CHAPTER II

SPORTS CARDIOLOGY

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1.Introduction

Today's sport expects top results from athletes, and in order to achieve great sporting success, athletes must train with extremely high intensity. The loads placed on the athlete require the adaptation of the organism to the efforts, and it includes the adaptation of the cardiovascular, respiratory and primarily the musculoskeletal system. In order to ensure the athlete's health, it is essential that each athlete undergo a detailed health examination that must be holistic and not focused on just one part. A medical examination should determine the athlete's general health and diagnose and examine any pathology if the examination reveals. Since sport does not only bring with it sports injuries in the form of musculoskeletal injuries, which is of course the most common, there can also be a problem with the cardiovascular system either due to heavy loads or if the athlete has a congenital heart defect that has not been previously diagnosed. Therefore, detailed cardiac treatment is very important for athletes in order to preserve the health of athletes and prevent sudden cardiac death, as the worst consequence.

2.Anatomy And Physiology of Heart Work

The structure of the heart

The heart is an organ located in the mediastinum and along with the brain is the most important organ in the human body. It consists of four chambers that perform the function of pumping blood into other organs

and tissues. The heart can be divided into a left heart and a right heart consisting of atrias and ventricles. Between the atria and the ventricles are atrial-ventricular valves that prevent blood from returning from the ventricles to the atria. On the left side of the heart is the bicuspid or mitral valve, named after the bishop's drop "mitral", while on the right side of the heart is the tricuspid valve. The function of the left side of the heart is to collect oxygenated blood that comes from the lungs through the pulmonary vein and flows into the left atrium, then passing through the mitral valve it flows into the left ventricle at the end of which is a large aortic blood vessel. By expelling blood through the aorta, the blood reaches other organs and tissues, which is why the left-sided circulation is called the systemic or large bloodstream. The function of the right side of the heart is to receive deoxygenated blood that enters the right atrium through the superior and inferior vena cava and then passes through the tricuspid valve into the right ventricle, which sends deoxygenated blood to the lungs for re-oxygenation by expelling it through the pulmonary artery. Therefore, the circulation of the right heart is called the pulmonary or small bloodstream.
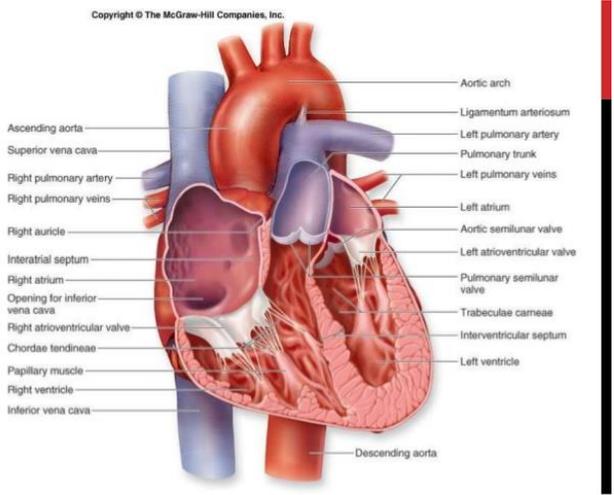

Figure1. Structure of the heart

The heart is made up of three walls, namely: the pericardium (outer layer), the myocardium (middle-muscular layer) and the endocardium (inner layer). The pericardium is a connective sheath that envelops the heart and consists of two leaves. The outer leaf consists of dense connective tissue and is attached to the tendon center of the shield and partly to the spine and large blood vessels coming out of the heart, while the inner leaf of the pericardium consists of a serous part and is attached to the heart surface. Between the inner and outer leaves of the pericardium is

a pericardial cavity containing a small amount of fluid that reduces friction between the leaves. The endocardium, like the pericardium, forms connective tissue, but unlike the pericardium, the endocardium is also covered with epithelial cells. The endocardium coats the entire interior of the heart as well as the heart valves, and continues into the endothelium of the large blood vessels. The myocardium is the middle layer of the heart wall, and at the same time a muscle without which the heart could not perform its basic function. It consists of three types of heart muscle: the atrial muscle, the ventricular muscle, and specialized excitable and conductive muscle fibers. Because their arrangement of actin and myosin fibers is very similar to that in skeletal muscle, the atrial and ventricular muscles contract in the same way as skeletal muscle, but with a much longer phase of contraction. The function of specialized excitable and conductive muscle fibers is the eruption of automatic electrical impulses and the conduction of action potentials through the heart, but their contraction is weak because they contain very few contractile myofibrils.

