

Evaluation of the Prevalence of Risk Indicators for Sudden Cardiac Death in Young Athletes

Antonio da Silva Menezes Junior*, Jutay Fernando Silva Louzeiro, Viviane Batista de Magalhães Pereira and Edésio Martins

School of Medical, Pharmaceutical and Biomedical Sciences, Pontifical Catholic University of Goiás, Goiânia, Brazil

*Corresponding author: Antonio da Silva Menezes Junior, School of Medical, Pharmaceutical and Biomedical Sciences, Pontifical Catholic University of Goiás, Goiânia, Brazil, Tel: +5562982711177; E-mail: a.menezes.junior@uol.com.br

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Abstract

Background: Sudden cardiac death (SCD) in athletes during physical activity is rare.

Objectives: To evaluate the warning signs for SCD in young athletes and correlate them with electrocardiographic data.

Methods: This was a case-control, prospective study, and comparing athletes with sedentary individuals. The Sudden Cardiac Death Screening of Risk Factors (SCD-SOS) questionnaire was applied and resting electrocardiography was performed.

Results: In total, 898 participants were included, 589 (65.6%) in the case group (athletes) and 309 (34.4%) in the control group (sedentary). Fainting episodes were significantly less frequent in athletes (odds ratio 0.252, $p < 0.001$). Heart rates were not significantly different. The most common electrocardiographic findings were sinus arrhythmia, right bundle branch conduction disorder, and early repolarization.

Conclusion: Young athletes had a lower frequency of risk indicators for sudden cardiac death. There was a positive correlation between fainting reported by athletes and the duration of the QRS complex.

Keywords: Sudden cardiac death; Athletes; Exercise; Cardiac arrhythmias

Introduction

Sudden cardiac death (SCD) in athletes during physical exercise is rare, but it can have a profound impact in the community, since an athlete is seen as a health reference [1,2]. Regular exercise promotes the enlargement of the cardiac chambers and thickening of the myocardium, which is known as the "heart of the athlete" [3,4]. These physiologic changes lead to alterations in the electrocardiogram (ECG) of an athlete when compared to that of the general population. However, some of these changes are common. Changes that are pathological can increase the risk of SCD up to five times during high-intensity exercise [5-8].

SCD is related to exercise when it occurs during or up to an hour afterwards. Reports in the literature consider up to 24 h after the onset of symptoms as exercise-related SCD [8-11]. The exact incidence of SCD in athletes is not known and varies according to age, sex, race, and study design [12,13]. There is a predominance in men, which is attributed to the higher rate of male participation in sports. In Brazil, the sport most associated with SCD is soccer [14].

SCD during or after exercise is related to cardiovascular alterations that correlate with a risk of fatality, since it is difficult to trigger SCD in a structurally normal heart. The suggestive symptoms are syncope, chest pain, palpitations, dyspnea, and fatigue disproportionate to effort [15].

In young people, the cause of SCD is related to cardiomyopathies and ion channel disorders. These are often asymptomatic diseases, and syncope or even SCD may be the first manifestation. In 10 to 20% of cases of SCD, no structural abnormalities are found [15-17].

Hypertrophic cardiomyopathy (HCM) is the most common disease associated with SCD, and frequent symptoms are dyspnea, precordial pain, and palpitations, but these findings are non-specific. HCM should be considered when syncope occurs in previously asymptomatic young persons. Often, pathological ECG findings are the first diagnostic finding [18].

There are ECG findings that are considered to be common in athletes (sinus bradycardia, sinus arrhythmia, right bundle branch conduction disorders, early repolarization) and there are also unusual findings (inverted T wave, pathological Q wave, right ventricular hypertrophy) [19-21]. This study aimed to compare the risk factors for, and indicators of SCD in young athletes, and to correlate them with ECG findings.

Materials and Methods

This research was approved by the Ethics and Research Committee of the Pontifical Catholic University of Goiás (PUC/Goiás/Brazil) in August 2015.

This was a case-control, prospective study, with the sample being adopted for convenience. The athletes who attended gymnastics academies in the city of Goiânia-GO were compared with sedentary individuals, students of PUC-GO. We consider athletes to be active

individuals who were regular gym attendants, defined as a minimum frequency of three times per week for a minimum and uninterrupted period of one year. Other inclusion criteria were age between 18 and 35 years and absence of previously diagnosed diseases.

