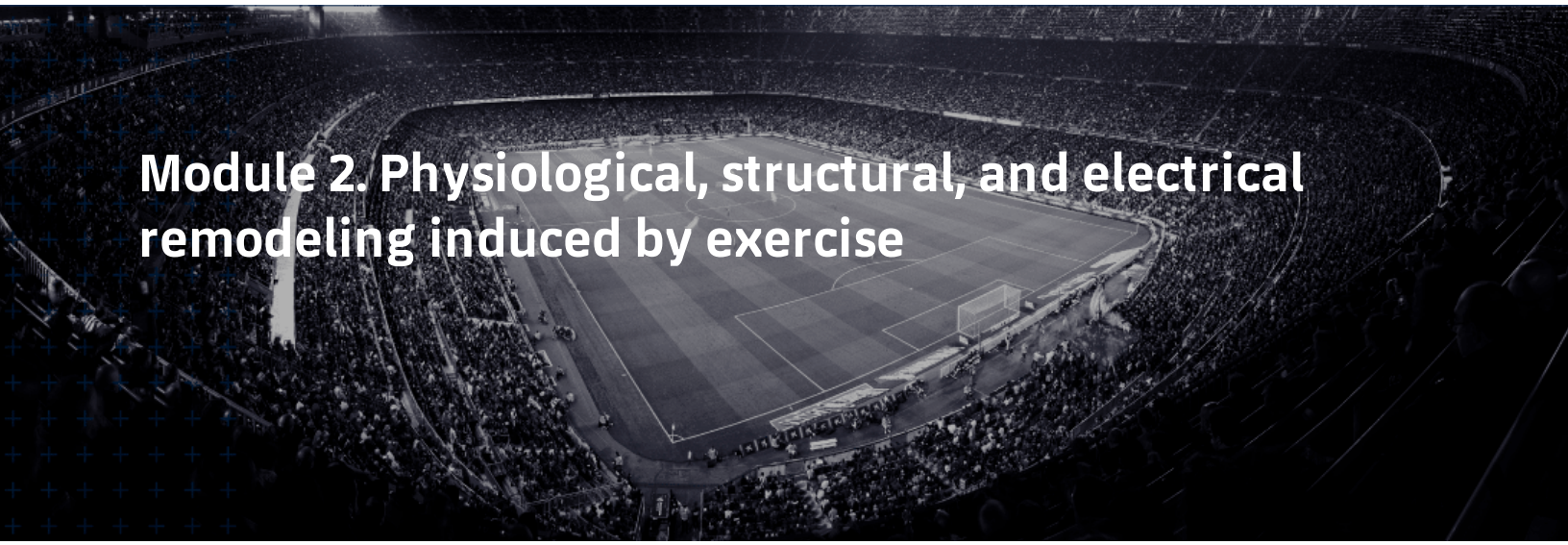

Module 2. Physiological, structural, and electrical remodeling induced by exercise

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Unit 2.1 Introduction

Regular and continuous physical exercise causes multiple structural and functional cardiac changes, responding to the demands imposed by physical activity. These changes are known as "athlete's heart." Although there is no precise definition of the athlete's heart, multiple descriptions have been made that integrate the structural and functional changes associated with this adaptation.

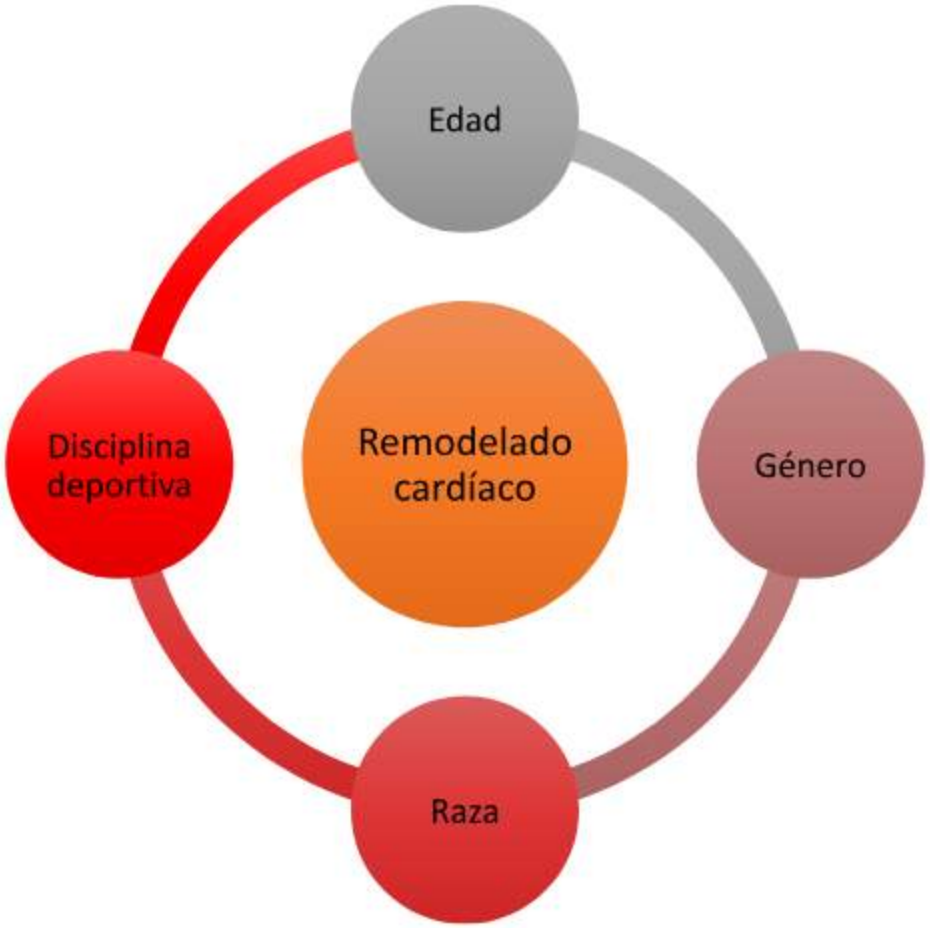
The first description of “athlete's heart” was made in 1899 by Salomon Eberhard Heschen, of the University of Uppsala in Sweden. Upon performing percussion on the thorax of Nordic ski athletes, he compared them to those of sedentary individuals, describing the difference in dimensions associated with the increase in myocardial work, and differentiating it from that related to heart disease (Henschen, 1899).

