

The incognita of the known: the athlete's heart syndrome

Bilinenin bilinmezliği: Sporcu kalbi sendromu

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ABSTRACT

Long-term athletic activity causes morphological and functional changes in the heart characterized as left ventricle cavity dimension changes, wall thickness and mass increase and rhythm conduction changes. This condition is identified as "athlete's heart syndrome". The changes that are seen clinically occur as a result of physiological adaptation to exercise. Cardiovascular adaptation depends on the exercise's type as well as its frequency, duration and intensity. In the athlete's physical examination, various changes can be seen that are mistaken with pathological conditions. In addition, there are changes present due to cardiac hypertrophy, increased vagal tone and repolarization. The knowledge and recognition of the organic and functional changes developing in the athlete's heart is being helpful to differentiate physiological changes from cardiac pathologies that can cause sudden death in athletes. (*Anadolu Kardiyol Derg 2011; 11: 351-9*)

Key words: Exercise, athlete, electrocardiography, echocardiography, hypertrophic cardiomyopathy, sudden cardiac death

ÖZET

Uzun dönem sportif aktivite kalpte, sol ventrikül kavite ölçülerinde, duvar kalınlığında, kitesinde artış ve ritim iletim değişiklikleri ile karakterize morfolojik ve fonksiyonel değişikliklere neden olur. Bu tablo 'sporcu kalbi sendromu' olarak tanımlanır. Klinik olarak görülen bu değişiklikler egzersize fizyolojik adaptasyon nedeniyle ortaya çıkar. Kardiyovasküler adaptasyon; egzersizin sıklığı, süresi, yoğunluğu kadar tipine de bağlıdır. Sporcuların fizik muayenesinde patolojik durumlarla karışabilen birçok değişiklik görülür. Ayrıca kardiyak hipertrofi, artmış vagal tonus ve repolarizasyon değişikliklerine bağlı birçok elektrokardiyografik değişiklik de mevcuttur. Sporculardaki bu fizyolojik değişiklikler, patolojik değişikliklerden her zaman için kolayca ayrılamayabilmektedir. Sporcu kalbindeki gelişen organik ve fonksiyonel değişikliklerin bilinmesi ve tanınması klinisyene, fizyolojik değişikliklerin sporcularda ani ölüm nedeni olabilen kardiyak patolojilerden ayrılmasında yardımcı olabilmektedir. (*Anadolu Kardiyol Derg 2011; 11: 351-9*)

Anahtar kelimeler: Egzersiz, sporcu, elektrokardiyografi, ekokardiyografi, hipertrofik kardiyomyopati, ani kalp ölümü

Introduction

From Henschen to today

Long term exercise done regularly and frequently affects the whole body and causes several physiological changes. Cardiovascular system is one of the systems these changes most frequently occur. The athlete's heart syndrome is identified as all of the structural, electrical and functional changes developed in the heart to help maintain cardiac output increase with

regular physical activity (1-6). The presence of this phenomenon first drew attention in 1899 with Henschen's examination in long distance skiers, determining the expansion of the cardiac borders with percussion (1, 7). After echocardiography went into use in clinics with these morphological changes more distinctively expressed, it has been seen that some physiological findings may be in an extent that will be mixed with pathologies leading to sudden cardiac death in athletes; which enhances the significance of the issue. The changes in the measures of cardiac

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cavity and thickness may sometimes reach a degree that imitates myocardial infarction.

The parameters of cardiac adaptation

These cardiac changes seen in athletes appear due to physiological adaptations to exercise. It is accepted that the intensity, frequency and the duration of the exercise and the gender, ethnicity and genetic features of the athlete are effective altogether during this adaptation period. Excluding ethnic, genetic and gender factors, the type of exercise is foreseen to be the utmost important determinant in cardiovascular response (8, 9). Simply, exercise is categorized under two titles as isotonic and isometric. Isotonic, also described as aerobic, exercise types are exercises that result in sarcomere length changes in big muscle groups without changes in muscle tension. Marathon and long distance skiing are sport genres that use this exercise type more often. Isometric, also described as anaerobic, exercise types are exercises that result in significant tension increase in smaller muscle groups without muscle size difference. Wrestling and weightlifting are the examples of sports that use this exercise type more often. Despite this differentiation, athletic activities usually use the combination of these exercise types (8, 9). Moreover, it is known that the parameters of intensity, duration and permanence of exercise, with variable levels of effects, also determine the adaptation, which develops in a different analogy.

