Structural changes in training aerobic soccer players

Abstract

The study aims to know the cardiovascular physiological adaptive response of players under rigorous training and long. The tests are carried out through non-invasive cardiology procedures, such as electrocardiography and echocardiography structural and functional changes that occur at heart level, in addition to obtaining data on the potential performance, adaptive responsiveness heart, physiologically or pathologically were evaluated and determine whether there is underlying pathology, congenital or acquired, which can be asymptomatic cause of sudden death.1

Keywords: pathology, lung foci, cardiology, heart, electrocardiogram

Introduction

Adaptation of the human heart to fitness has been a subject of medical-scientific interest during the last 100 years. The pioneer was the Swedish doctor Henschen in 1899 showed cardiac enlargement in skiers “crosscountry” using heart percussion and becoming the first researcher to describe the “athlete’s heart”.

Later, X-ray and electrocardiogram, allowed the advance knowledge of cardiac training adaptations; but it was with the advent of echocardiography in the 70s, which was a new and important impetus in this area of research.

Echocardiography in M-mode and have been used by numerous researchers in the field of sports medicine to study the cardiovascular changes produced by physical training of long duration and high intensity. Those investigations described in athletes’ enlarged ventricular cavities, a wall thickness and increased left ventricular mass.1–6

These anatomical findings explained by different theories, some related to physical training, as hemodynamic overload and / or endocrine factors, and other non-training, such as genetic and/or environmental influence.

The greater thickness of the ventricular walls is unobtrusive, but in some athletes, this thickness can be significantly higher generating a physiological hypertrophy in athletes. Previous studies have been developed with samples from patients, not athletes, relatively small and homogeneous size is counteracted when a population of athletes sports performance and very heterogeneous, which as patients have significantly different and different values of the limits taken normal physiological hypertrophy.4,5,6

The problem

From past and now, some researchers discovered that the rigorous physical training produces physiological changes of the whole organism. There are physiological responses that accompany acute exercise and chronic cardiovascular adaptations that occur in physical conditioning. Cardiovascular adaptations to physical training usually are not very obvious and sometimes are related with increases in size and cavitary parietal thicknesses.

The assessment of the athlete’s heart has been considered a challenge for the scientific community to consider a tendency as an example of a very effective and healthy physiological adaptation, which counteracts with another trend that consider it a disease, or at least in areas bordering on the pathological.

The review of the literature on anatomical data in the athlete’s heart research indicates a gap on the subject. The literature reviewed on these cases had been presented as more noticeable finding the presence of cardiac hypertrophy by physical exercise; which differs from the perception of some pathologists who did not accept such a statement.

The heart of the athlete trained on aerobic exercise has a relative correlation into body weight and larger hearts and shows the relation between maximum oxygen uptake and the practice in athletic discipline. It is estimated that the average cardiac output in relation to weight in the male is about 11ml/kg body weight, while in women is less in absolute and relative terms, 10ml/kg body weight.

Sports practice produce high performance adaptations in the cardiovascular system manifested through various electrocardiographic and Eco cartographic clinical changes, and can be detect in consideration of the athlete heart conditions. Cardiologist sport is serving a very important role not only in controlling the athlete with heart disease, but health assessments and aptitude for the sport. In this way the sport cardiologist helps to team trainers to understanding the various processes involved in preparation and incentives to maximize performance needed in different metabolic professional areas.

Changes in the athlete’s heart are manifested as a result of morphological, functional and autonomic nervous system changes. The electrocardiogram may present rhythm disorders, atrioventricular and intraventricular conduction, QRS voltage increases, and various alterations in ventricular repolarization. Morphological changes detected by echocardiography usually are not very obvious and sometimes are related with increases in size and cavitary parietal thicknesses.

Presentation of clinical, electrical and echocardiographic examination athlete findings could lead to diagnostic confusion with pathological conditions. The lack of uniformity of these changes in the same age group athletes with similar levels of training suggests no longer rule out the hypothesis of the involvement of genetic factors involved in its appearance.

In our society it is believed that the physical qualities of athletes are a reflection of their better health. However, sporadic and unexpected an athlete suddenly dies during training or competition, causing great concern among medical officials, coaches, and even among athletes.
and their families. In addition, the progressive increase in the number of practitioners of physical exercise increases the demand for accurate information about the human body aerobic conditions to practice certain sports with the least possible risk.

