INTRODUCTION

Favourable effect of continuous physical activity on functioning of cardiovascular system, as well as on prevention of primary and secondary cardiovascular diseases was well studied and confirmed by a great number of epidemiological studies (Leon et al., 1987; Paffenbarger et al., 1993). Athletes are considered the healthiest members of our society, therefore their unexpected death during training or competition draws high public attention. Sudden cardiac death (SCD) is the most frequent cause of death in professional athletes (Drezner et al., 2007; Maron et al., 1996a).

Although the first death of an athlete was recorded in 490 B.C., when after 42 kilometers of running from Marathon battlefield to Athens, Greek soldier and the first marathon runner Pheidippides...
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died, immediately after he had communicated the news about the great Greek victory over the Persians, the significance of SCD in broader medical public was recognized only in the middle of the last century (Lorvidhaya et al., 2003). In our country the issue started being discussed only after the World Basketball Championship held in Ljubljana in 1970 and the sudden death of our national team member Trajko Rajković.

Although there is no generally accepted definition, the sudden cardiac death can be classified as any non violent and unexpected death of cardiac origin, resulting from sudden cardiac arrest (Koester, 2001; Maron, 2003; Sharma et al., 1997). It is characterized by a sudden loss of consciousness in the course of one hour from the appearance of acute symptoms. Previous cardiac disease can exist, but it does not have to be an immediate cause of death (Koester, 2001; Lorvidhaya et al., 2003).

SCD in sport can be classified in three categories (Pedoe, 2000):
1. Commotio cordis (cardiac contusion) caused by a blunt hit with a sports apparatus in the athlete’s thorax and consequent fatal disorder of heart rhythm;
2. SCD in athletes younger than 35 due to structural, congenital heart diseases, which attracts the greatest attention of medical public;
3. SCD due to disease of coronary arteries is the most frequent in athletes over 35 (predominant risk in marathon and half marathon runners).

ATHLETE’S HEART AND REACTION OF CARDIOVASCULAR SYSTEM TO INCREASED PHYSICAL LOAD

Before we start discussing the SCD in athletes, it is necessary to say a few words on adaptive cardiovascular physiology. Already in 1935 it was proved that continuous physical training leads to haemodynamic and electro-physiological changes in normal miocard tissue (Wight, 1995). During an intensive aerobic training, a sudden increase of oxygen consumption occurs in muscular tissue, so the heart output increases proportionally to meet the requirements. During a longer period, aerobic training results in morphological and physiological changes on heart: increase of mass of the left heart, increase of heart frequency during work (and reduction of heart frequency during the repose), increase of stroke and heart output. Heart size depends on type, volume and intensity of training. The recent researches indicate the importance of genetic factor (gene for synthesis of angiotensin-converting enzyme) in the process of hypertrophy of left auricle in athletes trained according to endurance type (Maron, 2003).

Hypertrophy of the left ventricle is symmetrical, with equal increase of septum and paries of the left ventricle (maximally up to 13mm) (Futterman, 1998; Maron, 2003; Shephard, 1996). “Athlete's heart ” increases until it has reached critical mass of 350 grams for the left ventricle (normal mass of 200 grams). In the course of training or various situations under stress, when a sudden increase of circulating catecholamine occurring in blood, demands much greater quantities of oxygen. Such changes can result in miocard ischaemia or become a trigger for a heart arrhythmia, but only in a case with previous existing anomaly of cardiovascular system (Wight, 1995).

Even though ventricular hypertrophy can be linked to SCD in athletes, an overall number of death cases during physical activity is very small, so such link is far from grounded. Generally speaking, “athlete’s heart” is usual adaptation to continuous training, which increases working capacity of a person (Maron, 2003; Shephard, 1996).

EPIDEMIOLOGY

The accurate value of incidence of SCD appearance is hard to determine because the majority of studies relies on the media reports on number of death cases, which primarily concentrate on elite athletes, and not to the overall population of young athletes. It is especially hard to establish this number in our country since there are no statistical data related to cardiac death in athletes, so we used the data of European and authors from the USA, who in majority of studies, consider the frequency of SCD in athletes of 1 in 200.000 athletes annually (Epstein, 1986; Maron et al., 1995; Maron et al., 1996a).

In the study of Maron et al. (1996a), extensive data were provided, clinical, demographic and pathohystological on 134 young athletes who died of sudden heart arrest in the period between 1985 and 1995. Average life age was 17 (the youngest 12 years of age and the oldest 40), with significantly higher
frequency of male population (even 90%), of which 44% were Afro-Americans. Of all the dead, 68% were football and basketball players. The dominant cause of tragic death in those athletes was hypertrophic cardiomyopathy in 46% and anomaly of coronary blood vessels in 19% of the dead.