Electrophysiology of cardiac work

The work of the heart is manifested through the work of the heart muscle which "pumps" blood into various organs and tissues, in order to stimulate the myocardium to work at all, it needs an electrical impulse. This electrical impulse is created in the sinuatrial node or the so-called. SA-node, located in the posterior wall of the right atrium. The cells of the sinus node have the ability to generate electrical impulses, so they generate them rhythmically 60-80 times a minute in healthy people. Because the sinuatrial node is located in the right atrium, its cells are directly connected to the muscle fibers of the left and right atria and thus cause their contraction. An electrical impulse produced by the sinuatrial node by thin fibers reaches the interventricular septum near the right atrium where the atrioventricular node or AV node is located. The thin fibers between these two nodes conduct the impulse very slowly, so the impulses are retained and come to the ventricles only after the systole of both atria. This impulse retention allows the atria to empty normally and the ventricles to fill. The impulse after the atrioventricular node travels through the atrioventricular bundle, clinically called the His bundle to the upper part of the interventricular septum where it divides into left and right branches which then goes to the ventricles and ends up as Purkyni threads causing contraction of both ventricles.
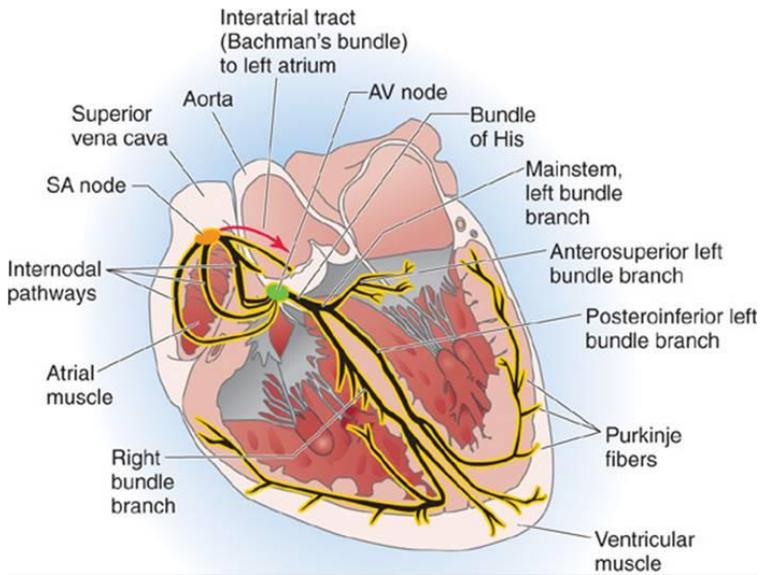
Figure 2. Representation of the electrophysiological system of the heart

The action potential of the heart muscle can be divided into four phases:

0. Depolarization phase - stimulation of the heart cell leads to the opening of voltage sodium channels that allow sodium to enter the cell and thus the depolarization of the cell itself

1. Initial repolarization phase - sodium voltage channels are closed, and potassium channels are opened through which potassium ions exit and thus initiate cell repolarization

2. The plateau-plateau phase of action potentials occurs after the initial repolarization as a result of the closure of potassium channels and the opening of calcium channels, ie reduced output of potassium ions, and increased entry of calcium ions

3. Rapid repolarization phase - includes closing of calcium channels and opening of slow potassium channels, thus ending the plateau phase and starting the phase of returning the membrane potential to the resting level

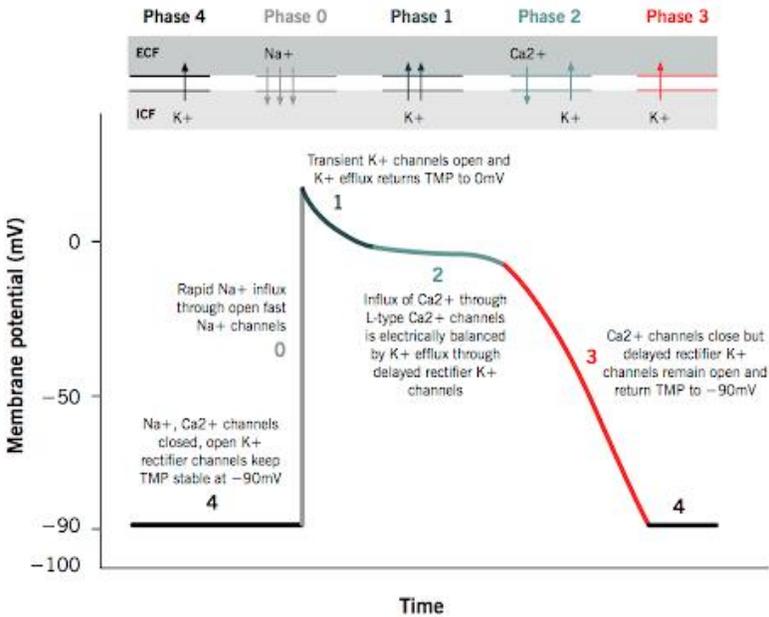4. Phase membrane potential at rest - approximately -90mV

Action potential of cardiac muscles

Figure 3. Phasis of action potential

3. The Role of The Cardiovascular System in Sports Activity

The role of the cardiovascular system in physical activity is based on the supply of nutrients and oxygen to the muscles. During sports activity, the diffusion capacity of the lungs is increased, which is manifested by greater blood flow through the lungs and thus allows faster gas exchange, which is important for biochemical aerobic processes to create ATP, which uses energy to gain muscle work. Thus, the cardiovascular system is the link between pulmonary ventilation and cellular oxygen consumption. A measure that demonstrates the ability to deliver oxygen during muscle work and cardiovascular endurance is oxygen consumption at maximum aerobic metabolism (VO2max).