This emerging approach emphasizes the importance of medical examination prior to the competition as a method to identify athletes with the greatest potential risk and make recommendations about the level of specificity of the tests to be included in each case.

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Sport activity induces a number of morphological and functional in the human heart directly related to the type, duration and intensity of training adaptations, and with years of sports. Its clinical expression depends on genetic, metabolic and largely on the type of training factors.15–16

In those sports in which the predominant dynamic exercise involving large muscle mass, the O2 supply to the muscles is active and is made primarily from aerobic metabolism and therefore results in a significant increase in O2 consumption. This volume overload conditions on the left ventricle and, therefore, predominantly a physiological hypertrophy eccentric type, with increased myocardial mass and ventricular dimension.

By contrast, sports predominance of static or explosive exercise and anaerobic energy demand mainly with barely increased O2 consumption and cause few changes in cardiac morphology. In these sports, hemodynamic changes related with fluctuations in blood pressure.

According to these considerations, the sports could classified under its dynamic and static components because they determine the type of cardiovascular demand and the necessary fitness to practice specific sporting activities.

Research objectives

The research purpose are assessing structural changes in aerobic training soccer players at Club de Futbol Loteria del Tachira in the Andean region of Venezuela. The specific purposes’ are: a) assess the cardiology high profile sportsman competition; b) recognized by medical history, physical examination, ECG and echocardiogram possible cardiovascular risk in our athletes; c) diagnosing the presence of ventricular hypertrophy in our athletes; d) recognize any illness, injury, or heart disease that may constitute a vital risk to the athlete; e) identify potential cardiovascular risk factors involved in sudden death in athletes in our study; f) discarding the presence of structural heart disease congenital.

Theoretical framework

History of the problem

The concept of exercise physiology include the study of changes in structures and bodily functions when exposed to acute or chronic stress of physical activity. Sports physiology applies the concept of exercise physiology training an athlete and improving physical ability, and sports cardiology field studies and heart disease aspects related to physical activity and sport.

It is for this reason that sports cardiology relates to clinical cardiology and in particular the differential diagnosis of cardiovascular changes produced by the exercise and heart disease.

This area of medicine began to develop in 1899 with work published by the Swedish physician Henschen, inspired by the first Olympic Games of the modern era in Greece (1896). Subsequently, the European School expands its research development labs exercise physiology in Germany, Italy and France later linking the development of research centers in North America.

With the advent of X-ray and electrocardiogram in the nineteenth century, the possibilities of studies for the advancement of knowledge about heart adaptations and training, improved with the emergence of the mono-dimensional echocardiography in the 70s extended in XX century. This scientific contribution was a major impetus in this area of research, with emphasis on the study of the left ventricular cavity. The subsequent introduction of two-dimensional echocardiography and MRI showed the athlete’s heart enlargement of all heart chambers and great vessels.17–19

The left ventricle shows an increase in all internal dimensions accompanied by a parallel increase in wall thickness to decrease wall stress (Laplace law). The resulting increase in volume accomplished with an increase of myocardial mass (hypertrophy appropriate) and mass index/volume remains unchanged. This sets up a dialog eccentric hypertrophy, characterized by an increase in number of sarcomeres. The ventricular cavity tends to assume a more rounded shape that, in theory, seems to be a more economical situation of the cardiac work.

Modification of left ventricular geometry depending on the work done by the heart is a derived concept of comparative physiology. A globular form is characteristic of a typical low resistance blood circulation, amphibian animals, in which a modest circumferential shortening mobilizes a high blood volume. A cone shape is typical of a high strength blood circulation, typical heart giraffe, adapted to a pressure overload and allowing tolerate high intracavitary pressure. In man, the shape of a truncated cone is an adaptation of the two previous
forms. By analogy, the left ventricle tends to assume a spheroidal shape in aerobic and/or dynamic athlete, in which the heart works primarily with increased volume.

This observation was the basis for the hypothesis Morganroth and collaborators who, using the monkey dimensional echocardiography were the first to describe two different models of training adaptation of the left ventricle. The first, with eccentric hypertrophy (increased left ventricular diastolic dimension without increasing the wall thickness), characteristic of subjects who practice aerobic and/or dynamic sports (runner). The second, with concentric hypertrophy (thickening of the septum and LV posterior wall), static characteristic of those who practice sports or strength (weights launchers and fighters).