SCD in female athletes occurs nine times less frequently when compared to male athletes, and the stated reasons for this are lower number of active female athletes when compared to males, better adaptation of cardiovascular system, less stressing requests at competitions, as well as rarer diagnosing of hypertrophic cardiomyopathy in general population females (Whang, 2006).

**ETIOLOGY**

The paradox of training is that it reduces the risk of SCD in undoubtedly longer period, while in the course and 30 minutes after exercising the risk actually increases (Ghosh, 2006). Therefore, in majority of (over 80%) athletes, the SCD occurred in this period, which could suggest that increased physical activity was a “trigger” of fatal cardiac arrhythmias (Maron et al., 1996a) in persons with undetected, congenital heart diseases (Firoozi et al., 2003).

Over 20 SCD causes in athletes were described in literature. It is interesting that there is a great geographic heterogeneity when it comes to the most frequent death causes. In North America the leading cause is hypertrophic cardiomyopathy while in Italy it is arrhythmogenous cardiomyopathy of right ventricle, in China it is Marphan syndrome, and in Germany it is most often myocardiitis (Pigozi, 2003).

There are numerous reasons for occurrence of SCD (Table 1). In athletes under the age of 35, the SCD is most often the consequence of hypertrophic cardiomyopathy, followed by the anomalies of coronary arteries with the predominance of the main left coronary artery starting from the right Valsalva sinus, while in those over 35 the most frequent cause of arteriosclerotic change of coronary heart arteries.

**Table 1. Causes of death in athletes**

<table>
<thead>
<tr>
<th>CAUSE</th>
<th>Under the age of 35 (N=387)</th>
<th>Over 35 (N=74)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertrophic cardiomyopathies</td>
<td>102 (26,4%)</td>
<td>1 (1,4%)</td>
</tr>
<tr>
<td>Anomalies of coronary arteries</td>
<td>77 (19,9%)</td>
<td>0</td>
</tr>
<tr>
<td>Commotio cordis</td>
<td>53 (13,7%)</td>
<td>1 (1,4%)</td>
</tr>
<tr>
<td>Idiopathic hypertrophy of left ventricle</td>
<td>29 (7,5%)</td>
<td>0</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>20 (5,2)</td>
<td>0</td>
</tr>
<tr>
<td>Rupture of aortic aneurysm and Mafan syndrome</td>
<td>12 (3,1%)</td>
<td>0</td>
</tr>
<tr>
<td>Arrhythmogenous cardiomyopathy of right ventricle</td>
<td>11 (2,8%)</td>
<td>1 (1,4%)</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>10 (2,6%)</td>
<td>1 (1,4%)</td>
</tr>
<tr>
<td>Arteriosclerotic changes of coronary arteries</td>
<td>10 (2,6%)</td>
<td>56 (75,4%)</td>
</tr>
<tr>
<td>Idiopathic dilatative cardiomyopathy</td>
<td>9 (2,3%)</td>
<td>0</td>
</tr>
<tr>
<td>Sarcoidosis</td>
<td>3 (0,8%)</td>
<td>0</td>
</tr>
<tr>
<td>Prolonged QT syndrome</td>
<td>3 (0,8%)</td>
<td>1 (1,4%)</td>
</tr>
<tr>
<td>Other causes</td>
<td>45 (11,3%)</td>
<td>13 (17,6%)</td>
</tr>
<tr>
<td>OVERALL</td>
<td>387 (100%)</td>
<td>74 (100%)</td>
</tr>
</tbody>
</table>

The data obtained by analysis of papers Lorvidhay & Stephen-Huang (2003) and Maron (2003).
The most frequent causes of sudden cardiac death:

1. Hypertrophic cardiomyopathy (HCM)

Hypertrophic cardiomyopathy is the most frequent cause of death in young athletes (30-50%) (Lorvidhaya et al., 2003; Maron, 2002; Maron et al., 1996a; Sharma et al., 1997). With the prevalence in general population of 0.1 to 0.2% (Maron et al., 1995; Pedoe, 2000) (meaning that the number of the affected persons in Serbia is around 10000), that is also the most frequent genetic cardiac disease. More than 400 different mutations on genes coding the synthesis of sarcomere were described (Michels et al., 2007). The consequence of the disturbed synthesis of these proteins is hypo functional sarcomere with reduced power and speed of contraction, which activates release of growth factors resulting in compensatory hypertrophy of miocard and proliferation of fibroblast (Hipp et al., 2004).

