Physical activity in the young: symptoms and ECG abnormalities, any relation?

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Abbreviations List

CBBB	Complete Bundle Branch Block
ECG	Electrocardiogram
ER	Early Repolarization
HCM	Hypertrophic Cardiomyopathy
IBBB	Incomplete Bundle Branch Block
LQTS	Long QT Syndrome
LV	Left Ventricle
LVH	Left Ventricle Hypertrophy
RBBB	Right Bundle Branch Block
SCD	Sudden Cardiac Death

Figures List

Tables List

Abstract

Extensive physical conditioning leads to structural cardiac remodelling, to rhythm and electrical conduction abnormalities. This cardiac remodelling is known by the expression athlete's heart and can be detected on ECG and echocardiography.

The aim of our study was to compare the prevalence of changes in the clinical history and in ECG between the individuals involved in regular physical activity and those who have a more sedentary lifestyle. For that purpose we used a sample of 241 young individuals participating in the Sudden Cardiac Death – Screening of Risk Factors trial, assessed by means of a questionnaire and an ECG.

No significant statistical differences were found in the prevalence of symptoms, previous cardiac disease and family history between the two groups. However, we found differences in heart rate, PQ, QRS and QT intervals. The prevalence of right bundle branch block presented was similar to the one reported for collegiate athletes. Early repolarization was two times more prevalent in the group that practice regular physical activity; more studies are needed to clarify the implications of this fact, since it is not clear if it is part of adaptive cardiac remodelling or not.

In sum, most of the cardiac remodelling associated with the practice of regular exercise occurs mainly in highly trained athletes and not as often in people that engage regular non competitive physical activity.

Keywords: sudden cardiac death; young athletes; exercise; ecg

Resumo

A prática regular de exercício físico conduz a um *remodelling* cardíaco estrutural e a alterações do ritmo e da condução elétrica. Este fenómeno é conhecido pela expressão coração de atleta e pode ser detetado através de exames complementares como o ECG e o ecocardiograma.

O objetivo do nosso estudo foi comparar a prevalência de alterações na história clínica e no ECG entre praticantes de atividade física regular e indivíduos com estilo de vida sedentário. Para esse efeito usámos uma amostra de 241 jovens que participaram no estudo *Sudden Cardiac Death – Screening of Risk Factors* através do preenchimento de um questionário e posterior realização de um ECG.

Não foram encontradas diferenças estatisticamente significativas na prevalência dos sintomas, doenças cardíacas previamente diagnosticadas e história familiar entre os dois grupos. No entanto, foi possível encontrar diferenças na frequência cardíaca e intervalos PQ, QRS e QT. A prevalência de bloqueio de ramo direito encontrada foi semelhante à reportada para atletas universitários. A repolarização precoce foi duas vezes mais prevalente no grupo que praticava regularmente exercício físico; são necessários mais estudos para que seja possível clarificar as implicações deste facto, uma vez que ainda não é claro se faz parte do *remodelling* cardíaco ou não.

Em suma, a maioria do *remodelling* cardíaco associado à prática de exercício físico regular ocorre sobretudo em atletas de alta competição e não tão frequentemente em indivíduos que praticam exercício físico não competitivo de forma regular.

Introduction

The beneficial effects of regular exercise in the prevention of cardiovascular disease are well established[1]. However, regular physical exercise can lead to physiological adaptation in cardiac dimensions, including increased LV wall thickness and LV cavity size, in order to enhance left ventricle filling during diastole and the overall cardiac output. Cardiac remodelling, in response to physical activity, is usually known as "athlete's heart" and can be detected trough ECG and echocardiography[1,2]. Extensive physical conditioning not only causes structural remodelling of the heart but it can also affect rhythm and electrical conduction (sinus bradycardia, first-degree atrioventricular block, early repolarization and premature ventricular beats)[2].

Athletes with cardiovascular abnormalities are at greater risk of exercise-related sudden cardiac death (SCD)[1,2]. Not only SCD is responsible for 50% of the deaths related to coronary heart disease affecting the older population [3–5], but it is also the leading cause of death in exercising young athletes[6,7]. In general young-adult population the incidence of SCD is 0,001-0,002% per year[8].

In general, SCD is the result of the combination of cardiac structural lesions and transient acute abnormalities that trigger the fatal event[8–10]. It is also demonstrated that individuals who practice physical activity less often present higher risk of SCD compared to those who practice exercise regularly[8].

SCD predominates in male gender (3:1) as well as in young athletes (9:1)[5,8,11]. The remodelling of cardiac structures that results from extensive exercise produces abnormal ECG's in up to 80% of highly trained athletes and is often consistent with LVH[2,7]. Some studies show that a 3 months period of restriction from exercise may decrease LV wall thickness by 2-5mm and is an effective method differentiating training-induced hypertrophy from the pathologic HCM, a well-known risk factor of SCD[2,7].

Abnormal ECG findings are more common in certain subsets of athletes such as those who participate in endurance sports (e.g. cycling, rowing and triathlons) or sports with high peak levels of activity (e.g. basketball, track and soccer)[7].

Family history of potential hereditary conditions such as HCM and long QT syndrome can be considered a risk factor for SCD; so, when assessing an athlete, identifying any diagnosed relatives with such diseases should lead to further investigation[2].

Personal history of chest pain, excessive shortness of breath, palpitations and syncope are positive findings for possible cardiac abnormalities that can lead to SCD. It is known that exercise-related syncope may indicate LV outflow obstruction, arrhythmias or congenital coronary abnormalities[2].