During physical activity, an increase of 5 to 6 times the basal cardiac output is required, which is a powerful stimulus for chronic structural changes; among which the most notable are dilation of cavities, ventricular hypertrophy, supranormal diastolic filling and changes in

the parasympathetic system. All these changes are usually modest and within the limits of normality (George et al., 2012). Nonetheless, a small group of athletes can often make major adaptations, showing adaptive changes that may overlap phenotypically with initial phases of pathological entities.

The degree and extent of cardiac remodeling is influenced by multiple factors, including age, gender, race, and sports discipline (Figure 1).

Figure 1: Main factors influencing the degree of cardiac remodeling



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Translation of Fig. 1

Cardiac remodeling

Age

Gender

Race

Sport discipline

Unit 2.2 Cardiovascular response to exercise

The acute response to exercise requires the proper functioning and interrelation of multiple systems, with particular emphasis on the autonomic nervous, cardiovascular, respiratory and motor (muscular) systems. Chronic physical activity requires structural adaptations and functional remodeling of organs and tissues, including increased plasma and red blood cell volume for adequate oxygen and carbon dioxide transport.

The cardiovascular effects of physical activity focus on providing an adequate amount of oxygen to the tissues. In basal situations, oxygen consumption is approximately 3.5 ml/min/kg (or one metabolic unit MET). In untrained individuals, maximum oxygen consumption can increase up to 10-12 times that of basal situations; meanwhile, in trained individuals, it can increase up to more than 20 times (80 ml/min/kg) (Saltin, 1967).

Cardiac output is the main determinant of oxygen consumption. At rest, the cardiac output of an average person is approximately 5 l/min, this being determined by heart rate and stroke volume. An untrained

person usually maintains a resting heart rate in the range of 70 bpm, while an athlete usually maintains a lower resting heart rate. During physical activity, the autonomic nervous system is responsible for increasing cardiac output. Although maximum heart rate is usually similar between two individuals of the same age, regardless of their training load, this mechanism alone would be insufficient if it were not accompanied by the significantly higher stroke volume in trained individuals associated with cardiac remodeling (Uusitalo, 1998).

During physical activity, oxygen demand is greatly increased, primarily by muscle tissue. To ensure adequate blood pressure perfusion, cardiac output, heart rate, and stroke volume are increased. Additionally, there is a decrease in blood flow to inactive muscles and capillaries in inactive regions (e.g., the splanchnic region). Blood flow is redirected to peripheral vascular beds (skin), which allow evaporation of the heat produced.

The onset of physical activity is characterized by an increase in heart rate, stroke volume driven by the autonomic nervous system, and oxygen required by the active tissues. The basal heart rate of an individual ranges between 60 and 90 bpm, but can decrease down to 30 bpm in highly trained individuals. The initial increase in heart rate is secondary to the inhibition of parasympathetic activity and to increases in sympathetic stimulation. On the other hand, the increase in stroke volume is secondary to the increase in venous return through the muscles, which, in turn, increases the end-diastolic

volume (EDV) and improves contractility by the Frank-Starling mechanism, reducing the end-systolic volume (TSV).

Cardiac output increases secondarily to the increase in heart rate and stroke volume, the latter being affected by preload (end-diastolic volume), afterload (resistance against which the heart pumps) and contractility (capacity of the myofibril to shorten). Systolic blood pressure increases in response to the increase in cardiac output; conversely, diastolic pressure remains similar, resulting in an increase in mean pressure. Vasodilation mediated by variations in intramuscular pressure and active compounds, such as nitric oxide, allows redistribution of blood flow to the active muscle tissue. This is associated with sympathetic vasoconstriction of the less active tissues, decreasing total peripheral resistance and increasing oxygen availability to the tissues.

Contractility and blood pressure increase at the onset of exercise in response to activation of the autonomic nervous system, due to the withdrawal of the parasympathetic system and the increase of the sympathetic system. The degree of these changes is proportional to physical activity and the muscle mass recruited. The suppression of the parasympathetic system increases heart rate, while sympathetic activity controls a series of modifications through three pathways: central command, exercise pressor reflex and arterial baroreflex (Dempsey, 2012). The central command is responsible for the activation of the motor and cardiovascular center in the central

nervous system in order to establish an autonomic activity response based on the intensity of physical activity. The pressor reflex controls sympathetic tone in response to mechanical signals originating in the active muscles. Together, these two—the pressor reflex and the arterial baroreflex—modulate the increase in heart rate, contractility and venoconstriction, increasing preload and, in conjunction, cardiac output. Thirdly, the arterial baroreflex is activated when blood pressure increases to between 60 and 180 mm Hg. This blood pressure is detected by baroreceptors located in the aorta and carotid arteries: these modulate an inhibitory effect to prevent sudden increases and variations in blood pressure.