This hypothesis has not received unanimous consensus from researchers. Some denied its validity, arguing that there is a single model of adaptation left ventricular hypertrophy characterized by body structures provided to the subject and intensity of training, but not his type. The distribution of hypertrophy would be unimodal, with increased left ventricular mass increased in the dynamic and minor athlete in the isometric athlete, in contrast to the bimodal distribution of hypertrophy proposed by Mongaroth.1-5

This controversy may have originated in the way echocardiographic variables normalized or scaled by different body structures, body surface (BS), lean body weight, etc., since these variables influenced by body size. Cardiac adaptation and particularly the left ventricle to the type of training showed structural modifications of the left ventricle with normal systolic ventricular function index (fractional shortening, velocity of circumferential shortening and ejection fraction).

All these advances in the understanding of the athlete’s heart have been of considerable value to the clinical cardiologist, demonstrating that cardiac physiological adaptation produces a mass/volume constant ratio. The opposite occurs in pathological Hypertrophic Cardiomyopathy, where volume overload produces a mass ratio/volume in exceeding unit (inadequate hypertrophy).

Athlete bradycardia is a common finding in trained athletes and is associated with an increased stroke volume. The origin remains in dispute, initially attributed to increased vagal tone or vagotonia, normal or decreased sympathetic tone. Badeer then hypothesizes vagal hypervagotomy relative and bradycardia would be due to a marked reduction of sympathetic tone with little or no variation in vagal tone.3,4

Bradyarrhythmia associated with a reduction in wall stress (Laplace law), results in decreased myocardial O2 consumption and leads to a high cardiovascular performance.

The above observations allow clearly differentiating between the physiological heart hypertrophy athlete and pathological hypertrophy, and considering physical activity as a therapeutic treatment for hypertension and ischemic heart disease. This provides a new view of cardiovascular physiology athlete and an improvement in the differential clinical diagnosis with pathological processes. Athlete’s heart is a beneficial adaptation produced by training, increases physical fitness and reduces dependency in old age.

Research background

Athlete ventricular hypertrophy, Faculty of Health Sciences, National University of Catamarca.-Argentina. The research demonstrate that there is a physiologic hypertrophy in athletes and probably an enlarged left ventricular cavity in response to hemodynamic overload imposed by physical training. This perspective is the emphasis for research as it considers functional and structural aspects to make specifically evaluations in athlete’s heart.15

Hypertrophy Left Ventricular Remodeling of Elite Athletes in Long Term After deconditioning. Circulation of the Institute of Sports Sciences in Rome. In this study, the authors wanted to show that intensive athletic conditioning long-term clinical consequences associated with left ventricular hypertrophy expressed, and that these changes are benign and physiological nature. The contribution to research is part of the methodology used by the authors, which based on two types of research: qualitative and quantitative addition to the technique for data analyses.21

Athlete’s Heart. Clinical, Electrocardiographic and Echocardiographic. Cardiovascular Prevention and Rehabilitation Division of the Institute of Cardiology and Cardiovascular Surgery and Sports Medicine Institute of Argentine Soccer Players. This research shows the most frequent findings in the cardiologic examination of the athlete, their prevalence and clinical significance. They note that in addition to considering cardiologists control the athlete with heart disease must continually assess health and fitness to specific disciplines to understand the different processes of training and incentives needed to maximize performance in metabolic areas of the player.22

Evaluation of structural and functional changes of the heart as effect of physical training. Personal Special Force units Infantry Navy of Peru (Naval Medical Center CMST). The statistical evaluation of this study involved the comparison of clinical variables, electrocardiographic and echocardiographic two groups, the trained group and the untrained, of which electrocardiographic and echocardiographic parameters took as reference for athletes of this research.23

Clinical Significance of Abnormal Electrocardiographic Patterns in Trained Athletes. Circulation. Sports Science Institute of the National Olympic Committee of Italy. In this study, the majority of athletes (60%) showed completely normal or minor alterations that generally corresponded to physiological cardiac remodeled electrocardiograms.17,23,24

Theoretical bases

Athlete’s heart

The athlete’s heart refers to clinical, electrical and structural changes secondary to myocardial intensive sports. Henschen in 1899 first introduced the term and described by chest percussion, increased heart size in skiers. He concluded that this increase was a physiological mechanism that allows better work for longer.25