Adaptation of the cardiovascular system is proportional to muscle requirements. Thus, the higher the intensity of training, the higher the hemodynamic parameters. During rest, 15-20% of the minute volume goes to the muscles, while during intense physical activity this percentage can reach up to 80-90%. With an increase in cardiac output, the heart rate and stroke volume increase, which affect changes in cardiac output. The stroke volume in athletes with high cardiorespiratory abilities is significantly higher at rest and during maximum load when the stroke volume can be up to 200 ml per beat. Of the structural adjustments, the most significant is

left ventricular hypertrophy, which is most exposed to stress and occurs in athletes who engage in sports that require endurance. During exercise, blood is redirected to the areas where it is most needed. In high-endurance training, the contracting muscles utilize 80% or more of the blood flow, while the flow in the liver and kidneys decreases. In contrast to exercise, during rest the muscles consume only 15-20% of the blood flow, while the liver and kidneys utilize half.
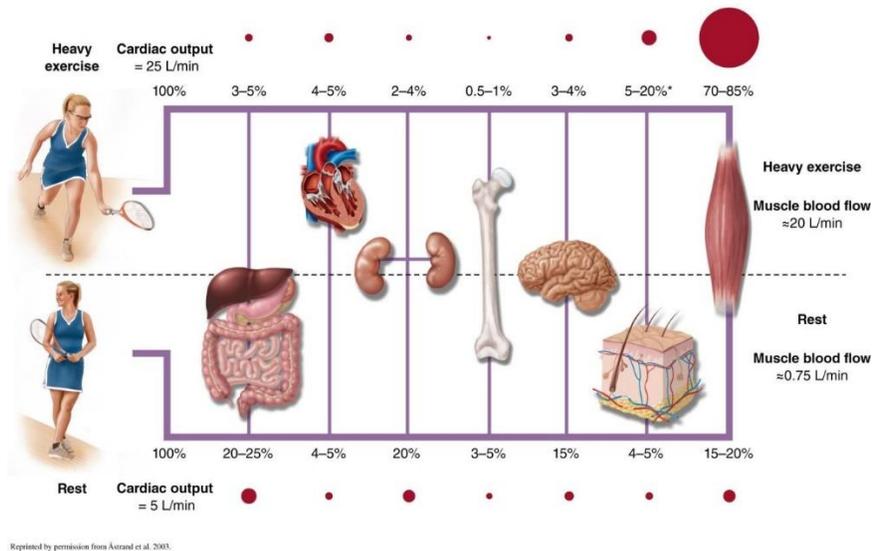

Figure 4. Distribution of blood flow during sports activity

Some studies have shown that hemodynamic parameters, structural, and functional adjustment of the heart can vary depending on the type of sport, which may be dynamic or static. Nowadays, it is difficult to say for a sport that it is predominantly static or dynamic, because athletes use different training approaches to improve their physical fitness. Close to an ideal example for a dynamic sport is long-distance running, while for a static sport an example is bodybuilding and weightlifting.

In athletes who predominantly engage in dynamic sports, an increase in the inner diameter of the left ventricle was observed by about 10%, while in athletes engaged in static sports or strength exercises, an increase of 2.5% was observed. In sports that required dynamic and static exercises and long-term training such as cycling, the wall thickness of the left ventricle was significantly more pronounced than in runners. The probable interpretation of these results is that the development of so-called eccentric or concentric left ventricular hypertrophy according to the type of sport cannot be considered an absolute or dichotomous concept, because training regimes and sports activities are not exclusively dynamic or static.

4. Cardiological Examination and Diagnosis in Athletes

History and examination
Cardiac examination of athletes places emphasis on the prevention of sudden cardiac death which is the leading cause of death in younger people (<35) during physical exertion. Therefore, cardiac treatment is important to identify undiagnosed conditions and adequately care for conditions that are already known. Guidelines for the cardiac examination of athletes were issued by the American Heart Association and European Society of Cardiology. After anamnesis and physical examination we usually perform ECG and echocardiography.

Electrocardiogram (ECG)
An electrocardiogram is a non-invasive cardiac diagnostic test that records the electrical activity of the heart. Athletes often experience changes in the ECG due to repeated exertion that causes electrical and structural adjustments that may overlap or suggest cardiovascular disease. Therefore, it is important to properly record and interpret the ECG findings to see if it is a pathological or normal physiological process. If the ECG finding is misinterpreted the athlete may be disqualified and may be referred to expensive diagnostic procedures that are unnecessary. Contrary to this situation, a misinterpretation of the ECG can replace the pathological process with a physiological one and put the athlete at great risk of a heart attack. Physiological adjustments of the autonomic nervous system of the heart, most commonly caused by increased vagal tone, cause changes in the athlete's electrocardiogram. Approximately 80% of athletes experience changes such as early repolarization, sinus bradycardia, or first-degree atrioventricular block. For example, athletes in endurance sports such as cycling and rowing have been shown to develop significantly greater changes on the electrocardiogram in the form of higher QRS voltage or sinus bradycardia compared to sports that do not require endurance but require strength and speed. Such changes on the electrocardiogram are due to higher heart capacity and left ventricular thickness. Most cardiac conditions that can lead to sudden cardiac death in young athletes are often asymptomatic and therefore not detected in time. The Italian screening experience, which lasted 25 years, concluded that in addition to physical examination of athletes, electrocardiogram recording should be included because it has significant value in detecting cardiac conditions and diseases that can lead to sudden cardiac death in asymptomatic athletes.