When SCD happens in an athlete it is difficult for the general public to understand it because athletes are perceived as the healthiest segment of the society[1,7]. There is the emerging need to separate between the cardiac remodelling that represents a physiological adaptation of an athlete's heart to exercise and cardiac pathologies that may predispose to SCD. This is relevant firstly because false-negative evaluations may be fatal as SCD is an event that often can't be reversed; secondly because false-positive diagnoses may lead to erroneous disqualification from a sport with loss of earnings and significant psychological distress to the athlete[1,7].

Our aim was to compare the prevalence of symptoms in clinical history and changes in the ECG between the individuals that practice regular physical activity and those that have a more sedentary lifestyle.

Materials and Methods

Our sample included a random group of 241 young individuals assessed trough the Sudden Cardiac Death – Screening of Risk Factors (SCD-SOS) questionnaire (Appendix 2)[12] and an ECG. The questionnaire is based on the recommendations of the European Society of Cardiology (ESC), International Olympic Committee and American Heart Association for pre-participation screening in competitive athletes and on the main cardiologic manifestations observed in clinical practice. Its language was simple and accessible and it consisted of eight multiple-choice questions, with blank spaces for further description of symptoms.

Participants are briefly asked about:

1. **Symptoms:**
    a) The ESC definition of **syncope** is a transient loss of consciousness;
    b) **Palpitations** are defined as abnormalities of heartbeat that ranges from accelerated heart rate to very noticeable changes of the heartbeat;
    c) C**hest pain** is defined by a precordial localization, related with exercise or emotions and relieved by rest or vasodilators;
2. **Previous cardiac disease:** had the individual ever attended a cardiology consultation or did he had been diagnosed with any cardiac disease.
3. **Family history:** did any of the family members of the individual die before 50 years in a sudden and unexpected way.

If participants' questions about the questionnaire were answered by the cardio-pulmonology technicians, which had had specific training about this questionnaire.

After that, standard 12-lead ECG were recorded using a portable electrocardiograph Mortara Eli 10 with a paper speed of 25 mm/s and amplification of 0.1 mV/mm.

The measurements taken from the ECG were the heart rate, the PQ interval, the QRS duration and the QT interval. All the intervals were automatically measured by the Mortara Eli 10 electrocardiograph.

The PQ interval was measured in all leads from the onset of the P wave to the end of the Q wave.

The QT interval was measured from the onset of the Q wave to the end of the T wave, as defined by the point where the steep down slope of the T wave crosses the baseline ("Teach the Tangent").

The QT interval was corrected according to the heart rate (QTc) and it was determined using the Bazett's formula: division of the QT interval by the square root of the preceding RR interval.

We considered that the values of reference of our variables were[13]:

- Heart rate: 60-100 beats per minute (bpm);
- PQ interval: 120-200 ms;
- QRS complex duration: ≤100 ms;
- QTc interval: 350-450 ms.

The ECG findings that were used in our study were bundle branch blocks, sinus arrhythmia, LVH, repolarization abnormalities, heart axis deviation, early repolarization, supraventricular and ventricular extrasystoles.

The QRS duration allowed us to determine which individuals had intrinsic impairment of conduction in either the right or the left bundle system (intraventricular conduction disturbances), since it leads to prolongation of the QRS interval. With

complete bundle branch blocks, the QRS interval is ≥120 ms in duration; with incomplete blocks, the QRS interval is between 100 and 120 ms.

Sinus arrhythmia was considered if there was a variation of the P-P interval of more than 120 ms, according to the phases of the respiratory cycle, with a constant P-R interval.

To determine LVH we used the Sokolow-Lyon criteria: SV1 + (RV5 or RV6) > 3.5 mV.

We defined ST depression with T-wave inversions (formerly called the left ventricular "strain" pattern) as repolarization abnormalities.

The QRS pattern in the extremity leads may vary considerably depending on the electrical axis of the QRS, which describes the mean orientation of the QRS vector with reference to the six frontal plane leads. Normally, the QRS axis ranges from -30° to +100°. An axis more negative than -30° is referred to as left axis deviation and an axis more positive than +100° is referred to as right axis deviation.

Early repolarization is characterized by ST segment elevation ≥0.5mm in two consecutive leads and by J wave or terminal slurring of R wave, most frequently in precordial leads.

Extrasystoles are defined as premature beats and their origin can be above the atrioventricular node (supraventricular extrasystoles) and below that level (ventricular extrasystoles).

For the purpose of this study we defined two groups, one that included individuals engaged in regular physical exercise (either participating or not in official sports competition) and the other composed by young adults with a sedentary lifestyle.

Statistical analysis

The quantitive variables were represented by the mean value and the standard deviation, and the categorical variables were represented by proportion in the total. We used the Chi square test with Fisher's exact test for the categorical variables and the unpaired Student's T-test for quantitate variables.

For statistical analysis the program used was STATVIEW, version 5.0, SAS Institute.

All the participants signed an informed consent (Appendix 1) and the anonymity of all the participants was guaranteed.

Results

Our population consisted of a random group of 241 young individuals with a mean age 25,8 ± 6,3 years, maximum 34 years. The majority of our population were females (n=136, 56,4%).

When asked about the practice of regular physical activity, 95 individuals were engaged in some sort of regular physical activity as opposed to 146 who were not (Fig. 1).