Morphological changes of heart are manifested athlete, in most cases, in individuals with high level of training. This condition achieved by stimulation of high intensity exercise, performed on a frequent basis, of longer duration and with the use of types of effort involving large muscle masses. It is difficult to find chamber dilation or hypertrophy of myocardial walls in occasional or low athletes training stimuli.26,27

The athlete are defined as the person involved in an individual sport or organized team and has a frequent confrontation with other subjects with similar condition, because it leads to require specific conditions in your training optimize the functioning of your body.
Since the morphological and functional changes may be different depending on the sport you practice, you need to consider the type and intensity of physical exercise involved in each sport.

An athlete classification field developed according to the mechanical activity and physiological mechanisms of energy production. Regarding the type of movement, they can be classified into: 1) dynamic exercises: generate changes in muscle length and joint movements caused by rhythmic contractions and develop low tension, which were called isotonic, and 2) static exercises: increments in muscle tension with little or no change in muscle length and joint movement (isometric). In most sports both types of effort with varying proportions depending on the activity involved.

Metabolic classification includes aerobic exercise, whose energy comes from oxidative processes, and anaerobic exercise, with energy production from the Phosphagens or anaerobic glycolysis. Energy production by either metabolism depends on the intensity and duration of exercise developed. In addition, most sports demand mixed forms of energy production.

Heart morphological adaptations and modifications stimulation by the autonomic nervous system, are different compared to training with predominantly dynamic and aerobics to those with higher static components (strength) and anaerobic exercise.

Sports training and generate dynamic cardiac changes secondary to volume overload. The increased diastolic volume can lead to an increase of myofibrils in series with enlargement of the cavity. This dilatation of the left ventricular diastolic diameter facilitates greater stroke volume with less shortening and lower energy loss by tension and friction.

The usual practice exercise generates decreased heart rate at rest and at submaximal efforts. This effect attributed to reflection factors and changes in the autonomic nervous system stimulation.

There is an increase of vagal tone in sleep with decreased influence of the sympathetic system and lower levels of circulating catecholamines. During submaximal efforts, stroke volume is greater in the trained subject, with attenuation of the baroreceptor reflex response with smaller increases in heart rate at similar workloads. Maximum heart rate, however, does not change (or does so only in a small proportion) for the training because it is based on the individual’s age.

Sinus bradycardia rest and lower heart rates at submaximal efforts are more common in sports that involve large muscle mass and have metabolic predominantly aerobic. These effects add up to greater stroke volume described to the heart of the athlete more efficient.

To match the increased intramyocardial secondary voltage to increase the radius of the cavity, proportional growth septum thickness and left ventricular free wall generated. Isometric exercises increase the thickness of the ventricular wall. Thus, it is possible to compensate for the increased wall tension by increased intraventricular pressure.

It demonstrated greater thickness of the left ventricular wall cyclists regarding endurance runners and bodybuilders regarding sedentary individuals. However, there are ongoing discussions about the existence of concentric hypertrophy as unique find in highly trained athletes in muscle strength.

Recent studies found no difference in the thickness of the septum and posterior wall of the left ventricle among high-performance athletes in strength and muscular endurance and control individuals. Most sports have mixed training components, which is why changes in the heart of the athlete are too. Soccer, for example, has a high dynamic component, although training exercises involve multiple muscle strength. A soccer match has high rates of exercise with anaerobic metabolism.

The leave the training, the reversal of the changes in the dimensions of the cavity was faster than that produced in the wall thickness. Both training and deconditioning did not influence the rates of ventricular function.

The changes described are not equal in all athletes at levels similar training. There are genetic factors involved in these differences. Heart Gene expression regulated by physiological and pathological stimuli. Physical activity is a major regulator of expression of metabolic and structural in the myocardium and in skeletal muscle through direct effects on the load or through changes in growth factors or proteins catecholamine levels.

The cardiac changes caused by the different loading conditions associated with electrical changes produced, mostly, by the autonomic nervous system changes. Regular training increases vagal tone idle and decreases the level of plasma catecholamines. Correlations exist in athletes between left ventricular mass and increased parameters of heart rate variability.

