Echocardiogram plays an important role in the diagnosis of cardiovascular disorders that may predispose young athletes to sudden death (SD) during sports-related activities.

With this technique, abnormalities involving the myocardium, aorta and cardiac valves can be detected and to be followed in their progression through time that may preclude safe participation in sports.

As an example, the athlete's heart hypertrophy, which is a benign and physiological adaptation to physical training, can be differentiated from the pathologic hypertrophy represented by the Hypertrophic Cardiomyopathy (HCM), a genetic origin disease associated with SD in young athletes.

Although echocardiography is widely used by modern medicine, echocardiographic screening to identify young athletes at risk for SD remains controversial.

Next a revision of the echocardiographic findings of the most common cardiovascular diseases that can cause SD in young athletes (younger than 35 years) will be made. SD occurring in older athletes is generally due to coronary disease and is not addressed.

**ATHLETE’S HEART**

Characteristics

The adaptation of the human heart to physical conditioning, has been a topic of medical and scientific interest since a century ago, when Henschen, a Swedish clinician, noted in 1899 [1] a heart enlargement in "cross-country" skiers, using heart percussion and becoming the first researcher in describing the "athlete's heart". Later on, the knowledge of the cardiac adaptations to training advanced due to the advent of radiography and electrocardiogram; but it was the introduction of echocardiography in the '70s that produced a new and important impulse in this researching area. The M-mode, Two-dimensional and Doppler echocardiogram, have been used by numerous authors to study the cardiovascular modifications produced by long-term and high intensity physical training. An enlargement of the ventricular cavities, a thickness of the ventricular walls as well as an increase of the left ventricular mass have been described in athletes [1-23]. In figure 1 it is observed an 18 year-old Brazilian cyclist with the typical modifications of "athlete's heart". These anatomical findings, have been explained by different theories, some related to physical training, like haemodynamic overload and/or endocrine factors, and others not related to training, like genetic and/or environmental influence [9,24]. The greater ventricular walls thickness is discrete [1-9], but in some athletes this thickness can be significantly greater, creating problems in the differential diagnosis between the athlete's physiological hypertrophy and the HCM [10]. This differential diagnosis is important, because it is probably one of the most frequent causes of SD in athletes younger than 35 years [11].
Echocardiographic Findings

The Health Sciences Faculty of the National University of Catamarca, Argentina, through the "Licenciatura en Educación Física" developed an investigation protocol in highly trained athletes that participated in three sport international competitions, carried out in Argentina during the years 1990-1995. This protocol included a clinical history and physical exam, blood pressure, kineantrometry, electrocardiogram and echocardiogram [25]. The electrocardiographic findings of the 444 studied athletes were the following (mean and standard deviation): interventricular septum wall 10.5±1.8mm (range from 6 to 18) exceeding the normal value of a non athletic population in 52 athletes (11.7%), left ventricular posterior wall 8.9±1.4 mm (range from 5 to 13); diastolic diameter of the left ventricle 52.3±4.7mm (range from 35 to 67), exceeding the normal value of 53 mm of a non athletic population, 213 athletes (48%), was observed that 32 athletes (4.3%) had a dimension±60 mm); left ventricular mass 191±51 g (range from 56 to 388) [exceeding the normal value of a non athletic population 28 athletes (6.3%)] of left ventricular mass index 102.5±22 g/m$^2$ (range from 55 to 176), [exceeding the normal value of +134 g/m$^2$ of a non athletic population 29 athletes (7%)].