CONTINUE

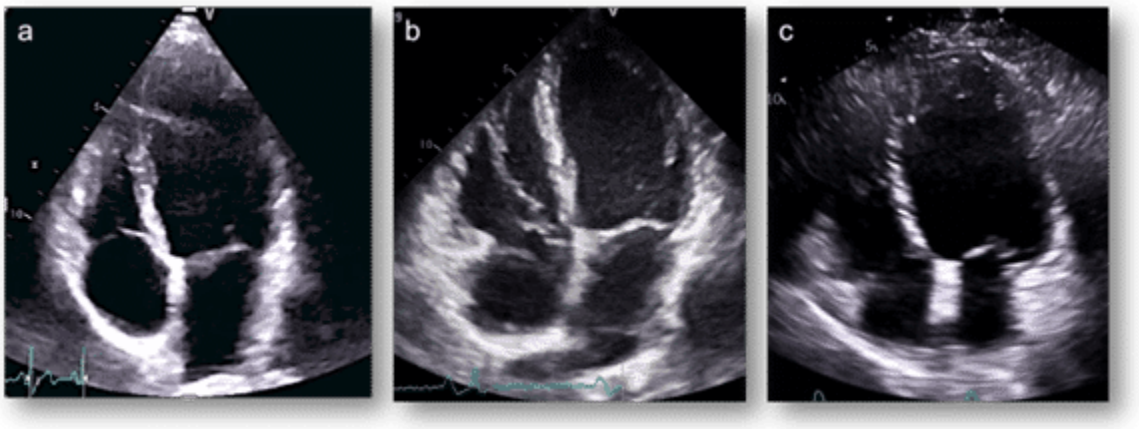
Unit 2.3 Structural remodeling

1 General aspects

Exercise-induced cardiac remodeling adaptively increases the size and thickness of the cardiac chambers. This is promoted by the volume and pressure loading required during physical activity. The pressure required during physical activity promotes structural, functional, and electrical remodeling, which allows for improved contractile function and efficiency, when compared to hearts from sedentary individuals (see Figure 2).

Figure 2. a. A 27-year-old woman who performs recreational aerobic exercise at low loads (< 3 hours/week); b. A 34-year-old man, a mountain racer with high training volumes (10 hours/week); the echocardiographic image shows a harmonic dilation of all the cardiac chambers; c. A 42-year-old man who performs low-intensity aerobic exercise daily (walking on flat ground); the effect of idiopathic dilated cardiomyopathy can be observed, and the

echocardiographic image shows a marked dilation of the left ventricle compared to the rest of the cardiac chambers.



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Increased stroke volume is the predominant mechanism by which athletes generate and maintain elevated cardiac output for long periods of time. Increased stroke volume occurs through a combination of increased LV end-diastolic volume (EDV), decreased end-systolic volume, and improved ventricular filling. Previous M-mode studies have shown a 10% increase in end-diastolic diameter and a 15-20% increase in left ventricular wall diameter in athletes, as compared to sedentary individuals (Maron, 1986).

These changes vary between different sports activities and levels of training; they are grouped into predominantly isotonic (dynamic) and isometric (static) activities (Levine et al., 2015). Isotonic (dynamic)

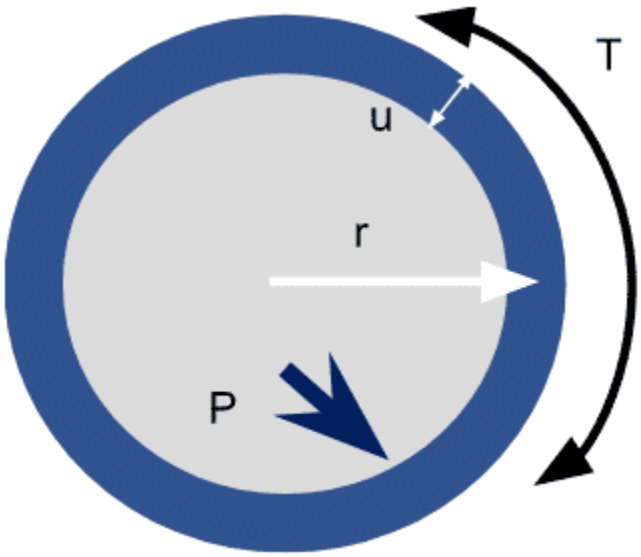
physical activity imparts a volume overload to the cardiac chambers, which induces their growth. On the other hand, predominantly isometric physical activity is associated with short periods of time with marked increases in peripheral resistance (afterload). Although these increases can be very intense, their short duration has less impact stimulating the growth of the chambers. This dichotomous classification is not always applicable; it will only be used as a means to simplify the wide range of sports disciplines with dynamic and static components, without diminishing the relevance of specific knowledge regarding the activity performed by each individual.

The stress exerted by the cardiac muscle is the most important determinant for ventricular remodeling. This is according to the principle of Laplace's law, in which the parietal stress is equal to the pressure, multiplied by the radius, over two times the thickness of the ventricular wall. This law describes the factors that influence cardiac remodeling, which is directly proportional to the pressure and the radius and inversely proportional to the thickness of the wall (Figure 3). The mechanical stress induced by hemodynamic overload stimulates the release of angiotensin II, growth factors and insulin that lead to the physiological adaptation of cardiac cells. Experimental models have been described where physical activity is related to an overexpression of the α isoform of myosin chains, associated with an increase in cardiac contractility (Herron and McDonald, 2002). Associated with this adaptation , experimental studies have described an improvement in the contractile function of

the LV in trained animals. This is secondary to the alteration of the regulation of the calcium channels responsible for the coupling and relaxation of muscle fibers (Kemi, 2003) .