Cardiovascular adaptation to regular exercise

Isotonic exercises increase cardiovascular volume load by increasing the venous return to the heart (8). As for the isometric exercises, they cause cardiovascular pressure load by increasing systemic blood pressure. The morphological and functional changes show diversity according to exercise type. A maximal isotonic exercise causes two times increase in stroke volume of the heart, around four times increase in stroke volume per minute, and around three times increase in oxygen consumption therefore as a result of regular exercise myocardial hypertrophy of proportional character is seen (4, 8). Approximately 10% of increase in left ventricle diameter and 33% of increase in left ventricle end-diastolic volume can be determined (10). The cardiac remodeling in athletes is correlated with maximal oxygen consumption, which is an indicator of aerobic performance (11). Ventricular wall thickness and muscle mass increase to maintain the normal wall tension of the dilated ventricle. Although both the wall thickness and the ventricle diastolic diameter increase, the mass/volume ratio remains stable. The physiological hypertrophy and dilatation is developed in quite a short time. Ehsani et al. (12) has shown that, the left ventricle mass have significantly increased after one week from the start of the exercise and while the wall thickness reaches the peak level after 3-5 weeks, the diastolic diameter of the left ventricle reaches its peak level approximately at the end of the first week. An exercise program of 3-4 times per week of 30-60

minute exercise duration at 60-70% intensity of maximal oxygen consumption level should be performed for these cardiac changes to appear (4, 8). The cardiovascular changes occurring because of isometric exercise are different. There is a sudden increase in blood pressure, myocardial contractibility and cardiac stroke volume as a result of the catecholamines that increase during exercise and the nerve signals caused from muscle reflexes (13). During isometric exercise, the blood pressure response can reach to higher blood pressure levels than seen in isotonic exercise (14, 15). Due to this pressure load, the wall of the ventricle thickens. During isometric exercise the venous return does not significantly change therefore, the diastolic diameter of the left ventricle does not significantly increase (13). Because of this with athletes whom does static exercises, the mass/volume ratio has increased (13, 16). Another possible mechanism of cardiac hypertrophy is that left ventricular mass appears to be more strongly related to the peripheral pulse pressure, measurements of hemodynamic pulsatile load during exercise in athletes (17) (Fig. 1). On the other hand, the maximal oxygen consumption does not significantly increase. The degree of the hypertrophy depends on the body type and gender of the athlete. Usually with athletes who have larger body mass, the cardiac volume and left ventricle muscle mass is greater. The older athletes have more significant myocardial hypertrophy due to the duration of active exercise time being longer (1, 2). It is determined that women athletes have smaller diastolic cavity measures and show less hypertrophy than male athletes on the same training level (1, 2, 18).

Apart from these, in athletes besides cardiac compliance, vascular compliance mechanisms also develop. The vascular reactivity, dependant on flow, proceeds in parallel compliance to

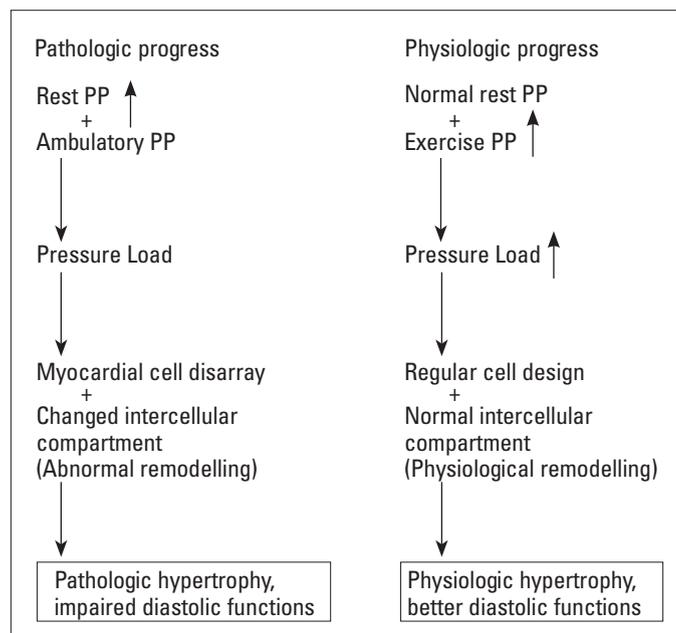


Figure 1. Possible mechanism of pulse pressure (PP) and cardiac remodeling in physiologic and pathologic conditions (reproduced from reference 17 with permission of the Anadolu Kardiyoloji Dergisi, Copyright 2005)

