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Differences in Heart Rate, Left Ventricular Wall Mass and Thickness, Diastolic Function, and Cardiac Output Index between Adolescent Athlete and Non-athlete Adolescents

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Abstract

BACKGROUND: Changes in cardiac function and structure in adolescent athletes increase the population's risk for sudden death. No studies in Bali, Indonesia, have examined changes in cardiac function and structure in adolescent athletes compared to non-athlete.

AIM: The objective of the study was to evaluate the cardiac function and anatomical structure of adolescent athletes.

METHODS: This study was a cross-sectional study of male adolescents aged 15–18 years at the high school of Denpasar, Badung, Gianyar, and Tabanan from September 2019 to November 2020, using consecutive sampling. Mann–Whitney U-test was used to assess differences in heart rate, mass and left ventricular (LV) wall thickness, and diastolic function, while independent t-test was used to assess mean differences in the cardiac output index.

RESULTS: A total of 88 subjects participated in this study. Heart rate in adolescent athletes was found to be slower than in non-athlete, with a median of 63.5 (range 46–107) beats per minute ($p < 0.001$). The mass and thickness of the LV (interventricular septum) in adolescent athletes were greater than non-athlete with a median value of 149.61 (range 101.02–280.80) g and 9 (range 6–12) mm with $p < 0.001$ and 0.005, respectively. There was no significant difference in diastolic function and cardiac output index among adolescent athletes compared to non-athlete.

CONCLUSION: Significant structural changes of the heart were observed in adolescent athletes (mass and thickness of the interventricular septum wall) but not in the function of the heart (diastolic function and cardiac output index). Adolescent athletes have a significantly slower heart rate than non-athlete.

Introduction

Cardiac function screening test program at the time of athlete selection and performance testing has not been implemented evenly to assess cardiac changes and defects in athletes. At the same time, the probability of sudden death is 2.5 times higher for athletes than for non-athletes [1]. Thus, identifying the athletes' cardiac function is an important endeavor to minimize the risk of cardiovascular diseases and sudden death.

A study conducted in Italy from 1993 to 2004 reported that the rate of sudden death among athletes aged 15–35 years before screening was 4.19 per 100,000, which decreased to 0.87 per 100,000. Screening can start after 12–14 years of age, that is, 15 years [2]. More than 40% of the cause of sudden death in young athletes are due to the presence of hypertrophic cardiomyopathy, in addition to congenital anomalies and acquired heart disease [3]. Several sports are associated with sudden death in the USA, including American football (30%), basketball (20%), soccer (6%), and baseball (6%), while in Sweden, these include soccer (44%), ice hockey (20%), indoor and ice-based bandy (16%), and athletics (8%) [1], [2].

The previous studies have reported changes in the function and structure of the heart in adolescent athletes aged 18 years, leading to an increased risk of sudden death in adolescent athletes. However, no studies in Bali, Indonesia, have examined changes in cardiac function and structure in adolescent athletes compared to non-athletes. It is necessary to evaluate the anatomical and physiological aspects of the heart in adolescent athletes, namely, heart rate, mass, LV thickness, diastolic function, and cardiac output index, especially when compared to non-athlete adolescents.

The aim of the study

Based on this background, the aim of this study was to evaluate the cardiac function and anatomical structure of male adolescent athletes in Bali.

Materials and Methods

This study was a cross-sectional observational analytic study evaluating cardiac structural and functional abnormalities in adolescent athletes aged 15–18 years old compared to non-athlete adolescents. The study is based at the Pediatric Cardiology Clinic in Sanglah Hospital, Denpasar, Bali, Indonesia. The study was conducted between September 2019 and November 2020. Adolescent male athletes and non-athlete adolescents aged 15–18 who attended high school in Denpasar, Badung, Gianyar, and Tabanan were recruited for the study. The criteria for inclusion include regular participation in training for one of the dynamic sports with metabolic equivalent ≥8, namely soccer, basketball, volleyball, badminton, running, tennis, or swimming with a minimum frequency of 3 times a week with a period of 1.5 h duration for each training for the last 4 years; consent to participate in the study and to participate in the training [4]. Criteria for exclusion include congenital heart disease or acquired heart disease, family history such as a father, mother, sibling with heart disease or sudden death, drug therapy for heart disease; chronic anemia; and hepatic dysfunction.

