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GUIDELINE IN SPORTS AND PHYSICAL EXERCISE CARDIOLOGY OF BRAZILIAN SOCIETY OF CARDIOLOGY AND BRAZILIAN SOCIETY OF SPORTS MEDICINE

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Guideline in Sports and Physical Exercise Cardiology of Brazilian Society of Cardiology and Brazilian Society of Sports Medicine

This guideline shall be referred as:

Ghorayeb N., Costa R.V.C., Daher D.J., Oliveira Filho J.A., Oliveira M.A.B. et al. Guideline in Cardiology of Sport and Exercise of the Brazilian Society of Cardiology and the Brazilian Society of Sports Medicine

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Summary

Guideline in Sports and Physical Exercise Cardiology of Brazilian Society of Cardiology and Brazilian Society of Sports Medicine

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If the last three years the author / developer of the Guidelines:

Names Members of the Policy	Participated in clinical studies and / or experimental trials supported by pharmaceutical or equipment related to the guideline in question	Has spoken at events or activities sponsored by industry related to the guideline in question	It was (is) advisory board member or director of a pharmaceutical or equipment	Committees participated in completion of research sponsored by industry	Personal or institutional aid received from industry	Produced scientific papers in journals sponsored by industry	It shares the industry
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Definition of grades of levels of evidence

Recommendations

Class I: Conditions to which there are conclusive evidences, and in its absence, general agreement that the procedure is safe, useful/efficient.

Class II: Conditions to which there are conflicting evidences and/or opinion divergences concerning the procedure safety and utility/efficiency.

Class IIa: Consideration or evidence/opinion favorable to the procedure. It is approved by most.

Class IIb: Safety and utility/efficacy less established, and there is no prevalence of favorable opinions.

Class III: Conditions to which there are evidences and/or agreement that the procedure is not useful/efficient and in some cases can be harmful.

Evidences

Level A: Data obtained from multiple significant randomized studies, according and/or of robust meta-analysis of random clinical studies.

Level B: Data obtained from a less robust meta-analysis, from only one randomized study, or from non-randomized (observational) studies.

Level C: Data obtained from specialist consensual opinion.

It is important to say that evidence levels classified as B or C cannot be interpreted as feeble recommendations. There are many consensual recommendations, therefore with recommendation grade I, with level C evidence (expert opinions). On the other hand, some indications considered controversial (recommendation grade II) can be founded on randomized clinical studies (level A evidence).

Presentation

In the year of 1986 in Belo Horizonte during the XLII Brazilian Cardiology Congress it was formalized, by gathering the signatures of all members, the creation of the Study Group in Sports Cardiology, which was in the beginning directly linked to SBC board, and afterwards a component of DERC.

The scientific fulfillment of this true practice of Brazilian cardiology was concluded with the first Guideline in Sports Cardiology. In the same rhythm of Societies of Sports Medicine and Cardiology all over the world, no doubt, the number of researchers and interested professionals that have their practice in sports and in the physical exercises of sports professionals and population in general will grow. The interest, even governmental, in heart disease prevention starts by the dissemination of physical activities for all. The current reality is the growth of people of all ages practicing physical activities in various modalities of sports.

The need of having a queries base compatible with our habits and usages was fulfilled with this text. The members are cardiologists and sports physicians, which facilitated the necessary integration and a legacy, the guideline, elaborated and within the difficulties of being the first one, to be optimized and periodically changed. The Sports Cardiology, being a new area of cardiology, is based in experience, but seeks to create its evidences in a global level.

Each day new modalities with increasing sports difficulties and intensities are "invented" and immediately incorporated to fitness centers, thousands of new long street races participants turn to us for technical evaluations and questioning, it all demands that the doctor keeps its practice updated, on behalf of more safety to sportsmen. It must be clear that there is no zero risk, despite the specialized emergency apparatus and acquired knowledge.

The fundamental is described in this Guideline, which was further studied in many heart disease aspects to facilitate reader's comprehension. Difficulties and lack of more consistent evidences in certain clinical situations will remain, but throughout the text you can find more pertinent solutions. Consistency with other BSC Guidelines was also sought.

This Guideline sought to approach the pre-participation evaluation and recommendations to prevent fatal and non-fatal events for the athletes, for people in general who practice sports and for para-athletes.

To conclude we would like to remember that, for the first time in a Guideline from Brazilian Society of Cardiology a chapter was added for disabled individuals, the cheered PARA-ATHLETES.

To the collaborators our best regards and congratulations. To the colleagues to use it we hope it can be useful for the good practice of Medicine.

Nabil Ghorayeb

1. Introduction

The pre-participation clinical evaluation (APP, PPE) for physical/sports activities should be understood as a systematic medical examination, uniform, able to comprehend the ample population of sports practitioners and athletes before their release to physical training. Its goal is to identify, or at least improve, the suspicion over cardiovascular diseases that are incompatible with the practice of physical activities seeking efficiency.

This evaluation, performed previous to the physical activity and periodically in its maintenance, has as its mean goal to prevent the development of cardiovascular system diseases (CSD) and sudden death by means of temporary or definite prohibition to practice physical activities or of treatment of the conditions that may be potentially fatal and triggered by physical exercise. The American Heart Association, as well as European Cardiology Society and Brazilian Society of Sports Medicine[1-3] agree on recommending the PPE for all professional athletes. There is also an indication to prescribe correctly exercises for non-professional sports practitioners that perform high intensity activities.

Currently, the main argument against PPE implementation is over the costs involved in this evaluation. Due to the low prevalence of conditions capable of triggering sudden death during sports activities and to the big population of sports practitioners in the country, , a lot has been discussed on which would be the evaluation model with best cost-effectiveness. While some societies as a American Heart Association defend the simple implementation of a questions form and physical examination, believing that the financial and psychological cost bounded to false positive results in the performance of complementary exams do not justify the benefits that might be found, others, like European Cardiology Society, reinforce the use of complementary diagnosis methods, as its use is capable of modifying the occurrence of sudden death in the athletes population[3-7]. Although we do not have randomized works comparing the two evaluation methods, we suggest, like Brazilian Society of Sports Medicine, that PPE should be associated to complementary methods for professional athletes, considering its indication is completely justifiable in the pursue of guaranteeing the athlete's physical integrity and all the costs involved in their training. Another controversial aspect concerns the PPE in children, over which until now there is no consensus about protocols to be followed[6,8,9].

These aspects will be approached in this document, which intends to establish the regulations for PPE in Brazil. For didactic purposes, and also because of the differences concerning physiology, epidemiology and clinical aspects, we have chosen to divide the individuals to be evaluated in three groups: one formed by sports practitioners, other by professional athletes and a third contemplating children and adolescents and also para-athletes or disabled athletes. There will always be an intersection zone among them, when several aspects of the exercise, like intensity, frequency, training volume, etc. are considered. The common sense of medicine practice and the individual experience of the evaluation doctor will be essential in the choice of a path for PPE performance in these cases.

2. Sports group

It is characterized by adult individuals who practice regular physical and sport activities, which intensity may vary from moderate to high, eventually competing, but without professional approach to the sport.

2.1. Anamnesis and clinical examination

The ideal would be that every individual who is candidate to practice sports in moderate/high intensity level be submitted, necessarily, to a medical examination that will allow the detection of risk factors, signs and symptoms of cardiovascular, pulmonary, metabolic or locomotor system diseases[1,10,11]. The classical techniques of clinical anamnesis must be employed with some differences, especially those that favor aspects related to exercise and to the history of family diseases or cardiovascular events related to the practice of sports.

During the anamnesis the Physical Activity Readiness Questionnaire (PAR-Q) developed in Canada, coupled with basic questionings applied by a doctor (Table 1), can be systematically applied. We must pay attention to sudden death or congenital heart disease cases in the family; family history for sickle cell anemia or other hemonoglobinopathies; provenience from endemic areas for Chagas disease or regions in which there is a prevalence of congenital diseases, like descendants from immigrants from the region of Veneto, in Italy, where there is a prevalence arrhythmogenic right ventricular dysplasia[3-12,13]. Special care must be taken when obtaining information related to the use of licit or illicit drugs that may be considered as doping, even involuntary, or that are hazardous to health or can cause sudden death[13,14].

Among the symptoms, we must pay attention to palpitations, syncope, precordial pain or thoracic discomfort; dyspnea on exertion; dizziness/lipothymy; asthenia; or any other symptom triggered by physical exercise. Accurate sensibility is required to evaluate whether the abovementioned symptoms are indicative of a pathological condition or are merely the consequence of a competition or of a more intense training. As for the syncope in athletes, a detail investigation is required, for until evidence to the contrary it should be treated as an episode of sudden death spontaneously aborted[14].

In the physical examination some clinical conditions should be highlighted, as: anemia; postural changes; infectious focuses (e.g., dental); systemic or severe infectious diseases; bronchial asthma, obesity, diabetes mellitus; systemic arterial hypertension; changes in cardiac and pulmonary auscultation; and special situations as pregnancy.

One must prioritize the search for characteristic signs related to the possibility of a heart disease, as: the presence of heart murmur, splitting in the third or fourth heart sounds, valve disease, changes in the palpation of superior and inferior limbs, Marfan syndrome physical features, besides the adequate measuring of blood pressure (on both arms in the first evaluation)[15,16].

Also, in the physical examination one must characterize the eventual muscle-tendon shortening, especially from some muscle groups: hamstrings, quadriceps, iliopsoas, calf, thigh adductors, iliotibial tract, back and shoulder muscles, as the adequate therapeutic guidance can help preventing and treating muscle injuries, improving the sport performance.

Recommendation Grade: I
Evidence level: C

Table 1 – Particularities that should be part of the athlete's personal and familiar history

Particularities that should be part of the athlete's personal and familiar history	
Has any doctor ever said you have some heart problem?	Are there cases of sudden death and heart disease in your family?
Pain or precordial discomfort in exertion or at rest	Do you feel chest pain when practice physical activity? Did you feel any chest pain while practicing physical activities last month?
Pre-syncope or syncope, especially if related to exertion	Do you feel unbalanced due to dizziness and/or loss of consciousness? Are there in your family cases of heart disease, premature sudden death before 50 years old or cardiac arrhythmia?
Arrhythmias	Do you notice palpitations (heart beats skips or accelerations)
Diagnosed Pathologies	Previous history of heart murmur Previous history of arterial hypertension Previous history of metabolic disorder Use of substances to improve training output/use of any medicine Proceeding from Chagas disease endemic zone
Do you have any bone or articular disorder that could be worsened by physical activity?	Do you currently take any kind of medicine? Ask directly about antihypertensive drugs, NSAIDs, anabolic, illicit drugs, alcohol consumption
Is there any other reason why you should not practice physical activity?	Occurrence of genetic diseases in the family; hypertrophic heart disease, dilated cardiomyopathy, canalopathies, arrhythmias, Marfan syndrome

2.2. Complementary exams (non-cardiovascular)

We highlight, among routine laboratory exams aiming health: complete hemogram, fasting glycaemia, urea and creatinine, complete lipidgram, uric acid, hepatogram (AST, ALT, gamma-GT, bilirrubines, PAT/INR), urine exam, feces parasitological exam.

Recommendation Grade: I
Evidence level: B

In individuals subject to practice exercises or compete in heights superior to 2.000 m, it is important the performance

of hemoglobin electrophoresis to discharge the possibility of hemoglobinopathies (e.g., sickle cell anemia). In our environment, especially on those with compatible epidemiology, the serology for Chagas disease is recommended[6,7] (IA). Thoracic teleradiography in PA (posteroanterior) and left profile must be routinely required, because of its low cost and by providing relevant information about cardiovascular diseases, pulmonary diseases and thoracic structure diseases.

2.2.1. Electrocardiogram

2.2.1.1. Introduction

It is known that there are controversies in the approach of younger athletes; the American guideline (AHA/ACC/ACSM) does not contemplate the ECG inclusion, but the Italian protocol, already followed by the European Society of Cardiology (ESC), justifies and recommends its performance.

As regards individuals classified as master or aged, the rest ECG is mandatory; add to that the method's wide availability and low cost. At this age there is a bigger prevalence of cardiovascular diseases, being coronary artery disease the main one. This indication applies even to asymptomatic individuals and/or individuals without knowledge of previous cardiovascular pathology.

2.2.1.2. Method

The conventional 12 lead ECG should be performed de 12 with the individual in supine position, registered in speed of 25 mm/s and obtained at least 24 hours after the last sport activity. Previous rest of at least 5 minutes is mandatory[17,18].

2.2.1.3 Analysis

The exam must be evaluated by a professional physician, cardiologist and with experience in the sport area, so that physiologic changes in the athlete's heart are not mistaken by eventual heart diseases.

2.2.1.4. Changes

The 12 lead ECG has limited diagnosis value for detection of coronary artery disease in population of asymptomatic individuals, mainly because of electrocardiographic patterns variations associated to physical training[17]. It is more useful when performed as part of a pre-participation evaluation, when it can identify non-expected changes, as previous myocardial infarction (in high age brackets), arrhythmias, and conduction disorders, among others.

It can also help in the diagnosis of less prevalent diseases, as MHCM, long QT syndrome, short QT syndrome, Brugada syndrome, WPW syndrome and pre-excitement, besides arrhythmogenic right ventricular dysplasia.

The 12 lead ECG may help in the diagnosis evaluation of aortic and mitral valvopathies, ectopic beatings (ventricular and supraventricular extrasystoles), bradyarrhythmia and tachyarrhythmia, other ventricular and supraventricular arrhythmias, disorders in the conduction system (LBB, RBB, AVB of varying grades). It can also detect changes in the ST segment, as early repolarization, St segment depression or elevation ST, T wave inversion in precordial and/or peripheral leads, raise in the R/S waves voltage suggesting LVE[2,19].

There may be variations in the prevalence of electrocardiography changes, those being greater in the female gender than in the male gender; regarding the age, in the population of masters or aged, we find a greater frequency of inverted T waves in precordial and/or peripheral leads, increased R/S waves suggesting LVE and stimulus conduction disorders.

The variations found in conventional 12 lead electrocardiograms are also statistically significant regarding the type of sport practiced and depending on the age bracket evaluated in table 2.

Recommendation grade: I
Evidence level: A

Table 2 – Different kinds of electrocardiography abnormalities related to age, in a non-selected population of 32,652 individuals[20] submitted to sport pre-participation evaluation in Europe.

	< 20 years old (n= 2430) %	20-29 years old (n=579) %	> 30 years old (n=844) %
Incomplete RBB, increased PR, early repolarization pattern	73.1	37.9	30.1
Inverted T waves	9.5	38.6	37.9
Voltage increase of R/S waves	3.1	4.6	7.2
RBB	10.9	12.1	10.9
LAHB, LBB	2.1	5.7	13.3
Pre-excitement pattern	1.3	1.1	0.6

2.2.2. Exercise testing

The exercise testing (ET) may be indicated in the initial evaluation of a sport practitioner in the early identifying of cardiovascular disease, as well as contribute to prognosis analysis in asymptomatic individuals or when there is reference to some symptom potentially indicative of this condition. Its indication also stands with goal to cardiorespiratory aptness evaluation in training evolution in some sport modalities, notoriously the ones with predominance of aerobic component[21-23].

The cardiovascular overload imposed by physical activity stress, performed as leisure, during the practice of some sport or in competition, requires a great metabolic demand and peripheral hemodynamic. To cover this demand, there is a progressive increase in oxygen consumption by the heart with the objective of making possible the elevation of cardiac debt. The identification of eventual limitations to cover this crescent demand of oxygen by the cardiac muscle can be evaluated by ET.

Although the ET should always be indicated in the PPE, in the specific condition of leisure activities of light and moderate intensities asymptomatic individuals can be released without the need of examination. In further conditions, the ET will always be recommended[24].

In adults up to 65 years old the coronary artery disease is the main responsible by mortality and the ET indication to those individuals aims predominantly the identification of myocardial ischemia, reflecting probable coronary disease (CAD). Also in asymptomatic people and even in those who have a known

CAD the indication of ET is sustained by the need of an adequate prescription of physical activity.

The presence of changes in the rest electrocardiogram, often recurring from the athlete's left ventricular hypertrophy, reduces the diagnosis capacity, in the electrocardiographic aspect, of myocardial ischemia in ET. The simultaneous analysis of other ET variables contributes to the evaluation[25,26].

The response evaluation of arterial blood pressure in the ET enables the adoption of early preventive and therapeutic measures, besides contributing to the correct exercise prescription.

The occurrence of palpitation during physical activity shall be investigated by means of ET with the objective of reproducing under monitoring the patient's complaint. The cardiac arrhythmia diagnosis may then be performed, the adequate treatment being oriented.

2.2.2.1. Variables to be evaluated in the exercise testing

2.2.2.1.1. Exercise capacity

The low exercise capacity expresses a bad prognosis. Its identification will deserve investigation as to the reason of its existence[27,28].

2.2.2.1.2. Thoracic pain

Reproduce by ET the complaint of pain or thoracic discomfort during the exercise will allow the probable myocardial ischemia diagnosis by coronary disease, with consequent investigation proceeding e withdrawal from physical activities. If during the ET completely non-ischemic characteristics of pain are identified and the pain is indicated by the athlete as similar to the one that motivated the ET and there isn't change in any testing variable, the coronary disease probability becomes insignificant, therefore the athlete can be released for competition[29].