the change in aerobic performance (19). The changes in vascular reactivity occurs both in central and peripheral arterial system. The relationship between aortic distensibility and the diastolic functions shows the functional result of the compliance in the athlete (20, 21).

These changes in the athlete due to exercise can be determined by physical examination, electrocardiography, and echocardiographic examination. These evaluation methods can also be important in diagnostics.

Physical examination findings

The physical examination findings of the athlete might be quite variable and nonspecific. Many findings that may be considered as pathologic in normal people can be seen physiologically in athletes. To determine the cardiac diseases that may result in sudden death due to exercise, it should be known how to distinguish which of these findings are normal and which are pathologic in the athletes. The cardiac stroke volume that's increased during rest causes the vagal tone to increase by creating myocardial tension and baroreceptor stimulation (1, 8). Therefore there's a significant rest bradycardia and a lower systemic blood pressure in athletes (1, 8). The pulse might be irregular due to sinus arrhythmia and often respiratory arrhythmia might be seen (5). The pulse beat has high amplitude. The left ventricle beat is significant, has changed its spot and can be palpable in a larger area. As a result of physiological hypertrophy, the 3rd heart sound and less rarely the 4th heart sound may be heard especially in child and adolescent athletes and it may not have a clinical importance in asymptomatic children (5, 8, 22). A soft, systolic ejection murmur might be heard as a result of the increased pulmonary blood flow. This innocent murmur in athletes can be best heard in lying in a supine position while it gets lighter or disappears in the auscultation done in standing position (8, 23).

Electrocardiographic (ECG) findings

The athlete's ECG may show quite a bunch of differences compared to a sedentary person's ECG. Pelliccia et al. (5) showed in a research done with 1005 athletes' ECG examination that approximately 40% of the athletes have abnormal ECG findings while 15% of them had changes that suggest cardiomyopathy. The changes seen in the ECG have developed secondary to increased vagal tone, decreased sympathetic tone and physiological hypertrophy (8, 24-27). The most frequent finding is sinus bradycardia at rest (8, 24). Especially in sleep recordings, less than 30 beats might be seen. In athletes, pauses for more than two seconds may be seen in 24-hour rhythm monitoring. It is accepted that unless the situation follows up with a clinical finding, it need no further investigation and treatment (24-27). With a significant existing bradyarrhythmia, escape junctional beats and rhythms might be seen. Supraventricular arrhythmias and ectopic beats of the A-V node are the changes that can be seen in athletes. In athletes; bradycardia, sinus pauses less than

three seconds, sinus arrhythmia, wandering atrial pacemaker or junctional rhythm don't need further examination (26, 27). If the sinus pause is more than three seconds further examination should be made. First-degree A-V block might be seen in 33% of the athletes. The mechanism here again depends on the deceleration of conduction velocity in the A-V node due to increased parasympathetic and/or decreased sympathetic tone. Second degree Mobitz type 1 (Wenkebach) A-V block can be seen in 10%. Mobitz type 2 A-V block and third degree A-V block are more rarely seen. An underlying cardiac pathology should be thoroughly investigated before interpreting a change as an adaptive change to exercise (24). If the QRS complexes are normal, the subjects that have a first-degree A-V block do not need further examination. If the PR interval time is longer than 0.3 seconds or the QRS complexes are abnormal, Wenckenbach type A-V block or congenital complete A-V block is present; exercise testing, 24 hour Holter monitorization and echocardiographic evaluation should be made (26, 27). When an atrial fibrillation is detected, attention should be paid and further examination should be made to rule out underlying heart disease.