All participants signed an informed consent form, answered a questionnaire, and underwent an electrocardiogram. The participants

were asked about their physical activity practices (type and frequency), symptoms such as fainting, fast heartbeat, and chest pain, comorbidities, family history of disease, and use of substances to increase physical performance, and other data as presented in Table 1.

		n
Sex	men	449 (50%)
	women	449 (50%)
Age	(years)	25.93 ± 5.68
Education	completed elementary school	48 (5.3%)
	completed high school	255 (28.4%)
	graduation	455 (50.8%)
	post-graduation	140 (15.6%)

Table 1: Demographic data of the study sample (N=898).

ECGs were obtained by the researchers in the gymnastics academies, using the Mortara Eli 10 Mobile device with 12 leads (Mortara Instrument, Inc., Milwaukee, Wisconsin U.S.A.).

Statistical analysis

SPSS software for Windows version 18.0 was used. The Kolmogorov-Smirnov test was performed to classify the normal variables. Continuous data are expressed as means and standard deviation, and the categorical variables are expressed as absolute numbers or percentages. Categorical data distributions were analysed by the chi-square test or Fisher's exact test, as appropriate, at a significance level of 5%. The Pearson or Spearman correlation test was also used to verify possible correlations between the questionnaire data and the electrocardiographic data.

Results

The study analyzed a total of 898 participants, 589 (65.6%) in the case group (athletes) and 309 (34.4%) in the control group (sedentary), with a ratio of 1.9 cases for each control. The demographic data of the study population are shown in Table 1.

Regarding the study variables, the SCD-SOS questionnaire answered by the athletes was adapted to include data as to the type of physical activity, and total time of practice, as well as the use of substances to increase physical performance, such as supplements and anabolics. The responses to the physical activity questions helped us to categorize the participants into three groups based on the following frequencies: 412 (45.98%) athletes participated in anaerobic activity, 134 (14.94%) athletes participated in aerobic activity and 350 (39.08%) athletes participated in both activities. The mean time of physical activity was 5.2 ± 4.71 years. Finally, 364 (40.61%) athletes affirmed the use of supplements.

The other responses to the modified SCD-SOS questionnaire are grouped as shown in Table 1, with respective p values, odds ratios (OR), and 95% confidence intervals. There was a significant difference in fainting episodes, with a lower frequency in athletes (OR 0.252, p<0.001). These were mainly reported during physical exertion (OR 0.251, p=0.017), fasting (OR 0.071, p=0.001), and stressful situations (OR 0.224, p=0.003). Regarding the symptoms that preceded the fainting, there was a prominence of a heart-throbbing sensation and falling to the ground, both of which occurred less frequently in athletes (OR 0.224, p=0.003 and OR 0.490, p=0.039, respectively) (Table 2).

	p	Odds ratio*
1. Have you ever "fainted"/"lost your senses"?	<0.001	0.252 (0.147-0.432)
In what context?		
After physical exertion	0.418	0.519 (0.103-2.608)
Heat	0.103	1.535 (1.428-1.650)
Prolonged standing	0.238	0.260 (0.023-2.889)
Seeing blood	0.304	1.529 (1.423-1.642)
During physical exertion	0.017	0.251 (0.074-0.849)