In relation to the increase in ventricular mass, an increase in coronary circulation, with a larger size and number of vessels, has also been described, as well as an improvement in endothelial function that results in an increase in myocardial oxygen supply (Pelliccia et al., 1990).

Figure 3. Laplace's law



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As we mentioned, the calculation of parietal tension is as follows:

parietal stress = (transmural pressure *radius) / (2* wall thickness)

Laplace's law allows the myocardium to make physiological adaptations when subjected to an increase in one of its components. That is, the larger the radius of the left ventricle, the greater the wall tension. On the other hand, if the thickness of the wall increases (ie. adaptations to the athlete's heart), the wall tension will decrease.

2 Left cavities

Possible exercise-induced physiological changes in the heart chambers are as follows:

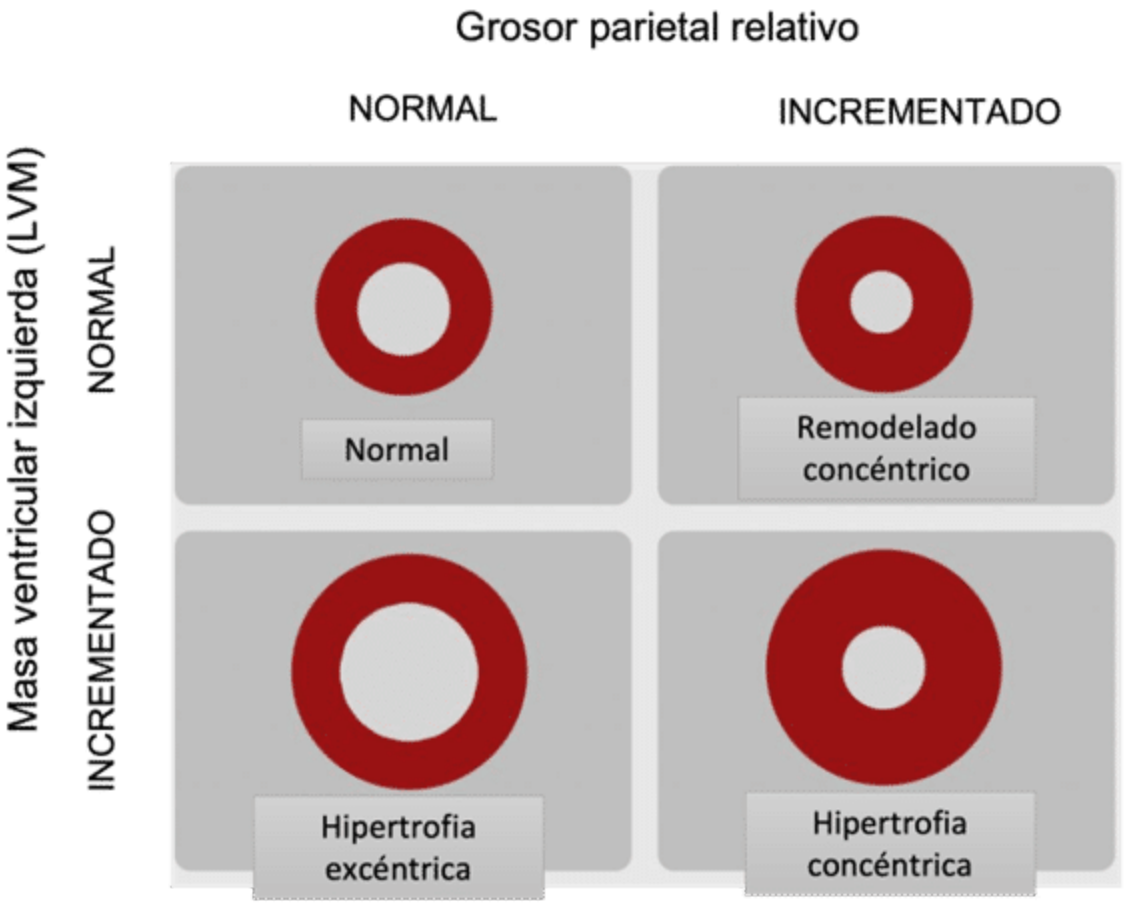
1. Increase in cavity diameter
2. Increase in the thickness of its walls
3. Decreased resting ejection fraction

The thickness of the left ventricle (LV) wall in athletes varies according to sex, duration of activity, race and sport, but in most cases it remains within normal limits. The highest degree of ventricular hypertrophy is usually found in male athletes, but values greater than 12 mm are uncommon (<2% in Caucasian athletes). On the other hand, in female athletes, the degree of hypertrophy is usually lower,

with values extending up to 11 mm. Furthermore, while Caucasian athletes rarely have an increase exceeding 12 mm, up to 18% of black athletes may present such an increase (Pelliccia et al., 1991).

The assessment of left ventricular geometry is based on relative wall thickness (RWT) and indexed ventricular mass (LVMI). Hypertrophy is defined as LVMI greater than 95 g/m2 in women and greater than 115 g/m2 in men; in addition, ventricular geometry must be assessed to classify the patient in one of the four possible scenarios (see figure 4). Athletes with abnormal ventricular mass (LVMI) may have eccentric hypertrophy (RWT ← 0.42) or concentric hypertrophy (RWT > 0.42).