Table 1: Characteristics of study subjects

Variable	Group	
	Adolescent athletes, n = 44	Non-athlete adolescents, n = 44
General characteristics		
Age, median (min-max), years	17 (15–18)	18 (15–18)
Weight, median (min-max), kg	64.50 (42–108)	60.50 (42–108)
Height, mean (SD), cm	171.40 (5.15)	169.76 (6.64)
Systolic blood pressure, median (min-max), mmHg	110 (95–140)	110 (90–140)
Diastolic blood pressure, median (min-max), mmHg	70 (60–90)	70 (50-90)
BSA, median (min-max), m^2	1.75 (1.52–1.92)	1.67 (1.38–2.30)
Sport type, n (%)		
Soccer	39 (88.64)	1 (2.27)
Badminton	2 (4.54)	5 (11.36)
Futsal	0	2 (4.54)
Basketball	3 (6.82)	0
Swimming	0	1 (2.27)
Run	0	4 (9.10)
No exercise	0	31 (70.45)
Frequency of exercise, median (min-max), hours/week	9 (6–14)	2 (0–6)
Duration of competitor, median (min-max), years	3 (0–6)	0 (0)
Electrocardiography characteristic		
PR interval, median (min-max), seconds	0.16 (0.12–0.20)	0.16 (0.12–0.24)
Duration of QRS, median (min-max), seconds	0.06 (0.04–0.08)	0.06 (0.04–0.08)
QTc interval, mean (SD), seconds	0.39 (0.03)	0.38 (0.03)
S in V1, median (min-max), mV	1.1 (0.40–2.50)	1.1 (0.20–2.20)
R at V6, median (min-max), mV	1.75 (0.90–3.10)	1.3 (0.80–2.40)
Sinus rhythm, n, (%)	19 (43.20)	35 (79.54)
Other variants, n, (%)	25 (56.8)	9 (20.45)
Sinus bradycardia, n, %	8 (18.18)	3 (6.81)
Sinus arrhythmia, n, %	4 (9)	1 (2.27)
LVH's, n, %	11 (25)	4 (9)
Incomplete RBBB, n, %	2 (4.54)	0
First-degree AV block, n, %	0	1 (2.27)
Echocardiography characteristics		
Ejection fraction, mean (SD), %	67.36 (4.69)	68.65 (6.02)
Shortening fraction, mean (SD), %	37.62 (3.86)	38.50 (4.81)
Stroke volume, mean (SD), mL	71.35 (13.46)	56.67 (13.20)
Cardiac Output, mean (SD), liter/minute	4.53 (1.02)	4.30 (1.17)
E/e' ratio, median (min-max)	5.80 (3.40-8.10)	5.80 (2.80-10.70)
LVH, n (%)	4 (4.54)	0

SD: Standard deviation, BSA: Body surface area, LVH: Left ventricle hypertrophy, RBBB: Right bundle branch block, AV: Atrioventricular, E/e' ratio: Ratio of mitral peak velocity of early filling to early diastolic mitral annular velocity, QTc: Corrected QT, mV: Millivolt.

A total of 88 subjects were recruited using consecutive sampling method, performed sequentially throughout the study period the previously determined sample size was reached. Screening was conducted at high schools in Denpasar, Badung, Gianyar, and Tabanan, Bali, Indonesia, to identify subjects that meet the inclusion criteria and are willing to be tested. Parents of the subjects who had signed the informed consent form were asked to come to the Pediatric Cardiology Center at Sanglah Hospital, Denpasar, while adhering to health protocols and schedules set forth due to the COVID-19 pandemic. Subjects were interviewed and underwent physical examinations, including electrocardiography and echocardiography. Electrocardiography was performed with a 12-lead ECG. Echocardiographic examination using a two-dimensional echocardiogram. Two pediatric cardiologist consultants interpreted the electrocardiography and echocardiography results, including ejection fraction, LV mass and thickness, diastolic function, and cardiac output index. Kolmogorov–Smirnov test was used to test data normality for all study variables with a numerical scale. Data distribution test is required if the test results are found to be $p > 0.05$. Independent t-test was conducted for numerical variables with normal distribution. Mann–Whitney U-test was used for a numerical variable normality with non-normal distribution. $p < 0.05$ is considered statistically significant. This study was approved by the Research Ethics Committee of Faculty of Medicine, Udayana University/Sanglah Hospital.

Results

During the period from September 2019 to November 2020, 90 consecutive subjects were registered. Eighty-eight subjects, consisting of 44 athletes and 44 non-athletes, met the inclusion criteria and had no complaints of tightness, palpitation, chest pain, and fainting history during exercise. Two subjects did not meet the inclusion criteria due to family history of heart disease. The main characteristics of the study subjects are shown in Table 1. The distribution of cardiac output index in the adolescent athletes and non-athlete adolescents is shown in Table 2. There was no significant difference in cardiac output index among adolescent athletes compared to non-athlete adolescents. The distribution of heart rate, LV

Table 2: Analysis of cardiac output index for adolescent athletes compared to non-athlete adolescents

Variable	Athletes	Non-athletes	Mean difference	95% CI	p-value
Cardiac output index, mean (SD), liters per minute per square meter	2.63 (0.57)	2.50 (0.57)	0.12	(-1.12–0.37)	0.292[a]

CI: Confidence interval, SD: Standard deviation. [a]Unpaired t-test.

wall mass and thickness, and diastolic function in the adolescent athletes and non-athlete adolescents is shown in Table 3. The diastolic function evaluated in this study is the E/A ratio. The LV wall thickness studied was the thickness of the interventricular septum (IVS) during diastolic phase. Heart rate in adolescent athletes was found to be slower than in non-athlete adolescents, with a median of 63.5 (range 46–107) beats per minute ($p < 0.001$). The mass and thickness of the LV (interventricular septum) in adolescent athletes were greater than non-athlete adolescents with a median value of 149.61 (range 101.02–280.80) g and 9 (range 6–12) mm with $p < 0.001$ and 0.005, respectively.