Figure 1

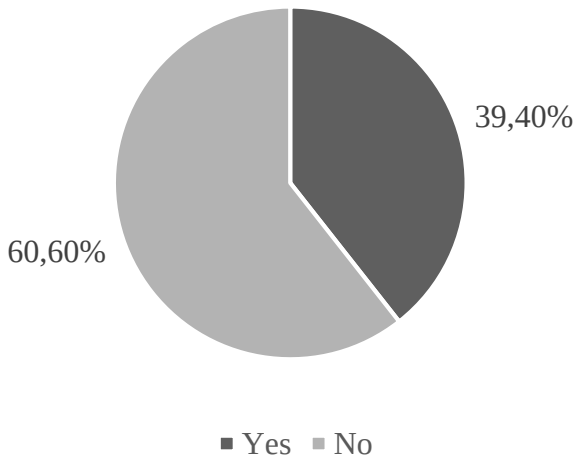

Fig.1 – Regular physical activity distribution in the studied sample

The results obtained in the clinical history are summarized in Figure 2 and Table 1. All the individuals answered the questions about previous episode of syncope and if they had ever felt palpitations or chest pain.

240 individuals (99,6%) answered the question of having or not a previous diagnosed cardiac disease and of attending to a cardiology consultation at least once (Table 1). Only 225 (93,4%) answered the question about having a relative that died unexpectedly before 50 years old (Table 1).

Figure 2

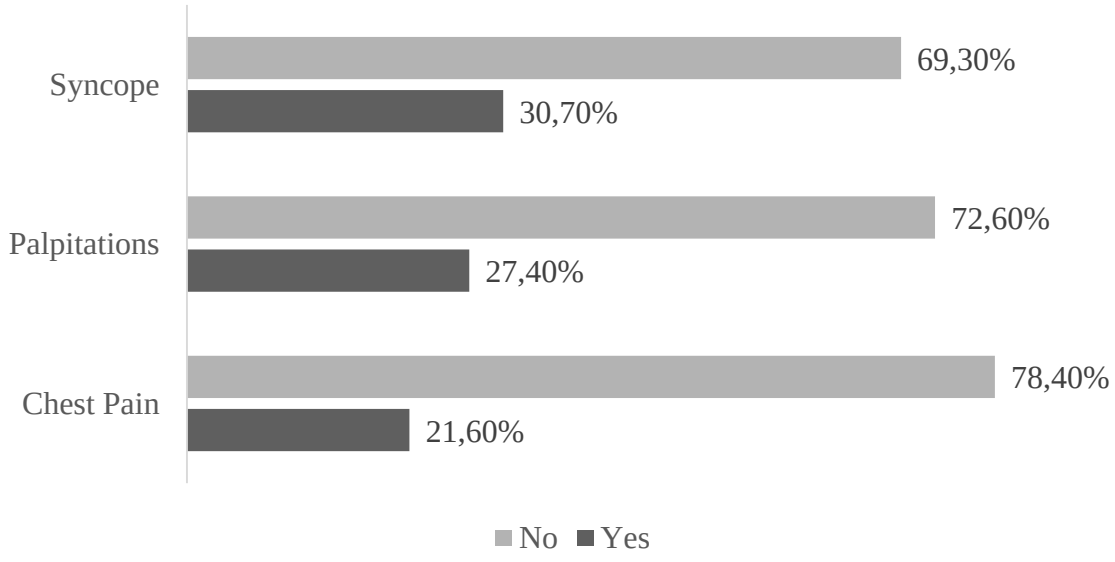

Fig.2 – Symptoms

Table 1 – Previous cardiac disease and family

	Yes		No	
	n	%	n	%
Cardiac Disease	14	5,8	226	94,2
Cardiology Consultation	76	31,7	164	68,3
Sudden death in a <50 years old family member	18	8,0	207	92,0

The measurements taken from it are presented in Table 2. The mean of the 5 variables studied were included in the expected interval of reference.

Table 2 – ECG measurements

	Mean ± Standard Deviation
Heart Rate	69,6 ± 11,8
PQ	148,3 ± 21,3
QRS	94,8 ± 11,7
QT	377,7 ± 28,2
QTc	394,7 ± 18,3

In our sample we found only one case of complete bundle branch block in the group of the participants that practice some sort of regular physical activity and none in the group of participants with a more sedentary lifestyle (Table 3).

The 15 cases presented as incomplete bundle branch block are, in fact, right incomplete bundle branch block (Table 3).

Table 3 – ECG findings in the studied sample (n=241)

	Yes		**No**	
	n	%	n	%
Complete Bundle Branch Block	1	0,4	240	99,6
Incomplete Bundle Branch Block	15	6,2	226	93,8
Sinus arrhythmia	52	21,6	189	78,4
Left Ventricular Hypertrophy	20	8,3	221	91,7
ST Abnormalities	15	6,2	226	93,8
Left Axis Deviation	3	1,2	238	98,8
Right Axis Deviation	10	4,1	231	95,9
Early Repolarization	21	8,7	220	91,3
Supraventricular Extrasystoles	1	0,4	240	99,6
Ventricular Extrasystoles	0	0	241	100,0

On Tables 4-8 we present the results obtained in the studied sample, compared between two groups: exercise practitioners and non-practitioners.

The majority of our sample who practiced some sort of regular physical activity were males and we found a significant statistical relationship between gender and physical activity (Table 4).