Another finding encountered in athletes is high voltage in ECG. In a study by Pelliccia et al. (5), around 60% of the athletes have been detected to have left ventricle hypertrophy when Sokolow-Lyon index is used for left ventricle hypertrophy. Left ventricle hypertrophy is detected in athletes that does bicycle, long distance skiing and rowing and usually into endurance sports. Again using Sokolow-Lyon index amongst 0.6-12% of the athletes, right ventricle hypertrophy is detected (24-27). The increased muscle mass cause's incomplete right bundle branch block amongst the 35-50% of the trained athletes and this finding disappears by terminating the regular exercise (8, 24-27). It is thought that these electrical changes of right heart in athletes are the result of geometrical compliance, which causes variably preceding functional effects in the right heart.

The ST-T wave changes, consistent with early repolarization, are seen in more than 50% of the athletes and are closely related with the athletic activity's duration and intensity (24-27). J point elevation can be seen in several derivations although it is mostly seen in V3-V4 derivations. These repolarization changes in athletes can be mixed with the findings of Brugada syndrome. Negative T waves might be seen in 2-3% of the athletes without any underlying heart disease (24-27). These changes are thought to happen as a result of heterogeneity of the myocardial action potential caused by vagotonia (24-27). Having these significant repolarization changes is accepted to have a situation that needs further examination. Horizontal and down-sloping ST depression is rare in athletes and thought to be occurring because of underlying pathologies (24-27). The ST, QT segment and T wave changes in athletes go back to normal during exercise due to vagal tone being inhibited. With the termination of regular exercise, these changes go back to initial levels (24-27).

Apart from these, the athletes, with a long QT interval, certainly must be distinguished from cases with congenital long QT

syndrome, which can be a reason for sudden cardiac death. Although the QT measures of the athlete are longer compared to non-athlete group, due to lower heart rate, the QT measures corrected with heart rate are amongst normal limits.

Echocardiographic findings

In the echocardiographic examination of the athletes, some changes that are caused by the same adaptation mechanisms have been determined. In the analysis of several echocardiographic studies performed, they are reported that when athletes are compared to control group, the left ventricle mass is approximately 46%, left ventricle cavity measures 10%, posterior septum thickness 14% and anterior septum thickness is 19% higher detected (1-5, 14, 18, 28-34). In most of the studies, in dynamic exercising athletes, an increase in the diastolic diameter is noted in left ventricle compared to control group (1-5, 14, 18, 28-34). In a study of Pellicia et al. (4) where 1309 elite athletes were echocardiographically examined, the diastolic diameter of the left ventricle was found in normal limits in 55% of the athletes (mean value ≤ 54 mm). In 14% of the cases, the left ventricle cavity was found so significantly dilated as to be mixed with dilated cardiomyopathy (in values up to 70 mm in men, 66 mm in women). In another study this ratio was found 19.7% (35). However, in these cases, the normal levels of systolic and diastolic functions of the left ventricle and the non-impaired wall movement make it difficult to differentiate it from dilated cardiomyopathy (4, 14, 36). Generally left ventricle septum and posterior wall thickness increase accompanies the dilation of ventricle cavity dimensions in athletes (1-3, 36). Again, in another study of Pellicia et al. (3) where 947 women and men athletes from various sport groups were examined, the ventricle septum thickness was found below 12 mm in 98% of the cases. In only 12% of the athletes, who were included in the study, ventricle septum thickness was detected over 12 mm; however athletes whom had values over 16 mm had not been detected. (3) It is utterly important to differentiate the septum thickness increased cases from hypertrophic cardiomyopathy because it is the most frequent cardiac reason for exercise related sudden deaths in young aged athletic groups (37-39). For individuals who have had this diagnosis, competitive and high intensity sports must be forbidden. The additional detection of the large left ventricle cavity (55-63 mm) in athletes, who have a septal thickness of 12 mm and over, is a finding, which suggests that the hypertrophy is physiological and it is important in differential diagnosis. That is because in most of the patients with hypertrophic cardiomyopathy, the diastolic cavity measurements are smaller and usually under 45 mm (5, 40). In athletes, the ventricle wall thickening is generally symmetrical and septum/free wall thickness ratio is in normal standards (<1.3) (3, 14). Apart from that the left atrium, right ventricle and left ventricle end-systolic cavity measurements are found higher compared to normal sedentary individuals (27, 41-43).