Figure 4. Assessment of ventricular geometry, according to relative wall thickness and left ventricular mass



Source: own elaboration

Translation of Fig. 4

Left Ventricular Hypertrophy

Left Ventricular Mass (LVM)

Relative Wall Thickness (RWT)

Normal / Increased

Normal / Concentric Remodeling

Eccentric Hypertrophy / Concentric Hypertrophy

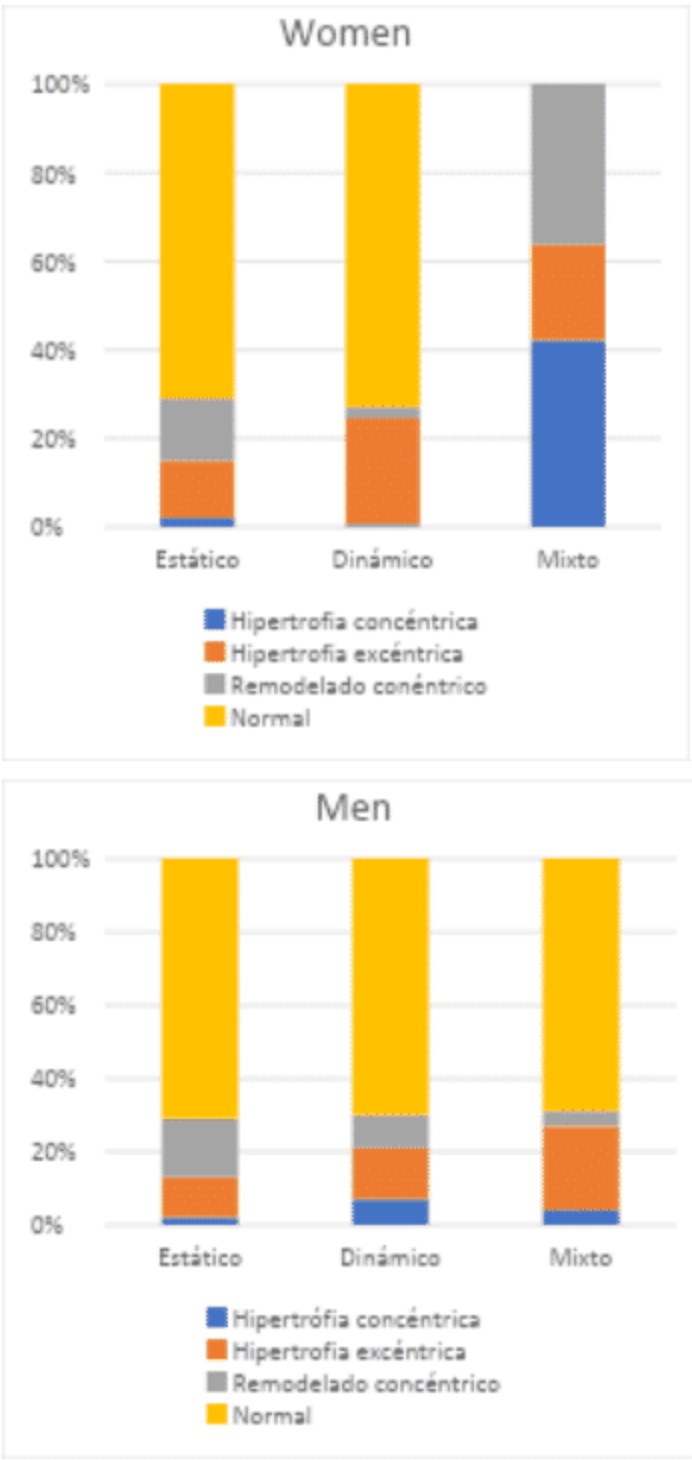
The increase in the dimensions of the ventricular cavity in highly trained athletes is well known. The dimensions usually vary according to the type of sport and the gender of the athlete. Telediastolic diameter values greater than 60 mm have been described in up to 15% of highly trained athletes (Pelliccia and Culasso, 1999), with canoeing, rowing and cycling being the activities most related to ventricular dilation. The growth of the cavities is usually associated with an increase in the absolute thickness of the ventricular walls, though this rarely exceeds 13-15 mm.

The group of Makan et al. (2005) studied 900 predominantly Caucasian elite athletes, with a mean age of 15.7 years, and compared the results with a study of a control group of 250 healthy sedentary persons. The athletes demonstrated a larger left ventricular end-diastolic diameter (50.8 vs. 47.9 mm); dimensions greater than 54 mm were present in 18% of the athletes, compared with zero patients in the control group. Thirteen percent of the athletes had a left ventricular cavity outside the accepted normal range (55-60 mm).

In a study by Sharma et al. (2002) involving 720 elite athletes, echocardiographic left ventricular wall thickness (LVWT) and LV dimensions of athletes were compared with those of 250 sedentary subjects. On average, athletes had a 13% greater LVWT and a 6% greater LVWT than those of the healthy control group. Although LVWT exceeded normal limits in 5% of athletes (38), only 0.4% had a LVWT greater than 12 mm (all men); in all cases, this was associated with harmonious remodeling of the rest of the chambers.

In a study by Finocchiaro et al. (2017) of 1083 Caucasian elite athletes (41% women), in the dynamic exercise subgroup, women demonstrated greater eccentric hypertrophy as compared to men; men, in turn, presented greater concentric hypertrophy. This demonstrated that sex also influences adaptive remodeling (see figure 5).

Figure 5. Left ventricular geometry in highly trained athletes



Source: Own adaptation based on Finocchiaro et al., 2017

Translation of the Fig. 5

Static / Dynamic / Mixed

Concentric Hypertrophy

Eccentric Hypertrophy

Concentric Remodeling

Normal

Normal geometry is the most common, regardless of the type of physical activity. In the dynamic activity group, women show greater eccentric hypertrophy, while men show greater concentric hypertrophy/remodeling.