Table 4 – Physical activity according to gender

	Yes				**No**				**p**
	Male		**Female**		**Male**		**Female**		
	n	%	n	%	n	%	n	%	
Physical Activity	55	57,9	40	42,1	50	34,2	96	65,7	≤0,0005

We didn't find statistical differences between those involved in regular exercise and those who were not in terms of the prevalence of syncope, palpitations or thoracic pain (Table 5), as well as in terms of previous diagnosed cardiac disease or significant family history (Table 6).

Table 5 – Prevalence of symptoms compared between exercise practitioners and non-practitioners

	Answers	Exercise Practitioners		Non-Practitioners		p
		n	%	n	%	
Syncope	74	27	28,4	47	32,2	ns
Palpitations	86	22	23,2	44	30,1	ns
Thoracic Pain	52	20	21,1	32	21,9	ns

Table 6 – Previous cardiac disease and family history compared according to level of regular physical activity

	Answers	Exercise Practitioners (n=95)		Non-Practitioners (n=146)		p
		n	%	n	%	
Cardiac Disease	14	6	6,3	8	5,5	ns
Cardiology Consultation	76	36	37,9	40	27,6	ns
Sudden Death in a <50 years family member	18	8	8,7	10	7,5	ns

Table 7 – ECG measurements compared between the group of exercise practitioners and non-practitioners

	Mean ± Standard Deviation		**p**
	Exercise Practitioners (n=95)	Non-Practitioners (n=146)	
Heart Rate	65,5 ± 10,4	72,2 ± 12,0	≤ 0,0001
PQ	152,2 ± 22,5	145,8 ± 20,1	≤ 0,05
QRS	96,7 ± 11,2	93,5 ± 11,9	≤ 0,05
QT	387,5 ± 28,4	371,4 ± 26,3	≤ 0,0001
QTc	397,2 ± 19,8	393,6 ± 17,1	ns (0,08)

In our sample we were able to find a significant statistical difference in the heart rate and in the duration of the QT interval between the individuals that practice regular physical exercise and those who didn't ($p \leq 0,0001$) (Table 7). However, that relationship wasn't present in the comparison between the QTc, where there was no statistical difference.

The PQ interval and the QRS complex were longer in those who practice some form of regular exercise ($p \leq 0,05$).

There were no other significant statistical differences in the ECG findings between the active group of individuals and the sedentary ones in our sample (Table 8). Despite

of that fact, the early repolarization proved to be two times more frequent in the practitioners of regular exercise and those who didn't.

We didn't compare the prevalence of CBBB, axis deviations, ventricular and supraventricular extrasystoles due to the low number of cases found in our sample.

Table 8 – ECG findings compared between the individuals that practice regular physical activity and those who don't

	Answers	**Exercise Practitioners (n=95)**		**Non-Practitioners (n=146)**		**p**
		n	%	n	%	
Bundle branch block	15	7	7,4	8	5,5	ns
Sinus arrhythmia	52	20	21,1	32	22,1	ns
Left Ventricular Hypertrophy	20	7	7,4	13	8,9	ns
ST abnormalities	15	7	7,4	8	5,5	ns
Early Repolarization	21	12	12,6	9	6,2	ns

Discussion

Symptoms, previous cardiac disease and family history

Among endurance athletes found to have structural heart disease, at least 25% had previously complained of syncope, palpitations or angina pectoris before their sudden demise[7].

Although syncope is a common phenomenon in all age groups, less is known about syncope in competitive athletes[14]. The prevalence in the general population is up to 40%[15]. In our sample we found a similar prevalence of syncope: 30,7%. The estimated prevalence of syncope is 15% in children (<18 years), 16% and 19%, respectively, in men and women aged 40-59 years and 23% in people with more than 70 years[14].

A common final mechanism of any syncope is a transient cerebral hypoperfusion[16]. We can distinguish three types of syncope that predominate in competitive athletes: neurocardiogenic syncope, orthostatic syncope and cardiac syncope[14,16,17].

Neurocardiogenic syncope is due to the sudden inability of the body to maintain blood pressure at a level necessary to maintain cerebral blood flow. It includes vasovagal syncope, vasodepressor syncope, carotid sinus syndrome, cardioinhibitory syncope and situational syncope[14]. Syncope can occur at peak exercise or immediately after stopping exercise; the latter ones may be due to overwhelming vagal input during sympathetic withdrawal[15,16].

Orthostatic syncope may be due to orthostatic intolerance, which can happen more frequently in long distance athletes[16]. Orthostatic hypotension may occur due to volume depletion and also due to the increased contractility of the athlete's heart[16].

The cause of cardiac syncope must be elucidated in order to determine if the syncope is a harbinger of SCD[15,17].This group is characterized by a syncope that occurs predominantly during exercise[15]. Some examples of sources that can lead to SCD in young athletes are HCM, commotio cordis, coronary artery anomalies, electrical abnormalities (arrhythmias) and mechanical factors (structural abnormalities).[14,17,18]

In our sample we didn't find a significant statistical relation between syncope in the active young population and the sedentary groups. One of the reasons that may explain this result is the fact that in many cases, the syncope referred by the participants, according with their descriptions, were vasovagal syncopes, which is, in fact, the most prevalent type of syncope[19]. Besides, part of our sample population was composed of medical students in whom it is known that the prevalence of syncope is higher[19].