2.2.2.1.3. ST-T Segment

ST unlevelling

ST segment sub-unlevelling or supra-unlevelling in slow, horizontal or descendant ascension may characterize CVD, in special CAD, especially when there is concomitantly the reference to thoracic pain or other manifestation that contributes to myocardial ischemia diagnosis[30].

In asymptomatic athletes, when there are ST changes during ET, even if they are not accompanied by thoracic pain and occur in high double product and in high exercise capacity, associated to frequent cardiac arrhythmias, the temporary withdrawal or the postponement of the physical activity start and the proceeding in the cardiovascular disease investigation are justifiable.

This approach may be based on the bigger cardiovascular demand and the bigger risk of cardiovascular events, including sudden death, during exercise or during the practice of sports, noticeably for those over 35 years old and when one or more risk factors for CAD are present.

The ST segment analysis should consider:

The morphologic characteristics, being descendent more serious than horizontal and this more serious than slow lenta[31,32].

The earliness of its appearance during exertion and these changes persistence lately in the recovering phase are conditions that indicate bigger risk and more severe[33,34].

The reason between the depression variation in the ST segment concerning the heart rate variation [(delta ST in mm x 100) / delta HR]. It is considered potentially ischemic when equal or bigger than 1,6[35,36].

The ST depression when it is bigger than 10% in relation to the previous R wave amplitude. This adjustment in the ST segment evaluation deserves to be considered mainly because of the habitual presence of high R waves, expressing left ventricular hypertrophy physiologically present in the athletes[37].

Still related to the ventricular repolarization we must pay attention to the fact that, when starting from a variation in rest ECG, these variations normalization during exercise is an important sign of benignity and good prognosis. Inversely, variations that do not normalize or even get worse with the raise in HR and of double product are often a sign of the presence of subjacent coronariopathy or myocardiopathy[38-40].

Ventricular depolarization

In the presence of a ST segment depression during exertion, an increase in R wave amplitude and simultaneously reduction or maintenance of Q wave amplitude in lateral leads (V4, V5, CM5, V6), increase the diagnosis efficacy for myocardial ischemia[41,42].

2.2.2.2. Systemic arterial blood pressure

Excessive high arterial blood pressure values during the exercise test enable to infer that during the exercise or sport practice the arterial blood pressure shall be very high as well. Sports that require a bigger static component will possibly be accompanied by even higher systolic, diastolic, arterial blood pressure.

The arterial hypertension laboratorial investigation and treatment should begin in sequence, and the sport activities should be interrupted temporarily until better control is reached[43]. On the other hand, progressive fall or systolic arterial blood pressure during ET, especially when systolic arterial blood pressure values inferior to pre-exertion are reached should deserve proceeding in the investigation of presence of cardiac disease.

2.2.2.3. Heart rate

Chronotropic incompetence indicates a bad prognosis and has been associated to endothelial malfunction, change in the autonomic modulation, elevated values in the inflammatory markers and coronary disease.

The heart rate inadequate response may be considered by the inability of reaching 85% of the maximum estimated heart rate. The function 208 (age x 0.70) may be used to predict the maximum heart rate. Topic index under 80% is another method to identify chronotropic incompetency, and it has prognosis value. It is obtained by the reason [(Obtained HR Reserve /Estimated HR Reserve) x 100][44].

The heart rate reduction in the recovery first minute related to the heart rate in the peak of exertion enables to infer the cardiac vagal modulation. This observation was first identified in athletes comparatively to cardiac insufficiency patients[45,46].

Reductions equal or inferior to 12 beatings per minute have been associated to bigger mortality incidence[47].

2.2.2.4. Cardiac Arrhythmias

Cardiac arrhythmias of a lesser level of complexity, as eventual ventricular extrasystoles on ET, often express increase in the sympathetic autonomic modulation imposed by graduated exercise. Such conditions taken isolated, without the presence of other variations do not justify greater restriction to physical activities in asymptomatic individuals.

Symptomatic or asymptomatic individuals, who develop complex ventricular arrhythmias, as ventricular tachycardia, sustained or not, will deserve investigation previous to release to physical activities.

The presence of seven or more ventricular extrasystoles exclusively in recovery phase, or in recovery and exertion, express a potentially bigger risk of future cardiac events[48].

Recommendation Grade and Evidence Level

Physical activity as leisure, of light or moderate intensity, in asymptomatic individual without cardiovascular risk factor: perform exercise test when initiating physical activity program.

Recommendation Grade: IIa
Evidence level: A

Physical activity as leisure, of light or moderate intensity, in asymptomatic individual with cardiovascular risk factor: perform exercise test when initiating physical activity program.

Recommendation Grade: IIa
Evidence level: A

High intensity leisure activity, sport and competition: perform exercise test when initiating physical activity program.

Recommendation Grade: IIa
Evidence level: A

At any moment:

Reference to thoracic pain or discomfort, tiredness or dyspnea of non-defined cause, palpitation, identification of previously inexistent arrhythmias, pre-syncope or syncope related to exercise or arterial blood pressure elevation in rest, compromising or not target organ: perform exercise test.

Recommendation Grade: I
Evidence Level: A

Note: the exercise test requirement will depend on initial clinical evaluation and may be contraindicated or eventually anteceded or succeeded by other complementary exams, if necessary.

2.2.3. Echocardiogram with Doppler

It should be used in the cases of heart disease familiar/clinical history or suspect physical exam, as well as for cases of rest electrocardiogram with positive criteria for cardiomyopathy (Table 3)[49]. Also in known cases of congenital heart diseases, especially those of low complexity, in which the physical activity and even sports practice seeking efficacy are not contraindicated, the periodical echocardiogram performance helps in the evolutive evaluation and in the correct management of the condition in question. We should reinforce the importance of echocardiogram with Doppler associated to physical exertion in situations in which the cardiac function verification during exercise can help in diagnosis and conduct[50].

Recommendation Grade: IIa
Evidence level: B

Table 3 – Criteria to consider a 12 lead electrocardiogram as suggestive of heart disease and of indication of echocardiogram European Cardiology Society (adapted from Pelliccia A)[18].

P Wave
Left atrial enlargement: negative portion of P wave in V1 lead of 0.1mV or less deep and during 0.04 or more seconds.
QRS Complex
Deviation from QRS axle: to the right 120° or more or left -30° to -90°.
Voltage increase: R or S wave amplitude in standard lead of 2 mV or more, S wave in V1 or V2 lead of 3 mV or more, or R wave in V5 or V6 of 3 mv or more.
Pathologic Q waves: during 0.04 seconds or more, or 25% or more of the subsequent R wave height, or QS pattern in 2 or more leads.
Left or right bundle branch block with QRS ≥ 0.12 seconds.
R or R' wave in V1 lead of 0.5 mV or more in amplitude and R/S relation of 1 or more.
ST Segment, T wave and QT interval
ST segment depression, flattened or inverted T wave in 2 or more leads.
Enlargement of QT interval corrected for heart rate bigger than 0.44 seconds in men and more than 0.46 seconds in women.
Variations in rhythm and conduction
Early ventricular beatings or complex ventricular arrhythmias.
Supraventricular tachycardias, atrial flutter or atrial fibrillation.
Short PR interval (<0.12 seconds) with or without delta wave.
Sinus bradycardia with resting heart rate <40 beatings/min or less*.
Atrioventricular block of first (≥ 0.21 seconds†) second or third degree.
*Increasing less than 100 bpm during exertion test.
†Non-shortening with hyperventilation or exertion test.

Regarding athletes with age superior to 35 years old, the standard protocol associated with question form for coronary artery disease specific evaluation should be applied. In men >40 years old and women >55 years old and in individuals with more than 2 risk factors for coronary artery disease (besides gender and age) [51,52] the performance of functional test with exercise should be considered.

In case of positive in the initial evaluation for coronary artery disease the investigation must go deeper with more accurate exams, like echocardiogram at rest, stress echocardiography or myocardial tomographic scintigraphy, evaluating each case individually.

Recommendation Grade: IIa
Evidence level: B

2.2.4. Other complementary exams

The use of other diagnosis tools, be it laboratory, charts, graphics methods, invasive or not, should comprise to clinical criteria and scientific evidences established in literature, as function of those found along the PPE.

Specific details on the indication and interpretation of those complementary exams will be found on the topic about sudden death and cardiovascular events prevention in physical and sports activity in this guideline.

3. Athletes group

This group is characterized by individuals who practice physical and sports activities in a regular and professional basis, systematically competing, with professional attachment to sports through clubs and/or sponsors of any kind. This group works always in pursue of records and limits overcoming, often submitting themselves to high intensity training load which places them invariably under intense physical and psychological stress, with frequently hazardous consequences.

3.1. Anamnesis and physical examination

The athletes' pre-participation evaluation (PPE) demands a manageable strategy regarding economics, especially in a country in development such as Brazil, with continental dimensions, well-populated and with great social and economic diversity. The cardiologic approach deserves special attention, as the event of sudden death depends mainly on cardiovascular causes. In this context, it is fundamental a correct anamnesis and physical examination[53,54].

It should be considered in the anamnesis, contributing to a correct interpretation of the physical examination, that the cardiac adaptations to physical exercise depend on its frequency, quantity and intensity, varying according to different demands observed in several sports modalities, different training systems and also individual responses[55]. This last aspect is what makes it possible that individuals submitted to similar physical activities have different cardiac characteristics. Therefore, there are HR and SAP variations that depend on the habitual physical activity and also on characteristics proper of each individual. It should be noted that the variations that produce the athlete's heart syndrome should, for starters, be considered as normal physiologic adaptations to physical exercise, transitory and with no negative results on health[56,57].

Laboratory exams are not required at first; its requirement should come from the clinical data obtained, with emphasis on cardiovasculatory aspects. The laboratory exams listed in the first part of this document (sports group) should also be considered in the athletes' group.

Recommendation Grade: I
Evidence level: A

3.1.1. Considerations on overtraining syndrome (OTS)

Many times the magnitude and the clinical repercussions of cardiovascular variations configure a health problem as the overtraining syndrome should be considered. Because it is a profile that is difficult to ascertain and depends essentially on the clinical analysis for its presence to be suspected, we have chosen to include here its description and the recommendations to manage it. In the confirmation of diagnosis, the laboratory sometimes has a fundamental role.

There are two types of overtraining: the sympathetic, or Basedow type, and the parasympathetic, or Addison type.

The OTS of parasympathetic type is more common in veteran athletes. It is not always followed by decline in sports efficiency. Many times it manifests only by some lethargic state, consequence of great vagal predominance. The sympathetic reflex responses are inhibited, which can lead to orthostatic hypotension, dizziness, faintness and Stokes-Adams episodes in daily life situations.

The OTS of sympathetic type, which is more common, usually causes decline in sports efficiency, irritability, insomnia, loss of appetite, profuse sweating, persistent tachycardia and other tachyarrhythmias, slow return to basal heart rate after physical exertion and arterial hypertension. It may indicate an accentuated catabolic process, with loss of weight by loss of muscle mass, and even osteoporosis and stress fractures. Laboratory variations may occur, with increase in urinary nitrogen excretion, reduction of anabolic hormones, like testosterone and growth hormone, and defense factors depletion with less immune resistance. By configuring the catabolic process, the anabolic hormones reduction is accompanied by increase of catabolic hormones, like cortisol. Thus, the reason serum cortisol / serum testosterone usually presents values above normal. The decrease in efficiency and the related symptoms demand immediate reduction or even interruption of physical exercises, in long resting period. The recovery time is variegated, requiring monitoring to indicate the best time to return to activities.

3.2. Electrocardiogram

Sports practice promotes morphofunctional variations in the heart related to training time and intensity, which are demonstrated in ECG at rest, like ventricular overload, atrioventricular conduction and rhythm disturbances, imposed by the exercise's vagal exacerbation. Abnormalities that can put the athlete in risk during sports practice can be detected, representing the expression of a subjacent heart disease. When an athlete's ECG is analyzed, the main goal is to discern between physiological patterns and those that require action and/or additional tests to exclude (or confirm) the suspicion of subjacent cardiovascular disease leading to increased risk of sudden death in sport[58].

Mistakes in the differentiation between physiologic and pathologic may have serious consequences. Athletes can be unnecessarily disqualified for competitions because of ECG variations which are inserted in the normal interval for athletes. On the other hand, they can be submitted to unnecessary exams, raising in much the evaluation costs. This is particular relevant for the professional, as the competitor disqualification generates psychological and eventually financial troubles. Alternatively, potentially fatal cardiovascular disease signs can be wrongly interpreted as variant from normal in an athlete's ECG.

The peculiarities of athletes' electrocardiogram in rest imply that the exam interpretation should be performed by cardiologist doctors with experience in the exercise physiology area or sports doctor with training in the cardiology area.

Currently available data enable to define the ECG usefulness and cost-effectiveness of use in pre-participation evaluation[59,60]. The Sociedade Brasileira de Cardiologia (Brazilian Cardiology Society) recommends it in every initial cardiologist appointment. The European Cardiology Society and the International Olympics Committee, the FIFA, indicate its performance in everyone who wishes to ingress in a leisure or competitive physical exercise program[61]. The American Heart Association does not indicate it as mandatory

in pre-participation evaluation for the practice of exercise or sports[62]. In Brazil, in many institutions where athletes are evaluated its performance is mandatory.

3.2.1. Variations considered physiologic X suggesting heart diseases

The efforts concentrate on better understanding interpretation basis, defining ECG criteria to discern between athlete's heart and actual heart diseases. More studies are necessary to test if the accuracy, usefulness and cost-effectiveness of ECG presents criteria variations regarding gender, age, ethnics and different levels of training and/or type of sport[63,64]. The variations in athletes' ECG may be divided in two groups: commons and/or related to sports training (group1) or sporadic and/or suggestive of heart diseases (group2), as listed in table 4.

Table 4 – Variations considered physiologic X suggesting heart diseases

Variations related to training	Suggesting heart diseases
Sinus bradycardia/arrhythmia	T wave inversion
First-degree atrioventricular block	ST segment depression
Second-degree atrioventricular block (Mobitz I)	Pathologic Q waves
Isolated left ventricular overload	Left atrial overload
Right bundle branch conduction final delay	Second-degree atrioventricular block (Mobitz II)
Early repolarization	Ventricular pre-excitement
	Right or left bundle branch block
	Short or long QT interval
	Suggesting Brugada syndrome
	Right ventricular hypertrophy
	Electrical axis deviation

Athletes often (up to 80%) show electrocardiographic variations as sinus bradycardia/arrhythmia (13-69%), first-degree atrioventricular block (35%), early repolarization (50-80%), resulting from physiological adaptations recurring from the increase in vagal tone. They can also present voltage criteria (i. e., based only in QRS amplitude measurings) for physiological left ventricular hypertrophy that reflects the remodeling of this cavity, without the concomitant presence of pathologic Q waves, electrical axle deviation, atrial overload and repolarization variations[65,66].

Those physiological variations in the ECG must be clearly separated from patterns suggestive of heart disease recognized by repolarization variations, pathological Q waves, electrical axle deviation, intraventricular conduction defect, pre-excitement, short/long QT interval and Brugada. Those findings are rare (5%), but may be the expression of heart diseases, genetic inheritance and canalopathies, which predispose to sudden death[67].

Variations caused by cardiac adaptation to physical exertion (group 1) should not cause alarm and the athlete may be released to practice competitive sports without further evaluation, as should continue under investigation athletes with signs suggestive of heart diseases even though asymptomatic, in the absence of positive family history or abnormal findings in the physical exam[68-70].

Recommendation Grade: I
Evidence level: A

3.3. Exercise test

The exercise test (ET) may be indicated in the initial evaluation of athletes in any age bracket as part of the strategy for early identification of cardiovascular disease, as well as to contribute in the prognosis analysis, in asymptomatic or when there is reference to any symptom potentially indicating this condition[71,72].

Your requirement will depend on the initial clinical evaluation and may be contra-indicated or eventually anteceded or succeeded by other complementary exams, if necessary.

The considerations on method made in the chapter dedicated to sports practitioners are totally applicable for professional athletes. We suggest, therefore, that the text quoted in this document be read.

Recommendation Grade: I
Evidence level: A

3.4. Cardiopulmonary test

For a long time, including in Brazil, athletes and individuals who participate in high efficiency physical activities have been submitted to the maximum cardiopulmonary exercise test (MCET), aiming performance evaluation and aerobic training prescription[73]. As described in table 5, MCET differs from conventional exercise test primarily by the addition of measuring and analysis of exhaled gases[74,75]. According to its primary objectives, when performed in athletes apparently healthy, contrary to exercise tests aiming clinical diagnosis, many times the arterial blood pressure measurings and even electrocardiogram are not performed, and the heart rate measuring is in this case obtained by the use of frequency meters.