Gray zone problem in athletes

The genetic abnormalities most associated with sudden cardiac death are cardiomyopathies, especially hypertrophic. While the changes, consequent to training compliance in athletes, are not frequent; yet they may be mistaken with cardiomyopathies. The most frequently met one amongst these problems is the differential diagnosis of the hypertrophy in athletes from hypertrophic cardiomyopathy. The clinical detection of athletes with hypertrophic cardiomyopathy can be challenging, given that many athletes may present with the non-obstructive form, which can make the disease clinically silent (44). Since the phenotypic expression of hypertrophic cardiomyopathy is variable, and not uncommonly includes patients with mild and localized left ventricular hypertrophy, the differential diagnosis with physiological remodeling of athlete's heart not uncommonly arises (45). Hypertrophic cardiomyopathy is camouflaged by left ventricular dilation due to volume overload in endurance athletes (46). Although several algorithms have been suggested, in some of the cases, differential diagnosis may not always be as simple (47) (Fig. 2). Most of the studies show that, in athletes with physiological hypertrophy, diastolic functions do not change or improve. In

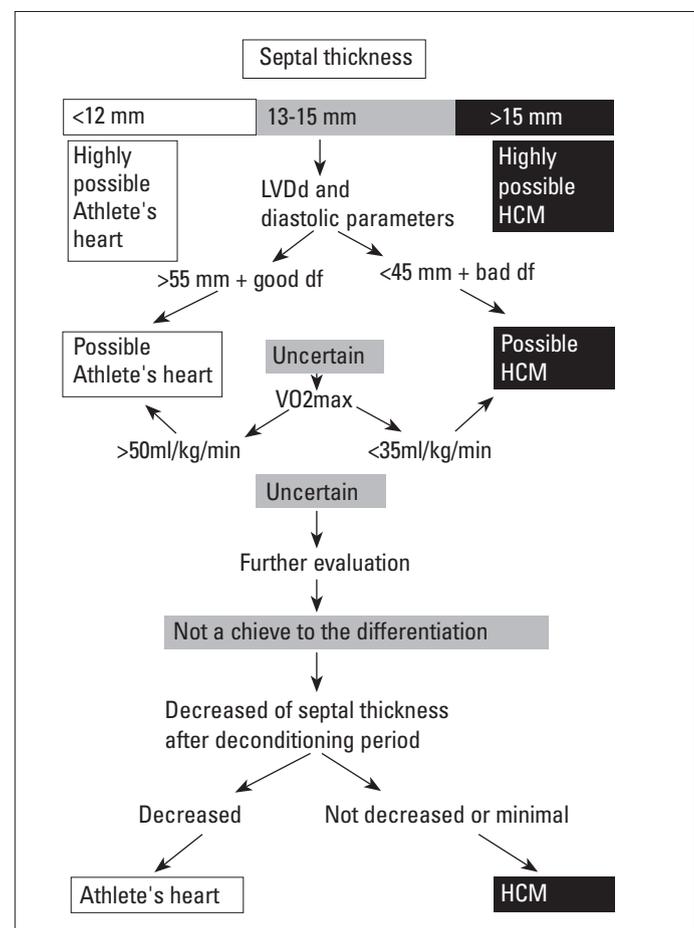


Figure 2. An algorithm for distinguishing hypertrophic cardiomyopathy from athlete's heart (modified from reference 47, with permission of the Anadolu Kardiyoloji Dergisi, Copyright 2007)

HCM - hypertrophic cardiomyopathy, LVDd - left ventricular diastolic dimension, df - diastolic filling

these studies, the left ventricle filling pattern and the diastolic flow ratio are detected normal (4, 8, 14). The normal or mildly improved diastolic function of the athlete may help to differentiate the athlete's heart from other pathological conditions that lead to left ventricle hypertrophy (31, 45). These cardiac morphological changes in athletes start with exercise and recede with the termination of the physical activity (12) (Table 1).

The left ventricle systolic functions are normal even in athletes showing high levels of cavity dilatation (4). The diastolic parameters measured by tissue Doppler imaging may be used successfully to distinguish pathologic and physiologic cardiac enlargement if left ventricular cavity diameter is within gray scale (35).