Cardiac causes of palpitations are responsible for approximately 43% of all the palpitations, while 31% are due to anxiety and panic disorders, 6% to drugs and 4% to other causes.[20] Although there are no large-scale epidemiological studies that define the aetiologies, it is expected that the incidence of palpitations and rhythm disturbances vary according to the age, the training, the competitive level and the type of sports participation of the athlete[20]. Some studies showed that palpitations or symptoms occurring at rest may be of a more benign nature than those occurring with exercise[20]. Furthermore, epidemiological studies of HCM, right ventricular dysplasia and dilated cardiomyopathy indicate that palpitations and SCD can and do occur at rest[20].

It appears that palpitations and ECG findings of rhythm disturbances are not frequent in the young general active population and are a lot more common in very highly trained athletes as well as in the elderly athletic population.[20] This fact may explain why we didn't find any differences between the assessed groups. Another aspect is that the

great majority of the rhythm disturbances occur more often as an asymptomatic manifestation on an ECG or on examination rather than felt as palpitations[20].

Chest pain is a common problem in the general population, accounting for 20-30% of hospital admissions[21]. Some causes of chest pain may affect athletes more frequently than the general population owing their sport and training patterns or less frequently owing their unique anatomy and physiology[21]. Among the general causes we can highlight musculoskeletal causes as being the most common, cardiac causes (angina/ischemic pain), gastrointestinal causes and respiratory causes[21]. Despite this list of aetiologies, it's not possible to order causes of chest pain related with exercise by incidence because of the lack of available data and the different patterns or conditions according to the sport in question[21]. In our sample, however, we found no evidence that the prevalence of these symptoms is related to regular physical activity. This may be due to the fact that we didn't had a sufficient large sample or because of the distribution of our sample: the majority of the individuals were females and young people with a sedentary lifestyle. It is known that the exercise-related differences are more pronounced in males, due to the fact that usually men practice more exercise than women.

Other important aspect is the fact that our sample of active individuals was composed by people practicing some sort of regular physical activity, which probably, in a lot of them, was not enough for the development of the typical exercise cardiac remodelling. This fact may as well explain the inexistent statistical differences in terms of cardiac diseases previously diagnosed and in the ECG findings that we obtained.

A cardiology consultation may sometimes be one of the requirements of pre-participation screening programs in some sports. That's why we found a large number of participants practicing regular physical activity that had been, at least once, in a cardiology consultation. Besides, some of the practitioners of regular physical activity

may have been submitted to an ECG that was reported by a cardiologist and might have took that as a cardiology consultation.

Some of the diseases related to SCD that have a family heritage are long QT syndrome, Brugada syndrome, HCM, arrhythmogenic cardiomyopathy and non-ischaemic dilated cardiomyopathies[18,22,23]. HCM is the most common genetic cardiovascular disorder[8,18]. Most of the cardiomyopathies that may evolve into SCD have a familial genetic component and most of them are autosomal dominant pattern of inheritance[24]. On the other hand, in terms of arrhythmic abnormalities, family screening is complicated by substantial variability in penetrance, such that the same mutation can cause frequent arrhythmias in one individual, but be asymptomatic in another[18,24]. This justify the question about family history. In general, there was no differences in what regards the this issue between the two groups.

ECG findings and measurements

Incorporating ECG into a screening protocol improves efficacy in identifying conditions capable of causing SCD[1,18]. It is the gold-standard investigation for detection of electrical abnormalities, ion channelopathies and cardiomyopathies[1,18].

Several ECG findings are seen in athletes, some of which are normal variant findings not suggestive of underlying cardiac disease. Many ECG entities mimic certain disease states such as ventricular hypertrophy with significant QRS-complex, ST-segment and T-wave abnormalities worrisome for HCM, for example[7].

In the ECG, the most frequent changes found in athletes are sinus bradycardia, first degree heart block, second degree Mobitz I and repolarization abnormalities[11,25,26]. In our sample, the most frequent abnormality was sinus arrhythmia, followed by early repolarization and then left ventricular hypertrophy.

Heart Rate and Sinus Arrhythmia

Sinus bradycardia is almost constant in an highly trained athlete and sinus rates as low as 25 beats per minute have been documented[7,25]. Langdeau et al tested 100 elite athletes and found that the average heart rate was approximately 52 beats per minute (bpm) vs 65bpm in healthy, sedentary control subjects[7]. Jouven et al reported that heart rate profile during exercise may serve as an independent predictor of SCD; they documented that the risk of SCD was increased in subjects with a resting heart rate >75bpm, <25 bpm, as well as the ones with a decrease in heart rate in recovery period of less than 25bpm[4].The strongest risk predictor for SCD was low increase in heart rate during exercise[4].

Sinus bradycardia depends on the type of sport and the level of training/competition[11]. The heart rate is usually lower in endurance sports like long-distance running[11]. Although we didn't find such a low mean heart rate, we did find a very significant statistical difference between the group with an active lifestyle mean heart rate and the group of sedentary individuals.

Like bradycardia, sinus arrhythmia is believed to reflect an increased vagal tone[11]. Asymptomatic sinus pauses greater than 2s are commonly detected in 24h athlete's ECG, particularly during sleep[11]. In our sample we weren't able to find a statistical difference between those involved in regular exercise and those who were not.

PQ, QRS duration and Bundle Branch Blocks

Variable degrees of atrioventricular block are common. The most common are first-degree (10%) and second-degree Mobitz type I (8%)[7].