Table 3: Analysis of heart rate, left ventricular wall mass and thickness, and diastolic function in adolescent athletes compared to non-athletes

Variable	Group	Median (-max)	Mean rank	p-value
Heart rate, bpm	Athletes	63.50 (46–107)	32.98	0.001[b]
	Non-athletes	80 (55–115)	56.02	
Left ventricular mass, grams	Athletes	149.61 (101.02–280.80)	55.73	0.001[b]
	Non-athletes	116.51 (66–243.40)	33.27	
Interventricular septal thickness, mm	Athletes	9 (6–12)	52.14	0.005[b]
	Non-athletes	7.85 (5.80–11.9)	36.86	
E/A ratio	Athletes	1.89 (0.50–2.89)	47.26	0.307[b]
	Non-athletes	1.80 (0.90–2.80)	41.74	

E/A ratio: Ratio of early-to-late diastolic velocity. [b]Mann–Whitney U-test.

Discussion

The median age of the athlete group was 17 (15–18) years. This study included only male participants due to the fact that cardiac output was affected by gender. Differences in the structure of the left ventricle can be detected in the early stages of adolescence. The mass, thickness, and diameter of the left ventricular cavity are more significant in men. Increased levels of testosterone in men during pre-puberty can stimulate an increase in the size of the cardiac myocytes [5], [6]. The present study observed sinus arrhythmia in adolescent athletes and non-athlete adolescents; this is in line with several previous studies reporting that sinus arrhythmia tends to substantially developed in adolescent athletes compared to non-athlete adolescents [7], [8], [9]. This study also observed first-degree atrioventricular block (AV block) in non-athlete adolescents which was not found in the adolescent athlete group. The previous studies have also found a difference in terms of a first-degree AV block between athlete and non-athlete population [7], [8], [9]. This difference may be due to the fact that electrocardiography is performed only once. Conduction prolongation in adolescents is usually due to increased vagal tone [10]. This study found an incomplete right bundle branch block in the athlete group. Several studies have reported a significantly incomplete right bundle branch block (RBBB) in adolescent athletes compared to non-athlete adolescents [7], [8], [9]. Cardiovascular adaptation in the form of incomplete RBBB occurs due to an increase in the right ventricular size, which causes a slight delay in conduction in the His-Purkinje system [11], [12].

There are variations in the effects of the left ventricular hypertrophy (LVH) in electrocardiography and echocardiography. Some studies have observed significant LVH in adolescent athletes relative to non-athlete adolescents [7], [8], [9]. Increased prevalence of LVH in non-athlete adolescents based on the Sokolow criteria occurred due to the fact that subjects in non-athletes were relatively slim and thin walled. Some electrocardiography analyses and echocardiographic measurements correlated with LVH but generally suggest that the Sokolow criterion is poorly associated with LVH echocardiographic measurements [13]. The limitation of the electrocardiography is that other factors can influence the measurement of the heart's electrical activity by electrodes on the body's surface [14]. Adolescent athletes with LVH as measured through electrocardiography have a poor correlation with LVH measured through echocardiography [15]. Pulsed tissue Doppler echocardiography has a sensitivity of 87% and a specificity of 97% for LVH differentiation in athletes [16]. The gold standard cardiac magnetic resonance test has a sensitivity of 80% and a precision of 99% for differentiating athletes' hearts from all pathological LVH [2].

In this study, the mean EF in the athlete category was lower than non-athletes but still within the normal range. Some studies found no substantial difference in the EF between the athlete and the control groups [17]. An ejection fraction that is still within acceptable limits may be triggered as a compensation mechanism for the cardiac changes that occur in the athlete. The athlete group had a higher mean value for stroke volume. There was a substantial increase in stroke volume in athletes aged 43–50 years who practiced consistently 4 days a week for 5 years relative to controls [18]. In endurance athletes, there was an eccentric formation marked by an increase in ventricular dimensions [18]. A lower heart rate indicates an increase in stroke volume. This compensatory mechanism results from maintaining a consistent resting cardiac output [19].