It is clear that there is still a long way to go for the discrimination of these two entities. However, it appears that evaluation of myocardial function by new echocardiographic techniques may be useful in solving this problem (48). Pathologic hypertrophy and dilation are probably related to a known characteristic of dia-

stolic dysfunction. Recently, an easily measured tissue Doppler index was proposed as a potentially useful method for distinguishing athlete's heart from structural heart disease (49, 50). In addition, tissue Doppler imaging should be used as a diagnostic criterion for differentiating physiological hypertrophy from the pathologic (51).

How can the cardiovascular events in athletes be reduced?

The best fragile question is that prevention of sudden cardiac death in athletes is dream or reality (52). Today, we have several major troubles for solving of the question. Large population, discordance between phenotypic and genotypic expression of genetic cardiovascular disease, no gold standard testing, standardization of evaluating, cost-effectiveness are some of the troubles. The one fact in the description of the athlete's heart that has not changed is that the utmost important factor in preventing cardiovascular events, especially sudden cardiac

Table 1. Clinical, imaging and laboratory parameters for distinguishing hypertrophic cardiomyopathy from athlete's heart

Differential parameters	Athlete's heart	Hypertrophic cardiomyopathy	Troubles
Characteristic			
Gender	Absent	Female	Need more study
Symptoms	Absent	Angina, syncope, dyspnea, Fatigue palpitation	Symptoms usually develop phenotypic expression of HCM, not all patients.
Family History	Absent	Positive clue	Phenotypic expression may differentiate in subjects, positive history
Physical examination	Ejection sound, not spread	Systolic murmur, spread, changing with maneuvers	Some patients have no obstruction and murmur
Electrocardiography		Prolonged QRS, deep Q waves, prominent ST depression negative T waves,	Not diagnostic
Peak oxygen uptake	>50 ml/kg/min	Usually <35 ml/kg/min	Not diagnostic
Echocardiographic examination			None of the parameters are patognomonic
LV diastolic diameter	>55	<45 mm	
Septal/posterior thickness	<1.2	>1.4	
Systolic anterior motion		Present, strongly suggest the diagnosis	
Diastolic function	Good	Bad	
Tissue Doppler imaging (systolic annular velocity)	Normal	Velocity <9 cm/s	
Coronary flow reserve	Normal/Increased	Decreased	
Magnetic resonance imaging (Gadolinium)		Reduced global and regional deformation, in association with fibrosis	Not patognomonic, cost-effectivity problem
Genetic analysis		Definitive	Usually false negative results, cost-effectivity problem
Deconditioning effect	Decreased cardiac hypertrophy	Not changed, or minimally	Valuable but losing the cardiac adaptation in athletes
HCM - hypertrophic cardiomyopathy, LV - left ventricle			

death, is the appropriateness evaluations. The right time for the evaluation of appropriateness of sports is pre-participation.

To avoid these unwanted events and serious health problems, it is important to complete the pre-season examination seriously (53). The first and most significant step of the method that needs to be followed is taking the detailed anamnesis of the athlete (Table 2) (53). While anamnesis taking, the athlete must be questioned whether she or he has a complaint. Even if the athlete does not have a complaint, thorough system query should be made. The positive findings should be noted in the athlete's file. The complaints of the athletes who have them should be thoroughly questioned and recorded. Complaints suggestive of heart diseases like chest pain, fatigue, palpitation, shortness of breath, dizziness and fainting must be persistently questioned. Within this query undergone medical problems, family history, habits and drug usage must be questioned.

After these, systemic examination must be done. The resting pulse and tension entities must certainly be noted. In cardiac auscultation it must be determined whether first and second heart sounds are normal and extra heart sounds are heard. If a murmur is heard, it must be noted whether it radiates or changes with maneuvers.

After physical examination, a resting electrocardiographic examination needs to be done in order to determine a hidden cardiovascular disease. In ECG, after determining whether the rhythm is sinusoidal or not; it should also be determined whether

there is hypertrophy, ischemia, and arrhythmia. It must be determined if the detected findings might be present with the normal limit changes or not. If the determined findings are going to be considered as abnormal, pre-diagnosis should be made. In athletes who have a risk factor or are suspected to have a coronary heart disease for any reason must be done an exercise test.