Pain	0.792	0.785 (0.130-4.754)
Fright/noise	0.304	1.529 (1.423-1.642)
Stress	0.003	0.224 (0.076-0.658)
Other	0.074	1.537 (1.430-1.653)
Fasting	0.001	0.071 (0.009-0.587)
Before fainting do you usually experience any of the following?		
Accelerated heart rate	0.003	0.224 (0.076-0.658)
Impending death sensation	0.226	0.345 (0.057-2.089)
Pallor	0.084	0.500 (0.225-1.111)
Nausea/malaise	0.061	0.392 (0.143-1.076)
Other	0.468	1.527 (1.422-1.640)
Did you fall to the ground?	0.039	0.490 (0.246-0.976)
If so, did you have any injuries?	0.792	0.785 (0.130-4.754)
Has your fainting been witnessed by another person?	<0.001	0.315 (0.174-0.569)
If so, did you have seizures?	0.202	0.387 (0.085-1.753)
2. Are you epileptic or have you ever used epilepsy medication?	0.510	0.521 (0.073-3.741)
3. Do you often feel "heart racing" or have "tachyarrhythmia attacks"?	<0.001	0.141 (0.087-0.228)
In this circumstance, have you ever measured your heartbeat?	0.522	0.649 (0.172-2.459)
In these episodes have you ever felt any of the below?		
Malaise	<0.001	0.221 (0.112-0.436)
Dizziness	<0.001	0.221 (0.112-0.436)
Feeling faint	0.007	0.248 (0.083-0.741)
Shortness of breath	0.003	0.357 (0.176-0.722)
4. Do you often feel "chest pain"?	<0.001	0.104 (0.06-0.176)
If so, in what context does it arise?		
Stress/emotion	<0.01	0.202 (0.103-0.397)
Home	< 0.001	0.092 (0.034-0.247)
Physical exertion	<0.001	0.185 (0.079-0.432)
If yes, how do you describe the pain?		
Sting	<0.001	0.021 (0.005-0.088)
Tightening	<0.001	0.207 (0.098-0.437)
Heaviness	0.039	0.254 (0.062-1.031)
Burning	0.238	0.260 (0.023-2.889)
Is the pain accompanied by another complaint, such as:		
Dizziness	<0.001	0.223 (0.094-0.532)
Malaise	0.041	0.415 (0.175-0.987)

Shortness of breath	<0.001	0.138 (0.050-0.382)
Nausea	<0.001	3.088 (2.673-3.567)
Perspiration	0.036	0.420 (0.183-0.964)

Table 2: SCD-SOS Questionnaire Responses modified (*SCD-SOS: Sudden Cardiac Death Screening of Risk Factors).

The study group had a lower frequency of tachyarrhythmia (OR 0.141, $p<0.001$) and a lower frequency of associated symptoms, such as malaise ($p<0.001$), dizziness ($p<0.001$), fainting sensation ($p=0.007$), and shortness of breath ($p=0.003$). Athletes reported a reduced frequency of chest pain (OR 0.104, $p<0.001$), that tended to occur during periods of stress or emotion, rest, and physical exertion ($p<0.001$) and was most commonly characterized as a fast heart rate and chest tightening sensation ($p<0.001$). Regarding cardiac murmurs, use of medications, and diseases in the family, there was no significant difference between the groups.

Among athletes, 351 (59.77%) had heart rates in a normal sinus rhythm, 82 (13.79%) had sinus arrhythmias, 61 (10.34%) had an isolated right bundle branch block (QRS<120 ms), 30 (4.98%) showed early repolarization patterns, 20 (3.45%) were in sinus tachycardia, 18 (3.07%) had left atrial enlargement, 11 (1.92%) had a first degree AV block, 6 (1.15%) had left ventricular enlargement, 5 (0.77%) had sinus bradycardia, 2 (0.38%) had a left posterior fascicular block (LPFB), and 2 (0.38%) showed a right axis deviation.

In the control group, 215 (68.89%) had heart rates in a normal sinus rhythm, 30 (9.63%) had an isolated right bundle branch block (QRS<120 ms), 28 (8.89%) had sinus arrhythmias, 10 (2.96%) had

sinus bradycardia, 6 (2.22%) had sinus tachycardia, 4 (1.48%) showed diffuse alterations of ventricular repolarization, 4 (1.48%) had a right bundle branch block (QRS>120 ms), 4 (1.48%) showed an early repolarization pattern, 2 (0.74%) had a left anterior fascicular block (LAFB), 2 (0.74%) had Wolff-Parkinson-White syndrome, 2 (0.74%) had right atrial enlargement and 2 (0.74%) had left ventricular enlargement.

