” heart. These may be: a) the thickening of the walls of the left ventricle as in hypertrophic cardiomyopathy and hypertension, and b) the dilation of the left ventricle as in diastolic cardiomyopathy (109).

To distinguish an “athlete’s heart” from one having hypertrophic cardiomyopathy, besides physical examination, or ECG, a good choice is echocardiography. Other methods of determining it are MRI, heart catheterization and endomyocardial biopsy. More complicated cases may require spiroergometry or genetic testing. In the athlete’s heart, there is an eccentric hypertrophy of both ventricles and the thickness of the walls is no more than 15mm, with a moderate dilation of the left ventricle. In HCM, there is an asymmetrical hypertrophy of the left ventricular with a reduced LV diameter. A feature of the athlete’s heart is the reaction due to absence from training. Within a few weeks or a few months, the morphological changes show a marked reduction, which is not the case in patients with HCM (58). Also, the distinguishment between the athlete’s heart and the sick one can be found by the existence or not of certain proteins such as or IGF-1 (109) or NT-proBNP (38). However, echocardiography is the only cost-effective, reliable method of imaging which is widely available and capable of the simultaneous quantification of anatomical variables and physiological characteristics (77). Regarding the clinical effect of long-term, intense, uninterrupted endurance training, it is not linked to improper remodeling of the left ventricle, or dysfunction or adverse clinical events, appearances of symptoms or new diagnosis of cardiomyopathies. The above training is considered safe, even at the highest level of play (elite Olympic endurance sports athletes) (89).

On the other hand, Hippocrates recognized the health benefits of physical activity, but also believed that the intense athletic competition had harmful effects on the heart and other organs, and reduced disease resistance (54).

**MECHANISMS THAT CAUSE SUDDEN DEATH DURING EXERCISE**

From a pathophysiology view, cardiovascular sudden death may be either mechanical or electric (arrhythmic). In the first case, the function of the heart is weakened due to an acute blockage of the blood circulation or by cardiac tamponade. Also considered cardiovascular SD is shock due to massive
hemorrhage such as extrapericardial aortic rupture, gastrointestinal bleeding, or due to adrenal septic apoplexy. However, in over 90% of the cases, the mechanism is of arrhythmic (electrical) nature, with acute pump impairment caused by asystole or ventricular fibrillation (9).

CONCLUSIONS

- Sudden death during sports is a relatively rare, yet tragic event with serious economic and social consequences.
- The annual incidence ranges from 3/100,000 to 1/250,000. Victims are mainly men with a ratio ranging from 18/1 to 9/1. The black athletes are proportionately more prone. Athletes have a 2.8/1 greater risk of sudden death than non-athletes, but this is usually due to an unnoticeable underlying cardiovascular disease.
- The main cause of sudden death in people older than 35 years with a big difference from the rest, is coronary artery disease. Regarding people under the age of 35, opinions differ. The studies of the US have as first cause Hypertrophic cardiomyopathy, while studies in Italy, Arrhythmogenic right ventricular cardiomyopathy.
- Other common causes of cardiac sudden death during exercise are Congenital anomalies of the coronary arteries, Commtotic Cordis, Myocarditis, Dilated cardiomyopathy, Mitral valve prolapse, Aortic stenosis, Aortic rupture, etc.
- Regarding the non-cardiac sudden deaths, Blunt trauma is the most frequent cause concerning about 22% of the total sudden deaths. Other causes are stroke, the use of illegal drugs, pulmonary diseases (1.5%) etc.
- The sports in which most frequently occur sudden deaths are football, basketball, running, cycling and swimming etc. This has to do with how demanding the sport is, but also with how many total people are involved with it.
- Pre participation cardiovascular screening of athletes saves lives and all agree that it is necessary. But there is a serious disagreement between the US and Europe about whether an ECG should be included in the procedure. The main reason called upon by the US for the non-use of the ECG is the high cost.
- The "athlete's heart" refers to the remodeling caused to it to meet the demands of exercising for a long period of time. It depends on the intensity and type of exercise, whereas in endurance athletes it mainly involves the increase of the inner diameter of the left ventricle and in power athletes, an increase of the wall of the left ventricle.
- The mechanisms that lead to sudden deaths are either of a mechanical or electrical nature with the latter consisting of over 90% of the cases.
ACKNOWLEDGEMENTS

No sources of funding were used to assist in the preparation of this review. The authors have no conflicts of interest that are directly relevant to the content of the review.

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