Echocardiography
Echocardiography or ultrasound of the heart is a non-invasive diagnostic imaging method used to detect morphological changes in the

heart. In sports cardiology, echocardiography can complement cardiac examination in two areas: screening before participating in sports and analysis of cardiac adjustments induced by training. The usefulness of echocardiography has been demonstrated in the differential diagnosis of various diseases that can cause sudden cardiac death, with special emphasis placed on left and right ventricular analysis. After all, some changes in the heart can be missed by physical examination and electrocardiogram, and can be easily diagnosed by echocardiography. The most common changes diagnosed by echocardiographic findings are divided into two groups, namely: physiological structural and functionally adaptive cardiac changes. Therefore, echocardiography can distinguish between physiological changes caused by training and pathological changes such as hypertrophic cardiomyopathy that can be the cause of sudden cardiac death. One 2013 study compared Doppler echocardiographic findings in soccer players, long-distance runners, and cyclists with the findings of the non-sports population, and the purpose of the study was to assess cardiac structure and function. The findings showed that athletes from these sports had a larger left atrial volume, left ventricular thickness, and diastolic diameter of the right and left ventricles as opposed to non-athletes. Among athletes, cyclists have been shown to have greater structural changes than footballers and runners. The systolic function of the left atrium recorded by Doppler technique did not show significant differences in athletes, while the systolic function of the right ventricle was higher in cyclists and football players compared to runners.

5. Cardiological Conditions and Diseases in Athletes

Sports heart

Sports heart is one of the scientific issues that sparked discussion among scientists for the reason that one part of them believed that sports heart is healthy heart, ie the heart adapted to constant intense loads, while the other part considered it to be cardiac pathology. But in 1889, the Swedish physician S. Henschen described hypertrophic changes in the heart in Nordic skiers caused by high load. Athletic heart is a physiological condition found in athletes who train at high intensity for more than five days a week for a minimum of one hour. Sports heart involves hypertrophy of the heart itself, as well as enlargement of the heart cavities and thickening of the myocardial wall leading to an increase in cardiac output which consequently results in a decrease in resting heart rate. Such adaptation of the heart to exertion allows for a greater minute volume which is very important for the supply of skeletal muscle with nutrients and oxygen, i.e. the more minute volume the larger skeletal muscle will receive more nutrients and oxygen. The sports heart can easily be confused with a pathological process and it is therefore important to recognize

whether it is physiological hypertrophy or pathological. If the sports heart is replaced with pathological hypertrophy, it can cause severe consequences in athletes who continue with high-intensity training. Therefore, detailed cardiac treatment of athletes is important to prevent side effects.

The diagnosis of sports heart is made on the basis of anamnestic data, clinical examination, electrocardiography (ECG) and other specific diagnostic tests if necessary in order to complete the clinical examination and remove the suspicion of a pathological condition. The sports heart is often asymptomatic, and is therefore only detected during routine examinations of athletes. Some athletes, however, develop some symptoms that are associated with adaptive changes in the heart, and may even suggest some heart disorder.

Cardiomyopathies

Cardiomyopathies are diseases of the heart muscle that can be inherited or acquired. According to the classification of the European Society of Cardiology, cardiomyopathies are classified into several groups:

1. Hypertrophic cardiomyopathy
2. Dilated cardiomyopathy
3. Arrhythmogenic dysplasia of the right ventricle
4. Restrictive cardiomyopathy
5. Unclassified cardiomyopathies

In sports cardiology, hypertrophic cardiomyopathy and arrhythmogenic dysplasia of the right ventricle are of the greatest importance, because they are the largest cause of death in young athletes.

Hypertrophic cardiomyopathy has been cited in numerous statistical analyzes as the leading cause of sudden cardiac death in athletes under 35 years of age. Left ventricular hypertrophic cardiomyopathy is the most common form of cardiomyopathy in athletes whose diagnosis is very challenging because it should be clearly distinguished whether it is a pathological hypertrophy or a physiological one caused by intense training. Because hypertrophic cardiomyopathy is the most common cause of death in athletes, the American Heart Association and the European Society of Cardiology have issued recommendations banning competitive sports with medium and high dynamic or static loads.

Dilated cardiomyopathy is characterized by dilatation and systolic dysfunction of the left ventricle, and its differential diagnosis can be difficult because a large proportion of athletes engaged in endurance sports show dilatation of the left ventricle. The diagnosis of dilated cardiomyopathy is made on the basis of these indicators: systolic function of the right ventricle with ejection fraction below 45%, right ventricular dysfunction, regional wall irregularities, positive family history, changes in the electrocardiogram in the form of wave inversion or some other

disturbances and tests such as magnetic resonance imaging and echocardiography are also very useful. Athletes who have dilated cardiomyopathy are considered to be at high risk for sudden cardiac death, and according to European and American cardiologist recommendations, such athletes should be excluded from competitive sports activities.