IRBBB morphology has been noted on 14% to 31% of ECGs of athletes, 35-50% of marathon runners and 9% in collegiate athletes[7,27]. In our sample of active young adults

we found a prevalence of 7,4%, which is close to the prevalence reported for collegiate athletes. IRBBB is the result of increased cardiac muscle mass and resultant conduction delay[27]. PQ interval is strongly influenced by the delay in conduction at the atrioventricular node.

IRBBB and also CRBBB appear to be markers of exercise-induced cardiac remodelling characterized by right ventricle enlargement[11], diminished relative right ventricle systolic function at rest and interventricular dyssynchrony[27]. The RBBB morphology has been shown to be reversible with deconditioning exercise[7,11]. The published recommendations for athlete ECG interpretation has characterized IRBBB as a benign, training-related finding. On the other hand CRBBB has been reported to be a marker of underlying cardiovascular diseases, such as arrhythmogenic right ventricle cardiomyopathy and Brugada syndrome[27].

We found the prevalence of IRBBB near the already published prevalence in collegiate athletes but we weren't able to find a significant relation with the individuals that practiced regular physical activity.

QT interval, QTc duration and Long QT Syndrome

In general, QT interval is longer in athletes because of the lower resting heart rate associated with athletic training, while the QTc of the athletic group is within normal limits[11]. Prolonged QTc interval may be due to long QT syndrome, genetic or acquired[22]. Acquired causes include anti-arrhythmics (e.g. amiodarone), antipsychotics (e.g. haloperidol and ziprasidone)[22], metabolic changes and electrolyte disorders associated with intense athletic activity[11,28]. The diagnosis of LQTS is based on the triad of prolonged QTc interval, syncope or polymorphic VT and a family history of SCD or LQTS. On the basis of this triad, the prevalence of LQTS is between 1 in 2500 to 10000[22].

Congenital long-QT syndromes are recognized as a cause of adrenergic-mediated polymorphic ventricular tachycardia (VT) and have been implicated in exercise-related sudden cardiac death in young athletes[11,22]. The identification of a prolonged QT interval corrected for heart rate (QTc) in an athlete raises the potential diagnosis of congenital LQTS and issues relating to disqualification from competitive sports. However, it should be kept in mind that an isolated prolonged QTc interval per se does not fulfil the proposed criteria for LQTS[22].

Basavarajaiah *et al* revealed that the prevalence of a prolonged QTc interval in elite athletes is 0.4%, which is similar to Mobitz type first-degree AV block, wandering atrial pacemaker and RBBB that are regarded as normal variants in athletes[22]. It is interesting to observe that the Italian pre-participation screening programme disqualified over 34000 (0.69%) of all athletes based on the identification of a prolonged QTc interval (>440 ms in males and >460 ms in females). Nevertheless, the results of both studies show that LQTS prevalence athletes is higher than other disorders, such as HCM, commonly implicated in exercise-related SCD in athletes[22]. The significance of an isolated prolonged QTc interval of <500 ms in athletes remains unknown but seems to represent a low probability of LQTS or a benign group in whom close monitoring rather than disqualification may be more appropriate in the absence a genetic diagnosis[22].

After the diagnosis of LQTS, subsequent genetic testing may be useful in confirming the genotype and facilitating cascade screening, if applicable[22].

In our study, we found a significantly higher QT interval in the individuals who practiced regular physical activity, but we didn’t find the same for QTc interval. We consider that these results are in consonance with the data reported so far.

Left Ventricle Hypertrophy

The most common changes in the structure of the athlete's heart are an increase in ventricular wall thickness and the diameter of the left ventricle (LV)[11,25,29]. The increase in cardiac muscle mass is an adaptation to the increased demands of the body during training. Up to 80% of athletes meet the Sokolow and Lyon index[7]. In our sample we obtained a significantly lower prevalence: 7,4% of the group that practiced regular physical exercise met the Sokolow and Lyon index.

The physiological LVH in trained athletes usually manifests as an isolated increase of QRS amplitude, with normal QRS axis, normal atrial and ventricular activation patterns and normal ST-segment T-wave repolarization[11]. Exceedingly tall R waves in the precordial leads are not uncommon in the nonpathologic athletes's ECG[7]. This type of LVH is associated to endurance sports such as cycling, cross-country skiing and rowing/canoeing and is also related with male gender[11].

When the LVH is associated with atrial enlargement, left-axis deviation, "strain" pattern and delayed intrinsicoid deflection, underlying cardiac pathology should be suspected[11]. The aetiology of pathological LVH includes chronic arterial hypertension, heart valve diseases and HCM. The latter occurs in 1 in 500 individuals and it is a prominent cause of sudden death before 35 years[11], which happens, most of the times, due to polymorphic ventricular tachycardia or fibrillation[8,18].

In our sample we didn't find any relevant statistical relation between the practice of regular physical activity and LVH.

ST Abnormalities

Repolarization abnormalities (ST depression with T-wave inversions, formerly called the left ventricular "strain" pattern) also may appear in leads with prominent R waves. As opposed to the common occurrence of ST-segment elevation in athletes, the presence of ST-segment depression is rare and should prompt the clinician to pursue pathological causes[7,11]. Its presence isn't associated with top-ranking training, with prolonged training or with any particular type of sport.[25]

T-wave inversion is also observed in the athletic population and the incidence can be as high as 30%. The mechanism of T wave inversion is unclear but likely related to the ST-segment abnormalities stated above. However, normalization of inverted T waves has been observed in the ECGs during stress tests of athletes whose subsequent cardiac catheterizations demonstrated widely patent coronary arteries[7]. It was also found that normalization of inverted T waves could be induced with maximal exercise or isoproterenol infusion[7].