Table 5 – Main differences between conventional ET and MCET

EXERCISE TEST		
Variable	Conventional	Cardiopulmonary
Functional capacity	Measured	Measured
Maximum aerobic power	Estimated	Measured
Anaerobic threshold	Undetermined	Determined
Ve/Q Reason	Non-evaluated	Evaluated
Inotropic answer	Limited evaluation	Excellent evaluation
Transients	Undetermined	Determined
Mechanical efficiency	Presumed	Measured
Protocol	More dependent	Less dependent
Actual maximum	Presumed	Probable/identified
Dyspnea etiology	Non- identified	Probable/identified

With hundreds of equipments available today (in laboratories, clinics, hospitals, sports clubs and centers) capable

of performing these measurings, it actually makes little sense to perform athletes' aerobic condition evaluation by a conventional exertion test, in which there is approximately 20% error when estimates based on formulae developed for clinical protocols for treadmill or cycloergometers. It is apparently consensual among physicians and physical education professionals that work with athletes that when performing a MCET is impossible, the tendency is to choose field tests like the 2,400 m or even the traditional Cooper test instead of the conventional exercise test.

In the clinical context of pre-participation evaluation of athletes and individuals who wish to start practicing intense aerobic exercises, the MCET has many advantages over the conventional exercise test, as illustrated in table 5, besides the objective and validated measurement of aerobic condition or maximum VO2. In certain clinical circumstances and for determined differentiated groups of athletes, for example master athletes or those individuals with cardiovascular and/or pulmonary infirmities who are involved with recreational sports competition (e.g., half marathon races, marathon races, high mountain climbing, road cycling competitions, water circuits, etc.) or professionally, the inclusion of MCET in their pre-participation evaluation may be recommended or even fundamental for the individual risk stratification, because of aggregated information by measure and analysis of exhaled gases. In these circumstances and whenever possible, the use of closest ergometer for the sports activity practiced should be preferred.

With the crescent involvement of middle-aged individuals in mass sports events[76], it is probable that MCET will be used to obtain objective and valid subsidies that orient the training more scientifically or to have more safety in the characterization of a low clinical risk for unfavorable cardiovascular events, especially those that have coronary risk factor.

Among the several possibilities of additional clinical information derived exclusively from a MCET in the physically active healthy athlete or individual, two stand out: a) more accurate and objective identification of limiting factor(s) for maximum exertion (cardiovascular, respiratory and muscular or metabolic) and b) evaluation of systolic volume behavior, obtained by the analysis of curves and of oxygen pulse maximum values (VO2/FC) and of ventilatory equivalents (VE/VO2 and VE/VCO2) when an incremental and maximum protocol is performed during at least 8 to 10 minutes (more commonly ramp protocol)[77].

In many cases, especially when considering the high relative prevalence of electrocardiography abnormalities of male athletes with long years of predominantly aerobic training, compromising at least in part the diagnosis specificity and sensibility of tracings obtained in exertion, the behavior of cardiopulmonary variables obtained during the MCET may contribute to solve doubts and adequately stratify the individual risk of a given athlete. In other situations, a systolic arterial blood pressure reduction in the MCET last minutes in an apparently healthy athlete, when accompanied by normal ventilatory responses, probably reflects an accentuated reduction of peripheral vascular resistance resulting from metabolic acidosis with a preserved or even crescent cardiac debt and not an acute dysfunction of cardiac inotropism/lusitropism recurring from myocardial ischemia.

The MCET is already incorporated in the cardiologic practice to evaluate heart failure patients[56] and to identify the etiology of exertional dyspnea[57] and recently considered as capable of identifying myocardial ischemia[58] or abnormal responses after cardiac surgeries[59].

With athletes, it is the procedure of choice when one wishes to obtain a valid measure and needs the aerobic condition and the determination of threshold heart rate to prescribe the exercise.

Recommendation Grade: I
Evidence level: A

It is for a more accurate stratification of the exercise limiting factor.

Recommendation Grade: II.a
Evidence level: A

When there are changes in the rest electrocardiogram that may interfere in its interpretation to exercise or suspect hemodynamic responses.

Recommendation Grade: II.a
Evidence level: B

Its routine use in apparently healthy children and adolescents, just for the purpose of stratify the risk of sudden death to exercise do not seem particularly useful.

Recommendation Grade: II.b
Evidence level: B

3.5. Echocardiogram

The pre-participation evaluation's goal is to prevent occurrences that could risk the integrity of individuals who practice recreational or competitive physical activities, prioritizing the identification of structural and/or functional cardiovascular variations that could increase the risk of morbidity and mortality during this practice[40,60,61].

The use of complementary methods in the routinely cardiovascular evaluation is controversial, and the most relevant aspect of this strategy is the cost-efectiveness[39]. In this scenery, echocardiography has an essential role, by the possibility of diagnosing the main diseases implied in athletes' sudden death (Table 6) and differentiating athlete's heart physiological variations from hypertrophic cardiomyopathy pathologic hypertrophy in an innocuous, fast and relatively low cost way.

Table 6 – Main causes of sudden death in athletes

Age > 35 years old	Age < 35 years old
Coronary artery disease	Hypertrophic cardiomyopathy
	Arrhythmogenic right ventricular dysplasia
	Anomalous origin of coronary arteries
	Myocarditis
	Valve disease
	Pre-excitement syndromes
	Conduction system disorder

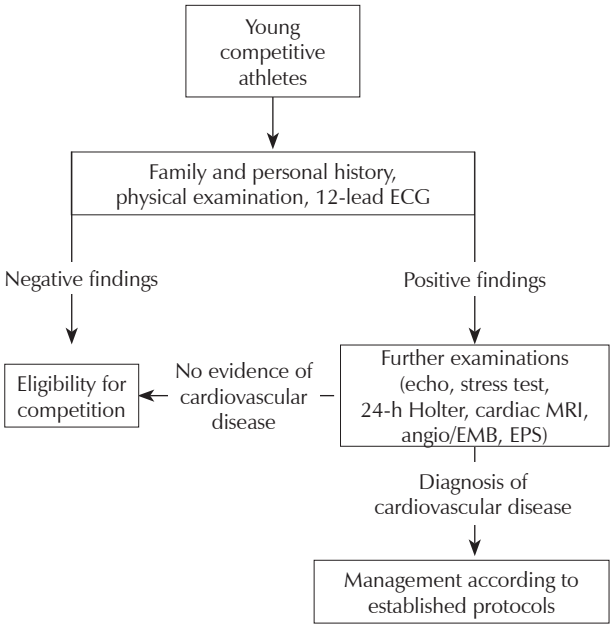
Adapted from: Herz Sports and Sudden Cardiac Death in Athletes. Can It be Prevented by screening. Ghorayeb e cols.[62]

The sudden death in young athletes (12-35 years old) affects, mostly, afro-descendants individuals (>50%) and of the masculine gender (1:9). However, the risk of sudden death do not depend on the competition level (school, amateur, professional)[63-65].

3.6. Recommendations

Institutions internationally known as the European Cardiology Society (ECS), the International Olympic Committee (IOC) and the American Heart Association (AHA) differ as to the definition of screening diagnosis exams to establish the public health investigation protocols[66-68]. This question's complexity lies in the relative infrequency of sudden death in young athletes (between 1:100.000 and 1:300.000 per year), besides the questionable efficiency in preventing its occurrence, being a fact that this event invariably causes repercussion and commotion in the society[3,5].

Considering this scenery, the European Cardiology Society (ECS) supported by the consistent Italian experience, which monitored athletes for 25 years, determines in its screening program for individuals between 12-35 years old an initial exam composed by family history, physical examination, 12-lead ECG, and additional exams will only be performed with positive findings during the initial evaluation. (figure 1)[17,69].



Adapted from: Thiene G, Corrado D, Basso C, Pelliccia A. Sudden cardiac death pre-participation screening of young competitive athletes for prevention of sudden cardiac. Pelliccia e cols.[69].

Figure 1 – Valuation protocol for young athletes.

This strategy was effective in reducing in almost 90% the annual incidence of sudden death in athletes in the Italian northeast[40].

The use of echocardiography in population programs in young asymptomatic athletes (12-35 years old) proved to be a high cost strategy and to the moment there is no population study with adequate follow-up to prove its efficacy[63-65].

Regarding athletes with age superior to 35 years old, the standard protocol associated to questionnaire for specific evaluation of coronary artery disease should be applied. In men >40 years old and women > 55 years old should be considered the performance of functional test with exercise and in individuals with more than two risk factors for coronary artery disease (besides gender and age)[1,70].

In case of positivity in the initial evaluation for coronary artery disease the investigation should proceed with more accurate exams, and echocardiogram at rest or stress echocardiography may be used, evaluating each individual case[40,41].

Several studies have suggested the introduction of limited echocardiographic modalities with restricted two dimensional examination being performed in 5 minutes. The results show a good sensibility and specificity for the diagnosis of several illnesses related to sudden death in athletes, especially the hypertrophic cardiomyopathy implied in more than 30% of the cases of sudden death in young athletes[71].

Table 7 – Recommendations according to age and competitive level

	Leisure	Amateurs	Professionals
Children / adolescent	Initial evaluation + 12-LEAD ECG	Initial evaluation + 12-LEAD ECG	Initial evaluation + 12-LEAD ECG
18-35 years old	Initial evaluation + 12-LEAD ECG	Initial evaluation + 12-LEAD ECG	Initial evaluation + 12-LEAD ECG
35-59 years old	Initial evaluation + evaluation of risk of CAD +12-LEAD ECG + (consider functional test)	Initial evaluation + evaluation of risk of CAD +12-LEAD ECG + (consider functional test)	Initial evaluation + evaluation of risk of CAD +12-LEAD ECG + (consider functional test)
>60 years old	Initial evaluation +12-LEAD ECG + functional test	Initial evaluation +12-LEAD ECG + functional test	Initial evaluation + 12-LEAD ECG + functional test

The recommendations in table 7 take into consideration the formulation of a population screening investigative strategy for public health policies and cannot be fitted to take decisions in singular cases, based in studies result and population records founded in cost planning programs.

Currently, professional athletes' associations and executive boards have their own protocols, due to legal and economic issues involved in the professional aspect[40].

The pre-participation evaluation based in initial medical appointment and 12-lead ECG enables the identification of athletes under greater risk of sudden death, however it is not a definite screening strategy in this segment. Other more accurate diagnosis methods will have more space as initial screening exam in the future[72-74,78].

Currently, echocardiography represents the confirmatory diagnosis modality to be performed after suspicion during initial pre-participation evaluation.

Recommendation Grade: I

Evidence level: A

There is no evidence for its routinely use in population screening programs in asymptomatic individuals.

Recommendation Grade: III

4. Children and adolescents group

Characterized by individuals who practice sports and physical activities in a very variable way, engaging mostly in recreational sports that reach moderate and high intensity levels, being as competitive as the athletic levels, even without systematic training, in certain situations they even surpass athletic levels. It is, therefore, impossible to discern between athletes and non-athletes in terms of intensity and energetic expenditure. They are children and adolescents that may compete systematically, sometimes already professionally linked with sports through clubs and sponsors of any nature. Sometimes they are submitted to intense physical and psychological stress, including, in some situations, by the parents or carers questionable positioning, when they should always be oriented as to inherent risks of such practice in this age bracket[12,75].

The main causes of sudden death more often related to physical activities in this age bracket[3] are: hypertrophic cardiomyopathy, congenital coronary artery anomaly, arrhythmogenic right ventricular dysplasia, Marfan syndrome cardiologic variations Marfan (aortic rupture), pre-excitement syndrome (WPW), Brugada syndrome, long QT syndrome, hemodynamic and arrhythmic repercussions of congenital heart diseases, myocarditis, comottio cordis, Chagas disease, infections and others like conduction dysfunction, hydroelectrolytic variations, sickle cell anemia, besides undetermined causes. In Brazil we don't have statistics epidemiologic records of sudden deaths related to sports practice in this age bracket, being restricted only to report of cases in publications. The above-mentioned order is therefore selective, and its prevalence in the population studied is not characterized.

A competitive athlete is one which takes part on an organized team or individual sport that requires systematic training and regular competitions aiming a goal, be it a prize or excellence in the modality. In general they are federates and/or members of sports clubs. Especially at this age bracket, the differentiation between "competitive" athletes and non-athletes, as mentioned before, is almost impossible.

The sports pre-participation evaluation has been developed and has become more formal, being a legal attitude in some countries, like Italy. An already established global consensus is that of the performance of a well-conducted clinical history, focusing mainly on symptoms and personal background as well as heart disease family history, mainly the occurrence of sudden death. The best strategy to perform the PPE on this age bracket is still a matter under discussion in several countries[3,75]. Such data may be obtained through validated questionnaires like the one of the American Heart Association (AHA) and of the Recommendations on Sudden Death of the International Olympics Committee (Lausanne), adapted on table 1.

4.1. Specific evaluation: Tanner stages I [79,80]

In the growth and development process, we have a sequence of pubertal events that culminate with the emergence of secondary sexual characters resulting from hormonal maturation, systematized by Tanner and classified in five stages, taking into account, in the feminine gender, the mammary development and the quantity and distribution of pubic hair, and, in the masculine gender, the genital organs aspects and also the quantity and distribution of pubic hair. The evaluation of these parameters is essential, as the increase in muscle mass, height, weight, besides motor specialization are related to them. Although there is constancy in the Tanner stages sequence, the time to pass from one stage to another is much variable, and thus the sexual maturation may last from 2 to 5 years.

The great increment in physical growth that occurs in puberty, the known growth spurt, is the phase in which the individual grows most. Usually, the growth acceleration in the feminine gender occurs in the beginning of puberty, between stages 2 and 3, and always antecedes the menarche, which usually coincides with the phase of growth deceleration, and with stage 4. In the masculine gender, the acceleration usually happens in stage 3 and its peak is in stage 4.

Electrocardiogram at rest in this group follows the same controversy already described as routine examination. Because of the extreme regional differences in the country[79,81] and taking into account the scientific evidences available, we recommend the ECG as follows:

Children and adolescents who are healthy and asymptomatic and have no important clinical fact observed in the initial medical evaluation with detailed clinical history and detailed physical examination, who meet the above-mentioned questionings.

Recommendation Grade: IIa
Evidence level: C

In the PPE of children and adolescents of 5 to 18 years old starting organized and competitive training in sports schools, fitness centers and clubs.

Recommendation Grade: I
Evidence level: A

In all children and adolescents with some suspicion of heart disease, detected with the data obtained during medical evaluation.

Recommendation Grade: I
Evidence level: A

4.2. Laboratory exams

Regarding the performance of laboratory exams, in the age bracket in question they assume some importance for the control of situations that are frequent in this population like parasitosis, anemia, alimentary mistakes and even some cases of malnutrition. We consider the exams listed below as passible of indication, always depending on previous clinical evaluation[76,77]:

Hemogram: hemoglobin analysis, associated to anemia and leukopenia, very common mainly in young people in training, and can cause an increase in the risk of superior aerial ways infection.

Iron and ferritin: it helps in the diagnosis of iron deficiency anemia and is also useful to detect overtraining syndrome, in which we find low levels of ferritin.

Sodium, potassium and chloride: detection of hydroelectrolytic disorders.

Lipid profile and glycemia: nowadays, due to constant mistakes in the nutrition of children and adolescents, it is becoming important in the primary prevention of hypercholesterolemia in adult age.

Chagas Disease Serology: it is still an endemic disease in Brazil and South America, cause of myocardiopathy, frequently sub-diagnosed, mainly individuals of a lower socioeconomic level.

Coprologic: identification of parasitosis.

Hemoglobin electrophoresis: evaluation of hemoglobinopathies like sickle cell anemia, important in the eligibility of competitive individuals.

Recommendation Grade: II
Evidence level: C

In selected cases with personal or family heart disease background, if variations in physical exam or electrocardiogram arouse, a deeper cardiologic evaluation is required before release for sports activity. This evaluation may include the performance of transthoracic echocardiogram, maximum exercise test and 24 hour holter[77].

In official statement, the Sociedade Brasileira de Medicina do Esporte (Brazilian Society of Sports Medicine) recommends that, from the public health point of view, children and adolescents may take part in recreational physical activities of low or moderate intensity without the need of a formal pre-participation examination. When the goal is competitive sport or high intensity physical activity, the youngster needs a medical and functional evaluation, including body composition and aerobic and anaerobic capacity[76].

Recommendation Grade: IIa
Evidence level: B

The American Heart Association, in document published in 2006 about exercise test in children and adolescents, describes among the main indications in this age bracket: 1- the specific evaluation of symptoms or signs induced or aggravated by exercise; 2- evaluate or identify abnormal response to exercise in children with heart diseases, pulmonary diseases or diseases in other systems, including the presence of myocardial ischemia and arrhythmias; 3- evaluation of the functional capacity for recreational or athletic activities[77].

Recommendation Grade: IIa
Evidence level: B

The 36th Bethesda conference and the Recommendations for competitive sports participation in athletes with cardiovascular disease refers that the exercise test performance is indicated in the pre-participation evaluation of individuals with congenital heart diseases, valve diseases, myocardiopathies, arterial hypertension, arrhythmias and other conditions of suspected or diagnosed disease with the goal of evaluating functional capacity, symptoms, arrhythmias and orientate the allowed intensity for exercise[81].