American Heart Association suggests that in high school and university students, appropriateness examinations should be done at least once every two years. Again the same organization suggests that it would be possible to identify individuals that gain risk by a 12 item rating scale, which consists of the athlete's medical history, family history and physical examination. On the suggested 12 items rating scale (54):

The athlete's medical history:

- Exercise related chest pain and discomfort
- Syncope and presyncope
- Unexplainable dyspnea or fatigue
- Heart murmur described before
- Detection of high blood pressure

Family history:

- Presence of early death from cardiovascular disease
- Disability before age of 50 due to cardiovascular disease
- Hypertrophic cardiomyopathy, dilated cardiomyopathy, Marfan syndrome, arrhythmia, channelopathy

Table 2. Cardiovascular risk assessment example query during pre-participation evaluation of athletes (Reprinted from the American Journal of Cardiology, Vol. 88/1, Vinereanu D, Florescu N, Sculthorpe N, Tweddel AC, Stephens MR, Fraser AG., Differentiation between pathologic and physiologic left ventricular hypertrophy by tissue Doppler assessment of long-axis function in patients with hypertrophic cardiomyopathy or systemic hypertension and in athletes, Pages No. 53-58, Copyright (2001), with permission from Elsevier)

- Have you ever had any chest pain during or after exercise? YES NO
- Have you ever fainted during or after exercise? YES NO
- Have you ever had any dizziness during or after exercise? YES NO
- During exercise, do you get exhausted before your team-mates? YES NO
- Have you had any palpitation or skipping heart beat complaints? YES NO
- Have you ever had your blood pressure (tension) measured? YES NO
- Have you ever had high blood pressure (hypertension) detected? YES NO

If yes, are you having any treatment?

- Have you ever had any blood analysis and blood fat measured? YES NO

If yes indicate the measurement results.....

- Have you ever been told that you have an abnormal sound or murmur in your heart? YES NO

If yes have you had further examinations?

- Do you have any family member or relative who has been diagnosed of heart disease? YES NO

If yes what is the diagnosis?

- Is there anyone in your family and relatives who has lost their lives before the age of 50 from any disease? YES NO

If yes what is the diagnosis of the disease?

- Have you ever been prohibited or restricted from attending sports by any physician because of heart disease? YES NO

If yes what is the diagnosis of the disease?

Physical examination:

- Hearing murmur in the heart
- Pulse features that suggest aortic coarctation
- Presence of the physical stigma of Marfan syndrome
- High blood pressure

On the other hand, European Society of Cardiology (55) suggests resting ECG to be used for appropriateness of sports examination as a routine. It is accepted that ECG is more cost-effective than other tests in detecting cardiac pathologies. The suggestion of European Society of Cardiology to this problem is to essentially and necessarily do detailed anamnesis, physical examination and resting ECG examinations. If a cardiovascular disease is suspected after these examinations than application of further examination methods are suggested. In our clinic practice, we have recommended that personal and family history, physical examination, rest ECG, and also exercise testing with 12-lead monitoring should be routinely used for evaluating of asymptomatic competitive athletes because of most of cardiovascular events may develop during or peri-exercise training interval (56) (Fig. 3). Echocardiographic examination is added in routine evaluation program when competitive athletes have any cardiovascular symptom (56) (Fig. 4). Furthermore, duration of repolarization heterogeneity should be measured during exercise testing for detecting high risk subjects (57).

Thiene et al. (52) recommend four tries for preventing of sudden death in the athletes consist of the following:

- Avoid triggers like high intensity exercise and training in athletes with cardiac disease
- Inhibit the onset of arrhythmias with drugs or ablation
- Switch off arrhythmias with defibrillator
- Hinder the recurrence of the disease with genetic counseling and/or therapy.

When determining the health appropriateness of sports within the athlete, family, trainer triangle, it should be primarily thought to not risk the athlete in terms of health. Furthermore, it should be kept in mind that unnecessary prohibition of sports may lead to a problem of loss of cardiovascular effects of exercise on health. Some cases of cardiac deaths in athletes may not be preventable by current practical means; however, the first step of main strategies must be to separate the high-risk athletes who have previous symptoms, a family history of sudden cardiac death at a young age and clinical or electrocardiographic abnormalities (58).