Visible to the naked eye, it is characterized by asymmetrical heart hypertrophy, with particular increase of the left ventricle (15 to 50mm, normally up to 13mm) and hypertrophy of interventricular septum (Picture 1). Additionally, there is no compensatory dilatation of ventricle, so that filling of ventricle in diastole is reduced. In 25% of the persons affected by HCM, it was determined that asymmetrically increased septum compromises the blood course in aorta during systole (Roberts et al., 2001). Consequently, haemodynamic disorders occur and based on them three forms of HCM can be identified (Maron, 2002):

1. **non-obstructive** – intracavit (subaortic) gradient in piece and provicable states < 30mmHg
2. **latently obstructive** - gradient in piece < 30mmHg, and under effect of provoking factors >30mmHg
3. **obstructive** - gradient in piece >30mmHg

In majority of persons, HCM does not manifest any symptoms. Only slightly over 20% of athletes who died of HCM had symptoms indicating heart diseases (syncope, palpitations, chest pain, dyspnoea during training, unconsciousness) (Maron, 2002). A disordered cell structure, ishaemia of miocard with creation of scar tissue, as well as proliferation of interstitial fibrose tissue are good arrhythmogenous substrate for electrical instability of miocard (Hipp et al., 2004).

The athletes with a diagnosed HCM are forbidden to professionally go in for sports, except for those sports with small physical load.
2. **Idiopathic Hypertrophy of left ventricle (IHLV)**

Idiopathic hypertrophy of left ventricle is a disease of unknown etiology, responsible for SCD in 7-10% of young athletes (Firoozi et al., 2003; Lorvidhaya et al., 2003; Maron et al., 1996a). From the autopsy report (Picture 2) a symmetrical increase of left ventricle paries was noticed, far greater than in "athlete’s heart" (Koester, 2001; Lorvidhaya et al., 2003; Whyte et al., 2008). As possible causes of expressed hypertrophy of miocard, the non diagnosed systematic hypertension and extreme physiological hypertrophy are given (Koester, 2001).

**Picture 2.** Idiopathic hypertrophy of left ventricle, extremely symmetrical increase of left ventricular paries

3. **Congenital anomalies of coronary arteries (CACA)**

During physical load, miocard has increased needs for oxygen. In persons with congenital malformations of coronary arteries, a hypo perfusion of miocard occurs during physical effort, due to impossibility of coronary arteries to adapt themselves and provide adequate oxygenation of miocard (Basso et al., 2000). The most frequent anomalies of are those of the main left coronary artery exiting from the right Valsalva sinus at acute angle, and passes between the aortic stoma and pulmonary artery (Picture 3). During exercises, when the aorta is dilated, a compression of aberrant artery and blood flow decreases, resulting in ischaemia of miocard and fatal heart arrhythmia (Epstein, 1986; Lorvidhaya et al., 2003; Maron, 2003).

**Picture 3.** Anomalies of main left coronary artery (Ao-aorta, RVOT-pulmonary artery)
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The remaining anomalies described in literature are the anomalies of the left coronary artery starting from the pulmonary stem, hypoplastic coronary artery, congenital agenesis of the right coronary artery and anomaly of right coronary artery starting from left Valsalva sinus (Basso et al., 2000; Futterman, 1998; Lorvidhaya et al., 2003).

The symptoms of myocard ischemiae are chest pain, dyspnoea and sudden syncope. They appear only in one of three athletes and are more frequent in persons with anomalies of the right coronary artery (Pelliccia, 2001). In their study, (Basso et al., 2000), indicate that the values of all cardiovascular tests, ECG included, in half of the athletes with prodromal symptoms of ischaemia tested during their life, were within normal limits.

4. Arrhythmogenous cardiomyopathy of right ventricle (ACRV)

Arrhythmogenous cardiomyopathy of right ventricle is a heart disease of unknown etiology, characterized by progressive loss of cardiomyocyte and their replacement with fat and fibrous tissue (Picture 4).


Fat infiltration leads to thickening (pseudo hypertrophy) of myocard, while in zones of fibrosis it is made thinner which causes formation of aneurysms of myocard paries (Indik, 2003; Sharma et al., 1997; Thiene et al., 2007) (Picture 5). The process is most frequently localized in right ventricle, but in certain persons it can also affect the interventricular septum as well as left ventricle. This disease is most probably genetically determined and can have two forms: autosomal-dominant and autosomal-recessive (more serious) (Thiene et al., 2007). By mapping genes, mutation of several genetic locuses for synthesis of proteins ventricle which mechanically connect cardiomyocyte.