Table 3 presents the numerical data concerning the ECGs, with means, standard deviations, p values, and 95% confidence intervals. There was no significant difference in heart rate between these groups, with athletes having an average heart rate of 78 ± 14 beats per minute. The duration of the QRS complex, although within the limits of normal, was longer in athletes (92.05 ms vs. 88.74 ms, $p=0.005$). The mean QT interval was longer in athletes (368.94 ms vs. 360.85 ms, $p=0.01$), as was the QT interval corrected (398.09 ms vs. 391.70 ms, $p=0.002$). Comparing QT interval values corrected by the Bazett formula (QTcB) and the Fridericia formula (QTcF), there was a significant difference only for QTcF, with higher values in athletes (400.04 ms vs. 392.56 ms, $p<0.0001$). The PR interval was significantly higher in athletes (150.54 ms vs. 140.39 ms, $p<0.0001$). The P, R, and T wave axes showed no significant differences between the groups.

ECG	Groups	N	Mean $\pm$ SD*	t	Mean difference	p	95% CI
HR	case	589	78.03 $\pm$ 14.11	-0.86	-1.24	0.38	-4.06 -1.57
	control	309	79.28 $\pm$ 12.56				
QRS DUR	case	589	92.05 $\pm$ 11.06	2.84	3.31	0.005	1.019 -5.60
	control	309	88.74 $\pm$ 11.04				
QT	case	589	368.94 $\pm$ 29.03	2.59	0.085	0.01	1.95 -14.21
	control	309	360.85 $\pm$ 30.60				
QTC	case	589	398.09 $\pm$ 20.27	3.089	6.39	0.002	2.32 -10.45
	control	309	391.70 $\pm$ 18.29				
PR INT	case	589	150.54 $\pm$ 26.81	3.983	10.158	<0.0001	5.144 -15.171
	control	309	140.39 $\pm$ 18.01				
P AXES	case	589	55.91 $\pm$ 23.50	1.764	4.447	0.079	-0.51 -9.004
	control	309	51.46 $\pm$ 24.54				
R AXES	case	589	58.77 $\pm$ 28.45	1.93	6.27	0.054	-0.115-12.65
	control	309	52.50 $\pm$ 34.80				
T AXES	case	589	37.49 $\pm$ 24.26	-0.103	-0.280	0.918	-5.63 -5.07
	control	309	37.77 $\pm$ 28.54				

AVG RR	case	589	788.49 ± 143.90	1.139	16.73	0.25	-12.140 -45.618
	control	309	771.75 ± 129.85				
QTCB	case	589	415.57 ± 33.12	1.44	4.57	0.15	-1.654 -10.803
	control	309	410.99 ± 22.98				
QTCF	case	589	400.04 ± 19.04	3.684	7.48	<0.0001	3.488 -11.472
	control	309	392.56 ± 19.62				
QTCF	case	589	400.04 ± 19.04	3.684	7.48	<0.0001	3.488 -11.472
	control	309	392.56 ± 19.62				

Table 3: Results of quantitative electrocardiogram variables.

We correlated the questionnaire data that presented the largest significant differences between the groups with respect to the ECG data, mainly in relation to PR intervals, QRS complex durations, and QT values. Fainting, a more frequent complaint in the control group, showed no correlation with ECG data (p values>0.455). In the athletes, a correlation was observed, with narrower QRS complexes in those who had confirmed fainting episodes (86.96 ms vs. 92.64 ms, p=0.011). For the remaining variables, such as accelerated heart rate (p values>0.222) and chest pain (p values >0.179), there was no positive correlation. Averages of all ECG values studied remained within the normal limits, but for all parameters athletes had higher values than controls.

Discussion

The exact incidence of SCD in athletes is not well known. Very different values have been reported in published studies, with variations identified according to age, sex, type of sport practiced by the athlete, study methodology, region, population, and race [1]. There is no national database to track SCD in athletes. Additionally, there is a lack of information from clubs, organizations, or institutions responsible for athletes who have suffered this type of fatality.

The estimated incidences of SCD in young athletes in Europe and the United States are, respectively, 2.1 deaths per 100,000 athletes/year and 0.96 per 100,000 athletes/year [2], with predominance in men that can be attributed to greater male participation in athletics. However, in this study there was no sex-related predominance, with 145 (55.6%) men and 116 (44.4%) women athletes which reflects the trends towards increasing physical activity among women and corroborates the most recent studies [1,3,8,16].