Recommendation Grade: I
Evidence level: A

4.2.1. Echocardiogram

Echocardiography represents a confirmatory diagnosis modality to be performed after suspicion during initial pre-participation evaluation. In children and adolescents it becomes a extremely useful exam in the presence of abnormalities in the physical exam that induce to suspicion of structural heart disease, especially when heart murmur is detected.

In cases in which ECG variations are detected, its performance is part of the arsenal for differential diagnosis of severe heart diseases that could potentially trigger sudden death during physical activity, as hypertrophic cardiomyopathy and arrhythmogenic right ventricular dysplasia[1,12,79,80].

Recommendation Grade: I
Evidence level: A

To the moment there is no evidence to justify its routinely use in population screen programs in asymptomatic individuals.

Recommendation Grade: III

5. Group of carriers of cardiomyopathies and myocarditis

5.1. Hypertrophic cardiomyopathy (HCM)

Hypertrophic cardiomyopathy is an autosomal dominant disease characterized by myocytes myofibrillar disarray, followed by hypercontractility, hypodyastoly, asymmetric septal hypertrophy with or without obstruction of left ventricle outlet. This disease is the main cause of sudden death related to exercise and sports in individuals under 30-35 years old in North America, while in Europe arrhythmogenic right ventricular dysplasia prevails as main cause of sudden death in this age bracket[3,82].

To the moment 12 genes are known which are implied in the genesis of this disease with more than 400 genetic mutations related to myocardial contractile proteins[83]. HCM carriers can be completely asymptomatic or present dizziness, syncope, especially related to exercise, dyspnea, palpitations and angina. Sudden death may occur to this patients due to ventricular and supraventricular arrhythmias (with pre-excitement or not), complete AV block, asystole and myocardial infarction[3].

5.2. HCM complementary exams

5.2.1. Electrocardiogram

About 95% of the patients with HCM present electrocardiographic variations, the ST segment and the T wave being commonly affected. Around 50% of the electrocardiograms present signs of left ventricular overload[84] and the pathologic Q waves are present in 30% of the cases. It should be noted that such finding may precede the pathologic hypertrophy that will be found lately in the echocardiogram[85]. The HCM Q waves arise by increase of the electrical forces generated in the hypertrophy areas. Its direction and magnitude are related to the vector that results from the areas with greater ventricular hypertrophy[84]. McKenna et al[86] proposed a series of five echocardiographic, electrocardiographic and clinical criteria for the diagnosis of this disease, especially when its origin is in the family. Table 8 synthetizes the main electrocardiographic variations in patients with HCM.

Table 8 – Electrocardiographic variations in HCM

Hypertrophic Cardiomyopathy	Electrocardiographic Variations
P Wave	1. Left atrial overload
	2. Right atrial increase: amplitude increase in DII, III or V1 > 0.25 mV
QRS Complex	1. QRS electrical axle deviation in frontal plane to right (+120°) or to left (-30° to -90°)
	2. Voltage increase: - of R wave in frontal plane > 2 mV or in V5 and V6 > 3 mV - of S wave in V1 or V2 > 3 mV - R or R' in V1 > 0.5 mV - Reason R/S > 1
Q Wave (except in aVR)	1. Duration > 0.04 min
	2. Reason Q/R > 25%
	3. Amplitude > 3 mm in two contiguous leads
	4. QS Pattern in two or more leads
	5. Absence of normal Q wave
QRS complex duration	Bundle branch blocks (right or left) during > 0.12 sec
Ventricular Repolarization	1. ST Segment - ST segment sub-unlevelling or supra-unlevelling in two or more contiguous leads
	2. T Wave - Plain or inverted in more than two leads, except in children - Amplitude > 10 mm
	3. QTc Interval - Duration > 0.44 min for men or > 0.46 min for women
Hypertrophic cardiomyopathy	Electrocardiographic variations
Variations in atrioventricular rhythm and conduction	- Complex ventricular extrasystoles or arrhythmias - Supraventricular tachycardia, atrial flutter or fibrillation - Short PR interval (< 0.12 min) with or without delta wave - Sinus bradycardia at rest (frequency < 40 bpm) - First degree AV blocks (PR > 0.21 min, except for athletes), - Second degree AV block and complete AV block

5.2.2. Echocardiography

The differentiation between hypertrophy recurring from MHCM and that secondary to physical training (ventricular hypertrophy of the athlete's heart) requires the integration of several information with the pattern and distribution of the hypertrophy, parietal thickness, the size of the cavities, the evaluation of diastolic function by tissular Doppler and yet the family history. Furthermore, it is important to evaluate the echocardiographic response of athletes under suspicion to a sports untraining[87]. Regarding the prognosis, the identification of a septum bigger than 30 mm is a bigger risk factor for sudden death, especially among adolescents and young adults[88]. The echocardiogram enables the characterization of the type and extension of the compromising of the hypertrophic ventricle in an expressive number of cases, but magnetic resonance may be useful in those in which the echocardiography was not enough to reach diagnosis[89,90].

5.2.3. Transthoracic Doppler echocardiogram

Class: I
Evidence level: B

Confirm the clinical suspicion of MHCM, determining the parietal thickness and the presence of dynamic gradient.

Investigate the presence of MHCM in first-degree family members.

Re-evaluation of clinical evolution and therapeutic interventions.

Myocardial contrast echo post-septal ablation to evaluate the size of the infarcted area.

Class II
Evidence level C

IIa: annual re-evaluation of family members of patients with MHCM between 12 and 18 years old. In individuals

who are more than 21 years old the re-evaluation should be performed every 5 years;

IIa: tissular Doppler to differentiate MHCM from athlete's physiologic ventricular hypertrophy or hypertension pathologic hypertrophy;

IIb: stress echo with exercise in patients symptomatic to habitual routine exertion that do not put in evidence significant gradients at rest or with Valsalva's maneuver.

5.2.4. Transesophageal Doppler echocardiogram

Class: I
Evidence level: B

Patients with inadequate transthoracic window;

To evaluate valve compromising, mechanism and magnitude of mitral regurgitation when it is not clear in the transthoracic echocardiography;

Intraoperative evaluation in myomectomy and in septal ablation by alcoholization.

Class: IIa
Evidence level: B

Clarify the mechanism of an atypical mitral insufficiency.

5.2.5. Exercise tests and (HCM)

The exercise test (ET) presents independent value for the identification of patients under increased risk of sudden death. The presence of marked variations of the ST segment in patients with HCM may indicate ischemia without necessarily representing coronary infirmity. In this case the ET presents little diagnosis value, but could be a prognostic marker that should be taken into consideration[84].

5.2.5.1. Indication of ET in patients with HCM

Class: I
Evidence level: B

Asymptomatic patients that do not present high risk criteria as associated element in prognostic stratification;

Asymptomatic patients that do not present high risk criteria and that wish to perform recreational physical activity;

ET as associated element in differential diagnosis between athlete's heart syndrome and HCM.

Class: IIa
Evidence level: C

Patients with doubtful symptoms not associated with other high risk criteria;

Sub-maximum ET in patients with implanted defibrillator that wish to perform low intensity physical activity aiming to evaluate functional capacity;

Sub-maximum ET in patients with implanted defibrillator that wish to perform low intensity physical activity aiming to evaluate heart rate response to exercise.

Class: III

Patients with high risk criteria without defibrillator;

Conventional ET aiming differential diagnosis between HCM and sports practitioner physiologic hypertrophy.

5.2.5.2. Role of exercise cardiopulmonary test in HCM

In athletes that are in the so-called gray zone the athlete's heart physiologic and the MHCM pathologic hypertrophy, the oxygen consumption and peak oxygen pulse measuring through the exercise cardiopulmonary test (MCET) may be of great help. Sports practitioners highly trained generally present values between 55–70 $mL.Kg^{-1}min^{-1}$. Sharma et al[89] demonstrate that those athletes with no factual disease had a peak oxygen consumption significantly more elevated than athletes with HCM (66.2 $mL.Kg^{-1}min^{-1}$ versus 34.3 $mL.Kg^{-1}min^{-1}$). It is important to highlight that there wasn't superposition among the groups. Therefore, the investigators established as cut-off the cypher of 50 $mL.Kg^{-1}min^{-1}$ to distinguish HCM pathologic hypertrophy from that secondary to the athlete's heart.

5.3. Sport and HCM

Why is there low prevalence of HCM in high level athletes? The answer seems to be related to a natural selection process that excludes individuals with functional and structural variations secondary to HCM from the high intensity regular training required for someone to be an athlete[89]. Being a competitive athlete who has HCM is synonymous of sudden death? The answer is no. We all know cases of athletes who received a diagnosis for this infirmity when they already had quit their professional competitive activity or even while they were still on it. The problem is that intense exercise may be a trigger to launch severe arrhythmias, increase in the obstruction of left ventricle outlet and/or ischemia by compression of small vessels (possible fibrosis by repetitive ischemias), during the training and competition periods. As a matter of fact, there are no clinical or genetic studies to ensure a good prognosis.

When this infirmity is diagnosed, the rule to be followed has been the exclusion from competitive sports[91]. However, it is possible that some exceptions be made for asymptomatic athletes without high risk criteria that compete in sports like golf, cricket, bowling and shooting.

The recommendation for exclusion from competitive sports remains for athletes with positive genotype, but with negative phenotype for HCM, although currently we do not have enough evidences available, as they do not have clinical diagnosis nor present risk criteria for HCM. However, some mutations in the troponin gene (chromosome 1) may not express themselves as macroscopic ventricular hypertrophy, but express histopathologic variations in the myocardial muscle fibers, being of bigger risk for sudden death[88].

In athletes with positive genotype and negative phenotype that practice acyclic sports (drastic variations in speed with unexpected accelerations and decelerations, e. g. soccer, basketball, tennis), there seems to be more risk than for those who compete in cyclic sports (jogging, swimming, cycling)[88].

5.4. SD risk stratification in hypertrophic cardiomyopathy

There are criteria suggested by Rickers et al[90] and Colin Lizalde[92] to establish risk factors for SD in HCM carriers. Those criteria are found in table 9.

Table 9 – Risk factors for SD in HCM carriers

Bigger risk factors
Survived cardiorespiratory arrest
Spontaneous sustained ventricular tachycardia
SD family history in individual <40 years old
Unexplained syncope or pre-syncope
Wall thickening of left ventricle > 30mm

Smaller risk factors
Abnormal pressoric response to exercise
Individuals under 30 years old
Non-sustained ventricular tachycardia

5.5. Recommendations for athletes with HCM diagnosis

Athletes with probable or unequivocal HCM diagnosis should be excluded from most competitive sports, with possible exception to those sports of low dynamic and static component (IA). This recommendation does not depend on age, gender, phenotypic appearance of the athlete, presence or not of symptoms, obstruction of left ventricle outlet, treatment with drugs, septal ablation, use of pacemaker or implanted defibrillator[86].

Class: I
Evidence level: C

Patients with HCM diagnosis should be excluded from sports competitions of competitive feature. Those classified as low risk may participate on golf, pool, bowling and shooting;

Recreational sports that require high intensity or drastic variations in intensity are not recommended;

Individuals with positive genotype and negative phenotype (without clinical evidence of disease) may participate in sports as long as they are periodically evaluated;

Patients without high risk criteria and with normal exercise test may perform cyclic recreational physical activity of low intensity;

Patients with implanted defibrillator should be excluded from contact sports.

Class: IIa

Patients without high risk criteria and with normal exercise test may participate on recreational contact sports of low intensity and low volume.

Class III

Asymptomatic patients with HCM, even if of low clinical and genetic risk, high functional capacity and without family history of sudden death may participate in high intensity competitive sports.

6. Group of carriers of arrhythmogenic right ventricular dysplasia (ARVD) or left ventricular cardiomyopathy

It is a variation in the heart muscle of genetic cause by variation in the desmosome formation that is characterized by pathologic fibro-fatty replacement of right ventricular myocardium, and it may manifest also in the left ventricle[92-94]. This infirmity is an important cause of sudden death in youngsters, including competitive athletes. This disease has been described as the main cause of sudden death in athletes in the region of Veneto, Italy[93,94]. As highly trained athletes may have right ventricular hypertrophy besides a variety of variations in depolarization, repolarization and conduction of nervous stimulus, the differential diagnosis between athlete's heart syndrome and ARVD must always be performed. The echocardiogram may present technical limitations to access right ventricle images, and its structural and functional analysis may be damaged[95,96]. The cardiac magnetic resonance is a non-invasive image technique that has shown itself to be promising[96]. The demonstration of global or segmental dysfunction of right ventricle or the substantial increase of this cardiac chamber cavity associated to myocardial tampering and presence of fibrosis (MR delayed equilibrium) gives support to the ARVD diagnosis[83,97-100].

6.1. SD risk stratification in arrhythmogenic left ventricular dysplasia

Some aspects must be considered in the SD risk stratification in arrhythmogenic right ventricular dysplasia.

Age Class: IIb, Evidence level: C

Age – There are divergent opinions on whether the appearance of symptoms in age to 20 years old would be of more risk.

Syncope Class: IIb, Evidence level: A

Syncope – great studies could not demonstrate a relation with increase in mortality.

ECG AR Class: IIA, Evidence level: C

High resolution electrocardiography – the presence of late potentials seems to positively co-relate with the seriousness of the disease and the occurrence of sustained ventricular tachycardia and ventricular fibrillation, when right ventricular dysfunction is present. However, new studies will be required to predict the development of ventricular arrhythmias.

Echocardiography Class: I, Evidence level: C

Echocardiography – sudden death is more frequent in patients with right ventricular diffuse dilatation and in those in who the left ventricle is involved, because its dysfunction seems to be a marker of risk of ventricular fibrillation and sudden death.

Programmed electrical stimulation Class: III, Evidence level: C

Programmed electrical stimulation - the induction of sustained ventricular tachycardia varies from 57% to 94% in patients with monomorphic sustained ventricular tachycardia, but is low in patients who present ventricular fibrillation or left ventricle involved. When the induction occurs in the presence of right ventricular dysfunction, the predictive value for sudden death is bigger.

Magnetic Resonance Class: I, Evidence level C

The MR identifies the right ventricular dysfunction, walls tapering and presence of delayed equilibrium (myocardial fibrosis) and the presence of microaneurysms.

Biopsy: little used in our milieu

24 hour Holter, exercise test and QT dispersion Class: III, Evidence level: C

24 hour Holter, exercise test and QT dispersion do not present value to identify patients in risk of sudden death as well as the type of ventricular arrhythmia.

Exercise – there are evidences that the exercise can be a triggering factor of potentially serious symptoms and arrhythmias, suggesting that these consequences depend on the sympathetic tone, especially in athletes[99,100].

Finally, it is important to highlight that the most efficient way of detecting silent cardiac pathologies that sometimes may be the cause of sudden death related to exercise and sports is a careful pre-participation evaluation and that many variations found in routine exams and that may be confused with heart diseases are physiological adaptations resulting from intensive and regular training, composing the characteristics known as athlete's heart syndrome, cardiomegaly, conduction and rhythm disturbances and variations of ventricular repolarization in the electrocardiogram[97,98,100,101].

Recommendations for athletes with arrhythmogenic right ventricular dysplasia diagnosis

Patients/athletes with unequivocal diagnosis of right ventricular dysplasia should be excluded from the practice of competitive activities[92,93].

7. Group of myocarditis carriers

It is an infirmity associated to a heterogeneous clinical profile, being the probable cause of sudden death in some athletes[73,102,103]. Usually, myocarditis is result of an infection, but it can be associated to alcohol or drugs abuse[103]. Sudden death may occur in its active phase or even when there is already a scar in the myocardium, consequence of complex arrhythmias deflagrated from an instability electric substrate. In the athlete we can have left ventricular increase by the disease per se, by hypertrophy secondary to physical training or by a mix of both. The differential diagnosis in this cases is made by the athlete's clinic (presence of arrhythmias, palpitations, pre-syncope or syncope or yet by systolic dysfunction). The cardiac magnetic resonance and the performance of endomyocardial biopsy in certain circumstances can help in the diagnosis. It is important to highlight that several studies have shown H1N1 flu as responsible for a kind of serious myocarditis, almost always accompanied by cardiogenic shock. Recently, the organizing committees of the Singapore Asian Games and of the Winter Olympics in Canada recommended that all athletes participating in these events came to these countries vaccinated against swine flu. We also remind here of the possibility of myocarditis by dengue virus in our country because of the constant outbreaks of this disease in Brazil.

7.1. Recommendations for athletes carriers of myocarditis

Athletes diagnosed with myocarditis should be removed from all competitive sports and undergo a recovery period of at least 6 months after the onset of clinical manifestations. These athletes may be released for training and competition after 6 months if:

Left ventricular function, ventricular wall motility and cardiac dimensions return to normal (based on echocardiographic and radionuclide studies at rest and with exertion);

Frequent or complex forms of ventricular and supraventricular arrhythmias and clinically relevant arrhythmias are absent;

Inflammatory markers and heart failure are standardized;

The ECG at rest is standard, although the persistence of ST alterations criterion alone is not impediment to return to training and competitions.