Structural changes of cardiac muscle are clinically manifested by appearance of ventricular tachyarrhythmia and haemodynamic disorders due to ventricles dysfunction. The first symptoms are usually manifested between the age of 15 and 35 and can vary from feeling of irregular and accelerated heart work (palpitation), weakness, fatigue, lack of air (dyspnoea), short fainting (syncope), to a sudden cardiac arrest (SCD) (Sharma et al., 1997; Thiene et al., 2007). Although it is considered that great physical effort is one of the most important factors for emergence of fatal cardiac arrhythmias, the affected by ACRV usually tolerate well physical activity, because the function of left ventricle is well preserved (Sharma et al., 1997).
5. Myocarditis

Myocarditis is an inflammatory heart disease associated with cardiac dysfunction. It is most often of viral etiology (coxackievirus B in more than half of the cases, Adenovirus, Herpes viruses), while bacterial and fungus infections are rarely causes of myocarditis (Futterman, 1998; Lorvidhaya et al., 2003). Inflammation of myocard with infiltration of lymphocyte and focal necrosis of the tissue, was found after post-mortem histopathological examinations, proved to be significant cause both for electrical instability of heart and origin of arrhythmias, especially during hard physical efforts (Coskun et al., 2006; Sharma et al., 1997).

In majority of persons, myocarditis does not give any symptoms, so often SCD is the only manifestation of illness (Futterman, 1998; Koester, 2001). Some of the early symptoms which can arouse suspicion of illness existence are the signs of deterioration of cardiac function (fast tiring during physical activity, palpitations, dyspnoea and orthopnea), accompanied with the signs of viral infection (headache, vomiting, rise in temperature, pains in muscles) (Ng et al., 2007).

Training during the acute phase of disease leads to virus replication, more expressed necrosis of myocyte, and consequently increase of risk of SCD (Martin et al., 1994). Therefore, athletes are advised to be on a sick leave and are forbidden to be engaged in physical activity for at least six months (Lorvidhaya et al., 2003).

6. Rupture of aorta and Marfan syndrome (MS)

Marfan syndrome is an autosomal dominant disease of connective tissue which requires more organic systems. The most frequent cause of a sudden cardiac death in athletes is rupture or dissection of aorta, caused by a congenital reduction in number of elastic fibers in muscular layer of aorta (Glorioso et al., 2002; Michael, 2001).

Athletes with MS have characteristic phenotype features (Table 2) (Koester, 2001), so it is easy to clinically diagnose it.

<table>
<thead>
<tr>
<th>Table 2 Characteristics of Marfan syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive family history with MS</td>
</tr>
<tr>
<td>Murmur of aortic stoma</td>
</tr>
<tr>
<td>Unusually high increase with regard to age</td>
</tr>
<tr>
<td>Gothic palatum</td>
</tr>
<tr>
<td>Long fingers and toes (arrachnodactylyia)</td>
</tr>
<tr>
<td>Deformities of thorax (pectus exavatum) and vertebral column (kyphoscoliosis)</td>
</tr>
<tr>
<td>Loosen joints</td>
</tr>
<tr>
<td>Myopia and ectopic lens</td>
</tr>
</tbody>
</table>
7. Brugada syndrome (BS)

Brugada syndrome is a genetically determined disease characterized by ECG findings of elevation of ST segment in right precordial offakes V1-V3 (Picture 6). All clinical manifestations of BS are connected to sudden ventricular arrhythmias (Matsuo et al., 1999). The disease is more frequent in males (8:1 male: female ratio), and appearance of syndromes i.e. arrhythmic events ranges from the 3rd to 7th decade of life (Wilde et al., 2002). In one third of the deceased, SCD can be the first and the only symptom of the disease. Most of the arrhythmic events occur during night time, which could indicate that misbalance of sympathicus and parasymphaticus tonus is an important trigger of cardiac arrhythmias (Eckardt, 2005).

BS patients are not allowed to go in for sports.

8. Prolonged QT syndrome (PQTS)

Emotional stress and physical activity can cause occurrence of lethal arrhythmias (torsades de pointes and ventricular fibrillation), in situations when repolarization of ventriculas is prolonged (Lorvidhaya et al., 2003). The clinical entity characterized by frequent appearance of syncopes and high incidence of SCD mostly occurred at stressful events when a concentration of catecholamine in blood is increased, is called a prolonged QT syndrome (Goldenberg et al., 2008). PQTS is a hereditary hearth disease, with prevalence of 0.4% in elite athletes. QT segment longer than 500 ms (normally up to 440±60 ms) points to LQTS with high certainty (Bonny et al., 2008).