Koike et al., in their study, affirmed that the regular practice of physical exercise improves cardiovascular system function, causing a reduction in heart rate [6]. However, in the current study, no significant difference was observed between groups (mean heart rate of 78 ± 14 beats per minute), which does not indicate an abnormality, but perhaps this group of athletes is not excessively physically conditioned. This might be due to numerous factors, such as regularity and intensity of physical activities.

The protective and cardiovascular risk paradox of physical activity was not seen in our study population, suggesting only a possible "protective effect" since the athletes showed a lower number of

symptoms suggestive of cardiac problems (fainting, chest pain, palpitation) in comparison to the control group [15].

Siebra et al. stated in their research that ventricular tachycardia is generally the most common etiology of sudden death, and that in young athletes it is related to left ventricular hypertrophy. [16] However, in this study, the heart rates were within the normal range for both groups. Additionally, findings related to HCM in both the questionnaire and the ECG was not significant. It is important to note that the "athlete's heart syndrome" [20] was taken into account in the differential diagnosis of HCM, and no relevant findings were observed.

Common or physical training-related ECG findings were similar to those found in previous research, with sinus arrhythmia, right bundle branch conduction disorder, and early repolarization being the most common in young athletes and the most frequent in the studied group. Sinus bradycardia is present in more than 80% of highly trained athletes; however, in our study, only 2 cases (0.77%) were identified, perhaps because the sample studied was not composed of highly trained individuals [17,18,20-25].

The right bundle branch conduction disorder can be considered a variant of normal, and has little clinical repercussions. The expression "final conduction delay" can be used when the disorder is very discreet. The presence of right-hand conduction delays can be expressed by RR-filled R waves, and S-waves in the D1, aVL, V5, and V6 leads (delayed duration ≥ 30 ms). Conduction delay sites have been attributed to the right ventricular free wall, particularly in the presence of supraventricular ridge hypertrophy, delayed conduction in the terminal Purkinje network of the right ventricle, or physiological variability of the wall thickness and mass distribution of the right ventricle [22-25].

Early repolarization is characterized by an elevation of the J point ≥ 1 mm, causing the end of the QRS to not coincide with the baseline. This generates an upper concave ST segment in at least two adjacent precordial leads with values also ≥ 1 mm [22,24]. In the current study, this ECG change was more frequent in the athlete group (4.98% vs. 1.48%). Although some authors consider this a common finding that can be related to physical activity, its clinical significance has been questioned in recent studies because of its correlation with fatal cardiac arrhythmias, especially in individuals without structural heart disease [22,24]. Sinner et al, in 2010, conducted a case-control study with 6213 participants between 35 and 74 years of age. The prevalence of the PR interval was 13.1% in the population, demonstrating an increased risk of mortality in the general population [17].

Despite the existence of studies in this field, further research is needed for better elucidation of this topic. Wolff-Parkinson-White syndrome (WPW) is another cardiac change related to SCD. It is characterized mainly by a PR interval less than 120 ms during the RS phase in adults, in addition to a notch of the initial portion of the QRS complex (delta wave). A case was identified in the control group, and due to its magnitude, we highlight its occurrence. It is important to remember entities that simulate the presence of ventricular pre-excitation (false WPW) such as HCM itself and familial forms of septal glycogen deposition (Fabri's disease) [22]. We could not identify any studies in the literature that correlated the responses of the SCD questionnaire-SOS with ECG data [25].

The present study has limitations that need to be considered. The studied athletes, for the most part, were not of high performance, therefore increasing their similarity with the control group. In addition, the limited sample size may have influenced the outcomes.

Conclusions

The present study demonstrated a lower frequency of risk indications for SCD in young athletes, as well as higher values for QRS duration, PR interval, and QT interval on ECG, despite being within normal limits. In addition, there was a positive correlation between the presence of fainting episodes and the narrower QRS in athletes.

Data availability

The data is available upon request, through direct request from the author; Antonio da Silva Menezes Junior, PhD or co-author: Jutay Fernando Silva Louzeiro, MD by e-mail: a.menezes.junior@uol.com.br and fernando_louzeiro@hotmail.com, respectively.

Conflict of Interest

We declare that there is no conflict of interest.

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