The percentage of athletes with ventricular walls thickness (LVW) compatible with HCM >13 mm was almost the12% of the studied population. Of this group the greater LVW of 18mm corresponded to two athletes, a soccer player and a volleyball player. Figure 2 and 2bis, shows an 18 year-old volleyball player from Paraguay with maximum thickness of the LVW. It was also observed that most of the athletes of this group belonged predominantly to dynamic or aerobic-type sports disciplines. It is highlighting that the maximum thickness of the LVW of the 129 female athletes studied, was 13 mm. This suggests that women increase the LVW thickness to a lesser degree that men in response to physical training (genetic and hormonal sex-related factors). Other authors like Pellicia and col. [18] studied near a thousand Italian athletes and they hardly found 2% of athletes with LVW thickness >13 mm and with a maximum LVW thickness of 16mm. They also described rowers, canoeists and cyclists like the only ones with physiologic hypertrophy. These differences with our research are probably due to the study that we perform on athletes in the maximum training level period, that is to say during sports competitions (greater cardio-vascular adaptation level to effort) and that our studied population was heterogeneous. Likewise other authors like Van Camp and Reguero [21-22] conveyed similar results to ours.
It was found in the LVW group of > 13 mm thickness, a left ventricular cavity enlarged with a mean 53±5 mm (range from 43 to 66), suggesting a hemodynamic overload effect imposed by physical training. A small left ventricular diameter is frequently found in HCM, although in our study it was observed in three athletes with physiologic hypertrophy, a same or smaller diameter than 45mm. Therefore the differential diagnosis between the athlete's hypertrophy and HCM based on the left ventricle diameter (LV), is suggestive but not decisive of such pathology. The maximum thickness of the LVW was found in the anterior and posterior region of the interventricular septum wall, being the other walls of the left, lateral and posterior ventricle of smaller thickness, homogeneous and they did not present asymmetry, figures 2 and 2bis.
It is highlighting that all athletes were completely healthy, without HCM antecedents, or SD in their relatives.

**HYPERTROPHIC CARDIOMYOPATHY**

**Characteristics**

HCM is a dominant autosomic genetic disease, clinically heterogeneous [26-28], and its prevalence in the general population is very low of 0.2% [29-30]. It is characterized by left ventricular hypertrophy, predominantly of the interventricular septum in absence to other hypertrophy causes like high blood pressure or valve pathologies [31]. The histopathology study shows its main characteristic which is a pronounced myofibrils disorganization. These are abnormally short and wide, they extend in different directions, and show abnormal bridges between the fibers with abnormal cellular contacts, forming spirals. Myocytes are hypertrophic with hyperchromatic and bizarre nucleouses. Interstitial fibrosis and abnormal thickness of the coronary intramural walls are also observed [31]. The natural history of this disease is characterized by a pronounced anatomic-functional diversity, and presents itself in a mild or massive, focal or diffuse, concentric or asymmetric way. Similarly the clinical manifestations or the natural disease history varies in the affected individuals. It may also elapse without signs or symptoms, in these individuals the diagnosis is carried out in a routine medical exam [32]. Many affected individuals refer dyspnea or angina, symptoms that slowly progress through time. Palpitations are common and may announce an auricular fibrillation development or SD that may occur in asymptomatic patients or with little symptomatology. Heart failure and embolic events may contribute to a premature morbi-mortality [20].

**Echocardiographic findings**

HCM should be diagnosed as obstructive or nonobstructive for all patients identified with this disorder. LV outflow tract obstruction in HCM typically occurs as a result of systolic anterior motion of the mitral valve (SAM) or chordae and a narrowing of the LV outflow tract [34]. The mechanism of LV outflow tract obstruction is related to the Venturi effect. Accelerated flow in the LV outflow tract results in a suction effect in which a portion of the mitral apparatus is drawn into this region. [35]. Figure 3 shows a 17 year-old basquetball player with HCM with severe asymmetric hypertrophy of the interventricular septum and SAM. Classification of patients with nonobstructive HCM requires that a provocative maneuver be previously performed such as exercise, isoproterenol or amyl nitrate inhalation. These maneuvers should be performed if the LV outflow tract is narrow in combination with a significant hypertrophy of the proximal septum or an elongated mitral leaflet. Amyl nitrate is easy to administer and due to its transitory effects is very safe [36]. Stress-echo is helpful in detecting outflow tract obstruction in patients who have little or non obstruction of the outflow tract in rest. The high incidence of reported nonobstructive HCM may be the result of patients not evaluated with provocative obstruction maneuvers [36].