This study found significant sinus bradycardia in adolescent athletes compared to non-athlete adolescents. This result is consistent with the study of McClean *et al*., Sharma *et al.*, Makan *et al*., and Galanti *et al*. that showed significant sinus bradycardia [7], [20], [21], [22]. Systematic analysis found that frequent use of athletic modality and exercise form resulted in various cardiac adaptations [23]. Endurance exercise may increase parasympathetic activity and decrease sympathetic activity [24]. A significant difference between the median value of LV mass in athletes and non-athletes has also been reported. The present study's findings are consistent with the previous studies that reported higher LV mass in male athletes aged 18 years than non-athletes and LVH in 76.9% of adolescent athletes compared to non-athlete adolescents [4], [7], [25].

In Italy, a study observed an increase index LV mass in male athletes compared to female athletes (100 ± 18 g/m^2 vs. 79 ± 12, p < 0.001) [26]. Another study on adolescent tennis athletes found that LV mass in athletes differed significantly from non-athletes, due to an increase in the LV systolic and diastolic diameters and the thickening of the ventricle wall [15]. There was an improvement in the mean value of the LV mass index for football athletes compared to non-athletes [27]. In the present study, LV wall thickness was measured as the diastolic thickness of the interventricular septum (IVS). We found a significant difference in the median value of the IVS thickness between adolescent athletes and non-athlete adolescent. Several previous studies have reported differences in the IVS thickness of adolescent athletes relative to the non-athlete [7], [22]. A significant thickening of the IVS in a professional football team compared to young athletes has been previously reported. This study shows that physical activity has a significant effect on both the functional and structural aspects of the cardiovascular system [28]. Continuous exercise induces the release of growth factors and neurotransmitters that activate different cell types of receptors mediating biological reactions. The growth factor in the coronary sinus blood of professional soccer players produced insulin-like growth factor 1 at a high level relative to safety controls. In addition, insulin-like growth factor 1 levels were positively associated with the LV mass index and the LV end-diastolic dimension index [29].

The diastolic function, namely, the ratio of early-to-late diastolic velocity (E/A ratio), was measured in this study with a median value that did not differ significantly between adolescent athletes and non-athlete adolescents. The ratio of early-to-late diastolic velocity of athletes to non-athletes was not significant [7], [22], [26]. Other studies have reported that the E/A ratio is increased in athletes relative to controls. The ratio of early-to-late diastolic velocity is not only determined by LV compliance but is also influenced by other factors such as preload, afterload, and heart rate. Slight heart rate decreases LV filling due to diastolic prolongation [17]. This study found that the average cardiac output value for adolescent athletes and non-athlete adolescents did not differ significantly and remained within normal limit. A previous study reported negligible increase in cardiac output in athletes aged 43–50 years who swim 4 days a week for 5 years compared to non-athletes [18]. Similar cardiac output values may be due to bradycardia and increased stroke volume. In this study, we found no statistically significant difference in mean cardiac output index between adolescent athletes and non-athlete adolescents. A previous study has also reported no significant difference between the cardiac output index for athletes and non-athletes [30]. The presence of bradycardia, increased stroke volume, and a larger body surface area in the athlete group produced almost the same cardiac output index as the non-athlete group.

Sudden death in athletes in the USA is often caused by hypertrophy cardiomyopathy (36.4 %) and in Italy mainly due to arrhythmogenic right ventricular (RV) cardiomyopathy (26 %) [2]. This study found changes in cardiac conduction in the form of cardiac rhythm, cardiac structure, that is, the mass and thickness of the LV wall, which, in turn, did not result in any changes in cardiac function, that is, diastolic function and cardiac output index.

There are several limitations to this study: First, this study did not measure the heart rate, LV mass and wall thickness, diastolic function, and cardiac output index using cardiac magnetic resonance so that they could not be deemed equivalent to the gold standard for analysis. Second, subjects in the study were athletes who had been training consistently for at least 4 years. Thus, it may take longer to determine the improvements and compensations in the heart of the adolescent athletes group. Third, this study did not evaluate the existence of genetic factors, and thus, there was no established genetic abnormality that played a role in affecting changes in the athletes' hearts. Fourth, most of the athletes in this study played soccer, which is an endurance sport, therefore necessitating the categorization of sports types to achieve more precise results. Finally, the subjects were examined only once using electrocardiography and echocardiography, and therefore, no previous abnormalities were identified. In Bali, Indonesia, no studies comparing adolescent athletes to non-athletes examined changes in cardiac function and structure. This study is being conducted in Bali for the 1st time. Thus, this research can serve as a foundation for future research on the same subject using a different methodology.

Conclusion

This research concluded that there were changes in the structure of the heart, but not the cardiac function of adolescent athletes aged 15–18 years compared to non-athlete adolescents. Differences in heart rate, LV mass, and IVS wall were observed, but there was no difference in the diastolic function and cardiac output index of adolescent athletes aged 15–18 years compared to non-athlete adolescents.

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