Arrhythmogenic dysplasia of the right ventricle is a disease characterized by the finding of fibrous and fatty tissue in the myocardium of the right ventricle, and can extend to the left ventricle and consequently can cause ventricular tachycardia and sudden cardiac death. It is diagnosed using non-invasive and invasive medical procedures that assess heart rhythm and structure. Treatment is based on the prevention of sudden cardiac death by the use of antiarrhythmic drugs, and in the more severe phase by the use of an implantable cardioverter defibrillator. Athletes with diagnosed arrhythmogenic dysplasia of the right ventricle are recommended to engage in sports of reduced intensity.

Athletes with any diagnosed cardiomyopathy should be excluded from high-intensity competitive sports, and exceptionally some athletes may engage in low-intensity sports such as golf or billiards. The ban on competition also applies to asymptomatic athletes who have been diagnosed with any form of cardiomyopathy, regardless of whether they use some form of treatment or not.

Myocarditis
Myocarditis means inflammation of the heart muscle, which can later lead to heart dysfunction and arrhythmias. The most common type of myocarditis in the world is viral myocarditis, while in young athletes the presence of toxins such as catecholamines and cocaine should be examined first during the examination. Although myocarditis is not very common unlike other heart diseases, it accounts for 5-22% of sudden cardiac deaths in younger athletes. It is believed that the onset of myocarditis first involves suppression of the athlete's body in response to intense physical exertion during training. Excessive inflammatory response of the body can lead to deterioration of the myocardium and skeletal muscles and catabolism due to lack of necessary energy.

The cause of myocarditis in athletes can be different depending on the type of sport in which the athlete is engaged. For example, contact sports such as boxing develop a higher risk of transmitting droplet infections, orienteering sports such as orienteering run have a higher risk of developing an infection such as ticks. According to the recommendations of the European Society for Preventive Cardiology (EAPC) and the American Heart Association (AHA), athletes with myocarditis should be excluded from competitive and amateur sports activities for six months. After six months, athletes with myocarditis should be evaluated before continuing with sports activities that must

initially be weaker. It is important for professional athletes to state the duration, intensity, frequency and mode of activity, as they have a different perception of lower physical activity as opposed to the rest of the population.
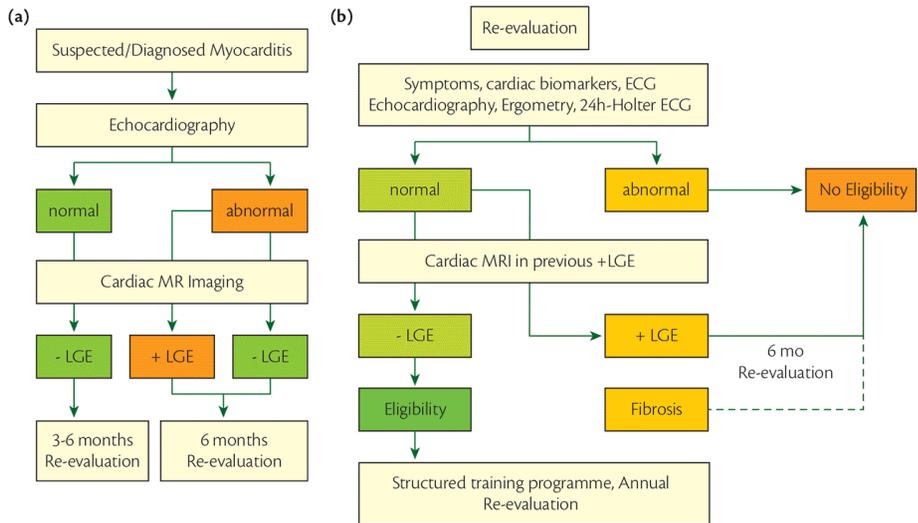

(a)
Suspected/Diagnosed Myocarditis → Echocardiography → normal / abnormal → Cardiac MR Imaging → - LGE / + LGE / - LGE → 3-6 months Re-evaluation / 6 months Re-evaluation

(b)
Re-evaluation → Symptoms, cardiac biomarkers, ECG Echocardiography, Ergometry, 24h-Holter ECG → normal / abnormal → No Eligibility
normal → Cardiac MRI in previous +LGE → - LGE / + LGE → Eligibility / Fibrosis → Structured training programme, Annual Re-evaluation
6 mo Re-evaluation

Figure 5. Assessment of myocarditis (a) and ability to play sports after 6 months (b)