LQTS patients are not allowed to go in for sports.

9. Cardiac contusion or Commotio cordis (CC)

Commotio cordis is a syndrome manifested by disorder of rhythm or SCD, as a consequence of a non-penetrating thud of a projectile in thorax or a clash of bodies of two athletes, without injuries of ribs, sternum or heart. The hit is of small energy (except if made by hockey puck), and when it leads to ventricular arrhythmia it must occur in the period of repolarisation of ventricula immediately before the
peak of T-wave, in the so called “vulnerable” period of heart cycle which amounts to only 10ms. Mechanical energy of the projectile at the moment of the hit is transferred to myocard and transformed into an electrical impulse, sufficient to initiate emergence of a fatal ventricular fibrillation. Apart from time, the appearance of ventricular fibrillation is significantly determined by hit localization, force and speed of projectile hitting, as well as changes at molecular level of Na⁺ and K⁺ in ATP depending channels (Madias et al., 2007; McCrory, 2002).

Differently from the previous causes of SCD in athletes, the victims of CC are healthy persons without previous case history of heart disease or other cardiac illnesses (Futterman, 1998; Madias et al., 2007).

Although CC very rarely occurs in sport, its significance is great because death is almost sudden, and death rate is over 90% (McCrory, 2002). A small number survives thanks to rapid measures of cardiopulmonary reanimation with use of automatic external defibrillator.

Prevention of SCD in CC is possible in two ways. Firstly, by wearing adequate protective gear of the highest standards. An upsetting data, that even 38% of athletes who died of CC had some sort of protection, tells that protective gear should be adapted to each individual. The other way is the change of competition rules and training techniques (McCrory, 2002).

PREVENTION OF SUDDEN CARDIAC DEATH IN SPORT AND PREPARTICIPATION SCREENING - TRYING TO FIND THE NEEDLE IN THE HAYSTACK

Athletes with a diagnosed cardiovascular disease (even a clinically silent one), when compared to other healthy individuals, due to the intensity of their regular training and participation in sport, have an increased risk of SCD or emerging of clinical aggravation of the disease. Two basic aims of preparticipation screening are: a) early identification of athletes with structural heart diseases and b) formulation of indications which represent balance between risk and benefit from participation in competitive sport (Pigozzi et al., 2003).

Taking into consideration significance of the problem and the data, that 55-80% of athletes who died due to SCD did not manifested any symptoms of heart disease (Drezner et al., 2007), a question is asked: “What else can be done to identify persons with increased risk?”. How is the incidence of SCD 1:200.000, even when applying a screening test whose sensitivity and specificity are 99%, we would get only one athlete with really positive and 1999 with false positive result. Another defect of the screening are “abnormalities” detected during examination, which in fact are normal variations. Such changes were found in persons with athlete’s heart syndrome, in which electrocardiograph, radiographic and echosonographic changes were detected caused by enlargement of cardiac muscle and reduced vagus tonus. While similar criteria for differentiation of athlete’s heart when compared to pathological states are well studied, the differentiation of normal findings when compared to physiological variations can be very hard.

A good screening test must be a cheap, valid, secure and generally accepted procedure, which would enable an early detection of disease in asymptomatic phase (Inklaar et al., 2007).

Athletes with prodromal symptoms, syncope and palpitation, dyspnoea during training and chest pain in peace or during physical activity, should be submitted to a detailed follow-up (Firoozi et al., 2003; Pedoe, 2000). European Association of Cardiologists (Corrado et al., 2005) even gave recommendations, according to which, all professional athletes should be included in screening in compliance with the Lausanne protocol in two acts. The first step is a basic screening which includes taking of a detailed history (personal and family) by filling up of a sports – medical form, followed by a physical examination and a 12-channel electrocardiogram (ECG). In athletes with a reasonable suspicion of heart disease, the examination is to be continued (second step) by using all available diagnostic procedures, echocardiography, stress ECG, computerized tomography (CT) and nuclear magnetic resonance (NMR).