There are particular characteristics in the examination of the heart of the athlete:

1. **Physical exam:** Peripheral pulses are usually large and normal blood pressure at rest. Possible observation shock tip, even in thin athletes. Palpation found outside the fifth left intercostal space. Cardiac auscultation shows normal noises presence of third and fourth noise in a low proportion of athletes. Often the presence of systolic murmurs in lung foci and the mesocardium. They are protosistólicos, modified with changes in decubitus (usually disappear in the standing position) or breathing movements and can increase with Valsalva maneuver. In dynamic training young athletes were described in 30% to 50% of cases.

2. **Electrocardiographic findings:**
   
a. **Sinus bradycardia:** Sinus bradycardia described in up to 91% of electrocardiograms recorded in endurance athletes, although the prevalence is lower in most series. Bradycardia is secondary to vagal hypertonnia and decreased sympathetic tone. However, after standing at low training heart rates detected in subjects hearts pharmacologically enervated and heart transplanted. It is feasible to think of an intrinsic heart component involved in the mechanism of bradycardia. The decreased sensitivity to catecholamines and changes in neural drive to sinus node suggested as additional mechanisms for the production of sinus bradycardia in the athlete. The incidence of bradycardia and heart rate values, relate to the type of exercise, training stage controls and intensity performed; the highest prevalence observed in endurance athletes. Lower heart rates appear as a result of individual responses of vagal tone training and are not always associated with higher volumes or ventricular dimensions.

b. **Driving conditions:** Vagal tone increased at rest and training response predominates sinus node level. However, in some athletes, this predominance appear at the level of the atrioventricular node and causes the appearance of varying degrees of blockage. While
the increased prevalence of AV conduction disorders in the athlete is attributable to a vagal hypertonus, it is often not possible to differentiate minimal organic changes revealed by an intense workout.

i. **AV block first degree**: The first-degree AV block happens at rest athlete. The AV conduction delay triggered by the effort should suggest a pathological modification.

ii. **AV block second degree Mobitz type I (with periods Wenckebach)**: This blockage may occur sporadically in the same subject and are attributable with the present heart rate at the time of evaluation (its appearance inversely related to sinus rhythm at the time of examination). It is present at rest and disappears with exercise. The degree of blockage is attributable with intensive training and goes to leave.

iii. **AV block degree Mobitz II second**: Their presence should suggest organic conduction disturbance. However, isolated cases reported with night breaks up 6 seconds without organic disease and cardiac conduction normalization abandoning training.

iv. **Complete AV block**: The AV conduction disturbances disappear with exercise if the disorder is secondary to training. The stress test is an invaluable element in the differential diagnosis of pathological states.

v. **Intra-atrial and intraventricular disorders of driving**: It is possible to observe nicks or higher than normal P-wave which could mean disorders secondary to intra-atrial conduction increased vagal tone durations. This inferred from the lack of correlation of these findings with increased dimensional echocardiography headaches. The QRS complex nicked V1 and V2 and on the underside, has attributed to delays secondary to increased muscle mass of the right ventricle ventricular depolarization. The presence of complete right bundle branch block is not related to sports. Neither the complete left bundle branch block nor left hemi blocks.

c. **Rhythms of the atrioventricular junction**: Depression can lead to sinus rhythm AV junction. AV dissociations can be observed by isorhythmic powers between sinus bradycardia and AV junctional rhythm. They usually are intermittent rhythms that appear lower sinus rates at 55 beats per minute. Ventricular escape rhythms are rare appearance.

d. **Voltage electrocardiographic waves and ventricular repolarization**: Increased QRS voltage is a common finding in the electrocardiogram. Much of athletes and present it may related into the thoracic constitution. Changes in ventricular repolarization are also very common. The J-point elevation in precordial leads with ST segment supra uneven is a common finding. The described morphology can lead to diagnostic confusion when the athlete develops chest pain suspicious. CPK increases and CPKMB, which can occur after intense exercise, help to interpret the symptoms as an acute coronary event. Aliasing in the QRS and ST segment elevation in the right precordial can lead to confusion with the morphology found in Brugada syndrome.

e. **QT**: While the QT interval may be longer in the athlete, in assessing the QT corrected these differences disappear. Analysis of high performance athletes with physiological left ventricular hypertrophy showed no QT prolongations.