Congenital heart defects

Congenital heart disease is a congenital defect, which can limit some athletes from playing sports. The most common congenital heart defects include ventricular septal defect, atrial septal defect, and open ductus arteriosus. Since there is no demonstrative evidence that these forms of heart defects cause sudden cardiac death, if children with these congenital heart defects do not have significant changes in hemodynamic parameters, they can continue to play sports. If congenital heart defect is accompanied by pulmonary hypertension that causes significant hemodynamic changes, acute symptoms may occur that reduce the ability to exercise, lead to chest pain, cause syncope or arrhythmias, and may result in sudden cardiac death. In untreated ventricular septal defect, it is recommended that athletes with a small ventricular septal defect with normal heart size and without pulmonary hypertension can participate in all sports, and in the case of a large defect, surgical intervention is required. After treatment for 3-6 months, asymptomatic athletes may engage in sport if pulmonary hypertension, atrial or ventricular tachyarrhythmia, or myocardial dysfunction are ruled out. For untreated atrial septal defect, it is recommended that in minor defects (<6 mm) with normal right heart volume and confirmed pulmonary hypertension, they should not

participate in competitive sports, while in major defects without pulmonary hypertension, sports are recommended. If the treatment is performed, athletes without pulmonary hypertension, arrhythmia or myocardial dysfunction can continue playing sports for 3-6 months after the intervention. Athletes with small untreated ductus arteriosus, normal left heart dimension and pulmonary artery pressure can play sports, and in moderate or large defects with persistent pulmonary hypertension and enlarged left ventricle, temporary exclusion from sports is recommended until the defect closes.

Valve diseases

Nowadays, the incidence of heart valve disease, the so-called valvular disease is on the rise in both the elderly and the younger. Changes that occur in the heart valve caused by a pathological process are stenosis of the natural flow of blood and a disorder in the one-way flow of blood through the heart valve.

The most common cause of mitral stenosis is rheumatic fever, which in turn causes fibrosis and calcification of the heart valves, and the tendon cords shorten and thicken. The hemodynamic consequences that occur due to mitral stenosis cause a decrease in blood flow and thus minute volume and an increase in left atrial pressure in diastole to increase minute volume. Due to the insufficiency of the mitral valve, the phenomenon of mitral regurgitation occurs, which means the return of blood from the ventricles to the atria.

Aortic stenosis can occur below, above or on the valve itself, so we distinguish between supvalvular, supravalvular and valvular aortic stenosis. The most common aortic stenosis is valvular, which can be the result of rheumatic fever, atherosclerotic changes and degenerative calcifying process, and the basic hemodynamic feature is the difference between the pressures of the left ventricle and the aorta. Aortic insufficiency is characterized by damage to the valves, so the valves leak blood back to the left ventricle. The most common causes of aortic insufficiency include infectious endocarditis and aortic prosthesis dysfunction, while less common causes may be arterial hypertension, trauma, or aortic dissection. Athletes suspected of having valvular disease should have an echocardiographic examination to see the function of the heart valves and the flow of blood through them. Those athletes who have mild to moderate mitral and aortic regurgitation without structural changes in the form of atrial or ventricular dilatation have no barriers to further exercise, while severe forms of regurgitation require exclusion from competitive sports and corrective surgical intervention.

Athletes with conduction disabilities

Sinus rhythm disorder in athletes includes asymptomatic bradycardia whose frequency is usually between 40-50 beats. Bradycardia

occurs in athletes due to neurovegetative changes caused by intense training, and especially in sports that require high aerobic capacity such as cycling and marathon. Severe bradycardia is rare, but can be found in older athletes over the age of 40 during the night. Bradycardia occurs physiologically in athletes in response to exertion, so it is considered a benign phenomenon if it is asymptomatic and if the heart rate increases normally during sports activity.

Early repolarization is considered by athletes to be a benign phenomenon caused by increased vagal tone as a result of intense training. Athletes who have asymptomatic early repolarization but an increased risk detected by a family history need to undergo additional cardiac treatment to rule out J wave syndrome. Since intense training leaves changes on the autonomic nervous system and thus on the primary ion channels, the fact cannot be ruled out that early repolarization can cause arrhythmias. Most athletes develop early repolarization with a growing ST segment, but the prevalence of malignant early repolarization has also increased including subtypes that may increase the risk of arrhythmias, such as inferior J waves and J waves that may be accompanied by a horizontal or descending ST segment. A study conducted for 90 days in rowers and football players showed that the prevalence of early repolarization increased in endurance sports, ie rowers, and remained the same in football players and strength sports.

In addition to sinus bradycardia and early repolarization, first-degree atrioventricular block is one of the most common changes in an athlete's electrocardiogram. First-degree atrioventricular block is often seen in athletes, and is marked by PQ prolongation at the electrocardiogram finding and leaves no specific consequences. If the PQ interval on the electrocardiogram is extended by more than 0.3 seconds and the QRS complex is altered, additional diagnostic processing such as ergometry, 24-hour holter electrocardiogram, and echocardiography should be performed to suspect possible structural heart disease. Asymptomatic athletes who do not experience a worsening of the PQ interval during exercise can engage in all sports, and if structural heart disease is detected during additional diagnostic processing then sports activity depends on the disease itself.