Caution should be given in regards to the dismissal of ST-segment elevations and T-wave inversions as inherently benign changes seen in the highly trained athlete. Although these ECG changes have been described in nonpathologic hearts, authorities recommend using this information with caution[7,25]. T-wave inversion in young and apparently healthy athletes may represent the initial phenotypic expression of an underlying cardiomyopathy, prior to the development of morphological changes detectable on cardiac imaging[11]. The ECG must be interpreted within the context of the clinical presentation[7]. Some studies discarded heart disease with echocardiography, stress test and T1 perfusion studies[25]. If ST abnormalities in ECG suggest heart disease, the athlete should be questioned about family history of SCD or other heart disease, symptoms at rest and during exercise and an echocardiography should be performed. If

there are any pathologic findings, it is necessary to determine whether the athlete should continue physical training[25].

In our sample ST abnormalities were not related to exercise.

Early repolarization

It is known that the early repolarization pattern, also known as J wave pattern, is extremely common in young competitive athletes[7,30,31], especially in subjects engaged in basketball, volleyball and soccer[31]. The characteristics of ER that differentiate it from potentially pathological ST-segment elevation include diffuse ST-segment elevation, upward concavity of the initial portion of the ST segment, notching or slurring of the terminal QRS complex, and concordant T waves of large amplitude[7].

It is estimated that the prevalence of ER in general population range from 20 to 50% [30] and ST-segment elevation has been found in 50% to 89% of resting athletes' ECGs[7]. In fact, some authors argue that ER is the rule rather than the exception[7,11,32,33]. However, there are no prospective studies investigating the SCD risk in athletes related to J-wave patterns. Athletes are generally symptom-free and do not show any decrease in their physical performance[25]. Benign early repolarization in athletes is thought to be related to modifications of the autonomic nervous system (increased vagal tone at rest) and myocardial structure[7,11,25,30]. In some studies, the early repolarization demonstrated no association with the adaptive cardiac remodelling that usually occurs in athletes[30,32], but there have been others that demonstrate that training causes in the development of ER[32]. As such, it seems that although ER may develop in parallel with structural features of the athlete's heart, it is not clearly caused by exercise-induced LV hypertrophy or dilation[32]. Despite these facts, it is a reversible phenomenon which reduces or disappears with deconditioning exercise[11,33]. The ST segments should also be expected to normalize

while engaging in exercise, during or immediately following[7,30] and this pattern may even disappear with aging[30].

Though the association between early repolarization and the practice of regular physical activity was not statistical relevant, but we found that this feature was two times more frequent in people that engaged in regular physical activity.

Conclusions

In what concerns the symptoms reported, we concluded that there were no differences between the studied groups. Giving the nature of our population it was not surprising the incidence of syncope, probably, nonpathologic vasovagal syncopes.

Since the cardiology consultation in a requirement for pre-participation screening programs in some sports, we found a large number of participants that reported at least one visit to the cardiologist.

Based in our study, most of the cardiac remodelling associated with the practice of regular exercise occurs in highly trained athletes and not as often in people that practice regular, non competitive physical activity. Eventhough sinus arrhythmia, early repolarization and left ventricular hypertrophy were found in that group.

The ECG changes, namely PQ and QT intervals, reflect the adjustment to lower heart rates in those who practice regular exercise.

Future Studies

Potential aggravating risk factors should be pursued to access the risk of SCD more accurately. These factors include electrolyte imbalance, stimulants such as caffeine and amphetamine analogues, and other drugs[18].

Another parameter that could be accessed in future studies is the evaluation of the systemic blood pressure. There is very little information on the relation between the conventional assessment of blood pressure and the risk of SCD; since the blood pressure is related to an increased risk of arrhythmias and LVH, a relation between these two parameters might be expected[34].

Some clarification about the exams that should be performed in the pre-participation screening of athletes should be target of future studies. Although the routine use of ECG in the pre-participation screening is the rule in some countries, there are many others where it doesn't fit the screening program. The European Society of Cardiology recommends a pre-participation screening that includes an ECG for all athletes in contrast with the American Heart Association and the American College of Cardiology. As more information is gained on the actual incidence of SCD, the true prevalence of cardiovascular disease in athletes and methods to improve the accuracy of ECG interpretation in athletes, data may soon favour routine use of ECG.[6]

The primary arguments against electrocardiographic screening include concerns regarding false-positive results, cost-effectiveness and psychological implications for athletes and their families[1,35,36]. SCD in the sports arena remains rare and ECG cannot identify all conditions associated with SCD[1,36].

Most nontraumatic deaths are attributed to conditions that can be identified during lifetime and managed with lifestyle modifications including abstinence from exercise of high or moderate intensity, pharmacotherapy and implantable cardioverter-defibrillators (ICDs)[1].

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Appendix 1 - Informed consent*

It is now known that heart diseases are among the leading causes of death in young people. We hereby request your assistance in a screening for heart disease in order to identify potential risk situations. This screening aims to detect cardiovascular disorders in young people, which may put them in a risk group, requiring monitoring by a cardiologist. We ask you to answer a short questionnaire of 7 questions (held in computerized form on a laptop that will be available to you) and an electrocardiogram. We ask you to give a contact number and the number of your Citizen Card if you want to be informed in case of risk. Therefore, this does not present any risks and the tests are quick and painless.