8. Group of carriers of coronary artery disease

The ischemic heart disease has declined over the decades, in Brazil. In 1980, the standardized death rate from coronary artery disease (CAD) was 65/100,000 inhabitants, and in 2005 the data showed 46/100,000 inhabitants[104]. Coronary artery disease accounts for the majority of the events of sudden death associated with exercise in people aged over 35 years old[105]. The mechanisms responsible for triggering coronary events during vigorous physical activity result from increased sympathetic activity and increased release of catecholamines, platelet adhesion and activation (leading to risk of thromboembolic events), electrolyte abnormalities by elevated potassium (serving as a trigger for ventricular tachyarrhythmias) and complications as subendocardial ischemia, leading to plaque rupture[106, 107] or possible plaque erosion[108, 109], facts implicated as immediate causes of exercise-related events in adults. In the young individual, the leading cause of sudden death during sports is hypertrophic cardiomyopathy[110].

In the last decade, the practice of physical activity has been recommended as part of intervention in primary and secondary prevention of coronary artery disease by providing a steady improvement in functional capacity (Class I) alleviating angina at rest (Class I), with improvement in the severity of exercise-induced ischemia (Class IIa) and the reduction of some cardiovascular risk factors (Class IIa). Proposed mechanisms and described the benefits of physical exercise in CAD are: increment of endothelial function, regression of atherosclerotic plaque formation and increased flow of collateral circulation, vasculogenesis, remodeling and decreased apoptose[108]. However, for this population start a physical practice is needed as to the stratification chance of having an event cardiovascular [109, 111]. The algorithm for the stratification of the patient begins with history taking and physical examination and the subsequent application of a progressive exercise test and maximum for the induction of cardiac arrhythmias, ischemic stress induced ventricular dysfunction and atrioventricular conduction disturbances. Data provided by exercise testing will guide the prescription of physical practice, you must follow some basic principles, such as frequency, duration, intensity and type of exercise[112, 113].

Type of exercise: aerobic exercise program are those that yield better benefits for the cardiovascular system and control of risk factors. This type of program is characterized by cyclical exercises of large muscle groups, such as walking, jogging, swimming, cycling, dancing, aerobics, among others.

Frequency of exercise: the recommended exercise frequency is 3-5 times a week, and in some groups (hypertensive and obese) the frequency can be up to 7 times a week.

Exercise intensity: the best way to measure the intensity of physical exercise is through heart rate, determined by maximum exercise testing (ET). The training heart rate should be between 70 and 85% of maximal HR obtained in ET and can be used to Karvonen formula (HR training = (HR maximum – HR at rest) x% of HR reserve recommended + HR at rest) or the corresponding heart rate between the anaerobic threshold and respiratory compensation point, assessed by spirometry. In beginners patients the range 50-60% HR reserve and 60-80% HR reserve for contingent should be used. Patients on beta-blockers should associate with HR training determined by functional testing to feel exertion by the Borg scale.

In muscular endurance exercises for the major muscle groups, you should use 40-60% of maximum voluntary contraction (low to moderate intensity) with 8-15 repetitions, three sets to one, or empirically, starting with low loads and evolving until the sense of effort is low to moderate.

Duration of aerobic exercise: 30-60 continuous minutes

8.1. Anamnesis and physical examination

Data supplied by anamnesis and physical examination are essential for the evaluation of coronary artery disease, whether it is stable course or not, helping to stratify the individual and exercise schedule (Class IIa, Evidence level B).

8.1.1. Clinical history

The clinical history is extremely important in the stratification of candidate patient with physical practice supervised or not, guiding the physician regarding the evolution and the current stage of the disease. You can identify the presence or absence of symptoms (pain, fatigue, shortness of breath) and with what degree of effort they are triggered. One should make an inventory regarding medications, as they may influence the dynamics of programming and practice exercises, must abide by certain characteristics of the symptoms that drive the probability of the presence of angina as:

Location: chest, retrosternal shoulder, epigastrium, cervical, hemithorax, back;

Type: constrictive, tightness, heaviness, tightness, discomfort, burning, stabbing;

Irradiation: upper (right, left, both), shoulder, jaw, neck, back, epigastrium;

Trigger factors: physical exertion, sexual activity, position, power, breathing, emotional component, spontaneous;

Factors relief, rest, sublingual nitrate, analgesic, food, antacid, position and apnea;

Associated symptoms: sweating, nausea, vomiting, pallor, dyspnea, hemoptysis, cough, pre-syncope and syncope.

The classification of chest pain becomes fundamental, and divide this into three groups: typical, atypical and not cardiac[114, 115].

Typical angina (definite)

Triggered by exercise or emotional stress;

Retrosternal discomfort or pain;

Relieved by rest or nitroglycerin

Atypical angina (probable)

Presence of only two factors above

Non-cardiac chest pain

Presence of only one or none of the above factors

Besides clinical history of chest pain risk factors for CAD should be considered in the stratification of individuals, such as diabetes, hypertension, dyslipidemia, smoking, family history of premature CAD (<55 years for men and <65 years for women), inactivity and stress.

8.1.2. Physical examination

Physical examination may be normal or show some changes identified by ectoscopy, palpation and auscultation, such as a stroke deflected to the left, the presence of an accessory heart sound (and B3 or B4), identifying a blow that may give clues about " status "of the heart. A blood pressure measurement should be performed in both arms, and in orthostatic, sitting and lying and at least one lower limb, with the goal of postural hypotension or identify certain problems that can cause there is a significant difference in blood pressure among members. The physical examination is usually normal in patients with stable angina[27]. Presence of atherosclerosis at other sites can provide data for an investigation of CAD in some individuals with lower limb pulses decreased, arterial stiffening and abdominal aortic aneurysm. Despite the physical examination in subjects with CAD be little instructive, complete workup, particularly the cardiovascular system should be performed carefully, because it can provide important information on other associated conditions such as valvular disease, hypertrophic cardiomyopathy and other.

8.2. Echocardiography in chronic CAD:

The transthoracic echocardiography has been used as an important diagnostic tool in coronary artery disease as well as a method for the evaluation of prognosis in these pacientes[30]. In patients clinical history and electrocardiogram are inconclusive, echocardiography can demonstrate reversible abnormalities of wall motion or not, identifying areas of hypokinesia, acnesia, dyskinesia and aneurysms (Class I) and extent of these changes, providing also an important variable in assessing left ventricular function, ejection fraction (Class I) [116].

8.3. Benefits of exercise on coronary disease

There are several physiological benefits elicited by exercise in patients with stable CAD. Table 10 summarizes the benefits for level of evidence.

Table 10 – Benefits of exercise on coronary disease.

Recommendation Grade: I	Recommendation Grade: IIa	Recommendation Grade: IIa Evidence level: B
Enhancement of angina at rest.	Attenuation of seriousness of ischemia induced by exertion.	Physical training associated to a low-fat diet can reduce the atheromatous plaque progression after 1 year of follow-up.
Enhancement in functional capacity.	Control of some risk factors to CV disease.	

Note: according to the Guideline on SBC cardiac rehabilitation, improvement of myocardial ischemia due to the increase in stroke volume (level A), attenuation of tachycardia during submaximal exercise loads effort (level B), improved endothelium-dependent vasodilator response (level B) and increased perfusion in coronary microcirculation (level B)[117].

8.4. Risk stratification for inclusion of patients in exercise and cardiac rehabilitation programs

The risk of patients in exercises and cardiac rehabilitation programs can be categorized according to table 11[117].

Table 11 – Risk stratification for inclusion of patients in cardiac rehabilitation programs.

	Risk stratification for inclusion of patients in cardiac rehabilitation programs.
Low	- Functional capacity = 7 METs absence of myocardial ischemia at rest or in exertion test with intensity smaller than 6 METs. - Ejection fraction of left ventricle = 50%. - Absence of significant ventricular ectopy after the 3rd day after-IAM. - Adequate response of arterial blood pressure to exertion. - Capacity of auto-monitoring the intensity with which exercises.
Moderate	- Presence of myocardial ischemia. - Depression of ST segment = 2 mm. - Reversible abnormalities, during exercise, in thallium myocardial scintigraphy. - Ejection fraction of left ventricle= 35-49%. - Absence of complex ventricular ectopies. - Absence of fall of arterial blood pressure during exercise.
High	- Recurrent angina with ischemic changes in ST segment beyond 24 hours following admission. - Signs and symptoms of congestive heart failure. - Ejection fraction of the left ventricle = 35%. - Complex ventricular ectopy (multifocal ventricular premature beats, ventricular tachycardia, R on T phenomenon, ventricular fibrillation). - Functional Capacity = 5 METs on exercise testing limited by angina, ST segment depression or inadequate response of blood pressure. - Decreased or failure to increase systolic blood pressure during exercise. - Changes persistent ischemic ST-segment and / or angina during exercise.

8.5. Computed tomography, magnetic resonance and athlete's heart

Despite the great usefulness and practical applicability of Doppler echocardiography in the evaluation of different heart diseases, there are situations where it is necessary to use other imaging modalities for the diagnosis and also for risk stratification. In particular in the evaluation of athletes with suspected heart disease using these methodologies can be important, such as the distinction between mild hypertrophic cardiomyopathy and myocardial hypertrophy secondary to sport activity[118, 119].

The complexity of the cardiac movements requires that equipment intended to produce image be sophisticated enough to reproduce the anatomy of the heart without artifacts resulting from the mobility of the heart itself [118]. The magnetic resonance equipment equal or greater than 1.5 Tesla can be used for this end[118].

Tomography data available in the literature are based on studies of equipment of 64 rows of detectors. Systems with at least 32 rows of detectors can be used effectively, there is only a need for more adequate preparation of the patient, to improve the quality of images obtained[118].

The professionals involved should have received appropriate training in specialized centers, to ensure greater safety and better quality of examinations performed[118].

The use of magnetic resonance imaging and computed tomography for the risk stratification of asymptomatic indivi-

duals and release for sports is still limited. There are no jobs available in the literature assessing the potential contribution that the resonance could have that effect. In addition, CT is at least in part, its limited application because it uses ionizing radiation, which restricts its indiscriminate use, especially in the younger population. However, this technology has definite role in some conditions that may be present in the evaluation of asymptomatic wanting to do sports activities[118, 120,121].

The presence of signs suggestive of myocardial ischemia in young people may be related to the existence of anomalous origin of a coronary artery. This is a condition in which the use of CT or MRI is fundamental and well defined[122].

In older individuals who wish to practice physical activities, CT can also help, especially if there is conflict between the results of other noninvasive tests with regard to the existence of silent myocardial ischemia. The most common situation is when there is stress test compatible with the presence of ischemia and myocardial scintigraphy normal or doubtful. In this condition, the high negative predictive power of the test may have a fundamental role to exclude the presence of obstructive coronary disease or to indicate the need for if further invasive diagnostic investigation.

The high spatial resolution of CT also enables it to be used for diagnosis of other conditions such as congenital heart disease, cardiomyopathies, and vascular anomalies, but also those ends are achieved with MRI, which has the added advantage of not using ionizing radiation[118] .

Other medical conditions that may involve the use of MRI for release for sports involve the existence of underlying heart disease that has not presented clinical manifestation previously. In these cases, the clinical recommendations for the use of resonance are within the regulations established in habitual cardiology[118].

For evaluation of heart disease in athletes who compete in categories aimed for people over 40 years and with conflicting non-invasive tests[118], tomography can be a useful test, because it has a negative predictive value greater than 97% and may be useful for rule out obstructive disease. Other clinical conditions may need further investigation, such as arrhythmias or the development of changes to electrocardiogram[120].

The heart of the professional athlete or amateur elite to resonance is characterized by having larger dimensions mass, end-diastolic volume and end-systolic volume. Such changes, however, occur without altering ejection fraction, regional contractility or contraction pattern of the ventricles. This aspect should always be considered as cases of hypertrophic cardiomyopathy usually show changes in the pattern of cardiac contractility, even if there is slight increase in thickness of the ventricular myocardium. Thus, the shape of ventricular twist in both the longitudinal and in the transverse plane, is preserved in athletes, even elite, which does not occur in cases of hypertrophic cardiomyopathy[119].

In cases of heart adaptation to stress found in elite athletes the degree of hypertrophy that affects the left ventricle differs from that which interests the right ventricle, being more pronounced in the cavity systemic. Furthermore, when there is a relationship between physiological hypertrophy volumes, dimensions and ejection fraction of the left ventricle compared to the right ventricle are comparable in athletes and sedentary individuals without structural heart disease[119, 120].

A major cause of death in young athletes is hypertrophic cardiomyopathy condition that should always be remembered and researched evidence of abnormalities that arise for this subgroup. The literature reports cases there are up to 13 mm thickness in the left ventricular myocardium as an adaptive response, especially in the case of elite athletes male. It is important to remember that in different sports among them weightlifting, rowing, cycling, swimming, marathons and professional football athletes may also have significant increases of cardiac dimensions, without presenting heart disease[121].

Hypertrophic cardiomyopathy resonance can also understand the presence of delayed enhancement, which may indicate the existence of fibrosis and be present in varying degrees, regardless of the clinical manifestation of heart disease. Some studies have associated this image pattern to the occurrence of arrhythmias and changes to the dilated form of the disease, a finding that should raise caution in case driving[118, 119.]

The arrhythmogenic also shows up as a challenge in athletes, especially due to the wide variety of image patterns that it can present. It must be remembered that the simple presence of fat tissue in the myocardium is not pathognomonic of that entity, since the presence of fat in heart muscle may be a consequence of other inflammatory conditions, degenerative and ischemic besides being a usual finding in patients over 65. The diagnosis of dysplasia is made from the existence of regional abnormalities in the ventricles, and the existence of areas of delayed enhancement in the ventricular walls, reflecting the presence of inflammation and / or myocardial fibrosis. The combination of these findings, with or without the presence of fat associated, allows for the identification of dysplasia. Important data such cases arises from the fact that due to the evolving nature of this entity, the diagnosis involves the removal of sports activities[121, 122].

Another condition that has clinical importance is myocarditis. This entity can have your event facilitated by the existence of conditions that lead to decreased immune activity, such as occurs when there is excessive training for a long period, habitual condition of elite athletes. The resonance has been shown to be an effective evaluation of these patients revealed the presence of late enhancement, even in cases where there is no change in ventricular contractility, which allow defining the presence of these changes. Some authors mention that the image pattern may even allow them to infer the causative agent of entidade121. Changes in the presence, extent and intensity of delayed enhancement in these conditions are important prognosis[121, 122].

Resonance and computed tomography can be important allies in risk stratification and evaluation of structural heart disease in athletes. Although its routine use is to essentially complement evaluation by clinical examination, ECG and echocardiography may, under some conditions such as anomalous origin of the coronary arteries, in search of arrhyth-

mogenic dysplasia and myocarditis, they can compose the line front and are arguably important stages of the diagnostic evaluation table 12.

Table 12 – Options of diagnosis evaluation by CT and MRI

Diagnosis evaluations options Clinical condition	CT	MRI
Release for sport practice without suspicion of structural heart disease	III	III
Athlete's heart characterization	III	IIa
Diagnosis of coronary artery anomalous origin	I	IIa
Hypertrophic cardiomyopathy diagnosis	IIb	I
Arrhythmogenic dysplasia	IIb	I
Arrhythmias study	III	IIa
Myocarditis	III	IIa

9. Risk stratification of SD-related exercise / sport in arrhythmogenic genetic syndromes.

9.1. Canalopathies

The canalopathies are inherited arrhythmogenic heart disease, no structural, caused by genetic alterations that result in dysfunction of cardiac ion channels, exposing their patients to a risk of sudden death[123]. Canalopathies The best known are the long QT syndrome (LQTS), short QT syndrome, Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia TVPC. The sinus node disease and disease Lenegre or conduction system disease are also examples of canalopathies.

The ion channels, ion currents passing through these channels, proteins that bind to cell membrane structure myocardial and the junctions between these structures are part of electrical impulse formation and transmission of these impulses throughout the heart muscle so synchronous generating the potential cardiac action[124, 125]. The performance of each of these functions is determined by different genes. Mutations that occur in these genes cause specific dysfunctions and diseases known to cause canalopathies[126, 127]. The ion channels present on the cell membrane allow entry and exit of ions, following a voltage gradient. The change caused by genetic mutation in each of these channels may generate a gain or loss of function. There are ion channels responsible for depolarization and repolarization of the cell membrane. The first channel from right to left, called electrogenic channel, allows entry of three sodium ions (Na +) to each output of calcium ion (Ca2 +) across the membrane. The depolarization channels are: 1) INa, sodium channel carried by SCN5A gene whose mutations that cause gain of function resulting in LQTS 3, while the loss of function causes Brugada syndrome and congenital sinus node disease, 2) channel iCal L-type calcium; 3) INa / Ca channel sodium and calcium channel called electrogenic. The channels involved in myocardial repolarization of the cell are: 1) IK1 potassium channel KCNJ2. This channel is responsible for the output of potassium at the end of phase 3 of the action potential. The gain of function of this channel results in short QT syndrome type 3. while the loss of the function of this channel results in long QT syndrome type 7 and Andersen syndrome. 2) ITo1 and ITo2 responsible for early repolarization - phase 1 of the action potential, 3) IKr - Potassium Channel - Gene KCNH2/KCNE2. This channel is responsible for the output of potassium in the beginning of phase 3 of the action potential. The gain of function of this channel results in short QT syndrome type 1. 4) IKs potassium channel gene KCNQ1/KCNE1. This channel is responsible for the output of potassium in the middle of phase 3 of the action potential. The gain of function of this channel results in short QT syndrome type 2. The loss of function of the KCNQ1 gene (the alpha subunit which forms pores in the cell membrane) and the KCNE1 gene (which encodes the protein which forms the slow component of the IKs channel) causes long QT syndrome type 1.