![Fig. 3: Basquetbolista de 17 años con MHE. Plano del eje mayor paresial del VI que evidencia la hipertrofia asimetrica del tabique interventricular de 37 mm. La flecha indica el movimiento anterior sistolico de la válvula mitral (SAM).](image_url)
LV outflow tract obstruction may produce mitral regurgitation (MR) in many patients with HCM where MR is caused by lack of leaflet coaptation as a result of their anterior motion. In many patients both leaflets are involved in the SAM, but they may also be one or another. Variability of the valves length and mobility may lead to an unequal coaptation, therefore to vary the MR degrees [37]. The severity of MR is estimated though color flow Doppler. MR jet is frequently directed posterolaterally. The outflow tract gradient (p) is calculated using continuous Doppler imaging, converting the flow velocity (v) in meters per second to mmHg using the modified Bernoulli’s equation (P = 4v²) [38]. Care must be taken to assure that the continuous Doppler imaging measures the outflow tract velocity and not of the MR. Color Doppler imaging the time and the shape of continuous Doppler help to differentiate both flows [39]. The diastolic function in HCM is frequently abnormal. This is produced by altered relaxation and a LV rigidity increase. Coexistence of LV may represent a problem in the diastolic function interpretation. Significant degrees of MR may mask the abnormal filling in some patients by left auricle elevation pressure and pseudo normalization of the mitral flow patterns [35]. HCM is associated with decrease compliance, therefore, there is an abnormal increment of the LV pressure for a given ventricular volume level. These findings are echocardiographically reflected by a high amplitude and prolonged duration of the pulmonary veins flow compared to A mitral wave duration. Although it is clear that there are abnormalities of the diastolic filling, there is not a good correlation between the LV filling measured by Doppler and the LV structure [40].

The main difficulty is to distinguish between the athlete's physiologic hypertrophy and the HCM, since this disorder causes SD in young athletes. Maron [41] proposed a strategy to distinguish HCM of athlete's heart when the LVW thickness is in a gray zone (13 to 15 mm) compatible with both diagnoses.

**Strategies to distinguish between the athlete's physiologic hypertrophy and the HCM.**

**Thickness of the left ventricular walls:**
In most athletes, the LVW absolute thickness value is within the normal limits (<12mm). In some athletes; however, this thickness may be greater, between 13-15mm, suspecting a HCM. In patient with HCM the increase of the LVW thickness is significantly greater, the mean value reported by different studies of this disease is around 20mm and reaching 60mm. Nevertheless an important group of patients with HCM show a mild LVW hypertrophy with a thickness in the range of 13 to 15 mm [41]. In highly trained athletes, the thickness prevalence region always implies the anterior septum, even though the increased thickness on other segments of the wall is similar, with a difference of 1 to 2mm. In patient with HCM, the anterior septum is always the most hypertrophic segment, however the hypertrophy pattern is frequently heterogeneous, asymmetric and occasionally it may present itself with greater hypertrophy in other walls and in lesser degree in the septum. In summary the LV contiguous walls, show different hypertrophy degrees, and the transition between those areas is abrupt [42]. HCM diagnosis is echocardiographically based on the hypertrophy magnitude, quantifying the thickness of a LVW segment. Be on the alert that, in doubtful cases, such circumstances represent a fertile field of the disease overdiagnosis. Since the hypertrophy increment occurs during adolescence in patients with HCM, young athletes with this disease (<16 years) may not present the maximum hypertrophy expression until they reach the top physical development and maturation [43]. Therefore an athlete with HCM, may be initially evaluated and echocardiographically submit a mild hypertrophy or to be in the normal ranges, difficulting the differential diagnosis with athlete's heart. However this problem can be solved if serial echocardiograms through time, months or years are performed until the definitive hypertrophy that allows to make HCM diagnosis appears.