The second stage block, the so-called The Mobitz I type or Wechenbach block is often found in highly trained athletes due to increased vagal tone and does not carry significant consequences for the athlete. The diagnostics used to detect this condition are an electrocardiogram, a 24-hour holter electrocardiogram and ergometry, and some athletes also use a heart rate monitor during training. The electrocardiogram is characterized by a prolonged PR interval, a blocked P-wave, and a discharged QRS complex. If a branch block is diagnosed in the electrocardiogram finding, an electrophysiological examination should be performed to detect the level of conduction disturbance. Asymptomatic

athletes who have a structurally healthy heart or some of the structural defects of the heart, and with stagnant or improved atrioventricular conduction are allowed to engage in all types of sports. Whereas in asymptomatic athletes who develop a first-degree block during or after exercise, the intensity of training should be reduced, and such athletes should be additionally electrophysiologically examined.

A second-degree Mobitz II-type block is characterized by a conduction disturbance below the level of the atrioventricular node, usually at the level of the His bundle. It is recognized on the electrocardiogram by intermittent non-conducting P-waves. It occurs very rarely in athletes, but if an athlete has this conduction disorder, he needs the implantation of a permanent pacemaker before sports activities.

Second Degree AV Block Mobitz Type 2

Lead II

25mm/sec 10mm/mV   © Jason Winter 2016 - @ ECG Educator

Figure 6. AV block second degree Mobitz type 2

Third-degree block or total atrioventricular block denotes an abnormal heart rhythm resulting from impaired conduction through the atrioventricular block. That is why the atria and ventricles knock separately, because the atria are led by the sinuatrial node, and the ventricles by one of the distal latent conductors, which later cause atrioventricular dissociation. Athletes who have a third-degree block should be excluded from competitive sports and need a pacemaker implant.

Arrhythmias

Most arrhythmias that occur in athletes occur due to undiagnosed cardiac structural changes and canalopathy. Signs of arrhythmia include morphological changes and changes in the electrocardiogram, and symptoms are often overlaid by morphological changes caused by training. The most significant arrhythmias are divided into supraventricular tachycardias and ventricular extrasystoles and ventricular tachycardias. One of the most common arrhythmias that occurs in athletes is atrial fibrillation, which belongs to the group of supraventricular tachycardias and is often associated with sports that require endurance.

The mechanism of atrial fibrillation is unclear, but it is assumed that there are three causes, namely a specific trigger that includes atrial ectopia,

sports supplements and illicit drug use, then a substrate caused by genetic predisposition, inflammation, fibrosis and heart remodeling and a modulator that includes autonomic activation, electrolyte disturbance, and gastroesophageal reflux. Treatment of atrial fibrillation initially requires a reduction in physical activity because temporary interruption can reduce or prevent recurrence of atrial fibrillation. Some athletes also need treatment with antiarrhythmics, but speed control agents, such as beta-blockers or calcium channel blockers, that may impair the athlete's performance should be avoided.

The second group of arrhythmias includes monomorphic or catecholaminergic polymorphic ventricular tachycardia and ventricular extrasystoles. Monomorphic ventricular tachycardia is a fairly common occurrence in athletes that most commonly occurs as benign as part of physiological changes occurring in the athlete's heart, however it can also occur as idiopathic and may not be associated with athlete status. Monomorphic ventricular tachycardia is often asymptomatic and is usually detected by electrocardiogram or cardiac treatment of symptoms, most often palpitations. Although monomorphic ventricular tachycardia is most often benign, it is important to identify all factors that may indicate structural heart disease and to reduce athletic activity to prevent sudden cardiac death.

Catecholamineric polymorphic ventricular tachycardia is rare and is often associated with hypertrophic cardiomyopathy and coronary anomalies and has been identified as a cause of sudden cardiac death. It most commonly occurs as syncope during emotional stress or intense physical activity in the presence of palpitations. All forms of ventricular tachycardia are treated with beta-blockers, and athletes are advised to discontinue competitive sports.

Ventricular extrasystoles are relatively common in athletes, and some studies have even shown that the incidence of ventricular extrasystoles is higher in highly trained athletes compared to the population engaged in recreational sports. Ventricular extrasystoles are not considered a risk for ventricular tachycardia or sudden cardiac death, and they occur in both structural heart disease and those who do not have structural heart disease. In a study by Biffi and colleagues who investigated the incidence of ventricular extrasystoles in athletes who had about 2,000 ventricular extrasystoles in 24 hours, it was shown that deconditioning athletes within 3 months can reduce the number of ventricular extrasystoles in 24 hours.

Heart comfort

Heart coma or heart agitation in athletes most commonly occurs in ball sports such as baseball, and can be caused by a blow to the ball or part of the body in the left chest area. When struck in the left side of the chest,