9.2. Long QT syndrome

The long QT syndrome is the prototype of canalopathies. Reported for more than 50 years, initially as autosomal recessive syndrome of Jervell and Lange-Nielsen128 (congenital deafness, increased QTc interval and syncope or sudden death) and subsequently autosomal dominant Romano-Ward syndrome (deafness without prolonged QTc), the LQTS showed an inherited trait of sudden death in patients without structural heart disease. Currently, there are several types of known LQTS, mutations in certain genes that cause distinct changes in ventricular depolarization and repolarization (Table 13).

Table 13 – Types of known LQTS

Canalopathies				
Ionic channels mutations/variations	Clinical syndromes	Gene	Functional variation	Types of inheritance
I_{Ks}	LQTS 1	KCNQ1KvLQT1	Loss of function of I_{Ks}	AD
I_{Ks}	Jervell-Lange-Nielsen 1	KCNQ1	Loss of function of I_{Ks}	AR
I_{Ks}	Atrial fibrillation	KCNQ1	Gain of function of I_{Ks}	AD
I_{Ks}	Short SQT	KCNQ1	Gain of function of I_{Ks}	AD
I_{Ks}	LQTS 5	KCNE1 /MinK/	Loss of function of I_{Ks}	AD
I_{Ks}	Jervell-Lange-Nielsen 2	KCNE1	Loss of function of I_{Ks}	AR
I_{Kr}	LQTS 2	KCNH2 /HERG/	Loss of function of I_{Kr}	AD
I_{Kr}	Short SQT	KCNE2	Gain of function of I_{Kr}	AD
I_{Kr}	LQTS 6	KCNE2 /MiRP1/	Loss of function of I_{Kr}	AD
I_{K1}	Andersen Syndrome LQTS 7	KCNJ2 /Kir2.1/	Loss of function of I_{K1}	AD
I_{Na}	LQTS 3	SCN5A	Gain of function of I_{Na}	AD
I_{Na}	Brugada Syndrome	SCN5A	Loss of function of I_{Na}	AD
I_{Na}	Lenegre disease	SCN5A	Loss of function of I_{Na}	AD
I_{Na}	Sinus node disease	SCN5A	Loss of function of I_{Na}	AD
Variations in actions of pacemaker cells (If)	Sinus node disease	ANK2	Loss of function of If	?
Variations in binding proteins	LQTS 4	ANK2		

Canalopathies				
Mutations/variations in ionic channels	Clinical syndromes	Gene	Functional variation	Types of inheritance
Variation in release and retention of intracellular calcium	TVPC	RyR2	Gain of function of calcium release	AD
		CASQ2	Loss of function of calcium reserve	

Different mutations in the same gene may result in loss or gain of function and, consequently, different syndromes. LQTS: long QT syndrome, AD: autosomal dominant, AR: autosomal recessive.

The typical clinical features of LQTS include syncope or sudden death associated with QTc interval prolongation and ventricular tachyarrhythmia presence and torsades de pointes. Classically, there are triggers of syncope in LQTS, adrenergic activity in LQTS 1 and sudden awakening or auditory stimulus in LQTS 2. However, there is a wide variety of presentations, the carrier of the mutation may be asymptomatic since, with no increase in QTc, even have syncope or sudden death in the first days of life. Therefore, we developed diagnostic criteria for scoring changes on 3 main criteria: clinical history, family history and electrocardiographic variation[129] (table 14).

Table 14 – Diagnostic Criteria of long QT syndrome

Criterion	Variation	Points
ECG variation: QTc	≥480ms	3
	460-479 ms	2
	450-459 ms	1
	Torsades de pointes	2
	T wave alternation	1
	T wave notch	1
	Low HR for age	0.5
Clinical history	Syncope to stress	2
	Syncope without stress	1
	Deafness	0.5
Family history	Relative with LQTS	1
	Sudden death < 30 years old	0.5
≥ 4 points: high probability of LQTS; 2-3 points: intermediate probability of LQTS; 1 point: low probability of LQTS (133).		

9.2.1. Genetic alterations of the long QT syndrome (LQTS)

The genetic alterations are known in approximately 60% of clinical cases of LQTS. They are currently 7 known genes responsible for these syndromes. However, hundreds of mutations have been described, which is the cardiac canalopathy with the highest number of mutations described[130].

The most common mutation occurs in the gene KCNQ1, responsible for LQTS 1 (40-50%) and the syndrome of Jervell--Lange-Nielsen type 1. This mutation causes a loss of function of the potassium current IKs, which plays an important role in cellular repolarization and QT interval adaptation to heart rate. The KCNE1 gene, whose mutation causes LQTS5, is much less frequent (2-3%) and is also responsible for the function IKs [123, 131, 132].

The KCNH2 gene (HERG) encoding the alpha subunit of potassium channels rapid and KCNE2, which encodes the beta subunit are responsible for the rapid entry of potassium during the third potential action[131, 133]. The loss of function of the alpha subunit represents 40% of the genotyped LQTS and is responsible for LQTS 2[134].

The LQTS 3 represents approximately 10% of all diagnosed mutations in LQTS occurs by changes in the SCN5A gene, which produces a gain of function sodium continuous input during the plateau phase, facilitating premature depolarizations of the cardiac cell. Hundreds of other mutations have been described. However, clinical applications of these mutations to identify treatment and monitoring of patients and relatives of LQTS are restricted to known forms[125].

9.2.2. Risk Stratification in LQTS

Genetic analysis has been widely used in risk stratification and specific therapeutic interventions in patients with prolonged QT interval, as well as their families, to identify patients with normal QT interval.

The recommendation of strategies for risk stratification and treatment for medical conditions are infrequent, and therefore the data obtained on the disease cohort studies, some with large number of participants, with follow along, others with more limited or Following narrower limits the level Evidence for the recommendations to B.

The most robust risk marker identified is a previous episode of aborted sudden cardiac death and its most common cause, are episodes of polymorphic ventricular tachycardia, or not degenerating into ventricular fibrillation. Patients who experience this condition are 12.9 times greater risk of new episodes of sudden death. Prior Syncope is also a risk marker extremely disadvantageous[135].

For asymptomatic patients were identified as risk factors: QTc> 500 ms for patients with LQTS 1 and 2 male patients in LQTS 3, regardless of the QT interval. Mutations involving a gene segment encoding the pore of the channel are also linked with poor prognosis. So once again it is clear the value of genotyping in patients with this syndrome. The presence of sudden death in the family was not a marker of increased risk of events[136].

9.2.3. Recommendations

Restriction of physical activity: although in LQTS 2:03 episodes of ventricular arrhythmia are not related to effort, it is recommended that all affected patients avoid physical exertion competitive. This recommendation is particularly important in the case of swimming for LQTS1. In 2 patients with LQTS should avoid acoustic stimuli especially during sleep, such as alarm clock and telephone. Obviously, drugs

known to prolong the QT interval should not be used, as well as drugs which reduce the plasma levels of potassium and magnesium (interventions Class I).

All patients with prolonged QT interval should receive beta-blockers, although the protection is incomplete for patients with LQTS 2 and 3 (intervention Class I). For patients with mutation, but with normal QT interval, also recommended the prophylactic use of beta-blockers (IIa)[137, 138].

The pacemaker has been considered an adjuvant therapy, always in conjunction with beta-blocker [139, 140]. It is effective in preventing recurrence of torsades de pointes, although its role is not well defined. The mechanisms involved are suppression of pauses preceding and the precipitate torsades de pointes, and by shortening of the QT interval. In general, is indicated for the prevention of severe bradycardia or pauses resulting from the use of beta-blockers in patients who cannot tolerate adequate doses, who remain symptomatic despite its correct usage or when there is evidence of pause-dependent arrhythmias. Certain genotypes of LQTS may benefit more than others with pacemaker implantation. The effect of bradycardia on transmural dispersion of repolarization was more pronounced in LQTS3 and LQTS2 and inexpressive in LQTS1. The correct programming of the pacemaker is important in preventing arrhythmias in LQTS. The stimulation at higher frequencies shorten the QT interval and reduces the dispersion of ventricular repolarization. The minimum frequency limit should be set to 70 to 80 bpm and can be increased in situations of higher risk, such as in surgical procedures and post--partum period. The occurrence of atrioventricular functional in some patients (especially children with very prolonged QT) immediately before the start of torsades de pointes causes the DDD pacing mode most widely used. There is no consensus on the best programming on the frequency response and time delay atrioventricular. Some of the programming features that enable drops low heart rate should be turned off, as hysteresis and sleep function.

The implantable cardioverter-defibrillator is recommended for all survivors of sudden death with good functional status and life expectancy of greater than 1 year (Class I recommendation). Patients may present syncope despite beta-blocker therapy may also benefit from defibrillator (IIa). The defibrillator may be considered in patients at high risk for sudden death, such as LQTS 2 and 3, even asymptomatic (Class IIb recommendation)[141, 142].

The sympathetic denervation may be considered for patients with syncope or polymorphic ventricular tachycardia that are already on beta-blockers (IIb)[143, 144].

9.3. Short QT syndrome

In this disease there is a shortening of repolarization, favoring the genesis of ventricular arrhythmias by reentry. Characterized by a short QT interval QTc <320 ms with peaked T waves, which can have increased amplitude, with its ascending phase and normal phase descending rapidly[125, 127,145,146]. As clinical parameters for diagnosis are still unclear, genetic analysis is useful to confirm the diagnosis in suspected cases. Mutations in three genes were described KCNH2, KCNQ1 and KCNJ2 all resulting in a gain of function channel IKr, IKs and IK1, respectively, and determining the short QT syndrome type 1, 2 and 3. As the number of patients with a confirmed diagnosis is low, is not yet determined whether any specific type of mutation determines a worse prognosis. Risk factors for the occurrence of arrhythmias are not known.

Treatment for this condition is still a matter of controversy. In patients with mutations in the KCNH2 gene, showed the use of quinidine prolong refractoriness and suppress arrhythmia induction during electrophysiological study [146] for the other mutations, their utility has not been established. The disease seems to have high lethality, however, there may be a diagnostic bias only in serious cases. The use of the defibrillator may be considered, however, one must keep in mind the downside of the possibility of inappropriate shocks for the phenomenon of double counting (QRS complexes and T waves)[147, 148].

9.4. Brugada Syndrome

This syndrome is characterized by the occurrence of syncope or sudden death caused by polymorphic ventricular tachycardia in structurally normal hearts. His most peculiar aspect is the occurrence of ST-J point in right precordial leads V1 to V3, although this phenomenon has already been described in inferior leads[149]. Perhaps influenced by gender 90% of cases occur in men. Until now, only been identified in the SCN5A gene mutations that are present in about[123] 1/3 of affected[123, 150]. Clinically, the disease manifests with syncope or sudden death, mainly in the third or fourth decades of life. Fever is a trigger for arrhythmias.

Risk stratification in individuals with ST-J point that occurs spontaneously have a worse prognosis compared to those in the typical pattern was only observed after infusion of flecainide, procainamide or ajmaline. The occurrence of syncope associated with the occurrence of spontaneous elevation, increased risk of sudden death six times. The presence of sudden death in the family has not proved useful for risk stratification as well as the detection of gene mutation SCN5A.[151, 152]

The use of programmed ventricular stimulation for risk stratification is controversial. In the series of Brugada et al [152], the absence of inducible arrhythmias during electrophysiological study had a negative predictive value of 93% at 3 years for clinical events. However, the number of patients and cols.136 Priori, electrophysiological studies had low accuracy for predicting events. Currently, the use of programmed ventricular stimulation is Class IIb indication for risk stratification in asymptomatic patients.[153, 154]

As therapeutic recommendations, the use of defibrillator is indicated for Class I patients recovered from sudden death. It is Class IIa indication for patients with ST spontaneous point that J expressed or syncope who had previously documented ventricular tachycardia.

The infusion of isoproterenol can be useful in the treatment of thunderstorm (Class IIa) as well as the use of quinidine for that situation (Class IIb)

9.5. Catecholaminergic polymorphic ventricular tachycardia

The disease is characterized by episodes of polymorphic ventricular tachycardia, triggered during stress or emotions in children and young adults who have hearts structurally

normal[155, 156]. The ECG at rest is normal, except for a relative bradycardia for age and frequent presence of wave U. It manifests clinically as syncope triggered by physical or emotional stressful situations[155, 156]. Two mutations were described in RyR2 and CASQ2 genes that encode the ryanodine receptor, protein responsible for calcium release from the sarcoplasmic reticulum, and calsequestrin protein which binds to calcium in the sarcoplasmic reticulum. The genetic analysis does not contribute to risk stratification, but it is important to identify carriers of the mutation that has not yet manifested symptoms.

The typical manifestation is the occurrence of arrhythmias during exercise, most often when they reach 120-130 beats per minute, starting with isolated ventricular premature beats, progressing to episodes of non-sustained ventricular tachycardia and sustained, if effort is maintained (Figure 3).

The use of beta-blockers is very effective in reducing symptoms, and indication Class I to patients with clinical manifestations, IIa Child carriers of the mutation, but that did not exhibit symptoms, and Class IIb for adults in the same situation[157, 158].

The defibrillator use in Class I indication is retrieved from Class IIa and sudden death in patients who remain with sustained ventricular tachycardia or syncope despite beta-blocker therapy.

It has been recently suggested treatment with cardiac denervation friendly left as a therapeutic alternative for patients who cannot make use of beta-blockers. The mechanism of action of this therapy seems very attractive for these patients, since it reduces cardiac catecholaminergic stimulation, which in theory would reduce the triggering stimulus arrhythmia. Some authors have reported promising initial experiments, but there is little evidence that this alternative is incorporated routine, currently only recommended in cases of failure or contraindication of beta-blockers.

The recommendations regarding the release for sports activities take into account the type of activity and the diagnosis established from the pre-participation evaluation and any additional tests that may be necessary. Sports are categorized into static and dynamic according to table 15. The recommendations are summarized in Tables 16,17 and 18.

Table 15 – Sports classification[12,13,51]

	A. Low dynamic	B. Moderate dynamic	C. High dynamic
I. Low static	Bowling Golf Shooting	Fencing Table tennis Tennis (double) Volleyball	Race (marathon) Race walk Squash
II. Moderate static	Motorsport Diving Equestrian sports Motorcycling Gymnastics Judo/karate Sailing Archery	Athletic Jump Ice-skating Lacrosse Race (dragster	Soccer Basketball Race (track) Swimming Tennis (individual)
III. High static	Climbing Weight lifting Windsurf Water skiing Shot put	Wrestling Physiculture Snow skiing (mountain) Body boarding	Boxing Canoeing Rowing Cycling Triathlon

Table 16 – Athletes with congenital heart diseases

Pathology	Eligibility criterion	Recommendation
Atrial septal defect (closed or small, non-operated) and patent foramen ovale	Defect <6mm or 6 months post-closing, with normal pulmonary artery pressure without significant arrhythmia or ventricular dysfunction	Release for all sports. In patients with patent foramen ovale, percutaneous closure may be considered before the regular practice of diving.
Ventricular septal defect (closed or small, non-operated)	Restrictive defect (left to right gradient <64mmHg) or 6 months post-closing, without pulmonary hypertension.	Release for all sports
Atrioventricular septal defect	With no or only mild insufficiency of the atrioventricular valves, no subaortic stenosis, or significant arrhythmia, normal maximum gas exchange measurements.	Release for all sports
Partial or complete anomalous venous drainage	No significant systemic or pulmonary venous obstruction, without pulmonary hypertension or atrial arrhythmia induced by exercise.	Release for all sports
Persistent ductus arteriosus (operated)	6 months post-closing and no residual hypertension.	Release for all sports
Pulmonary stenosis (native mild or treated)	Native or 6 months post-intervention/post-surgery; peak transvalvular gradient between 30 and 50 mmHg, normal right ventricle, normal ECG or only mild right ventricular hypertrophy.	Release for sports low dynamic and static low to moderate.
Aortic coarctation (native or repaired)	Without systemic hypertension; peak pressure gradient between upper and lower limbs<21mmHg, peak systolic blood pressure<231mmHg during exercise without ischemia on exertion ECG without left ventricular overload.	Release for sports dynamics and sports low and moderate static (IA, B + II A, B). If the presence of graft, avoid risk of collision with sport body.
Aortic stenosis (moderate)	Transvalvular mean gradient <21mmHg, with no history of arrhythmia, without syncope, dizziness or angina pectoris.	Release for all sports except high static and dynamic sports.
Aortic stenosis (mild)	Transvalvular mean gradient between 21mmHg and 49mmHg, with no history of arrhythmia, without syncope, dizziness or angina pectoris.	Release for low dynamic and static sports (IA).
Tetralogy of Fallot	With no or only mild obstruction of the outflow tract of the RV, no more than mild pulmonary regurgitation, normal or near normal biventricular function and no evidence of arrhythmia.	Sports of low to moderate dynamic and static

Table 17 – Athletes with valve disease.