**Cavity dimensions of the left ventricle:**
An increase of the left ventricle final diastolic diameter (LVDD) (>55 mm), is a frequent finding on athletes, in our research the LVDD was 52.3±4.7mm (range of 35 to 67) (they exceeded the normal value of 53 mm of a non athletic population 213 athletes (48%), being observed that 32 athletes (4.3%) had a dimension> 60 mm). On the other hand patients with HCM, the LVDD is usually small (<45 mm). Figure 4 shows an 18 year-old soccer player with HCM, interventricular septum wall hypertrophy and a small LV diastolic diameter. While LVDD is > 55 mm only those patients with HCM that evolved to the final phase of the disease with progressive heart failure and systolic dysfunction. Therefore it is possible in some cases to distinguish between athlete's heart and HCM, based on the left ventricular cavity dimension. However, in those athletes whose ventricular size is not submitted within these values, the mere dimension do not solved the differential diagnosis [41].
Transmitral Doppler:
Abnormal diastolic functions of the left ventricle have been identified in a non invasive way with pulsed Doppler echocardiography or with radioisotopic angiography, in patients with cardiac diseases associated with left ventricle hypertrophy. Many patients with HCM, including those with mild hypertrophy (that may be confused with athlete's heart), present abnormal Doppler indexes of the diastolic function, independently the presence of symptoms or LV outflow tract obstruction. The early peak velocity of the transmitral inflow is typically reduced ("E" due to quick filling) and the deceleration timing of the E wave is prolonged. The late peak is increased ("A" due to atrial contraction), and the normal relation E/A is inverted. Figure 4bis is observed with an 18 year-old soccer player with HCM and altered mitral pulsed Doppler. On the other hand athletes always show normal diastolic function patterns. Therefore in a trained athlete suspected of HCM, abnormal Doppler indexes of the LV diastolic function, present this diagnosis, while a normal Doppler pattern is compatible with both diagnosis, athlete's heart or HCM [44]. Myocardial ultrasonic reflectivity.
Initial observations suggest that patient with asymptomatic (or with little HCM symptomatology) show an increase of the septum ultrasonic signal intensity and of the LV posterior wall (including patients with mild and located hypertrophy) while highly trained athletes with physiologic hypertrophy show normal reflectivity of the myocardial tissue. However it is not known for certain that the differences found between the groups may be applied to one particular subject [45].

**Type of sport training:**
The specific nature of the athletic training itself has a major influence in the type and magnitude of the left ventricular dimensions. In our study of 444 athletes from different countries and races, we have found in almost the 12%, ventricular walls thickness > 13 mm (in the gray zone between physiologic hypertrophy and HCM), mostly in dynamic type sports and the greater thickness 18 mm, was in a soccer and volleyball player. Conversely to weight lifters and judokas, isometric sport types, the maximum thickness of the ventricular walls was13 mm [25].

**Gender:**
It has been identified in athletes, sexual differences, in relation to modifications in the heart's dimensions and left ventricular mass. In our study we could hardly identify 5 women with maximum thickness of the ventricular walls 13 mm. These observations suggest that in those women athletes with ventricular walls thickness > 13mm (with normal or small left ventricular cavity) HCM is suspected.

**Regression of left ventricular hypertrophy with deconditioning**
It has been observed in athletes that deconditioning decreases the ventricular cavity and the ventricular walls thickness [47]. In the pathological forms of ventricular hypertrophy as HCM, the physical deconditioning does not produce changes on the left ventricular walls thickness. Identification of such changes on the ventricular walls thickness of the left ventricle requires along with the physical deconditioning, an adherence and motivation so that the athlete suspends the physical training and serial echocardiographic studies of optimum technical quality [48].