LV end-diastolic volumes have been studied in endurance athletes, and are rarely higher than those described in the general population. When assessing cavity dilation, a practical fact is that the increase in this is almost invariably associated with an increase in ventricular mass. This suggests that the modifications are always related in a harmonious and balanced way. The increase in end-diastolic volume allows athletes to achieve greater stroke volumes, and this, associated with the increase in vagal tone, allows for lower resting heart rate values.

The left ventricular ejection fraction (LVEF) of highly trained subjects is usually normal or minimally reduced. In the case of finding an LVEF of less than 45%, it is necessary to perform extension studies that include tests that increase heart rate, such as hand-grip or exercise, to assess the normalization of the LVEF with the increase in heart rate.

A study by Abergel et al. (2004) with 286 professional cyclists showed that 6% of them (17) had an LVEF of less than 52%. In addition, all the athletes displayed left ventricular dilation.

The increasing use of myocardial deformation techniques, especially speckle tracking techniques, allows for easier assessment of myocardial deformation during the cardiac cycle for indirect assessment of myocardial contractility. This technique often shows slightly differentiated deformation patterns in both highly trained athletes and in sedentary individuals, as well as in pathological situations, although more evidence is needed in this regard.

Diastolic function is a good indicator of pathology in highly trained subjects, since, regardless of the degree of left ventricular remodeling, it tends to be normal. Commonly, in young athletes, transmitral flow is characterized by a significantly higher E/A ratio than in sedentary people, but it is always greater than 1. Therefore, a transmitral flow ratio E/A <1 suggests an underlying pathology. In young athletes, it is common to find a giant E wave associated with a small A wave. This is due to the fact that, at rest, the atrial contribution to stroke volume is

usually minimal when compared to sedentary controls, and most of the ventricular filling occurs during the beginning of diastole (E wave). During exercise, the increase in the A wave helps to increase stroke volume. Another method of evaluation is tissue Doppler of the middle and lateral mitral annulus. In this case, its values are usually normal (> 8 cm/s); values lower than this cut-off have been related to some pathology.

Magnetic resonance imaging (MRI) has emerged as a useful tool in cardiology due to its ability to assess regional myocardial motion, volumes, and possible fibrosis through late gadolinium enhancement. Studies have been conducted in highly trained athletes that demonstrate late enhancement patterns by MRI that suggest myocardial fibrosis. The mechanisms of this fibrosis and its consequences are not yet fully understood; however, there could be increased risk of arrhythmias and sudden death (Schnell et al., 2016). It is important to recognize patterns of fibrosis that may denote a simple adaptation to training, such as fibrosis located at the insertion point of the interventricular septum, and those found in other locations and that—generally speaking—indicate underlying pathologies.

Atrial remodeling is common in athletes with high training loads, especially those who practice endurance sports. Upon completion of an endurance competition, a transient decrease in atrial function has been observed (Martínez et al., 2020).

An anteroposterior left atrial diameter equal to or greater than 40 mm is present in 20% of young athletes, while diameters greater than 45 mm are present in as many as 2%. An important difference is that physiological atrial growth is always accompanied by changes in other cavities—such as the left ventricle and/or right cavities—unlike pathological growth, which is usually isolated (Iskandar et al., 2015). The assessment of atrial function using deformation patterns is a novel method that allows qualitative assessment of the degree of dysfunction. Nonetheless, to date, there are no normal cut-offs for athletes.

3 Right cavities

Similar to what has been said about the left cavities, the right cavities also tend to show remodeling in the form of dilation as a consequence of training for long periods of time (D'Andrea et al., 2015). Male athletes tend to have larger cavities, according to Sanchis et al.; although, in our own studies, we recognized further differences.

The most relevant dilation has been described in endurance athletes, with few changes described in strength activities (D'Andrea et al., 2012).

It is important to differentiate physiological and adaptive right heart dilation from the pathological counterpart found in early stages of arrhythmogenic cardiomyopathy. Athletes with physiological remodeling have normal RV systolic function, without alterations in segmental mobility. In addition, as previously mentioned, global cardiac remodeling is relevant, always being harmonious and generalized in all cardiac chambers and proportional to the load and type of training.

Table 1 summarizes the different normality cut-offs of cardiac dimensions considered for athletes.

Table 1. Normal limits for left ventricle and right ventricle in adult athletes based on sex and ethnicity

Group of athletes	Gender	DdVI (mm)	Septum VI	RV Dimensions					
				TSpVD (PLAX)	TSdVD	Basal VD	VD average	VD long	VD wall
White adult	Man	≤64	≤12	≤40	≤32	≤55	≤47	≤109	≤6
	Women	≤57	≤11	≤37	≤29	≤49	≤43	≤100	≤5
Black adult	Man	≤62	≤15	≤40	≤32	≤55	≤47	≤109	≤6
	Women	≤56	≤12	≤37	≤29	≤49	≤43	≤100	≤5

Source: Own adaptation based on Pelliccia et al., 2019.

- LVD: left ventricular diastolic diameter.
- VI: left ventricle
- RVSpT: proximal right ventricular outflow tract
- TSdRV: distal right ventricular outflow tract
- RV: right ventricle
- Long : longitudinal.

4 Aorta and great vessels

Physical activity-related changes in the aorta and large vessels are secondary to chronic hemodynamic load. Increased stroke volume and blood pressure lead to increased stress on the aortic wall, resulting in increased dimensions.

When comparing trained individuals with sedentary controls, the presence of aortic root dilatation has usually been described, always of a mild degree (< 40 mm). A larger dimension should raise suspicion of pathology, and extension studies are recommended for adequate assessment (CT), although body size should always be taken into account and indexed values should be used.