The answer to this questionnaire could provide important health gains of the participants, since in the light of current knowledge, about 1 in every 333 young people have heart disease risk, potentially treatable and that will be detected this way. This is an opportunity for a cardiac evaluation that is completely free and totally voluntary.

You may, at any time, declare your intention to leave the study. The data provided will be under the tutelage of researchers and will remain completely confidential and will be used only for study purposes, in view of the health of the participants.

I declare that I agree to participate in the SCD-SOS study and I was given me the opportunity to clarify all my doubts concerning this subject:

Name: _____

Date: _____

No. of Identity:: _____

Telephone No.: _____

I wish to be informed in case of alterations in the examinations	
Yes	No

**This informed consent was reviewed and modified according to suggestions made by the ethics committee of the Hospital of Coimbra.*

Appendix 2 – SCD-SOS Questionnaire

Identification:

Gender: ☐Female ☐Male Age: _____

First and Last Name:_____

No. of Identity:_____ Telephone No. :_____

Occupation: _____ If student, indicate the area:_____

Do you practice any sports/physical activity regularly? ☐Yes ☐No

Which one(s)? _____ How many hours per week?_____

Have you ever practiced competition sports/pre-competition/federated?_____

How long have you stopped?_____

1) Have you ever fainted/lost consciousness?

☐No ☐Yes (if not, go strait to question 2)

For how long did you lose consciousness? (Choose the best option)

☐Did not lose consciousness

☐Less than 30 seconds

☐Between 30 seconds and 5 minutes

☐Between 15 and 30 minutes

☐More than an hour

How old were you when you first fainted?_____

How many times have you fainted in the last 5 years?

☐None ☐Once ☐2 to 5 times ☐Monthly ☐Weekly

In what context? (choose one or more as necessary)

☐During physical exertion ☐After physical exertion

☐Fright/Loud noise ☐Stress ☐Pain ☐Hunger ☐Drugs

☐Heat ☐Prolonged standing ☐Sight of blood/injection

☐Alcohol ☐Other – specify _____

Prior to fainting, do you usually experience one or more of the following?

☐Racing heart ☐Nausea/feeling unwell ☐Pallor ☐Sweating

☐Sensation of intense malaise ☐Change in vision ☐Disturbed hearing

☐None. The fainting occurred without any warning.

☐Other – specify_____

Did you fall down?

☐No ☐Yes – If yes, were you injured?

☐No ☐Yes – specify_____

If you didn't fall down, explain why:_____

Have your fainting episodes been witnessed by others?

☐No ☐Yes – If yes, did you have a seizure?

☐No ☐Yes

2) Do you have epilepsy or have you ever taken medication for epilepsy?

☐No ☐Yes

3) Do you ever feel your heart racing or have tachyarrhythmic episodes?

☐No ☐Yes – If yes, how long does it last?

☐Seconds ☐Up to 5 minutes ☐Up to 15 minutes ☐More than 1 hour non-stop

When this happens, have you ever measured your pulse/heart rate?

☐No ☐Yes – Indicate the measurement_____

During these episodes, have you ever felt any of the following? Select the appropriate(s)

☐Feeling unwell ☐Dizziness ☐ Feeling faint ☐ Trouble breathing

During these episodes, have you ever had to:

☐Stop everything you were doing ☐Seek medical help?

4) Do you ever experience chest pain?

☐No ☐Yes

If yes, in what context does the pain occur?

☐Physical exertion ☐Stress/emotion ☐At rest ☐Other – specify_____

If yes, how would you describe the pain?

☐Stabbing ☐Tightness ☐Crushing ☐Burning

Is it accompanied by any of the following symptoms? Select the appropriate(s)

☐Feeling unwell ☐Dizziness ☐Trouble breathing ☐Nausea ☐Sweating

What effect does the pain have on your activity? Select the appropriate

☐You have to stop what you were doing ☐You carry on

How long does the pain last?

☐Less than 10 seconds ☐1 minute ☐Up to 5 minutes ☐15 minutes ☐1 hour

5) Do you take any medication?

☐No ☐Yes – which one(s)? _____

6) Have you ever been diagnosed with heart disease? (Among others, pay attention to the

list below)

☐No ☐Yes – which?_____

7) Have you ever been seen by a cardiologist?

☐No ☐Yes – In what context? _____

8) Do you have any relatives who:

– Died suddenly before the age of 50? ☐No ☐Yes

Indicate the cause: _____ ☐Don't know ☐Not known

Relationship_____ Age_____

– Died in a car accident before the age of 50? ☐No ☐Yes

– Type of accident: ☐Collision whit other vehicle ☐Crashed alone ☐Other

Relationship_____ Age_____

– Died of drowning before the age of 50? ☐No ☐Yes

Relationship_____ Age_____

– Has a pacemaker? ☐No ☐Yes

Relationship_____ Age fitted with device_____

– Has an implantable cardioverter-defibrillator (ICD)? ☐No ☐Yes

Relationship_____ Age fitted with device_____

Has a known heart disease (see list below)

☐No ☐Yes – which disease? _____

Relationship_____ Age_____

List of diseases for question 8: hypertrophic cardiomyopathy, Marfan syndrome, arrhythmogenic right ventricular dysplasia, dilated cardiomyopathy, left ventricular non-compaction, aortic aneurysm, Wolff-Parkinson-White syndrome, anomalous origin of coronary arteries, Brugada syndrome, long QT syndrome, catecholaminergic ventricular tachycardia.