Pathology	Eligibility Criteria	Recommendation
Mitral valve stenosis	Mild stenosis, stable sinus rhythm	All sports, except for high dynamic and static (IIIC).
	Mild stenosis in atrial fibrillation and anticoagulation	Low moderate dynamic and static (IA, B + II A, B), no contact sport practice
	Moderate and severe stenosis (sinus rhythm or atrial fibrillation)	Low dynamic and static (IA), not to practice contact sport
Mitral valve regurgitation	Mild to moderate regurgitation, stable sinus rhythm, normal left ventricular function / size, normal exertion test	All sports, but in atrial fibrillation, avoid contact sport.
	Mild to moderate regurgitation, mild left ventricular dilatation (end-systolic volume <55ml / m²), normal left ventricular function in sinus rhythm.	Low to moderate dynamic and static (I, B + II A, B)
	Mild to moderate regurgitation, left ventricular enlargement (end-systolic volume> 55ml / m²) or left ventricular dysfunction (ejection fraction <50%) or severe regurgitation	Not to practice competitive sports
Stenosis of the aortic valve	Mild stenosis, left ventricular function and size normal at rest and stress, without symptoms, without significant arrhythmia.	Low to moderate dynamic and static (I A, B + II A, B).
	Moderate stenosis, normal left ventricular function at rest and stress, frequent/complex arrhythmias.	Low dynamic and static (IA).
	Moderate stenosis, left ventricular dysfunction at rest or stress, presence of symptoms or severe aortic stenosis	Not to practice competitive sports.
Tricuspid valve stenosis	No symptoms	Low to moderate dynamic and static (I A, B + II A, B)
Tricuspid valve regurgitation	Mild to moderate regurgitation.	Low to moderate dynamic and static (I A, B + II A, B)
	Any degree, with right atrial pressure> 20 mmHg.	Not to practice competitive sports
Multivalvular disease	See more relevant defect.	In accordance with the most relevant defect.
Aortic valvular or mitral biological prosthesis	Normal valve function and normal left ventricular function in stable sinus rhythm	Low to moderate dynamic and static (I A, B + II A, B). If atrial fibrillation and anticoagulation, do not practice contact sports.
Aortic valvular or mitral metallic prosthesis	Normal valve function and normal left ventricular function and anticoagulation	Low to moderate dynamic and static (IA, B + II A, B). Do not practice contact sports.
Post-valvuloplasty	Assess the severity of residual lesion (stenosis or regurgitation).	In accordance with residual lesion.
Mitral valve prolapse	In the presence of unexplained syncope or family history of sudden death or arrhythmias complex supraventricular / ventricular, or long QT interval, or severe mitral regurgitation.	Not to practice competitive sports
	In the absence of the above conditions	All sports

Table 18 – Athletes with myocardiopathies, miocarditis and pericarditis

Pathology	Eligibility Criteria	Recommendation
Definitive diagnosis of Hypertrophic cardiomyopathy	High risk profile.	Not to practice competitive sports
	Low risk profile: no history of sudden death among relatives without symptoms, mild left ventricular hypertrophy, normal blood pressure response to exercise without ventricular arrhythmias	Low static and low dynamic (IA).
Definitive diagnosis of dilated cardiomyopathy	With a high risk profile	Not to practice competitive sports
	No family history of sudden death, with no symptoms, moderately depressed ejection fraction (≥ 40%), normal blood pressure response to exercise without complex ventricular arrhythmias.	Low to moderate dynamic and low static (I A, B).
Definitive diagnosis of cardiomyopathy / arrhythmogenic right ventricular		Not to practice competitive sports
Athletes with active myocarditis or pericarditis		Not to practice competitive sports
Athletes after the resolution of myocarditis	Without symptoms, normal left ventricular function without arrhythmias	All competitive sports
Pathology	Eligibility Criteria	. Recommendation
Marfan Syndrome complete phenotype	Absence of the following conditions: aortic root dilatation greater than 2 standard deviations, moderate to severe mitral regurgitation, family history of sudden death or aortic dissection	Low to moderate dynamic and low static sports (IA, IIA). Echocardiography should be performed every 6 months for evaluation.
	Grade 1 hypertension without other risk factors	All sports
	Hypertension up to 1 degree two risk factors Grade 2 hypertension with or without to risk factors	All sports, except those with high dynamic and static (IIIC)
	Hypertension with grade 1 or 2 With three or more risk or damage factors in target organ or diabetes Hypertension grade III without other risk factors Stage 1 or 2 hypertension associated with other cardiovascular or renal conditions Hypertension Stage 3 with one or more risk factors, or target organ damage or diabetes, or other cardiovascular or renal conditions	All sports, except for high static sports (III-C). Low to moderate dynamic and low static (IA-B). Evaluate specific recommendation for other associated conditions.

Table 19 – Athletes with ischemic heart disease

Pathology	Eligibility Criterion	Recommendation
Ischemic heart disease	High probability of cardiac events.	Competitive sports are not allowed.
Ischemic heart disease	Low probability of cardiac events: no exercise-induced ischemia, no bigger symptoms or arrhythmias, non-significant coronary lesions (<50%), ejection fraction >50%.	Only dynamic low to moderate and low static sports (I A-B).

Table 20 – Athletes with arrhythmias and arrhythmogenic conditions

Pathology	Eligibility Criterion	Recommendation
Marked sinus bradycardia (<40bpm) and / or sinus pauses ≥ 3s	Symptoms	Temporary interruption of sports and re-evaluation
	No symptoms	All sports
1st and 2nd grade, type 1 atrioventricular block	In the absence of symptoms and heart disease, with improvement of conduction disorder during exercise	All sports
Atrioventricular block 2nd degree, or advanced type 2	In the absence of symptoms, heart disease, ventricular arrhythmias during exercise and at rest and if heart rate is> 40 bpm and improves with exercise	Sports of low to moderate dynamic and static (IA, B + II A, B)
Ssupraventricular extrasystoles	No symptoms, no heart disease.	All sports
Paroxysmal supraventricular tachycardia (tachycardia by atrioventricular nodal reentry or tachycardia by atrioventricular reentry by concealed accessory pathway)	Ablation is recommended After ablation catheter if no recurrences after 3 months and without heart disease	All sports
	If ablation is not performed, and tachycardia is sporadic, without heart disease, no hemodynamic consequences and no relation to exercise	All sports, except Those with high risk if there is a syncope episode
Ventricular pre-excitement Wolff-Parkinson-White (WPW) Syndrome	A) paroxysmal tachycardia by atrioventricular reentry. B) Atrial fibrillation or flutter	A, B) ablation is mandatory. After catheter ablation: if there are no recurrences and no heart disease, release for for all sports
	Asymptomatic pre-excitement standard - ablation is recommended, but not mandatory	All sports, except those with high risk if there is a syncope episode
Pathology	Eligibility Criterion	Recommendation
Atrial fibrillation	After paroxysmal atrial fibrillation: the absence of heart disease and WPW, in sinus rhythm for more than 3 months.	All sports
	Permanent atrial fibrillation in the absence of heart disease, and WPW: evaluate heart rate and left ventricular function in response to exercise.	Individual evaluation
Atrial flutter	Ablation is mandatory; after ablation: absence of symptoms for more than 3 months, absence of heart disease or WPW and without therapy.	All sports
Ventricular extrasystolis	In the absence of: arrhythmogenic cardiac disease or condition, family history of sudden death, symptoms (presyncope, fatigue, dizziness), relation to exercise, frequent extrasystoles and / or polymorphic and / or paired frequent with short RR interval.	All sports
Non-sustained ventricular tachycardia	In the absence of: arrhythmogenic cardiac disease or condition, family history of sudden death, symptoms (presyncope, fatigue, dizziness), relation to exercise, multiple episodes of non-sustained ventricular tachycardia with short RR interval.	All sports
Slow ventricular tachycardia, fascicular ventricular tachycardia, right ventricle outlet tachycardia	In the absence of: arrhythmogenic cardiac disease or condition, family history of sudden death, symptoms	All sports, except those with high risk if there is a syncope episode
Syncope	Neurocardiogenic	All sports, except those with high risk if there is a syncope episode
	Arrhythmic or primary cardiac	See specific cause
Long QT syndrome	.	Not to practice competitive sports
Brugada syndrome		Not to practice competitive sports
Implanted pacemaker	Normal increase in heart rate during exercise, without significant arrhythmias, normal cardiac function	Sports low to moderate dynamic and low static (IA, B), except those at risk of bodily collision.
Implantable cardioverter-defibrillator	No malignant ventricular tachycardia, normal cardiac function, at least 6 months after implantation or ICD intervention last.	Sports low to moderate dynamic and low static (IA, B), except those at risk of bodily collision.

10. Basic life support in the athlete

10.1. Sudden death in athletes

Although rare, sudden death (SD) in sports is an event that causes public outcry, especially when it involves high--performance athletes. Statistics from different countries show that its prevalence varies from 0.28 to 1 per 100,000 athletes[159, 160]. Different structural and arrhythmogenic variations are responsible for cases of sudden cardiac arrest in athletes, however, the most common is hypertrophic cardiomyopathy, accounting for 25-36% of cases[159, 160]. Vigorous exercise linked to heart disease seems to be the hidden trigger that triggers the arrhythmia responsible for cardiopulmonary arrest (CPA).

Guidelines nacionais3 internacionais161 and advise the pre-participation cardiovascular screening, which is, in general, in a detailed history, physical examination and 12-lead electrocardiogram. Such a screening would be able to detect approximately 90% of athletes who had a fatal event, although there are reports of the sportsmen SD victims who did not have any structural or condução162 isolation, screening is not able to significantly decrease the number of deaths from sudden cardiac arrest in this risk group, mainly by not performing this evaluation by athletes and the possible false-negative results, necessitating the establishment and strengthening of a second pillar, basic life support.

10.2. Initial care to the athlete

Care for better emergency care are summarized as a set of actions taken in the first minutes that follow after a sudden event. These actions can be summarized in two pillars: the organization and planning of the emergency care team, at the places where physical activity is performed, and training of first responders in CPR and handling of automated external defibrillator (AED). Sites where physical activities, such as training centers, schools, colleges, gyms, etc.. must have an emergency response plan well organized, with professionals trained in basic life support (BLS) and communication services with fast and effective prepared to perform advanced life support in cardiology (ACLS).

Effective treatment of an athlete who suffers a sudden PCR depends on a sequence of interdependent actions, so that, when linked together, form a chain effect, increasing the survival rate of victims, named by the American Heart Association, the "current survival, "comprised of the following links: fast access, early CPR, early defibrillation and early ACLS.

Most sudden PCR in athletes occurs due to a tachiarrhythmia[163] (ventricular fibrillation) and therefore can be treated with defibrillation. The reduction in mortality in athletes surprised by such an event requires CPR training programs and handling the DEA, and rescuers equipped and trained to recognize emergencies, activate the emergency system, providing quality CPR and use the AED. There are current guidelines for sports facilities that have more than 2,500 patrons or develop physical activity programs for individuals belonging to certain risk groups, such as heart disease and the elderly, have defibrillators strategically allocated[131].

It is already well established that for each minute without cardiopulmonary resuscitation (CPR) survival of a victim PCR witnessed decreases from 7 to 10%. However, in the context of ventricular arrhythmias structural diseases appear to be more susceptible to small delays compared to a defibrillation[129] context a structurally sound heart, which possibly brings to athletes victims of a sudden PCR a more significant decline in survival while waiting for an AED, stressing the utmost importance of the third link in the chain, early defibrillation.

The consequent increase in survival programs of public access to defibrillation has been well documented in numerous studies, including leases as casinos[164], and airplanes[165] airports [132] however, little is known about the impact of this initiative on the specific risk group of athletes, mainly by that this is a rare event.

Important risk marker, the pre-participation medical evaluation should be mandatory for practitioners of physical and sports activities, as it is able to detect cardiovascular abnormalities that predispose to sudden death. Despite different recommendations, there is a consensus that every athlete should do, necessarily, a medical history, physical examination and 12-lead ECG, complementing the research with other tests according to the degree of suspicion.

Sportsmen victims of SD should be handled promptly and need immediate CPR quality, providing a vital amount of blood flow to the brain and heart. The local emergency team should be able to accomplish an ideal time between collapse and defibrillation of 3-5 minutes, thus increasing the chances of success of shock. As in most cases the post-pace defibrillation is not capable of a perfusion effective CPR should be resumed immediately after the shock.

Finally, periodic medical evaluation, an effective local protocol for emergency responders and trained in BLS and able to provide quality CPR, early defibrillation and quick contact centers with qualified ACLS are the pillars of support for a common goal: reducing the number cases of sudden death in athletes and increased survival of victims.

10.3. Special aspects of prevention of SD related to exercise and sport

10.3.1. Doping: illicit substances in sport (www.wada-ama.org)

Some used as doping substances are able to cause deleterious effects, particularly cardiovascular system, including sudden death. Among the most commonly used substances include anabolic steroids themselves, ephedrine and amphetamine. Among the most used social drugs are cocaine, marijuana and 3,4-methylenedioxymethamphetamine MDMA, known as ecstasy.

10.3.1.1. Anabolic steroids (AS)

These substances cause various side effects, including unwanted cardiovascular effects. The EA may induce arterial hypertension (AH) and secondary nephrosclerosis. Testosterone may cause increased vascular response to norepinephrine and, as a consequence, promote fluid retention and increased peripheral vascular resistance leading to HA3.

Another important effect is described by Tagarakis et al[166], which demonstrated for the first time at the microscopic level, an adaptation of cardiac capillaries myocytes

and with concomitant use of anabolic steroids and physical training. That would cause a disproportionate increase in myocardial mass in relation to cardiac capillaries. The results of this study suggest that the use of EA could develop an imbalance between supply and O2 consumption, especially during exercise.

10.3.1.2. Ephedra

Stimulants in general cause tachycardia and increased myocardial oxygen consumption, which may cause arrhythmias and myocardial infarction in susceptible individuals.

Ephedrine can cause symptomatic ventricular tachycardia, ventricular extrassísstoles frequent atrial fibrillation and sudden death. Importantly, many so-called natural products and "derived from flora" have substances like ephedrine in its formula without being part of the bull.

10.3.1.3. Cocaine

Cocaine use causes widespread vasoconstriction, and its main consequence, hypertension. Although cocaine use cause intense vasoconstriction in the central nervous system, this may also impact on other organs such as the kidneys causing failure renal1. Cocaine use can cause, yet, MI, cardiac arrhythmias, congestive cardiomyopathy, myocarditis, subarachnoid hemorrhage, rupture of the aorta, hypertension, myocardial ischemia or induced by exercise and sudden cardiac death.

10.3.1.4. Amphetamines

It is the prototype of CNS stimulants. Have wide variety of salts and mixtures under various forms of presentation, the most used dextroamphetamine sulfate. This substance has a direct action by stimulation of adrenergic receptors in cortical level and ascending reticular activating system and an indirect action, displacing the endogenous sites of their nerve endings. Its side effects are more pronounced: general insomnia, dizziness, profuse sweating, shivering and euphoria; Cardiovascular palpitations, tachycardia, chest discomfort: neurological brain hemorrhage.

10.3.1.5. MDMA (3,4 - methylenedioxymethamphetamine) (ecstasy)

It is a hallucinogen similar to amphetamine. Due to its low cost and availability in tablets, its popularity and consumption have increased significantly. The ingestion of MDMA increases the release of serotonin, dopamine and norepinephrine by presynaptic neurons. Furthermore, prevents the metabolism of neurotransmitters by inhibiting monoamine oxidase. Its major cardiovascular effects are hypertension, tachycardia and arrhythmia, which can lead to SD[167, 168].

10.4. Evaluation of the athlete, organization and planning of care

Sudden death related to exercise and sport is a dramatic event and the doctors and some measures can and should be taken to try to prevent this rare but dreaded complication of sports / exercise.

We describe some preventive measures related to SD related to exercise and sport.

10.4.1 Aspects related to the athlete

10.4.1.1 - Pre-participation evaluation

Considering that in most cases the SD associated with the sport are caused by known or undiagnosed heart, every candidate for physical activity should undergo clinical examination prior, regardless of age. This clinical examination should be preceded by a good history with particular attention to the family history of cardiovascular disease and sudden death. The pre-participation evaluation in an attempt to detect these pathologies is the most efficient way to prevent one cardiovascular event fatal3. In a recent document, the International Olympic Committee ratifies the importance of periodic medical evaluations for athletes elite2.

While acknowledging that clinical examination alone can not fail to detect all heart diseases with potential to cause SD, this procedure with emphasis on the cardiovascular examination, preceded by a thorough history and past medical history and family history, is the first step to proper evaluation of the athlete.