Conclusion

Athletes who are going to get involved in an intense activity, must certainly have a periodic serious systemic examination before and after attending the activity. However, it is not always

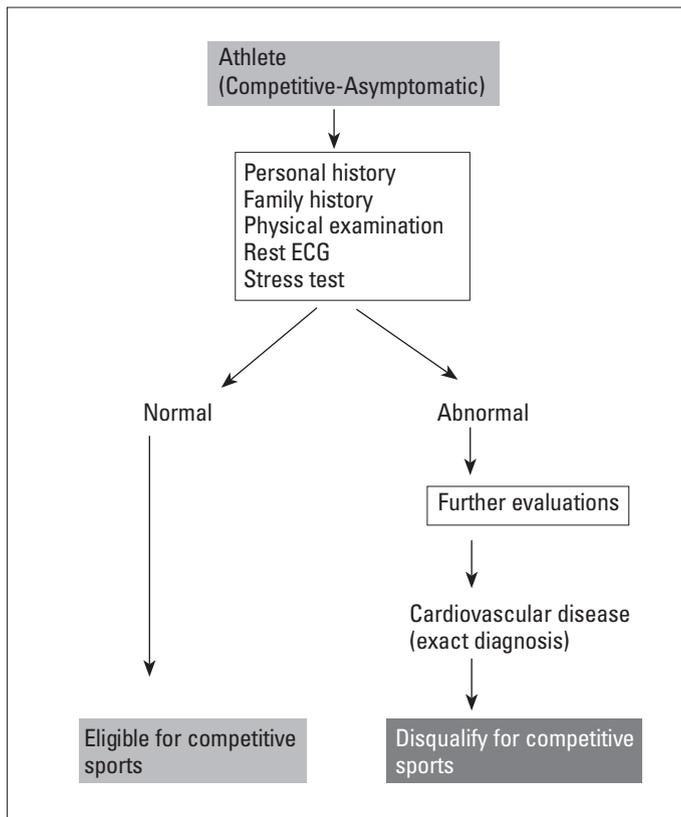


Figure 3. The cardiovascular risk evaluation protocol of competitive athletes without cardiovascular symptoms
ECG - electrocardiogram

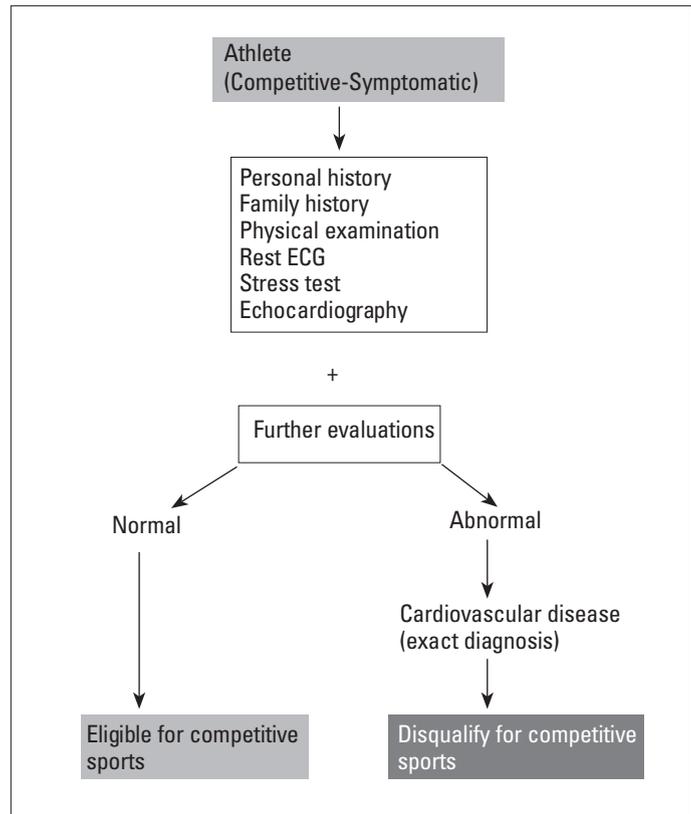


Figure 4. The cardiovascular risk evaluation protocol of competitive athletes with cardiovascular symptoms
ECG - electrocardiogram

easy to diagnose most of the diseases that led to unwanted cardiac events and the most important stage in diagnosis is to suspect. If the standard scanning tests are not sufficient, differential diagnosis has to be made with further examination methods. With some serious cardiac pathology that is detected after these examinations, individuals must certainly be prohibited from attending intense exercise programs and competitive sports.

Conflict of interest: None declared.

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