**Echocardiographic findings**

The dimensions of the cavities and ventricular walls vary according to the sport performed and the training level attained. Athletes with more intense workouts dynamic present the greatest increases in both the cavity dimensions and thickness of the walls. Weightlifters show higher relative changes in wall thickness to the size of the cavity, although the absolute values rarely exceed normal limits. When hoist weight can develop systolic blood pressures above 300mm Hg and diastolic up to 200mmHg. However, these overloads time is very short, so that few morphological changes generated. In conjunction with ball sports (soccer, basketball, volleyball, handball or water polo) is a balanced enlargement, although not over 60mm absolute increases in left ventricular cavity (usually, and paretial thickenings within normal parameters. In large populations of different disciplines athletes it has been observed that the thickness of the ventricular wall not increase beyond 16 mm, while average values are in the order of 11.5mm. The largest ventricular diastolic dimensions described left to reach 70 mm in high endurance athletes. Increases in the thickness of the ventricular walls come within an expansion of the cavity. This criterion is important in the differential diagnosis between physiological and pathological hypertrophy.

**Differential diagnosis**

While the morphological changes of the heart of the athlete usually not significant, in some endurance athletes need further studies to make the differential diagnosis with pathological conditions. This occurs in the presence of very overt electrocardiographic changes or to significant increases in cavity dimensions and/or paretial thicknesses. Pathologies that most commonly lead to diagnostic uncertainty are hypertrophic and dilated cardiomyopathies. As already stated, the electrical changes in right precordial leads (QRS nicked and ventricular repolarization) can, in rare occasions, generates some doubt related acute coronary syndromes or even with Brugada syndrome.

a. **Dilated cardiomyopathy**: The most important for the differential diagnosis of dilated cardiomyopathy feature is the absence of alterations in systolic and diastolic function. Older ventricular dilations occurred in men with elevated body surfaces and participating in sports with high dynamic training.

b. **Hypertrophic Cardiomyopathy (MH)**: It is necessary to take into account different variables to distinguish physiological hypertrophy of MH because there is no single, definitive method.

i. **Electrocardiogram**: The MH causes, generally of greater magnitude changes in QRS and ventricular repolarization that described in the athlete’s heart. The T wave high voltage are not among the usual modifications secondary sport. However, in many cases of MH, electrical changes overlap with those found in the athlete.

ii. **Wall thickness**: The septum and posterior wall of the left ventricle are within normal limits in the vast majority of athletes. Hypertrophic cardiomyopathy characterized by higher than those observed in the trained subject, although cases only moderate increases in wall thickness hypertrophies. In the anterior septum, the most pronounced values observed. At the heart of
the athlete, surrounding areas have similar hypertrophy, while the appearance of the MH asymmetries with sharp transitions between neighboring areas is feasible. In adolescents with MH may have mild hypertrophy confused with the physiological athlete and it is necessary to note that in that age group training can be very intense in high performance sports.

iii. **Left ventricular cavity:** In athletes, the cavity expansion accompanying increased wall thickness. By contrast, in a small cavity MH generally observed. Faced with a parietal hypertrophy, the lower cavity dimensions of 45 mm suggest the diagnosis of MH and over 55 mm suggest an athlete’s heart.

iv. **Diastolic function:** Diastolic function is normal physiological hypertrophy and often not be in the pathological. Doppler echocardiography, the E / A can be invested in the MH.

v. **Deconditioning:** The suspension can generate training regressions wall thickness of 2 to 5 mm at 3 months of abandoning sports. This measure may be useful in selected cases to define between physiological and pathological hypertrophy. However, the suspension of work on a temporary basis can be detrimental to the career of the athlete in the sport has their way of life.