In 1996, American Heart Association (AHA) adopted 12 recommendations for preparticipation screening of cardiovascular system (Table 3), of which 8 are related to anamnesis data, and 4 to physical examination (Maron et al., 1996b). If any of the aforesaid elements of screening is answered with “YES”, it is necessary to continue further examinations of cardiovascular system.
Cardiovascular Disorders as the Cause of Sudden Cardiac Death in Athletes

Table 3. 12 AHA recommendations for examination of cardiovascular system

<table>
<thead>
<tr>
<th>PERSONAL HISTORY</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Chest pain at effort</td>
</tr>
<tr>
<td>2. Lack of air at effort</td>
</tr>
<tr>
<td>3. Hypertension</td>
</tr>
<tr>
<td>4. Syncope</td>
</tr>
<tr>
<td>5. Heart murmur</td>
</tr>
<tr>
<td>6. Excessive fatigue</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>FAMILY HISTORY</th>
</tr>
</thead>
<tbody>
<tr>
<td>7. Cardiac death in family before the age of 50</td>
</tr>
<tr>
<td>8. Existence of cardiac diseases in close relatives younger than 50</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>PHYSICAL EXAMINATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>9. Auscultation of heart murmurs</td>
</tr>
<tr>
<td>10. Palpation of femoral pulses in order to discover coarctation of aorta</td>
</tr>
<tr>
<td>11. Stigma for Marfan syndrome</td>
</tr>
<tr>
<td>12. Measuring of blood pressure</td>
</tr>
</tbody>
</table>

At the 36th Conference of Bethesda held in January 2004 new recommendations for going in for sport for persons with diagnosed heart diseases (Table 4) were given (Maron, 2005). According to this panel, a physician who is examining athletes has ethical, medical and legal obligation to inform in details athletes on risks appearing in competitive sports and in the case of appearance of high cardiovascular risk, the physician is responsible for the final conclusion in order to prevent undesired event or to reduce the risk for progression of disease. The recommendations depend on nature and difficulty of cardiovascular disease, as well as of classification of sport (statistic and dynamic).

Table 4. Recommendations for going in for sports in persons with heart diseases that most often cause SCD (Bethesda 2004 )

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>Prohibition of professional sports career, with a possible exception of sports with small physical load</td>
</tr>
<tr>
<td>Arrhythmogenous cardiomyopathy of right ventricle</td>
<td>It is not allowed to go in for sports</td>
</tr>
<tr>
<td>Congenital anomalies of coronary arteries</td>
<td>It is not allowed to go in for sports</td>
</tr>
<tr>
<td>Athletes without signs of ischaemia at stress load test can go in for sports 6 months after surgical intervention</td>
<td></td>
</tr>
<tr>
<td>Prolonged QT syndrome</td>
<td>It is not allowed to go in for sports</td>
</tr>
<tr>
<td>Coronary arteries diseases</td>
<td>If low risk of SCD is established, can participate in sports of low and moderate intensity. Mandatory annual follow-up.</td>
</tr>
<tr>
<td>If high risk of SCD is established, can participate only in sports of low intensity. Mandatory follow-up every 6 months.</td>
<td></td>
</tr>
</tbody>
</table>
## CONCLUSION

SCD in athletes is extremely rare event, whose incidence ranges from 0.5 to 1.6 in 100,000 athletes. It occurs nine times more often in males, and the most frequent cause of death is HCM. The upsetting fact that majority of athletes who died from SCD did not have a previous case history of heart disease, or prodromal symptoms before death, places a hard task for physicians to detect persons with an elevated risk. The dilemma is even greater due to disagreement of American and European recommendations, about which screening test is the most economic and the most optimal for revealing the athletes with elevated risk. While the European Association of Cardiologists puts ECG examination as a golden standard of each screening test, the AHA categorically rejects it, due to its small sensitivity and great number of positive results. Substantiating the European recommendations are the data that in Italy in the last ten years not a single case of SCD was recorded in athletes subjected to preparticipation screening. They, depending on findings, were excluded from competitive activities and are submitted to therapy.

Taking into consideration moderate economic standard of the Republic of Serbia it is hard to determine which screening test is reliable and still cheap. Our opinion is that all active athletes should be legally obliged to undergo preparticipation follow-up in certain licensed outpatient departments and institutions. The follow-up should be standardized and prevent „overlooking”. In elite athletes it is necessary to perform once a year an ultrasound (US) heart examination. All participants in sport with a diagnosed disease are to be subjected to standard medical diagnostic and therapeutic procedure, and recommendation for volume and type of physical activity is given by physicians according to the valid world recommendations which were gathered and translated by the Association for Sports Medicine of Serbia (Dikic, Ostojic, Zivanic & Mazic, 2004).

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