A study by Abuli et al. (2019) on 2083 athletes showed an increase in aortic root diameters in men as compared to women. The diameters were all less than 40 mm. This study demonstrated the importance of indexing values to body surface area.

5 Electrical remodeling in the heart of a male athlete

The electrocardiogram (ECG) allows the assessment of the electrical activity of the heart and, indirectly, allows conclusions to be drawn about cardiac anatomy. In athletes, the ECG has become a crucial tool for evaluation and screening, as well as for the prevention of adverse effects and potentially lethal conditions associated with exercise.

There are electrical changes related to the continuous stimulus that physical activity imposes on the heart muscle. Multiple electrocardiographic changes have been identified in athletes; these are usually related to an increase in vagal activity and the growth of the heart cavities (Pelliccia, 2007). Up to 60% of athletes show physiological changes on ECG, mainly sinus bradycardia, sinus arrhythmia, incomplete right bundle branch block, low-grade AV block, and voltage criteria for ventricular hypertrophy.

A small group of athletes (5-17%) develop electrical changes that may overlap with those of pathology related to sudden death (channelopathies, calcium channel abnormalities, pathological Q waves, ST segment depression, among others). In these cases, it is vitally important to perform a differential diagnosis, as will be reviewed later. Distinguishing these changes from pathological ones is among the main challenges that sports physicians must face.

CONTINUE

Unit 2.4 Determinants of cardiac remodeling in response to training

1 Gender

As already mentioned, the influence of gender on the development of “athlete's heart” is independent and related to sporting discipline and age, but in general, women tend to have smaller changes as compared to men. The reason for this is still unclear; it could be due to hormonal factors, changes in body composition, changes in blood pressure, and differences in the intensity of the training performed.

Compared to men, women tend to have smaller indexed cardiac chamber sizes. In a study of 1,309 white Olympic athletes, LV dilation greater than or equal to 60 mm was reported in only 1.7% of women, compared to 18% of men (Pelliccia et al., 1999); furthermore, ventricular thickness was less in female than in male athletes (8.2 ± 0.9 cm vs. 10.1 ± 1.2 cm), with no thickness greater than 12 mm in any woman. On the other hand, it has been shown that male athletes tend to present a greater degree of dilation and adaptation of the

right chambers, when compared to their female counterparts (Sanz de la Garza, 2017).

A study conducted in male and female athletes showed that right atrial changes in size and deformation are usually smaller in women compared to those observed in men of the same age. Higher myocardial deformation values and smaller atrial volumes were observed in female athletes (Sanchis, 2017).

The right ventricle tends to show a similar behavior: when compared to sedentary women, female athletes tend to have larger cavities; however, when compared to male athletes, female athletes tend to have smaller cavities.

2 Impact of sports discipline on the athlete's heart

The cardiovascular response to prolonged exercise varies according to the type of activity. The recognition that a sporting discipline has an influence on cardiac structural remodeling was first described by Morganroth in 1973 using echocardiography under the so-called “Morganroth hypothesis”, which posits that dynamic exercise, involving the propulsion of the athlete through space, as opposed to the generation of muscular force, imposes a predominant volume load

on the myocardium, resulting in eccentric hypertrophy, characterized by an increase in end-diastolic volume.

On the other hand, isometric or static exercise involves the generation of force through muscular contraction, which increases arterial blood pressure with a minimal decrease in peripheral vascular resistance, little or no displacement in space, and which predominantly exerts a pressure overload on the myocardium. This results in concentric hypertrophy characterized by an increase in ventricular mass and thickness with normal or slightly increased volumes.

Multiple studies have confirmed Morganroth's hypothesis; however, other studies suggest that this hypothesis is too dichotomous, since most athletes have mixed training regimes. This classification is different from that of aerobic and anaerobic exercise. In general terms, isometric or static exercise (strength) is performed through anaerobic metabolism, unlike dynamic exercise, which is predominantly based on aerobic metabolism. Mitchell's classification divides sport into three categories, according to its intensity (low, moderate and high). Figure 6 shows the different end-diastolic diameters in healthy individuals, according to the sport they practice. Activities with a predominance of static or isometric (strength) are associated with a greater increase in the ventricular wall.

Figure 6. Morganroth hypothesis

Source: Own adaptation based on Morganroth et al., 1975

Translation of Fig. 6

Swimming

Running

Greco-Roman wrestling

Sedentary

Activities with a predominance of dynamic movement (endurance) are usually associated with an increase in the cardiac chambers; while predominantly static or isometric (strength) activities are associated with an increase in the ventricular wall.