**ARRHYTHMOGENIC RIGHT VENTRICULAR DYSPLASIA**

**Characteristics**
It is an autosomic dominant genetic origin cardiomyopathy. It has been described as a cause of SD in the Veneto region of northwest Italy. According to Thiene [49-50] that reported 60 young individuals death between 1979 and 1986, 20% died of this disease. Most of them died during exercise and had antecedents of palpitations, syncope or both. Ventricular arrhythmias were detected in most of them. Echocardiographically the disease diagnosis is possible but requires an appropriate knowledge and specific search. The echocardiographic signs of the Arrhythmogenic Right Ventricle Dysplasia (ARVD) reflect the pathological
process of adipose and fibrous infiltration of the myocardium. Frequently affecting the right ventricle outflow tract (anterior infundibulum), the apex and the infero-basal wall, this anatomical area has been denoted the triangle of dysplasia \[51\]. Other diagnostic methods that have been used for the recognition of this disease were, magnetic resonance, cardiac radionuclear, contrast ventriculography and myocardical biopsy, though these studies are more expensive than echocardiography \[52-53\].

**Echocardiographic findings**

The echocardiographic sensitivity to the detection of the ARVD varies and depends on clinical history, disease prevalence in the studied population, disease stage and the quality of the obtained images. Dilatation of right ventricle with hypokinesis occurs in most patients with ARVD, however a normal echocardiogram does not exclude the diagnosis \[54-55\]. It should be kept in mind that the right ventricle enlargement and dysfunction is more frequently due to other cardiac and pulmonary diseases and not to ARVD. Therefore the echocardiography specificity for the diagnosis of this disease is low in unselected populations.

Enlargement of the right ventricle (RV) and wall motion abnormalities, in a focal or diffuse way is the most common echocardiographic profile suggesting the ARVD diagnosis. The inflow tract (parasternal short axis), outflow tract (parasternal long axis), and RV body (apical of 4 chambers) are the most typically enlarged segments. The RV outflow tract is the most frequently affected region \[54-55\]. RV function may be reduced to normal at rest but it decreases with exercise \[56\]. Regional or diffuse hypokinesis may vary from mild to severe and diskinesis or akinesis segments may also occur \[54\]. Follow-up studies of patients with ARVD showed enlargement and progressive dysfunction through time \[55\].

The pathognomonic findings of ARVD are aneurysms or sacculations of the RV free wall (triangle of dysplasia). Figure 5 shows a 27 year-old sprinter with ARVD, RV enlargement and aneurysm on the RV tip. These segments may be single or multiple and represent the infiltration and thinning of myocardium in those regions. RV prominent and irregular trabeculations may also be observed and the moderator band may be more evident \[54, 55, 57\].

![Image of echocardiogram](image)

**Fig. 5** - Velocista de 27 años con DAVD. Eco 2D en plano apical de cuatro cámaras. El VD está agrandado y se observa un aneurisma de la punta del VD, patognomónico de la DAVD.

Doppler exam of the tricuspid valve may show an inversion of the E/A relationship representing the RV diastolic dysfunction \[58\]. A premature pulmonary valve opening with an abnormal diastolic flow has also been
observed. Although this is not a disease-specific finding its presence suggests this diagnosis [59].

Even though ARVD is a cardiomyopathy that affects the right side, echocardiographic studies demonstrated abnormalities in the left side. Associated to the left ventricle enlargement it is observed a diffuse or focal wall dysfunction similar to RV that may be progressive in nature [54]. Left chamber dysfunction may be best evidenced with exercise [56].

Echocardiography is an effective tool to diagnose despite its following limitations, sub-optimal images, irregular shape of right ventricle and lack of echocardiographic standard criteria.

Some studies [55-57] showed the echocardiographic sensitivity to diagnose ARVD in asymptomatic patients with clinical suspicion, though the sensitivity is lower when the screening in asymptomatic sportsmen's population without clinical suspicion is performed.