the myocardium deforms and thus causes ventricular fibrillation, which in turn causes sudden cardiac death despite the normal structure of the heart and thus represents an emergency. If the stroke occurs during ventricular repolarization, the change on the electrocardiogram occurs in the form of a high T-wave, and if the stroke occurs later it is likely to result in an elevation of the ST segment, transient tostal heart block, or left branch block. Heart convulsions, although very rare with cardiomyopathies, are the leading cause of death, especially in childhood athletes. Therefore, a register of heart commotion has been launched in the United States, with an average reported age of 15, and a very small number of athletes are over the age of 20, which may be the cause of thinner chest wall thickness in children compared to adults. Since cardiac compression is characterized by ventricular fibrillation, the first step in treatment should be directed towards early defibrillation and cardiac massage, and if resuscitation is continued, medications to increase coronary perfusion flow and ventilation accessories should be introduced. If dull cardiac trauma has occurred that has caused dysrhythmia, treatment is based on stabilizing the electrical activity of the heart. Survival rates after cardiac compression improved, thanks to timely and early outpatient resuscitation for both other cardiac arrest and cardiac compression.
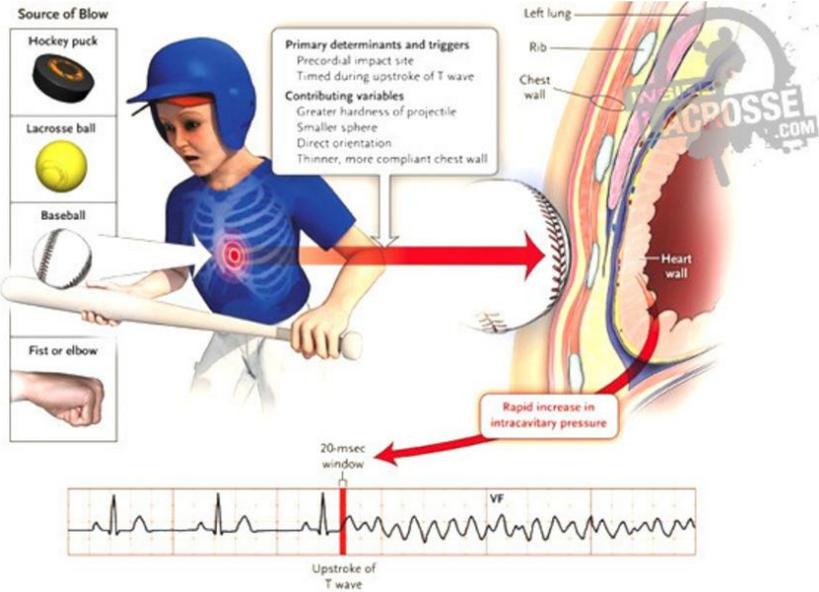

Figure 6: Heart confort and mechanism of injury

6.Sudden Heart Death in Athletes

Sudden cardiac death is the most common medical cause of athlete death, although its very rare occurrence in athletes leaves long-lasting emotional and social consequences on the athlete's environment.

Therefore, considerable efforts are being made to identify and understand as early as possible the causes that lead to sudden cardiac death. The definition of sudden cardiac death in athletes varies because some incidence estimates include only death during exertion or shortly after exertion, most often within one hour, while other estimates include death of athletes outside of exertion.

The most common causes of sudden cardiac death in athletes are due to:

Congenital diseases :
Structurally altered heart
Hypertrophic cardiomyopathy
Congenital long QT syndrome
Arrhythmogenic dysplasia of the right ventricle
Catecholaminergic polymorphic ventricular tachycardia
Dilated cardiomyopathy
Wolf-Parkinson-White syndrome
Brugada syndrome
Congenital anomalies of the coronary arteries
Ionic canalopathy
Aorthopathy
Valvular diseases

Acquired diseases
Coronary atherosclerotic disease
Heart convulsions
Kawasaki disease
Acquired prolonged QT wave
Myocarditis
Other environmental factors and substances

Since 1966, 1101 cases of sudden cardiac death have been reported in athletes under 35 years of age. The largest share is carried by cardiovascular diseases, of which 50% were caused by cardiomyopathies and congenital heart diseases, and 10% by atherosclerotic altered coronary vessels. In young athletes, the fact is that 90% of cases of sudden cardiac death occur in training or competitions that can be caused by physical exhaustion that can consequently cause pathological arrhythmias. The incidence of sudden cardiac death depends on the athlete's general health and the type of sport he is engaged in. In older athletes, the most common cause of sudden cardiac death is atherosclerotic altered coronary blood vessels, while in younger athletes, the most common cause is congenital heart defects. It is very difficult to detect a quality screening system that will be able to identify all the factors that can cause sudden cardiac death in athletes, so today there is increased access to automated external

defibrillators and increased education as well as the importance of community-based cardiopulmonary resuscitation. prevented sudden cardiac deaths on sports fields.

7.Conclusion

Nowadays, when medicine has advanced tremendously, it is possible to diagnose and treat almost all cardiovascular diseases. The vast majority of young athletes are asymptomatic, so a detailed personal and family history is very important, which is the first step towards the diagnosis of heart disease, then a physical examination and electrocardiogram, which are the basis of diagnosis, but if necessary, athletes can be sent for additional cardiac treatment. on the diagnosis and general condition of the athlete, a decision is made on whether the athlete can continue to play sports or not. It is far better to detect heart disease as soon as possible in order to start treatment on time, and thus reduce the possibility of sudden cardiac death. However, some heart diseases require athletes to stop playing sports, which can cause a negative emotional reaction in athletes, but the health of the athlete must come first. If an athlete is diagnosed with one of the inherited heart diseases, a life can potentially be saved for a close family member.

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