### Methodology

Observational research is descriptive and because intended to detail and specify the status of the players of Club Lotería del Tachira, its electrocardiographic features and cardiographic echo. Its main characteristic measurement variables with correlational hypothesis testing and application of statistical techniques to estimate the correlation. The research design correspond to field data collected process with the collaboration of the Andean Heart Foundation (Fundacor). As population census, have 34 players in the Club Lotería del Tachira. Technique and instrument, observation and data history collection as shows following. a) personal data and history: specifically recorded information on age, level of education, years of training, family background and factors relating to comorbidity, psychobiological habits of athletes personnel; b) physical examination: weight, height, BMI, test with 15-minute rest in the supine position and measurement of blood pressure by conventional mercury sphygmomanometer; c) electrocardiographic examination: remaining supine with electrocardiographic tracing 12 lead through standardized with a speed of 25mm/sec and voltage 1mV (=10mm) characteristic of the sinus or non-sinus rhythm (in this case was specified) electrocardiograph heart rate in beats per minute, measures the P wave duration in seconds and amplitude in millimeters, measured PR interval and QRS complex in seconds corrected QT interval with heart rate to obtain the corrected QT interval (QTc) by formula Bazet ty Kissin et al, diagnosis of left ventricular hypertrophy under Criterion SokolowLyon: SV1 + RV5 equal to or greater than 35mm, presence or absence of signs of early repolarization to show ST-segment elevation in precordial leads V1-V5 and/or T wave negative V1 ; d) cardiology Eco Test: Two-dimensional, M-mode and Doppler using echocardiographic Phillips transducer 2.5MHz and scanning speed of 50mm/sec, images parasternal long axis with incidence of the ultrasonic beam perpendicular to the left ventricle, in parasternal and apical four chamber,

Analysis techniques and data processing: For the analysis of the data obtained in this research, used the PASW Statistics 18 program

### Results

#### Simple frequency analysis

According to the graphs presented, all of the rated players had a left ventricular function within normal or preserved. Despite the mild hypertrophy of the septum in response to adaptive changes experienced by the heart as a result of years of physical training (Figure 1–8).

![Figure 1](image1.png) Weighted distribution by Age range.

![Figure 2](image2.png) Distribution weighted by Years of training.

Results Simple frequency analysis

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Figures 9 and Figure 10 show that 65% of the players have high electrocardiogram precordial T waves of V2 to V5, while most 95% have an elevated ST segment. Both electrocardiographic signs of early repolarization in the 12-lead electrocardiogram.
Structural changes in training aerobic soccer players

The effect of intense physical training evidenced by the presence of asymptomatic bradycardia observed in 21 of the 34 players evaluated (Figure 11).

Figure 4: Distribution weighted by size ranges.

Figure 5: Distribution weighted ranges BMI.

Figure 6: Distribution weighted ranges interventricular septum thickness.

Figure 7: Distribution ranges weighted thickness of the posterior wall of the left ventricle.

Figure 8: Ejection fraction of the left ventricle.

Figure 9: T High (Signs of early repolarization ECG).

Figure 10: ST segment elevation on ECG.

Figure 11: Presence of Bradycardia electrocardiogram.

Citation: Mogollon KRA, Guerrero JAO. Structural changes in training aerobic soccer players. J Lung Pulm Respir Res. 2018;5(6):215–223. DOI: 10.15406/jlprr.2018.05.00194
From 17 patients with Mild Hypertrophy only 6 of these had electrocardiographic signs of left ventricular hypertrophy given by S in V1 and V5 or V6 R greater than or equal to 35mm. Important: the only patient with moderate hypertrophy present no sign of Sokolow Lyon (Table 1).

Table 1 Ratio of thickness of the septum in the presence of index Sokolow Lyon

<table>
<thead>
<tr>
<th>Sokolowlyon</th>
<th>Sivdiastole</th>
<th>Normal 0.6 to 1.0</th>
<th>Mild hypertrophy: 1.1 to 1.3</th>
<th>Moderate hypertrophy: 1.4 to 1.6</th>
<th>Total</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>SI</td>
<td>No</td>
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<tr>
<td>Sivdiastole</td>
<td></td>
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<tr>
<td>Normal 0.6 to 1.0</td>
<td>2</td>
<td>14</td>
<td>16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild hypertrophy: 1.1 to 1.3</td>
<td>6</td>
<td>11</td>
<td>17</td>
<td></td>
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<tr>
<td>Moderate hypertrophy: 1.4 to 1.6</td>
<td>0</td>
<td>1</td>
<td>1</td>
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<tr>
<td>Total</td>
<td>8</td>
<td>26</td>
<td>34</td>
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Form 17 patients with mild septal hypertrophy; 16 showed ST segment elevation in the electrocardiogram, which would suggest that there is a direct relationship. Nevertheless, of the 16 patients with normal thickness of the septum 16 had elevated ST segment, creating us controversy regarding the fact infer that there is a direct relationship between the two variables (Table 2).