MARFAN SYNDROME
Characteristics
Marfan syndrome (MS) is caused by a genetic flaw that produces an abnormality on the connective body tissue. This disease may occur as a result of spontaneous mutation. It may affect several organic systems such as skeletal, lungs, eyes, heart and blood vessels [60].

The most noticeable physical sign is high stature and long extremities. Ironically these physical characteristics for sports such as basketball and volleyball are idealistically considered [60]. Cardiovascular involvement produces aortic dilatation and mitral valve prolapse in most patients. Abnormalities of the fibrillin of the aorta connective tissue and the valves myxomatous degeneration constitute the anatomic-pathological processes in this disease [61]. The natural history of this syndrome leads to an ascending aortic dilatation and the risk of aortic dissection, rupture and SD [62].

This syndrome is particularly difficult to identify for there are no specific laboratory tests for its diagnosis and for the variable characteristics of this disease.

Echocardiographics Findings
Echocardiography is useful in the evaluation of the aortic valve and proximal ascending aorta the most commonly affected places by MS [63]. The evaluation of the aortic valve should be centered in the detection of aortic insufficiency and the secondary effects on the left ventricle enlargement. Study of the aorta should include besides echocardiographic standard views, parasternal left region, right parasternal (ascending aorta) and suprasternal notch (aortic arch). Additional windows for the visualization of the descending aorta (modified apical and subcostal) may also be used, although these segments of the aorta are less affected by the MS.

Measurements of the aortic dimensions at the aortic annulus level, sinuses of Valsalva, sinotubular junction and ascending aorta should be performed on individuals undergoing screening for aortic pathologies. Standardized criteria have been described to measure the size of the aorta with M-mode and 2D echocardiography. These measures should be adjusted to age and corporal size. Applications of these corporal indexes for the correction of the aortic measures are useful to evaluate athletes of tall stature that may be affected by this disease [64].

Aortic enlargement is the most common finding of MS acquiring the shape of "onion bulb" that represents a malformation with aortic annulus dilatation, sinuses of Valsalva and proximal ascending aorta. Figure 6 shows a 20 year-old volleyball player with MS aortic dilatation and the classical "onion bulb-shape". Disappearance of the sinotubular junction may occur with or without aortic dilatation and it may be the only sign of this pathology [65]. Progression of aortic dilatation leads to aortic regurgitation and risk of dissection or aortic rupture. Aortic regurgitation may occur when the aortic dimension exceeds 50mm and the dissection risk for rupture is high when the dimensions are greater than 60mm [66]. When aortic regurgitation occurs without aortal enlargement, an aortic dissection should be suspected and investigations with transesophageal echocardiography or other techniques should be performed. A significant variation exists in the aortic enlargement in patients with MS [67]. Aortic dissection may occur even with a mild dilatation. Clinical or echocardiographic predictors of the aortic dilatation evolution are not well-known [68]. Therefore echocardiographic follow-ups should be conducted from every 3 months to 1 year [66]. Decisions to the athlete's competitive participation depend on these measures and clinical criteria.
Because MS affects the connective tissue, valvular insufficiencies as prolapse of the mitral valve and/or tricuspid and aortic regurgitation are manifested [69].

It has been observed that mitral regurgitation occurs as a result of mitral valve prolapse with elongation of the chordae and leaflets or as a result of annulus dilatation, produced by left ventricular enlargement, secondary to an aortic regurgitation [64-66].

Recognition of aortic dissection in patient with MS is sometimes difficult due to the transthoracic echocardiographic limits to detect its presence, localization and extension. For this reason the transesophageal echocardiographies as well as other techniques have greater sensitivity [64].

CONGENITAL ANOMALIES OF THE CORONARY ARTERY

Characteristic
This disease is another cause of SD in young athletes and it can be presented in different ways. The most common is an abnormal origin of the left coronary left artery of the right sinus of Valsalva. As a consequence the abnormal coronary artery emerges from the aorta with an acute angle and also runs between the aorta and the pulmonary trunk. These alterations during physical effort may decrease the coronary flow producing angina, arrhythmia and SD [50, 70].