3 Impact of race on the athlete's heart

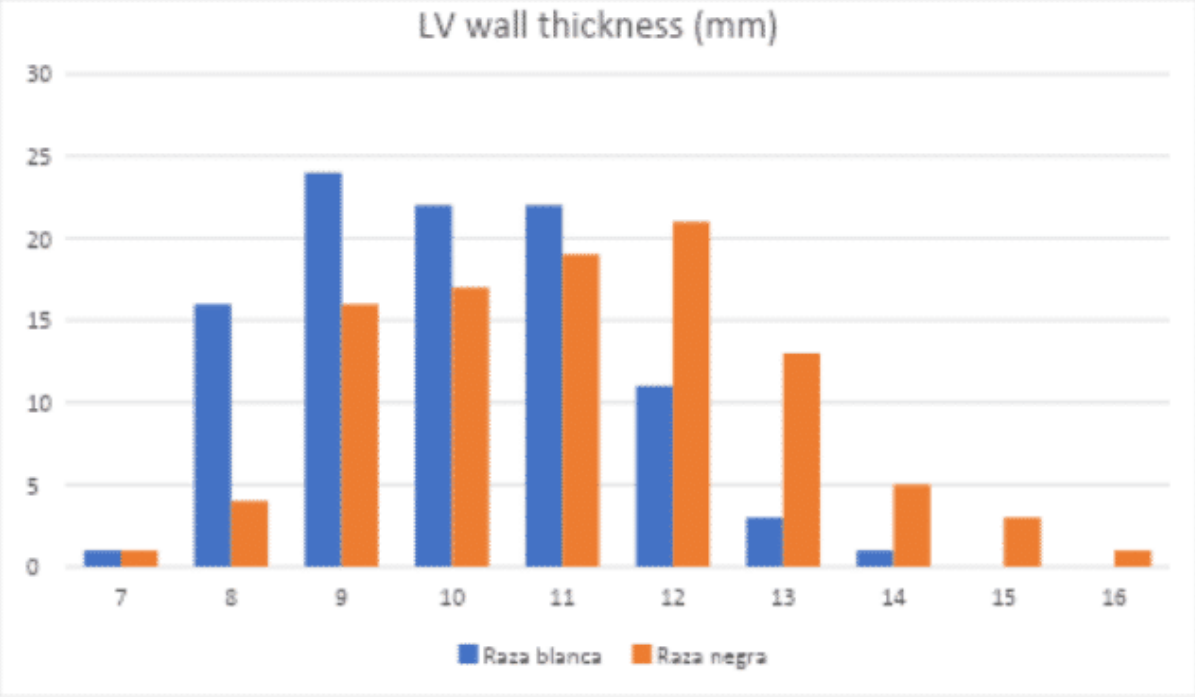
Multiple studies have demonstrated greater adaptive remodeling in African-American athletes, particularly in relation to myocardial hypertrophy and hypertrabeculation of both ventricles (see Figure 7).

A study by Lewis et al. (1989) of 265 predominantly Black athletes showed that 11% had a left ventricular thickness greater than 13 mm, within the range of hypertrophic cardiomyopathy, compared with 3% of White athletes. Another study in the United Kingdom showed that

when comparing the cardiac dimensions of 300 Black and 300 White athletes, 18% of Black athletes had a left ventricular thickness greater than 12 mm, and up to 3% had a left ventricular thickness equal to or greater than 15 mm.

On the other hand, there is a higher prevalence of T-wave inversion in precordial leads in Black subjects.

Figure 7. Distribution of maximum LV wall thickness in Black and White athletes



Source: Own adaptation based on Pelliccia et al., 2019

Translation of Fig. 7

White athletes

Black athletes

There are multiple factors that lead to major changes related to the athlete's heart, among which race, endurance activities, and male sex stand out as the most important (see figure 8).

Figure 8. The impact of the type of discipline, gender and age on the athlete's heart

Source: own adaptation

Translation of Fig. 8

Black athletes

Endurance activities

Men

Greater changes to the heart of the athlete

CONTINUE

Unit 2.5 Extreme situations

There are a number of adaptations to different extreme stimuli. The amount and magnitude of these will depend on factors such as the duration of exposure (acute vs. chronic), the inherent characteristics of the athlete (age, sex, gender) and the degree of exposure (medium altitude, extreme altitude).

- **Altitude**: The basis of the challenge to the body during altitude training is the gradual decrease in oxygen pressure as altitude increases. Physiological responses to this include:

    1. **Hyperventilation**: increased hypoxic drive and regulation of acid-base balance.
    2. **Increased cardiovascular response**: Submaximal heart rate and CO may increase up to 50% above sea level values.
    3. **Synthesis of hemoglobin and erythrocytes by hypoxic stimulus**: these mechanisms seek to

counter-regulate the decrease in oxygen pressure, in a chronic way.

Chronic exposure to extreme altitudes leads to a chronic state of alveolar hypoxia, hypoxemia, and polycythemia. Healthy individuals undergo various adaptations that allow them to increase oxygen transport to the tissues. A study by Doutreleau et al. (2022) demonstrated an increase in right ventricular dilation, left ventricular remodeling, and an increase in mean pulmonary arterial pressure in athletes exposed to extreme altitudes. Arias-Stella and Saldaña (1963) performed autopsies on natives of the Andes mountain range (3700-4540 m) and demonstrated greater hypertrophy of the distal pulmonary branches and right ventricular hypertrophy as adaptive responses to the altitude stimulus.

Furthermore, it has been shown that at higher altitudes (5100 m vs. 3800 m), despite similar training loads, heart rate is usually higher as an apparent consequence of the chronic hypoxic stimulus and adaptation to it (Doutreleau, 2022). It is necessary to continue studying altitude as a chronic hypoxic stimulus for athletes in order to provide better recommendations during their evaluation.

- **Temperature** : The hypothalamus, which is responsible for regulating body temperature, typically receives information from thermal receptors

in the skin that send information to central control. Stimulation of cutaneous cold receptors induces vascular constriction, decreasing blood flow to cooler surfaces of the body and directing it to the center. During cold training, adrenaline and noradrenaline production are increased to increase heat production. During hot training, the skin dissipates heat through perspiration and vasodilation.

Studies by Periard et al. (2015) in athletes exposed to extreme heat and cold demonstrated a decrease in basal heart rate, an increase in plasma volume, greater diastolic filling and an improvement in stroke volume, when compared to athletes not exposed to these stimuli. The magnitude of these adaptations depends on multiple factors, such as exercise intensity, duration, frequency and exposure time.

CONTINUE

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