Table 2 Relation interventricular septum thickness with ST segment elevation

<table>
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<tr>
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<tr>
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<td>0</td>
<td>1</td>
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<tr>
<td>Total</td>
<td>33</td>
<td>1</td>
<td>34</td>
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</tbody>
</table>

We can see that either presence of hyperacute T waves occurs the patient febrile hypertrophy interventricular septum or not, since we appreciate that the 17 patients with mild hypertrophy 11 if presented hyperacute T. Nevertheless, of the 16 patients evaluated with normal interventricular septum thickness 11 also presented this electrocardiographic variety (Table 3) (Table 4).

Table 3 Relationship interventricular septum thickness T and beaked high

<table>
<thead>
<tr>
<th>Sivdiastole</th>
<th>T. Hyperacute</th>
<th>Normal 0.6 to 1.0</th>
<th>Mild hypertrophy: 1.1 to 1.3</th>
<th>Moderate hypertrophy: 1.4 to 1.6</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SI</td>
<td>No</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sivdiastole</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal 0.6 to 1.0</td>
<td>11</td>
<td>5</td>
<td>16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild hypertrophy: 1.1 to 1.3</td>
<td>11</td>
<td>6</td>
<td>17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate hypertrophy: 1.4 to 1.6</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>23</td>
<td>11</td>
<td>34</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

61% of patients have asymptomatic bradycardia.

Table 4 Relationship of years of training with bradycardia

<table>
<thead>
<tr>
<th>Training Years vs. Brady</th>
<th>Brady</th>
<th>Do not</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 to 10 years of training</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>10 to 14 years of training</td>
<td>7</td>
<td>6</td>
</tr>
<tr>
<td>14 to 18 years of training</td>
<td>4</td>
<td>two</td>
</tr>
<tr>
<td>More than 18 years of training</td>
<td>4</td>
<td>two</td>
</tr>
<tr>
<td>Total</td>
<td>twenty-one</td>
<td>13</td>
</tr>
</tbody>
</table>

Conclusion and recommendations

Morphologic and functional at heart level as an adaptive response to exercise effects depend on the type, intensity and duration of training and athletes have substantial ventricular level that can be explained with the control mechanisms of ventricular remodeling structural alterations. The response to dynamic aerobic workout routine manifested in increased vagal tone, decreased resting heart rate and increased cardiac output. In this type of exercise volume overload it is imposed, with the resulting increased tele left ventricular diastolic dimension, proportional increase in the thickness of the sepal and free walls, and increased left ventricular mass.

In this paper, an increased thickness of the interventricular and lower level of the rear wall likely as a compensator septum is apparent physiological mechanism. The development of septal hypertrophy is a hallmark of hypertrophic cardiomyopathy. However, some athletes show hypertrophy as part of their physiological development. Certainly, it appears in subjects who engage in resistance to fatigue and increases as the training intensifies. The greatest thickening of the partition increases the posterior septum / wall ratio; this proportion athletes, usually is within normal limits, but may be exceeded slightly. In absolute values, the wall thickness in athletes is within normal limits or very little high.

In this group of players with intensive training it has not been demonstrated increased left ventricular mass.

The most common finding in the resting electrocardiogram is sinus bradycardia, related to the level of high competition training. Heart rates up to 38 beats per min found which pathophysiologically attributed to a vagal hypertonia and reduction of sympathetic tone.

Changes was also seen in the QRS complex and ST segment, which corresponds to ventricular hypertrophy. The increase in voltage QRS complex suggests left ventricular hypertrophy. The ST segment can be elevated appreciation was in 97% of cases, followed by a high T wave rises rapidly at 68% which is quite common. This explained in pathophysiologic sense of a functional syndrome called early repolarization observed in the precordial leads.

Note that the players evaluated fortunately not supra ventricular arrhythmias presence is appreciated and ventricular extrasystoles.

It is important to show that one of the evaluated players presented echocardiographic particular to appreciate a left ventricle compatible spongiform appearance with probable non-compaction cardiomyopathy of the left ventricle, but did not meet the criteria.
eco cardiographic to assert that were a serious case so recommended periodic evaluation.

As a final recommendation suggested soccer clubs, professional or amateur level, implementing programs cardiovascular assessment during recruitment of players and prior to the initiation of any training program. Also recommended developing programs for periodic evaluation of athletes in order to rule out diseases that may endanger his life.28,29

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None.

Conflicts of interest

Authors declare that there is no conflicts of interest in publishing this article.

References