Clinical examination should be accompanied ideally by a 12-lead electrocardiogram. Although there is a disagreement between the American school (this only recommends medical history and physical examination) and European School (this recommends adding the 12-lead electrocardiogram to anamnesis and clinical examination) 52, we remember that in the SBC guideline criteria for diagnostic test ordering Cardiovascular entity considers that the 12-lead electrocardiogram as a compulsory examination first visit cardiology (ECG-1-1096) 169. Moreover, it is known that numerous electrical heart diseases that can lead to SD are likely to be diagnosed by ECG at rest, including the long QT syndrome, Brugada syndrome, syndrome Lenegre-Lev syndrome and Wolf -Parkinson-White170. The European protocol that includes history, physical examination and electrocardiogram is adopted currently by the International Olympic Committee, the Italian Olympic Committee, FIFA and Football Union (UEFA) 171. Clinical examination should include a personal and family history and a specific screening for the syndrome Marfan172.

A more detailed evaluation before participation in physical activities and sports available in another section of this guideline.

10.4.1.2 For the preparation of the athlete

Tracking the athlete has to be done holistically. Therefore, to prevent cardiovascular clinical events, preventive measures are also important basic, adequate nutrition and hydration, respecting rest periods and avoiding training and competition in the hottest times of the day. Furthermore, it is important to monitoring and observation of athletes in training and competition for qualified medical staff, preferably with experience in sports medicine and basic first aid in cases of emergency clinical situations.

10.4.2 Issues related to training and competition venues

10.4.2.1 Emergency service and medical contingency plan

In addition to all the material needed to care for cardiac arrest should be prepared a contingency plan in medical

training and competition venues, with staff trained in cardiopulmonary resuscitation, in the event of an emergency or clinical cardiovascular, optimizing the transport of athletes hospital for more complex, as the case[173, 174].

10.4.2.2 Automated External Defibrillator

The automated external defibrillator (AED) should be available for use in less than 5 minutes in training and competition venues, clubs, arenas, stadiums, gyms and rehabilitation clinics with staff trained in cardiopulmonary resuscitation [174, 175].

Among young athletes cardiac arrest occur, usually after intense training sessions or during a competition. Although these events are rare (corresponding to 1% of those that occur in middle-aged or elderly), the value of a prompt service and successful resuscitation improves survival in long term[176].

11. Para-athletes or athletes with special needs.

11.1. Cardiologic evaluations: pre-participation and re-evaluations

All Paralympic athletes must undergo independent evaluation of age, sex and disability associated with, the pre-participation evaluation should include children, adolescents, adults, master / seniors, men and women, under the sole responsibility of the attending physician (Recommendation Grade: I , Evidence level: C).

The evaluation should be comprehensive, taking into account the organism as a whole, emphasizing the physical and somatic; should keep in mind the interactions between physical, comorbidities, and their sequelae in physical training and sports performance (Recommendation Grade: I, Evidence level: C) (Figure 4).

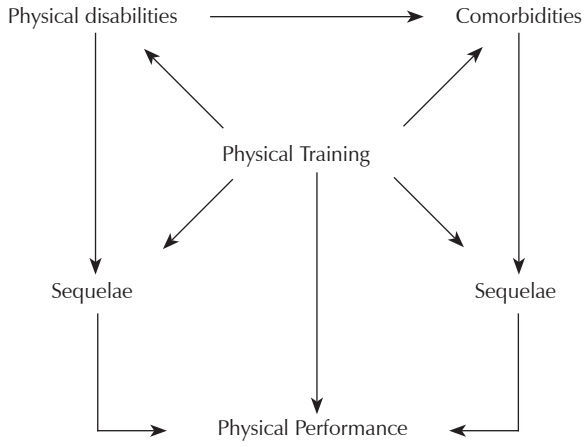


Figura 4 – Interações entre deficiência, co-morbidades, e respectivas sequelas no treinamento fisico e desempenho esportivo.

The frequency of re-evaluations should be at the discretion of the attending physician, according to the characteristics of each case, however, the frequency should have the primary aim of the sport safe (Recommendation Grade: I, Evidence level C).

The evaluation of Paralympians obeys defined protocol, summarized in Table 21

Table 21 – Protocol for the evaluation of paralympic athletes according to the Medical Department of Brazilian Paralympic Committee[10].

1. Application of standardized medical questionnaire, involving identification, personal and family history, sports history, eating habits and life;
2. Physical examination, standardized medical record;
3. Laboratory tests: blood count, serum iron, ferritin, folate, vitamin B12, blood type, total lipids, cholesterol, HDL, triglycerides, uric acid, glucose, parasitological, urinalysis, creatinine, urea, sodium, potassium, testosterone, free testosterone, insulin, cortisol, free T4, free T3, T3, T4, TSH, serology for Chagas, herpes, HIV and HCMV, total protein, AST, ALT, GGT, alkaline phosphatase, calcium and homocysteine;
4. Chest radiography;
5. Resting ECG and exercise stress test.

After the initial evaluation, according to the findings must be given specialized tests at the discretion of the attending physician, such as cardiopulmonary exercise testing (CPET), echocardiogram (ECHO), vectorcardiogram (VCG), computed tomography, magnetic resonance, ultrasound, hemoglobin electrophoresis (research SCD) and cardiac evaluations, ophthalmic (Marfan research, glaucoma, retinal detachment) and orthopedic[177, 178] (Recommendation Grade: I, Evidence level C).

In athletes with cerebral palsy can use the evaluation score of spasticity (QSFC Quantitative sports and functional classification), based on the condition of muscles of upper and lower limbs and trunk, for use in clinical research, clinical and physical training[179].

Athletes in wheelchairs and people with dentures should be thoroughly and then examined for pressure sores or scabs on the stump of the prosthesis implantation. The presence of ulceration in these places becomes temporarily ineligible athlete until they are restored local conditions of integument (Recommendation Grade: I, Evidence level C). The practice of urinary retention in wheelchairs should be ruled out, given the risk of large elevations of blood pressure and stroke. In cases of neurogenic bladder should be alert to the presence of subclinical urinary infections.

11.2. Cardiopulmonary exercise testing

The basics of cardiopulmonary exercise testing protocols include: 1) reproducibility of sports act according to the principle of specificity, 2) the adequacy sport and means of locomotion athlete, 3) perform the tests with stability and security, ensuring accuracy and reproducibility of measures[11] (Grade Recommendation: I, Evidence level: B).

Special care should be taken in relation to the type and degree of disability, the athlete's posture, at room temperature, prior to emptying, prevention of hypotension, the risk of seizures and accidents, the blood pressure measurements and the proper veiling masks. Several factors may limit performance in evaluations: 1) Clinical: intellectual and sensory disabilities (visual, tactile, auditory, epilepsy, autonomic dysreflexia, neurogenic bladder, deprivation friendly, post-polio syndrome, tachypnoea stress, malnutrition, 2) locomotion: reduced mass, muscle strength and flexibility, increased muscle tone, decreased joint mobility, motor incoordination, osteoarticular lesions secondary to sports, injuries from amputation stumps, 3) cardiovascular: any associated disorders, 4) physiological: reduction of VO2 PICO, anaerobic threshold, respiratory compensation point, early fatigue, physical inactivity, and 4) socioeconomic and cultural: social exclusion, lack of patrocínio177.

According to the principle of specificity, has been used to arm ergometers players, throwers, weightlifters, swimmers and fencers, bike cycling and treadmill to other modalidades177; cyclists can use your own equipment coupled to the system 850 Mag Minoura180 (Recommendation Grade: I, Evidence level: C).

The cardiopulmonary tests can be performed by several specific protocols, citing the example summarized in Table 22.

Table 22 – Protocols for cardiopulmonary tests in Paralympic athletes (Centro de Estudos em Fisiologia do Exercício – Unifesp / Escola Paulista de Medicina)[177].

ET Wheel Chair (in wheelchair) on treadmill	Initial speed 3-13 km / h, the initial inclination 0-2% increments from 0.5 to 1.0 km / h and 0.5 to 1.0%, every 3 minutes
ET on treadmill	Initial velocity 3-8 km / h, the initial inclination of 0% increments from 0.5 to 1.0 km / h and 0.5 to 5.0%, every 3 minutes
ET on exercise bike	Initial load of 25 to 50watt and increments every 25watts
ET in bicycle-roll	3 minutes
ET ergometer in arms:	Initial speed 30-33 km / h increments of 3 km / h every

The protocol Knechtle and Köplif (Institute of Sports Medicine Swiss Paraplegic Centre) for treadmill test in wheelchairs, starts at speed of 8 km / h and slope of 1%, with 0.5% increments every 2 minutes and speed constant until exaustão181, 182.

Field tests can also be performed with vantagens183, 184. Variations between 48-80% have been described in the regression equations for determining the physical capacity in paraplegics and quadriplegics. These variations can be explained by the level and degree of spinal cord injury, age, gender, physical activity and weight corporal183. In our environment, the values for the aerobic power of Paralympic athletes have been similar (Table 23)[180,185].

The pre-participation evaluation for leisure activities is similar, depending on the physical and mental stress. On many occasions, given the emotional burden involved and the lowest level of training, physical and psychological stress can be of great intensity, similar to the relative degree of competitions.

Table 23 – Aerobic power of Brazilian paralympic athletes participating in the Atlanta Games Silva AC, Oliveira Fº JA. Evaluation of Atlanta paralympic athletes. Non-published data. Unifesp-EPM, São Paulo, 2006 [186].

Modality/deficiency	n	$VO_{2\ PICO}$ $ml.kg_{-1}.min_{-1}$	Variation $ml.kg_{-1}.min_{-1}$	LA %
Soccer ♂ CP	18	50.6 ± 6.70	36.5 – 62.8	70 ± 9
Swimming ♂ tetra, PM, ML	7	36.8 ± 17.7	19.8- 59.0	64 ± 5
Swimming para, PM, ML	4	48.9 ± 9.90	35.3 – 61.4	56 ± 9
Basketball ♀ PM, ML, amp	14	30.0 ± 6.00	20.0 – 40.0	61 ± 8
Tennis ♂ ML	2		29.7 – 33.3	60
Table tennis ♂ ML, PM	2		31.0 – 34.5	64, 67
Judo ♂ VD	4	45.5 ± 12.0	36.0 – 62.0	59 ± 11
Field/wheelch ♂ tetra, PM, CP	3	32.8 ± 10.0	25.0 – 44.0	60 ± 2.9
Field/wheelch ♂ para, amp	2		39.0 – 42.0	47, 62
Pista ♂ VD	3	57.0 ± 7.0	50.0 – 65.0	80 ± 5
Pista ♀ VD	2		51.0 – 59.0	46, 72
Pentatlo/cad. ♂ Para, PM, amp	2		44.0 – 51.0	64, 81

$VO_{2\ PEAK}$ = peak aerobic power; AT = anaerobic threshold; ♂ = masculine; ♀ = feminine; CP = cerebral palsy; ML = medullary lesion; PM = poliomyelitis; VD = visual deficiency; amp = amputated; tetra = tetraplegic; para = paraplegic; wheelch = wheelchair.

12. Prevention of events / sudden death in sports

The event prevention should include prevention of accidents, injuries and aggravation of preexisting comorbidities and sudden death.

The goals of a protocol event prevention in sports based on pre-participation screening:

Identifying predisposing conditions, ie cardiovascular diseases that can potentially cause sudden death;

Set if there are steps that can be taken to reduce the risk of sudden death: What? How should they be developed?

Standardize the approach to be adopted in every heart and discuss the possible disqualification of the athlete's exercise of his profession.

Prevention of events and sudden death in sports and leisure is performed taking into account the early diagnosis and treatment of cardiovascular diseases, as well as the application of existing criteria and unenforceability specific to the various conditions described in Parts I and II of this document and properly applied to Paralympic athletes[187]. It is imperative that the resources exist venues doctors and paramedics, appropriately equipped for emergency care.

In many institutions, the clinical director and / or the physician responsible for the care answerable to the respective Regional Council of Medicine compliance with those standards.

Individuals newly hospitalized or sedentary longstanding need progressive training with gradual increments in the frequency and duration of sessions and the intensity of the exercises, besides the risk of injuries and physical sequelae, the appearance of these can be discouraging factor training, damaging self-image, and predisposing to cessation program (Recommendation Grade I, Evidence level C).

12.1. Ethical Aspects

The medical evaluation should include experts from various fields, highlighting the sport and exercise medicine, cardiology, orthopedics and physiatry.

When it comes to athletes with disabilities, it is important to emphasize that it is the exclusive competence of the physician directing the training, diagnose any diseases and sequelae, order tests, prescribe treatment and athletes away from sports, being forbidden to assign medical functions or delegate its exclusive competence for professionals not qualified to practice medicine. (Federal Medical Council, Resolution No. 1236/87). Moreover, the implementation of the training will be conducted by physical education teachers and therapists. The interaction between physicians, physical education teachers, physiotherapists, physiologists, nutritionists and psychologists is critical to the success of the program. The training prescription should be made in prescription doctor, aiming mode, frequency and duration of sessions, training intensity and other observations, at the discretion of the attending physician. This conduct is ratified by the Federal Medical Opinion 4141/2003: "For all the above, it is for the doctor, after diagnosing the disease, prescribe the appropriate treatment to the patient and even the prescription of physical activity in the face of pathology diagnosis or prevention of various pathologies."

In many institutions, the clinical director and / or the physician responsible for the care of these standards by responding to the respective Regional Council of Medicine.

The relationship between doctor and other professionals practicing in health care should be based on mutual respect, freedom and independence of each professional, always seeking the interest and welfare of the patient (Code of Medical Ethics 08/01 / 1988 Article 18). The interaction between doctors and para-medical physical education teachers, physical therapists, nutritionists, psychologists and coaches is critical to the success of the training program and should be encouraged at every moment.

12.2. Recommendations

Nowadays, given the paucity of reports in the literature, the criteria for answering Paralympians are based generally on expert consensus (Evidence level C).

The determination of eligibility sports must follow the protocol of the International Paralympic Committee Classification Code and International Olympic Committee and Standards188 Brasileiro189. Thus, the para-athlete may be eligible and ineligible to a modality to another. The eligibility criteria are defined for each sport Paralympic sport by federations internacional190.

The recommendations for the care of Para-athletes are listed in Table 24.

Table 24 – Recommendations for the care of para-athletes (Recommendation Grade: I, Evidence level: C).

1. All para-athletes should be submitted to evaluation regardless of age, gender and associated disableness:
2. The pre-participation evaluation should include children, adolescents, adults and aged, men and women, under sole responsibility of the assistant physician:
3. The frequency of re-evaluations should be at the discretion of the attending physician, according to the characteristics of each case, the frequency should have the primary aim of the sport safe:
4. Evaluations should follow the protocol of the International Paralympic Committee and should be specific to each modality and individualized for each athlete:
5. The clinical and cardiologic must be coordinated and implemented by physicians; involving physical education teachers, physiotherapists, physiologists, nutritionists and psychologists should be part of the evaluation, is of great value integration between physicians and paramedics:
4. The clinical evaluation must include all equipment and systems in the body, being carried out by multidisciplinary team and involving various medical specialties:
5. A cardiovascular evaluation follows the same eligibility criteria related to athletes in general:
6. The pharmacological prescriptions should always be oriented according to the most recent determinations of WADA which are updated periodically.

13. Appendix

13.1. Elaboration of aptness or release reports to exercise and practice sports[173]

The medical report to release for amateur and competitive physical activities is a part of the medical act and a patient right. It should be objective, clarifying as to the kind of physical activity allowed and preferably suggesting the physical training intensity. In cases in which there are restrictions to the practice of a modality, those should be clearly mentioned in the document. Any and every information concerning the clinical profile, physical examination and complementary exams should be in the medical report when required and authorized by the patient.

With basis in evidence-based medicine the individual risk stratification is more appropriate, considering clinical condition and physical aptness, gender, age and conditions under which this practice will be performed. For most situations it is possible to compare the risk of a given individual with that expected for their fellows of same age and thus characterize the risk as much smaller, smaller, similar, bigger or much bigger. Specifically, it may also be convenient to characterize the practice as therapeutic, recreational or competitive. In addition, environmental factors, notably climatic, may be relevant.

As to the issuing of the report, this should characterize or specify any clinical restriction, be it of locomotor or cardiorespiratory nature. Ideally, the conclusive statement should attain to the limits of what was actually examined or evaluated, avoiding generic and poorly funded sentences as "apt to sports practice", being probably more adequate to use something like "formal clinical contraindications to the practice of recreational or competitive physical exercise were not found".

When the decision is for the professional disqualification, the part most interested in the information should be the athlete or their legal representative (when under 18), even when the athlete is part of a team and his/her evaluation was offered by the club or association to which they are associated. In these cases it would be prudent that the report be signed by the physician responsible and at least two other physicians that follow the decision, besides the athlete himself/herself, in at least two copies, one of them filed in the athlete's medical records. Special care must be taken to keep the athlete's integrity, keeping the secret required in each case.

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15. List of Tables