Other anomalies of the coronary arteries are hypoplasia of the right coronary artery and/or the left circumflex artery, origin of the right coronary artery in the left coronary sinus and/or complete absence of the left coronary artery [71].

Another cause of SD attributable to congenital anomalies of the coronary arteries may occur as a result of a myocardial bridge. This occurs when a major coronary artery tunneled or is completely surrounded by a myocardial sheath encircling the intramural coronary segment in a portion of its course. As a result of this constriction the coronary flow is restricted and may produce angina and in some cases SD [72].

Echocardiographic Findings
Recently with the new echocardiographic technological advance, the coronary anatomy can be studied through this technique. The anatomy of the main epicardial branches of the two coronary arteries can be visualized [73]. This visualization particularly favours athletes of aerobic resistance for different reasons: optimum thoracic conformation, cardiac enlargement that brings the heart near to the thoracic wall, prolonged diastolic duration due to bradycardia and increase of the coronary arteries caliber due to training [74]. The
ostium of the left and right coronary trunk may be visualized in the plane of the short parasternal left axis of the aortic root and with mild transducer angulations it is also possible to observe the bifurcation of the left coronary, the initial tract of the circumflex artery and the anterior descending artery [75]. Figure 7 shows a 17 year-old basquetball player with an abnormal growing of the right coronary artery of the left sinus of Valsalva.

![Figure 7: Basquetbolista de 17 años con una anomalia congénita de la arteria coronaria. Eco 2D en plano parasternal del eje corto de la aorta. Se observa la flecha que indica el origen anormal de la arteria coronaria derecha (CD) del seno de Valsalva izquierdo. Ostium de la arteria coronaria izquierda (CS), aorta (AO), ventrículo derecho (VD).](image)

In cases of clinical suspicion, transesophageal echo is more sensitive to recognize coronary artery anomalies [76]. The sensitivity and specificity of this method will subsequently evaluated in prospective studies.

OTHER DISORDERS
Echocardiography can be useful in the detection of other congenital or acquired diseases that may produce SD. These are coronary artery diseases such as Kawasaki disease and atherosclerosis. Annuloaortic ectasia; valvular diseases such as mitral prolapse; aortic stenosis; myocarditis such as idiopathic and sarcoidosis, and cardiomyopathies such as restrictive and dilated may also be identified [20].

Some congenital disorders in young individuals such as valvular aortic stenosis or dilated cardiomyopathy such as Chagas disease are precociously detected by their symptomatology and inability of undergoing intense physical activity and they are not cause of frequent SD in athletes. These diseases also have typical echocardiographic characteristics that facilitate the diagnosis [77].

ECHOCARDIOGRAPHIC SCREENING IN YOUNG ATHLETES
Echocardiography is a highly sensitive and specific test to detect congenital or acquired coronary abnormalities in this population. Hypertrophic cardiomyopathy, MS and valvular disorders are easily identifiable with echocardiography.

But echocardiographic screening in big athlete's populations remains in controversy [78]. A group of authors proposed to use echocardiography as a universal method due to its short order examination and low cost. They consider the clinical history, physical exam and electrocardiogram are not sensitive enough to detect many cardiovascular abnormalities [79]. Other authors consider that the cost/benefit of the echocardiography massive evaluation in athletes is inadequate due to the low incidence of these diseases in the general population. It should be evaluated 200 thousand athletes to identify a thousand at risk and to prevent 1 SD, besides the need of technicians with expertise in this area [80].

CONCLUSION
SD in young athletes is an unexpected and rare event. Diseases such as HCM, MS, ARVD and coronary artery anomalies may be echocardiographically recognized and characterized by a cardiologist with specific knowledge of the most prevalent diseases in the sport population. The decisions-making to allow the athlete its sport participation should be significantly based on the echocardiographic findings. The convenience of a massive echocardiographic screening in young athletes still remains controversial.

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